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Socioeconomic Status and Health in Blacks and Whites: The Problem of Residual Confounding and the Resiliency of Race

Jay S. Kaufman, Richard S. Cooper, and Daniel L. McGee

A large number of epidemiologic studies have focused on racial/ethnic differences, particularly between blacks and whites. Because health endpoints and racial categorizations are associated with socioeconomic status, investigators generally adjust for socioeconomic indicators. The intention is usually to control for confounding, thereby making groups comparable and excluding socioeconomic status as an alternative explanation to hypotheses of innate physiologic differences. A threat to the validity of these analyses is therefore the presence of residual confounding. We identify four potential sources of residual confounding in this analytical design: categorization of socioeconomic status variables, measurement error in socioeconomic indicators, use of aggregated socioeconomic status measures, and incommensurate socioeconomic indicators. Using simulations and examples from the literature, we demonstrate that the effect of residual confounding is to bias interpretation of data toward the conclusion of independent racial/ethnic group effects.

Investigators often refer to possible "genetic" differences on the basis of models that control for socioeconomic status. We propose that such conclusions on the basis of this analytical strategy are generally unwarranted. Racial/ethnic differences in disease are a pressing public health concern, but the current approach does not often provide a basis for inference about putative biological factors in the etiology of this disparity.. (Epidemiology 1997;8:621–628)

Keywords: epidemiologic methods, race, socioeconomic status, confounding.

A substantial proportion of the public health and medical literature is devoted to racial/ethnic differences in health outcomes.[1,2] Although these comparisons have proliferated recently to include a variety of categorizations, the primary comparison in the United States has been focused on whites and blacks.[2,3] This *de facto* dichotomy in racial classification owes much to the enduring impact of slavery on our social and scientific institutions and the central significance of skin color in racial classification schemata.[3,4]

There do exist physiologic traits, such as sickle cell, that affect health status and are known to differ between blacks and whites.[1] These known traits are largely irrelevant, however, when making black-white health comparisons in the United States. Although blacks have a higher mortality rate for virtually every major disease,[5] the contributions of recognized physiologic mechanisms account for a minuscule part of this excess. Single gene disorders such as hemoglobinopathies collectively make up less than 0.3% of the differential mortality burden, for example.[6] The diseases that account for the largest proportion of excess morbidity and mortality among blacks are chronic diseases, especially hypertension, diabetes mellitus, stroke, renal disease, and common cancers. There is no consistent scientific hypothesis that would link these diseases to the African ancestral origin of the black American population.[7]

The lower average socioeconomic status (SES) of blacks, compared with that of whites, provides a plausible explanation for some or all of the excess prevalence of these conditions in blacks.[8] The relation between SES and numerous outcomes has been widely demonstrated,[9] and race is intimately linked to SES in the United States.[4,5] Potential confounding by SES of black-white comparisons is therefore the most obvious alternative hypothesis in any study that reports a biological difference between groups on the basis of observational data.

To address this problem, most investigators attempt to "control" for SES when making racial/ethnic comparisons. Reported estimates of an independent effect of race are therefore potentially biased by the presence of residual confounding. Given the established relation of the potential confounder with both the outcome and the

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group indicator, careful variable definition and analysis are required to reduce residual confounding. Unfortunately, SES is most often poorly conceived and operationalized in epidemiologic studies.

Sources of residual confounding are interrelated but are categorized here into four general classes: categorization of the SES variable, measurement error in the SES indicators, the use of aggregated SES measures, and incommensurate SES indicators. Whereas these potential biases are relevant to a wide range of epidemiologic situations, they pose a particular danger in studies that report black-white comparisons and claim to be adjusted for SES level. The statement most often made in the conclusions of such investigations is that the black-white difference *persists after adjustment for SES*, and that this indicates another source of susceptibility, frequently ascribed to "genetic" factors. This conclusion is based on the mistaken belief that the groups have been made exchangeable with respect to socially related exposures.

