REVIEWS AND COMMENTARY

The Bell Curve: On Race, Social Class, and Epidemiologic Research

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In a recently published book entitled The Bell Curve: Intelligence and Class Structure in American Life (1, henceforth The Bell Curve), the late Harvard professor of psychology Richard Herrnstein and Heritage Foundation researcher Charles Murray claim that the position of US citizens in the country’s class structure is, to a significant extent, the consequence of inherited differences in “g,” a general factor of intelligence that is measured by intelligence quotient (IQ) tests. Furthermore, these differences in “g” are responsible for racial differences in class position (i.e., some racial groups inherently have more or less “g”). Using a series of regression analyses of data from the National Longitudinal Survey of Youth, which include a proxy for IQ as an independent variable, Herrnstein and Murray suggest that inherited differences in “g” are responsible for black/white differences in behaviors such as crime and having children out of wedlock. The explanations put forward by Herrnstein and Murray regarding the fact that poverty disproportionately affects blacks in the United States (i.e., reflecting inherited differences in “g”) has made race the center of the controversies surrounding the book (2).

The aim of the present commentary is to briefly review the limitations of The Bell Curve claims with regard to racial differences in intelligence and health outcomes and to establish a parallel between the epistemologic stance of the book regarding race and main-stream epidemiologic research. Herrnstein and Murray claim that several conclusions regarding racial differences in tests of cognitive ability are “beyond significant technical dispute” (1, p. 22). Chief among their conclusions are that IQ tests are not biased against ethnic or racial groups and that racial differences in cognitive ability are substantially heritable. The main but unstated assumption behind these conclusions is that the variable “race,” as operationalized in social science and epidemiologic studies (i.e., “black,” “white”), has a fundamentally biologic interpretation.

CURRENT CONTEXT OF “BIOLOGICAL DETERMINISM”

The debate regarding the inheritance of racial differences in IQ is a long-standing academic battle that has sporadically surfaced in the broader public domain over the past several decades. In the late 1960s and early 1970s, similar arguments as those advanced by The Bell Curve were advanced by Jensen (3), Eysenck (4), and Herrnstein (5) himself. Efforts at debunking the claims of hereditarian explanations for racial differences in IQ measures (6–10) were not successful enough to thwart this research program altogether.

Thus, although The Bell Curve has been launched to reach a nonacademic readership (2, 11), its assumptions regarding the operationalization and interpretation of the variable race are frequently found in the academic world (12, 13), including public health (14). The Bell Curve represents an effort to reach large audiences, but its views are in many cases shared by standard research publications working in the “biolog-
 scholarly determinant" paradigm. For example, *The Bell Curve* defends research in developmental psychology that includes the extension of inherited racial differences in cognitive ability to brain size, rate of sexual maturation, length of the menstrual cycle, penis size, infant mortality, and mental health (15).

The research program on biological determinism is also represented in the recent history of epidemiology and public health. An extreme and rare instance is the enduring opposition to considering smoking as a major risk factor for lung cancer and cardiovascular disease, coupled with the attempt to explain away the risk attributed to cigarette smoking in terms of inherited personality traits differentially distributed among racial groups (16). A more prevalent case of biological determinism are studies on the inheritance of cognitive abilities as an explanation for the different location of minorities in the social structure as well as their undermining of welfare policies aimed at improving the health of African-Americans (17).

Given the increasing skepticism among biologists and anthropologists surrounding the use of race as a biologic category (18, 19), its use in epidemiology and public health as an implicit biologic category should be reexamined (20, 21). In biology and anthropology, the resiliency of the biological determinist research program has been explained by the influence of the broader social environment in defining what constitutes worthwhile research (7, 19, 22). In contrast, in epidemiology and public health, the "falsifiability criterion" toward scientific knowledge prevails (23, 24). Consequently, in epidemiology, cautious calls for additional studies allow the perpetuation of hypotheses of race as a biologic category predisposing to illness in major biomedical forums, even when more realistic alternative mechanisms have been suggested (18–21) and even tested (25, 26).

Our attempt is not to censor the authors' version of biological determinism. One of the ethical principles of scientific conduct is tolerance for the test of hypotheses and the use of methods that one dislikes (27). Nevertheless, as the sociology and history of science have shown, decisions about basic assumptions guiding research are social phenomena not understandable by simply monitoring the empirical progress of a given field (22). The scientific community determines in part the acceptability of hypotheses for inquiry, publication, and continued funding through a social process in which certain assumptions are uncritically accepted even in the face of empirical refutation (28). In the biomedical fields including epidemiology, one such assumption is that racial labels such as "black" and "white" classify human beings into groups with genetic homogeneity for health outcomes (20, 29, 30).

**RELEVANCE TO EPIDEMIOLOGIC RESEARCH**

**Significance of causal assumptions underlying the use of the variable race**

Race is widely used in biomedical research, often without any explicit indication of the theoretical construct that its use implies (20). Even basic pathophysiology mechanisms shared by different animal species are systematically studied in humans separately by race without a clear rationale. The underlying and often unstated assumption, however, is that racial differences are mainly genetically determined, which in turn can lead to conclusions that could have profound public health implications, as in the following examples.

