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ONE STEP BACK IN UNDERSTANDING RACIAL DIFFERENCES IN BIRTH WEIGHT*

TUKUFU ZUBERI

In recognition of the biological and social connections in demographic processes, demographers have integrated biological factors into their models of population variation. This new effort has tended to focus on the analysis of fertility and mortality. Edwin J.C.G. van den Oord and David C. Rowe's article, "Racial Differences in Birth Health Risk: A Quantitative Genetic Approach," published in the August 2000 issue of Demography, is part of this effort. These authors use race as a proxy for genetic variation, which subverts even the most positive attempts to understand the impact of genetic variation on demographic processes. The authors' statistical results restate their anachronistic theory of race using latent variables that are not open to empirical testing. Although new data increase the opportunities for the examination of the relationship between biology and demographic processes researchers must be vigilant not to commit the errors of the past by misusing race as a variable.

In "Racial Differences in Birth Health Risk: A Quantitative Genetic Approach," Edwin J.C.G. van den Oord and David C. Rowe (2000) argue that a combination of racial genetics and environmental factors causes the racial differences in birth health risk. Their effort to integrate biological and social factors into their analysis is commendable. Biological data are increasingly available for demographic research; these data allow researchers the opportunity to examine the relationship between biology and demographic processes. However, van den Oord and Rowe present an anachronistic theory of racial differences and model race inappropriately as a causal effect. These two errors are confounded when the authors gloss over the causal and racial concepts in their research.

RACIAL GENETICS

According to van den Oord and Rowe (2000), races are "genetic entities because generations of 'reproductive isolation' have led to differences in gene frequency across racial groups. During prehistoric times, the physical barrier of the Sahara Desert reduced gene flow between Europeans and sub-Saharan Africans. For many traits, including those physical traits that visibly differentiate the groups (e.g., skin color and physical features), gene frequencies are sharply different" (p. 286). The genetics of racial differences are the focus of van den Oord and Rowe's article.

The authors follow a line of research addressed by James C. Cramer (1995) in his attempt to explain racial differences in birth weight. After controlling statistically for sociodemographic factors, Cramer's model could not explain all of the large racial differences in birth weight. This failure is used by van den Oord and Rowe as justification for pursuing a "quantitative genetic approach."

Their genetic approach consists of a latent variable model of three "theoretical components of variation." This model is thought to represent the joint distribution of birth weight, length of gestation, and birth length (the observed variables) in terms of a simple structure involving heredity, shared environment, and nonshared environment (the unobserved variables). The authors assume that "the genetic and environmental factors do not correlate or interact" (p. 287) in their model. This model is governed by a racialized theory of genetic difference: van den Oord and Rowe believe that heredity corresponds to racial groups "because generations of 'reproductive isolation' have led to differences in gene frequency across racial groups" (p. 286). According to this perspective, people are born and die as members of a racial group. Shared environment refers to "environmental influences that operate to make siblings alike" (p. 285). Nonshared environment corresponds "to environmental effects that are unique to a particular child" (p. 285).

The race variable used by van den Oord and Rowe is measured indirectly and is based on the mother's self-reported racial identification. The authors cannot directly measure the so-called genetic aspect of race. In fact, the basis on which the NLSY mothers reported their racial identification is a social system that uses skin color as the criterion for classification. Theoretically we can group human populations on the basis of skin color. Such a grouping, however, is based on our arbitrary distinctions (Zuberi 2001). The difference in skin color between people of African and European origin is believed to be the result of three to six polygenes (Bowman and Murray 1990:76–77; Wilson 1998:146); yet even in the twenty-first century, the mode of inheriting skin color and the relationship of skin color to health outcomes remain unknown. The constructed measurements of birth weight, birth length, and length of gestation used by van den Oord and Rowe are thought to indicate the unobserved racial genetic and environmental variables.

Human biological variation is real (Marks 1995; Zuberi 2001), but race as a way of organizing that variation is false. This point is important because it contradicts van den Oord and Rowe's perspective concerning the essential and physi-

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cal reality of race. Racial stratification is real, but biology is not its root cause. Race is often referred to as either a biological (anthropological) or a demographic characteristic; in reality it is neither.