Residual Confounding Due to Categorization

Mortality has been consistently observed to decrease monotonically across SES values, whether the indicator is education,[10,11] occupation,[12] or income.[13,14] Investigators making black-white comparisons often categorize SES into only two or three levels, however, leaving considerable variation in risk within groups. The parameter estimate for SES and the degree of control achieved for SES as a confounder are functions of the cutpoints chosen,[15] and these vary widely between studies. Furthermore, if misclassification of SES is differential by race, which is likely given different distributions of SES variables in each racial group, then the resulting parameter estimates are biased. The greatest magnitude of error occurs when, as in this context, the effect of the factor of interest is weak compared to that of the confounder.[16]

For example, Brancati *et al*[17] studied 442 persons diagnosed with end-stage renal disease (ESRD) within a catchment area between 1980 and 1985.[17] The relative risk for blacks compared with whites was 2.70 [95% confidence interval (CI) = 1.89–3.86] after adjustment for clinical features, health care, and SES. The authors concluded that "excess incidence of diabetic ESRD among blacks is not fully explained by a higher prevalence of diabetes or hypertension. . . or by racial differences in age, socioeconomic status, or access to health care."[17,p.3079] The authors went on to propose that "the natural history of renal complications in diabetes. . . may differ by race. . . suggest[ing] an inheritable genetic predisposition."[17,pp.3083–3084]

The SES indicator used in these analyses was the percentage without college education among each of 13 ZIP postal codes in the catchment area (77.2% for blacks and 65.0% for whites). The contention that there was statistical control for SES in this analysis rests on the assumption that blacks and whites in each category had equal social positions. National data indicate otherwise. For example, 27.1% of blacks lacked a high school degree in 1990, compared with only 18.0% of whites, demonstrating an unequal distribution below the cutpoint.[18]

Residual Confounding Due to Measurement Error

The biases that result from variable misclassification have been documented, including the loss of control for confounding associated with confounder misclassification.[19] Since SES variables are interpreted as surrogates for myriad undefined exposures, the true confounder must therefore be measured with error. This error results not only from the fact that education, income, and occupation represent unspecified quantities, but also because of difficulty in measuring even the surrogates. Income, in particular, is subject to severe bias from differential nonresponse, underreporting, poorly specified operational definitions, and volatility over time.[20]

If exposure or disease variables are measured with nondifferential error but the confounder is perfectly specified, then the adjusted effect estimate is always closer to the null.[21] When measuring SES, however, substantial error can occur. This error is potentially more dangerous than measurement errors in exposure or disease, as a large exposure effect can be observed when the true relation is zero or even negative.[21,p.647] The effects of measurement error on the validity of analytical conclusions has been demonstrated previously by simulation, including the effect on confounding.[22]

A related problem is that researchers may rely on significance testing for the purpose of model selection and thereby exclude true confounders from the analysis. Coughlin *et al*,[23] for example, examined black-white differences in death from cardiomyopathy among 356,222 men screened for the Multiple Risk Factor Intervention Trial (MRFIT) study. Family income was estimated as the mean value for residents in the participant's ZIP code area and was included in the analysis as a dichotomous variable (≥$16,000 per year). Whereas "black race" was found to be a predictor of mortality (relative risk = 1.59; $P = 0.045$), the SES surrogate was not, and the authors concluded that "The apparent black race predominance may be due to. . . differences in genetic background."[23,p.170]

This analysis strategy and the resulting conclusion are problematic because a weak observed association between a confounder and an outcome may be due to measurement error.[24] Misclassification of an SES confounder may lead to poor control and a spurious association between the race variable and disease. If there is particularly poor SES measurement, however, it may be dropped from the model altogether, leading to a complete lack of control. Claims that SES confounding has been controlled in black-white comparisons, or that the relation was not observed to be confounded by SES, are therefore suspect, and conclusions about unspecified genetic differences would be unwarranted on the basis of this strategy.