**Race-specific standards for hematologic parameters**

Without any evidence from genetic studies, the observation that blacks tend to have lower leukocyte count than whites, for example, led scientists to the conclusion that "neutropenia is probably a normal genetically determined characteristic" in people of African descent (31, p. 1023). Similarly, African-Americans have been reported to have lower hemoglobin values than whites even after "controlling for socioeconomic differences" (32). However, this study, as well as many other epidemiologic studies, do not in fact adequately control for socioeconomic confounders. If racial differences persist after stratification or adjustment by surrogates of socioeconomic position and other risk factors, no matter how imperfect or partial these surrogates are, investigators often conclude that a genetic factor must be playing a role. A basic methodological principle, i.e., that adjusting for an imperfect surrogate of a suspected confounder leads to imperfect adjustment (residual confounding), is unfortunately rarely invoked.

Clearly, despite criticism of the genetic hypothesis to explain racial differences in hematologic parameters (33), the majority of scientists take at face value the "normality" of lower hemoglobin and neutrophil values in blacks. Moreover, some researchers recommend separate hematologic reference values for blacks (34) without concern for the biologic plausibility of the mechanisms linking skin color to hematologic parameters (i.e., the philosophy of pragmatism).

**Race-specific birth weight distributions**

A second example of implicit genetic determinism involves arguments regarding the potential genetic origins of low birth weight in infants from different racial/ethnic groups. Since the 1940s, recommendations for race-specific standards of black and white
infants to define low birth weight have been published
(35-37), and more recently differences in birth weight
distributions between white and Asian infants have
been studied (38). The proposals for race-specific
standards have been fueled by the consistent finding
that low birth weight blacks as defined with the
<2,500-g cutoff have lower perinatal mortality than
white counterparts (39). The subject has been recently
revived after the application of statistical models that
separately fit the population distribution of birth
weight (40) and birth weight-specific mortality for
blacks and whites (41). The result is a standardized
and different birth weight distribution for blacks and
whites. These models eliminate the "apparent para-
dox" of better survival in small black babies since with
these standardized distributions, blacks generally have
greater perinatal mortality (39, 42). These results have
been interpreted as suggesting that the differences in
population distributions of birth weight are genetically
determined (39). However, the authors did not take
full advantage of the information that was available on
social class, such as maternal education (39). The main
argument here is not derived from biologic theory but
is statistical (i.e., their model fits the apparent paradox
of better survival in small black babies as compared
with white babies). However, the predominant factors
causing low birth weight and prematurity may differ
because of unmeasured social exposures determined
by class position and racial discrimination, with dif-
f erent severity in terms of mortality. The mechanical
use of the variable race precludes an understanding of
why black immigrants show much lower low birth
weight rates than United States-born blacks, why
black-white differences in low birth weight rates are
not evident in other nations, or why paternal race does
not affect the birth weight distribution of those born to
white mothers and black fathers (e.g., 43-45). Al-
though adjustment of birth weight to population-
specific weight standards underscores racial differ-
ences in preterm delivery (46), the assumption or
suggestion that such a relevant risk factor might be
genetically linked to skin color (e.g., 38, 39) promotes
a biologic notion of race, providing justification for
more research on the biology of race and making
direct examination of race-associated social depriv-
ation secondary.

Racial differences in blood pressure

A third example of implicit biological determinism
is hypertension, in which an elevated prevalence is
still observed in blacks after adjustment for education
or income levels (47, 48). The genetic origin of the
elevated risk of hypertension in blacks is taken for
granted in epidemiology and medicine. In a recent
review by Cooper and Rotimi (47), the weakness and
inconsistencies of this prevalent dogma are high-
lighted. Critical psychosocial aspects of the lifelong
experience of racial discrimination are not taken into
account. For example, black women who responded
passively to the experience of racial discrimination are
more likely to have high blood pressure than those
who respond actively when faced with discrimination
(25). Studies have also suggested that denial of the
experience of racial discrimination and acceptance of
its associated belief system, in addition to the direct
experience of discrimination in the workplace, pro-
duce adverse effects on blood pressure (20).

Although the role of racial discrimination as a pos-
sible determinant of hypertension has been empha-
sized by a few epidemiologists (29), most investi-
gators continue searching for biologic mechanisms that
would explain an increased genetic susceptibility in
blacks. Increased susceptibility to sodium or sodium
retention is a popular one, although the results are far
from consistent (49). Even if some populations had a
higher sensitivity to sodium, this most likely would
represent a phenotypic difference (47). Speculative
historical hypotheses (e.g., Middle Passage) of this
suspected racial susceptibility to salt intake have been
discredited (47). For example, one may challenge such
hypotheses by noting the impropriety of solely using
US samples to make inferences about white and black
races (26).