Residual Confounding Due to Aggregation

It has become increasingly common in epidemiologic and public health research to assign to individuals the average SES value of the community (county, ZIP code, census track or census block group, for example) in which they live.[25,26] Although SES indicators are often absent from available records, this technique allows for SES to be analyzed whenever the addresses of individuals are known. This "ecologic" assignment of SES has been used in a wide variety of black-white comparisons, including the two studies described in the previous examples.[17,23]

In studies that aggregate not only the SES variable but also the outcome, the well-known "ecologic fallacy" can occur: parameter estimates for the relation between exposure and outcome may be biased in either direction.[27] In studies of individual outcomes, the univariate relation is theoretically unbiased for many model forms,[28] but problems arise because of reduced power and confounding. Power is affected because assigning to individuals the value of the group mean leads to loss of intragroup variation relative to intergroup variation.[28] Within-group variation is often large relative to between-group variation, which leads to considerable loss of power and therefore to erroneous exclusion of SES indicators from statistical models if significance testing is used to select confounders. Although inclusion of aggregated SES measures in the model is intended to adjust for confounding, bias may result if there is an independent group effect, or if the aggregate value differentially misclassifies the individuals in each group. These potential biases have been referred to as *specification bias* and *aggregation bias*.[29]

Specification bias occurs when both the individual and the group level variable are independent predictors of the outcome. For example, where individual income is a predictor of disease, a neighborhood indicator may also be an independent predictor because it captures information on additional exposures that are not characterized by the individual income variable. When both the group indicator and the individual SES variable independently predict disease, the aggregated SES variable will encode more than just mean individual SES; it will also encode information about group effects that are independent of the individual effects.[30]

Aggregation bias occurs when all subjects within the group are assigned to the mean, since blacks and whites within the group may have unequal average deviations from the assigned value. For example, if a hypothetical sample had equal numbers of blacks and whites, and every white in the group had higher SES than every black, then assignment of the mean to all subjects would lower every white score and raise every black score. The result of this differential error in the measurement of SES would be residual confounding; the lower individual-level SES of blacks and the higher individual-level SES of whites would be lost in the SES measure and reflected instead in the race term.

To gauge the degree of residual confounding that occurs because of the use of aggregated SES measures to adjust for confounding in black-white comparisons, Geronimus *et al*[30] compared models with individual-level education and income to models with aggregate measures. In analyses of the Panel Study of Income Dynamics, the pattern that emerged was that group-level SES indicators were poorer at controlling for confounding by SES in the relation between race and the outcome. For example, the estimated regression slope associated with race in a model predicting overall health was inflated by 38% when census tract level means for SES variables were used instead of individual values. The result of aggregating the SES variable was incomplete control, biasing interpretation erroneously toward spurious black-white differences.

Residual Confounding Due to Incommensurate Indicators

When a confounder is measured with error, incomplete control for confounding occurs. The problem is exacerbated, however, if the magnitude of error is correlated with the factor of interest. Specifically, measured SES variables are interpreted as surrogates for unmeasured aspects of social position. If the relation between the SES variables and these unmeasured attributes differs systematically between blacks and whites, then the problem changes from simple measurement error to a fundamental incommensurability of the SES indicators being used.

In fact, there is abundant evidence to demonstrate that standard SES measures are not commensurate between blacks and whites. In the case of education, the 1992 income associated with any number of years of schooling is less for blacks, as shown in Table 1.[31] The use of occupation categories produces similar discrepancies in income. Ratios of black to white income for categories of self-employed, lower-blue-collar, upper-blue-collar, lower-white-collar, and upper-white-collar are 0.56, 0.63, 0.74, 0.75, and 0.75, respectively, based on 1987 data from the Survey of Income and Program Participation (SIPP).[32,p.119]

The black-white difference in payoff for a given level of educational attainment or a given occupational level is even more striking for family net worth (total accumulated assets, minus debts) than for income. The ratio of black to white median family net worth by educational level ranged in 1987 from 0.02 (less than high school) to 0.23 (postgraduate education).[32,p.197] The ratios of black to white median family net worth by the five occupational levels described above were 0.18, 0.09, 0.23, 0.13, and 0.18, respectively.[32,p.119]

Even when income is used as the indicator, the racial disparity in the value of a unit of SES is considerable. Rather than units of standard value, income values are actually relative to average costs. Since blacks are more likely to live in neighborhoods in which equivalent housing, basic food costs, insurance costs, and loan interest rates are higher, a dollar buys fewer goods and

TABLE 1. Median Income (US$) Earned by Individuals Ages 25 Years and Older, by Educational Attainment, Sex, and Race, 1992*

Education	Black Males	White Males	Ratio BM/WM	Black Females	White Females	Ratio BF/WF
<9 years	9,836	12,444	0.79	8,213	7,863	1.04
9–12 years	13,168	16,718	0.79	9,359	9,902	0.95
High school degree	16,599	24,086	0.69	12,762	13,342	0.96
Some college	20,732	27,563	0.75	15,606	16,706	0.93
Associate degree	25,875	30,497	0.85	20,780	19,505	1.07
Bachelor's degree	29,392	37,360	0.79	25,451	23,871	1.07
Master's degree	36,651	44,289	0.83	32,052	30,888	1.04
Professional degree	—†	1,124	—	—	37,151	—
Doctoral degree	—	53,022	—	—	38,247	—

* Source: reference 31.
† Cell omitted because $n < 75{,}000$.

services, and a given income has less real value.[33,34] Wealth disparities arise or compound because accumulated net assets are a direct representation of lifetime disposable income—the cumulative residual value of income after accounting for differential costs.

Median family net worth for blacks and whites in 1991 is shown in Table 2, based on data from the SIPP.[35] The overall median for whites in 1991 was $44,408, whereas for blacks the figure was $4,604. Stratification by income suggests that only when black family income is at least 5–6 *times* as large as white family income can the measures be considered commensurate in asset value. Whatever the mechanism for this disparity, blacks have fewer material resources than whites at equal levels of education and income. Inclusion of SES surrogates will not control confounding if studies do not account for the fact that blacks need to earn considerably more than whites to achieve a comparable material position.

Most published reports of black-white differences would not endure a realistic adjustment for incommensurate SES indicators. For example, Cowie and colleagues[36] examined black-white differences in non-insulin-dependent diabetes mellitus (NIDDM) prevalence using data from the second National Health and Nutrition Examination Survey (NHANES II). Analyzing 471 blacks and 3,908 whites, the authors found a 60% higher prevalence of NIDDM among blacks, and this difference persisted, although only among the obese, after adjustment for several potential confounders. This finding led the investigators to postulate "racial differences in metabolic adaptation to obesity"[36,p.719] and to recommend that the next research step should be "studies of intrinsic and genetic differences by race."[36,p.727] The SES measures considered were annual family income and education level. Income was categorized as <$10,000 and was dropped from the multivariate analysis because the authors used significance testing to exclude potential confounders from the final model. Education was retained in the final multivariate model in scaled categories. The effect of education (>12th *vs* ≤8th grade) was to reduce risk by 30% (95% CI = 0–50%), whereas the adjusted NIDDM prevalence observed in blacks at the highest obesity level was approximately 70% higher than whites (95% CI = 10–280%).

Based on the values reported, we can make only a crude approximations of the degree of bias associated with incommensurate SES measures. Given that blacks receive approximately 90% of the income of whites at a given education level (Table 1), inflation of the education values by 11% for blacks would extend the lower limit of the confidence interval to include values contrary to the published conclusion. Adjustment to equivalent accumulated net assets, however, would be an order of magnitude larger and would therefore erase the adjusted prevalence excess entirely.