As the hypertension research illustrates, the disre-
gard for racial discrimination as an expla-
nation for racial inequalities in health is matched by the lack of
evidence supporting genetic explanations. The con-
sideration that a specific phenotypic trait such as skin
color is a marker for an increased susceptibility to a
wide variety of diseases and pathophysiologic traits
must be justified. Skin color is mostly a reflection of a
common history of exposure to ultraviolet radiation,
and its use for characterizing a discrete "package" of
genetic material has been by now widely discredited in
anthropologic research (19, 29, 47, 50). Since the
1950s, it has been known that human biologic charac-
teristics affected by migration selection or drift are
distributed in geographic gradations or clines such as
those for facial features, hair texture, epidermal mel-
in, ABO alleles, which do not correlate (19). For
example, the gene codes for type B blood increase in
frequency from west to east across Europe and Asia,
reflecting migrations out of Asia, whereas epidermal
melanin is distributed in a decreasing pattern from the
equator to northern latitudes in response to selection
for protection against ultraviolet B radiation. Thus,
discordant patterns of heterogeneity falsify descrip-
tions of populations as if they were genotypical or
even phenotypically homogeneous (i.e., biologic races; 19). Furthermore, 85 percent of human genetic variation is found within human populations rather than between the major populations socially labeled as "races" (51). Therefore, according to the "received view" in physical anthropology and evolutionary biology (19, 30), biologic races appear to be the outcome of social perception.

SUGGESTIONS FOR EPIDEMIOLOGIC RESEARCH

Now is the time for social explanations to constitute the first hypotheses when looking at racial inequalities in health. Different racial/ethnic groups might have different social experiences that affect health. Examples of this are presented in table 1, which is a summary of discrimination mechanisms that might operate as cumulative exposures over the lifetime. It has been empirically demonstrated, for example, that African-Americans are less likely to receive a mortgage loan than whites of similar education and income level (52) and are systematically denied access to middle and upper income housing developments (53). In addition, minority students with the same academic achievements as their white peers are less likely to be included in accelerated tracks in the public school system (54); and minority defendants with identical socioeconomic position as white defendants are subjected to a harsher administration of justice (55). These are examples of differential life experiences that are not accounted for by the standard surrogates of social class.

Another form of implicit biological determinism in epidemiology takes place when researchers use individual level categories (e.g., occupation, race, gender) without an explicit statement of the theory that might explain differences in health outcomes associated with these categories. Thus, some researchers may adopt a pragmatic approach to social epidemiology (56) and conclude that "education" is the preferred social stratification predictor of health. This practice leaves the explanation of what determines education to the reader's implicit theory of causality (57). Because people in general have a tendency toward attributing people's behavior to intrinsic properties of the person (57), biological determinism becomes a likely explanation among biomedically trained scholars in particular (7). This mechanism might be even more compelling when

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<th>Discrimination</th>
<th>Economic</th>
<th>Political</th>
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<td>Access to goods and services (e.g., loans, health insurance, health care, justice, schooling)</td>
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<td>Myers (63)</td>
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<td>Barbarin (67)</td>
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<td>Residential segregation (e.g., housing, environmental exposures)</td>
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<td>Brown (68)</td>
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<td>Access to labor markets (e.g., schooling, training, employment, promotions, primary labor markets) (i.e., &quot;good jobs&quot; with benefits); wages and salaries; workplace hazards; and psychosocial environment</td>
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scholars use categories such as race and gender, which have a long history of biologically determinist folk psychology (58).

The views presented in The Bell Curve are one extreme in what can be conceived as a continuum of racial discriminatory views that permeate all research including medical and public health. Most epidemiologists and public health practitioners probably find the arguments of The Bell Curve outrageous. However, many epidemiologists continue to use the variable race uncritically and with little attention to theory (20, 29, 59). That is, they fail to consider possible social determinants of racial inequalities in health, including mechanisms originating from exposure to multiple forms of racial discrimination (20). Even though considering racial and ethnic minorities to be inherently more susceptible to hypertension, leukopenia, and low birth weight may seem more acceptable than considering them more prone to low performance in “cognitive ability” tests, the rationale for these inferences does not differ essentially from the rationale behind the conclusions of The Bell Curve.

Sociologists routinely spell out their assumptions and mechanisms in their studies of the determinants of poverty (e.g., culture of poverty, biological determinism, social change, and social class (60)). The adoption of similar standards would certainly help the theoretical development of the use of race and social class in epidemiologic research. Even more desirable would be the replacement of topologies such as race or occupation for mechanisms (e.g., exposure to racism, racial identity; work-related stresses) and exposures (20) or social processes including not only economic inequality and power asymmetries (60) but also cultural relations (61) (see table 1, 62–73).

The history of the twentieth century has provided evidence that only a short time is required for beliefs about the biologic origin of differences in class structure to be translated into policy and that these social policies can have devastating consequences (9, 74, 75). The success of the ideas endorsed by The Bell Curve and similar efforts in shaping public health policy will partially depend on the energy with which they are discredited in epidemiologic forums, on methodological and empirical (13, 76) as well as ethical grounds.

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