Simulations

To describe the degree of residual confounding that occurs in the presence of categorization similar to that found in the report by Brancati and colleagues,[17] we used Stata statistical software[37] to generate simulations of 1,000 blacks and 1,000 whites, with years of education represented by a normal ($\sigma = 1$) random variable. The mean was set at −0.30 for blacks and 0.30 for whites to represent the approximate standardized difference in mean years of education between blacks and whites, ages 45 years and older, in the 1990 National Health Interview Survey (NHIS).[38] A "disease" was then assigned probabilistically, solely on the basis of education, using logistic slope parameters from −0.1 to −1.0. An intercept of $\alpha = 0$ was chosen for all simulations, so that prevalence of disease would be held constant across the range of slope parameters. We then defined a dichotomous education variable with a cutpoint at 0 and analyzed each of 1,000 simulation repetitions using a logistic regression equation that included 0–1 variables for race and education (Table 3).

TABLE 2. Median Family Net Worth (US$), by Race of Householder and Monthly Household Income Quintile (White or Black), 1991*

	Lowest 20%	Second 20%	Middle 20%	Fourth 20%	Highest 20%
Black	1	3,299	7,987	20,547	54,449
White	10,257	25,602	33,503	52,767	129,394
Ratio	<0.01	0.13	0.24	0.39	0.42

* Source: reference 35.

These simulations as-

TABLE 3. The Spurious Association of Black Race with "Disease" Owing to Categorization Bias in the Exposure Variable: Results for Simulations with 1,000 Blacks, 1,000 Whites*

β	Mean Crude OR for Black Race†	Mean Adjusted OR for Black Race‡	% of Repetitions with OR ≥ 1.0§
−0.10	1.07	1.03	60
−0.20	1.13	1.05	70
−0.30	1.19	1.07	74
−0.40	1.27	1.09	81
−0.50	1.33	1.11	86
−0.60	1.40	1.13	90
−0.70	1.47	1.16	92
−0.80	1.53	1.18	94
−0.90	1.59	1.19	96
−1.00	1.65	1.21	97

* "Disease" generated randomly as: $p(d) = \frac{e^{(\beta Z)}}{1 + e^{(\beta Z)}}$; 1,000 repetitions at each β; $Z_{white} \sim N(0.30, 1)$ and $Z_{black} \sim N(-0.30, 1)$, representing "education."
† From the model: logit(disease) = $\alpha + \beta_1$race + ϵ, where race is coded 1 = black, 0 = white.
‡ From the model: logit(disease) = $\alpha + \beta_1$education + $\beta_2$race + ϵ, where education is dichotomized at $Z \geq 0$ and race is coded 1 = black, 0 = white.
§ The percentage of replications with adjusted odds ratios for black race ≥ 1.0.

TABLE 4. The Spurious Association of Black Race with "Disease" Owing to Measurement Error in the Exposure Variable: Results from Simulations with 1,000 Blacks, 1,000 Whites*

β	Mean Crude OR for Black Race†	Mean Adjusted OR for Black Race‡	% of Repetitions with OR ≥ 1.0§
−0.10	1.07	1.04	63
−0.20	1.13	1.07	74
−0.30	1.20	1.10	83
−0.40	1.26	1.12	90
−0.50	1.33	1.16	95
−0.60	1.41	1.20	97
−0.70	1.46	1.22	99
−0.80	1.53	1.26	99
−0.90	1.59	1.29	>99
−1.00	1.65	1.32	>99

* "Disease" generated randomly as: $p(d) = \frac{e^{(\beta Z)}}{1 + e^{(\beta Z)}}$; 1,000 repetitions at each β; $Z_{white} \sim N(0.30, 1)$ and $Z_{black} \sim N(-0.30, 1)$, representing "log(income)."
† From the model: logit(disease) = $\alpha + \beta_1$race + ϵ, where race is coded 1 = black, 0 = white.
‡ From the model: logit(disease) = $\alpha + \beta_1$log(income)* + $\beta_2$race + ϵ, where log(income)* is the sum of log(income) and a random disturbance term which is $\sim N(0, 1)$, and therefore log(income)* is normal with $\sigma = \sqrt{2}$; race is coded 1 = black, 0 = white.
§ The percentage of replications with adjusted odds ratios for black race ≥1.0.

sume a normal distribution of education, whereas the true distribution is irregular. Other covariates, which might contribute additional residual confounding, are not included for the sake of simplicity. Furthermore, a constant effect of education is employed, whereas there are actually inherent discontinuities in the education scale at points of program completion. These simulations are therefore intended only as an illustration of the magnitude of error that might be encountered. With a slope of −0.50, for example, 86% of all repetitions yield odds ratio (OR) estimates greater than 1.0 for an independent effect of race, and this value increases with a larger education difference between groups or a larger β value.[39]

To simulate the degree of residual confounding that is likely in the presence of nontrivial measurement error, we recreated the simulated sets, with ln(family income) represented by a normal ($\sigma = 1$) random variable. The mean was set at −0.30 for blacks and 0.30 for whites to represent the approximate standardized difference in ln(family income) between blacks and whites found in the 1990 NHIS Family Resources Supplement.[38] "Disease" was assigned solely on the basis of family income. A random disturbance ($\mu = 0$, $\sigma = 1$) was added to each income to simulate measurement error. We then predicted disease using the correct model, but on the basis of the income variable containing random measurement error. Estimated coefficients for black race and income were obtained in each of 1,000 repetitions at each true value of β (Table 4).

Again, these simulations only demonstrate the bias that might be encountered under one set of plausible conditions. The magnitude of error encountered when actually measuring SES cannot be determined, since the quantity for which income or other measured values are surrogates is rarely stated. Even if one were willing to assume that income, for example, was the causal quantity, the error in measurement might easily exceed the level simulated here. Longitudinal studies use baseline income to assign risk, for example, whereas Duncan[40] observed that approximately one-third of households followed over a decade experienced drops in annual income of 50% or more at some point. These simulations demonstrate the inability to control for SES by using poor measures of distant surrogates for undefined causal processes. In these simplified circumstances, the probability of *erroneously* detecting an independent effect of race is considerable, and it may be even larger in actual practice. For a demonstration of the effects of aggregating the SES variable, we simulated five standard normal groups ($\sigma = 1$), centered at −3, −1.5, 0, 1.5, and 3 SES units, and the proportion of blacks was set in each group as: 1.00, 0.75, 0.50, 0.25, and 0.00, respectively. We generated values for 5,000 subjects in each repetition, 1,000 in each group. We arbitrarily set a group indicator at values of 0, 1, 2, 3, and 4 (Figure 1) and ran four sets of simulations to demonstrate the effects of specification

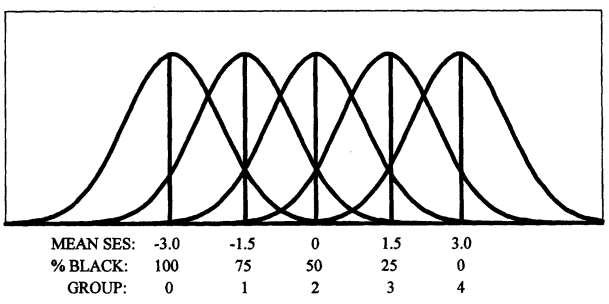

Figure 1. Simulated data: five groups with various mean levels of socioeconomic status (SES) and various proportions of black subjects. Each group is normally distributed, with $\sigma = 1$.

TABLE 5. The Spurious Association of Black Race with "Disease" Owing to Ecologic Assignment of SES: Odds Ratios for Black Race* from Simulations with 2,500 Blacks and 2,500 Whites†

Model	Mean Crude OR for Black Race	Individual SES Measure		Aggregated SES Measure	
		Mean Adjusted OR for Black Race	% of Repetitions with OR ≥ 1.0	Mean Adjusted OR for Black Race	% of Repetitions with OR ≥ 1.0
Model A No independent group effect; black-white distributed randomly within groups	3.72	1.00	50	1.00	50
Model B Independent group effect; black-white distributed randomly within groups	4.31	1.06	74	1.00	49
Model C No independent group effect; black SES < white SES within groups	5.82	1.00	49	2.25	100
Model D Independent group effect; black SES < white SES within groups	6.83	0.97	37	2.25	100

* From the model: logit(disease) = $\alpha + \beta_1$*SES + β_2*race + ϵ, where SES is individual or aggregate and race is coded 1 = black, 0 = white.

† "Disease" generated randomly as: $p(\text{disease}) = \frac{e^{(-1 + \beta 1 * \text{SES} + \beta 2 * \text{GROUP})}}{1 + e^{(-1 + \beta 1 * \text{SES} + \beta 2 * \text{GROUP})}}$; individual SES ~ $N(\mu, 1)$, where $\mu = -3, -1.5, 0, 1.5, 3$; group indicator = 0, 1, 2, 3, 4; 10,000 repetitions of each model (A–D); $\beta_1 = -0.50$ in all models; $\beta_2 = 0.00$ in Models A and C; $\beta_2 = -0.10$ in Models B and D.

and aggregation biases on ORs for race in models adjusted for either individual or aggregate SES. We used mean SES for each group as the aggregate SES variable and ran each of the four sets with 10,000 repetitions. In Simulations A and B, race was distributed randomly within groups. In Simulations C and D, all blacks had lower individual SES than all whites within the same group (Table 5).

In Model A, there was no group effect on risk of disease, and so adjustment for individual and aggregate SES measures both yielded unbiased parameter estimates. In Model B, both individual SES and group made independent contributions to disease risk, but SES was distributed randomly by race within groups. The aggregate model estimate was unbiased, but use of the individual SES indicator created a spurious association between race and disease, because confounding by group was not controlled by including individual SES (specification bias). For Models C and D, race and SES were no longer independent *within* groups. For Model C, the estimate using individual SES was unbiased, because group made no independent contribution to disease, but the estimate based on the aggregate measure was highly biased, leading to observed race effects in 100% of repetitions (aggregation bias). Finally, in Model D, there was SES confounding on both the individual and the group levels. The use of the individual SES variable biased the OR for black race downward, whereas use of the aggregate measure biased the OR for black race upward.

To demonstrate the effects of incommensurate SES variables on confounding control, we recreated the simulated datasets with ln(family wealth) represented by a normal ($\sigma = 1$) random variable. The mean was set at -1.1 for blacks and $+1.1$ for whites, representing the approximate standardized difference between blacks and whites from 1991 SIPP data.[35] A ln(family income) variable was derived by shifting each observation an equal distance toward 0 to yield a 0.6 standard deviation difference between groups. Whereas probability of disease was determined only by wealth, we predicted disease using the income variable and estimated coefficients for race and income in each of 1,000 repetitions at each value of β (Table 6).

In this example, we have assumed that wealth is the causal quantity but analyzed the data using income instead. This is another example of measurement error, with income being a poor surrogate of wealth, although the difference is that the error in this example is correlated with race, yielding a situation in which the income variables for blacks and whites are not commensurate. Ignoring the incommensurability between black and white income is like analyzing incomes for Canadians and Americans without taking into account the fact that a Canadian dollar is equal to about $0.75. In the case of blacks and whites in the United States, the "exchange rate" is apparently closer to about 0.20. Failure to account for this difference leads to results that are not interpretable. Even with relatively modest SES effects, these simulations produced results in which virtually 100% of repetitions showed substantial but completely erroneous race effects. In such instances, a researcher might conclude that race effects "persist" after control for SES and that these results therefore provide the basis for speculation on the biological differences between blacks and whites.

TABLE 6. The Spurious Association of Black Race with "Disease" Owing to the Use of Incommensurate Exposure Variable: Results from Simulations with 1,000 Blacks, 1,000 Whites*

β	Mean Crude OR for Black Race†	Mean Adjusted OR for Black Race‡	% of Repetitions with OR ≥ 1.0§
−0.10	1.25	1.18	95
−0.20	1.56	1.39	100
−0.30	1.90	1.62	100
−0.40	2.34	1.90	100
−0.50	2.86	2.25	100
−0.60	3.41	2.62	100
−0.70	4.04	3.09	100
−0.80	4.74	3.62	100
−0.90	5.49	4.26	100
−1.00	6.26	4.97	100

* "Disease" generated randomly as: $p(d) = \frac{e^{(\beta Z)}}{1 + e^{(\beta Z)}}$; 1,000 repetitions at each β; $Z_{white} \sim N(1.1, 1)$ and $Z_{black} \sim N(-1.1, 1)$, representing "log(wealth)."
† From the model: logit(disease) = $\alpha + \beta_1$race + ϵ, where race is coded 1 = black, 0 = white.
‡ From the model: logit(disease) = $\alpha + \beta_1$log(income) + $\beta_2$race + ϵ, where log(income) = log(wealth) + 0.8 if black, log(income) = log(wealth) − 0.8 if white, and race is coded 1 = black, 0 = white.
§ The percentage of replications with adjusted odds ratios for black race ≥ 1.0.

Conclusion

Previous authors have expressed skepticism over adjusting racial/ethnic comparisons for SES, including Cooper and David,[41] who were concerned about the failure of such a model to account for causal order.[41,p.113] Race is a determinant of SES, they reasoned, and therefore it is not sensible to compare racial groups by controlling for their unequal social positions. Since the confounder is causally subsequent to the exposure, the adjustment is being made on a component of the causal pathway between exposure and disease.

Several authors have also raised concern over the incommensurability of SES measures between blacks and whites.[33,42,43] Krieger *et al*[42] noted the additional deleterious socioeconomic conditions that accompany black racial status in the United States, including differential exposure to environmental toxins, more dangerous occupational conditions, and community-level and individual stressors.[42,pp.84–88] All of these present daunting measurement issues and are not captured by simple income or education categories.

Williams and Collins[33] considered the effect of differential political power, both in terms of individual levels of control and the allocation of societal resources.[33,pp.377–378] Although there exist psychological measures of the degree of control that people have over their lives, the deficit of political power in the hands of African-Americans at the societal level is not a characteristic of the individual and could only be assessed in terms of indirect consequences at the individual level. Adjusting at the individual level for an effect that occurs causally at the societal level cannot logically produce a meaningful model of disease etiology, no matter how refined the measures.

We have provided illustrative examples of residual confounding in studies that attempt to evaluate biological hypotheses about race by controlling for SES variables. This reasoning is common in medical and epidemiologic journals and yet is prone to suggest spurious independent effects for race. Many of these errors result from reliance on significance testing, both in the selection of models and in the interpretation of estimates. The compromise of statistical judgment by relying on significance testing is not sufficient, however, to explain the eagerness with which so many researchers rush to embrace racialist conclusions. We propose that this tendency reflects the influence of social ideology on the conduct of medical science. Questionable techniques may be retained if they provide what is believed to be the "right" answer, and in a society with deeply ingrained beliefs about racial difference, a scientific confirmation of these differences is the expected, and therefore the "right," answer.

The time has come, we believe, to think more carefully about the justification for this analytical interpretation and its consequences, both for epidemiology and for the society as a whole. The social distinction between blacks and whites is multidimensional and cannot be captured fully in a scalar such as education or reported income. To believe that one can account for all social distinctions between these groups by using such a variable and on this basis evaluate a hypothesis about innate physiologic difference is not science, but rather a leap of faith. The faith that our 19th century predecessors had in the innate inferiorities of blacks, women, and immigrants seems absurd to us today.[44] Perhaps we should ask ourselves how our present work will be viewed by the next generation.

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