RACE: NEVER A CAUSE

Interpretations of statistically based explanations can differ because of different causal theories or different understandings of causal effects. Genetic differences are presented by van den Oord and Rowe as a cause of the racial differences in birth weight. They mistakenly use unobserved entities in their causal inferences. Causation, however, is not a simple matter in statistical analysis of racial differences (Zuberi 2000, 2001). This issue is further complicated when we attempt to use variables that purport to "measure" the genetic basis of race.

Before proceeding further, let us briefly examine the difference between causal theory and causal effect. Causal theory offers a description of the various processes by which a treatment produces its effects. A causal theory can describe the relationship among biological, physical, chemical, and/or social processes. Causal theories should be based on known information about the subject under study. Consequently, causal effects should be regarded as an essential part of causal theories. Causal theories attempt to predict events on the basis of the known empirical information about the subject; this includes what is known about causal effects.

Causal effect refers to the effect of a factor or treatment on a given response variable. In experimental studies, causal theories can be questioned if they contradict the results of the study. In observational data, causal theory serves a more fundamental role: in observational studies, causal effects can be questioned if the causal theory is contradicted by empirical results of a study when the theory is supported by evidence from other studies and by well-developed arguments. The study by van den Oord and Rowe is not an experiment; their data come from the NLSY.

Further, van den Oord and Rowe's racial theory is contradicted by their results. In their conclusion, the authors state, "Our results suggested that the gap between black and white babies' birth weights may be explained by the shared environment of the fetuses, not by fetal genes nor by environmental influences that affect one pregnancy but not another. This finding implies that the relevant environmental factors are correlated with birth health risk, are stable across pregnancies, and have different average levels among blacks and among whites. It is clear that traditional socioeconomic variables such as poverty and years of education fall into this category" (pp. 295-96). Yet in the conclusion, the authors discuss the importance of maternal genes in explaining the difference in birth health risk. They justify the genetic interpretation of their findings despite what is shown in their results.

Racial ideas in demography historically have been influenced by eugenics (McDaniel 1996; Zuberi 2000, 2001). Eugenists argued that racial differences were the result of genetic differences. They sought to establish the connection between biology and behavior outcomes, and in this way to

connect race with population health. According to van den Oord and Rowe, a genetic interpretation of racial differences is supported by evidence from other studies (Chakraborty et al. 1992; Rowe, Vazsonyi, and Flannery 1994; Rushton 1995; Rushton and Bogaert 1987). These studies, however, are inconsistent with the present-day literature on race in health research. The research cited by van den Oord and Rowe is not representative of the literature on the genetic basis of race (for further discussion of this point, see Anderson and Armstead 1995; Chase 1980; Cooper 1984; Fortney 1977; Marks 1995; Terris 1973; Tucker 1994; Wilkinson and King 1987; Yee et al. 1993; Zuberi 2000, 2001).

Statistical analysis cannot be used to establish race as a causal effect. Because most social science researchers study causal effects to make inferences about the effects of manipulations to which groups of individuals in a population have been or might be exposed, causes are only entities which, in theory, can be manipulated or altered (Holland 1986; Sobel 1995). This recognition suggests that van den Oord and Rowe consider the ability of the individuals or units we study to vary. They correctly recognize that researchers can conceptually manipulate the environment effect. Heredity as defined by van den Oord and Rowe, however, cannot vary nor be manipulated for any particular individual; nor, it follows, can racial heredity vary conceptually (Holland 1986; Zuberi 2000, 2001). Yet, van den Oord and Rowe maintain that race is a causal effect of racial differences in birth health outcomes. They argue, incorrectly, that race is a proxy for a genetic relationship.

For latent variables it is necessary for any given unit that the value of the cause could be different (Holland 1986; Sobel 1994). In addition, the researcher must be able to explain how the causal mechanism produces this difference. In van den Oord and Rowe's study, the hereditary cause is an unobserved variable, and they cannot produce different values of racial identity by randomization. In principle, there is nothing problematic about the authors' modeling of covariates they think are sufficient for the treatment assignment to the latent variable (Sobel 1994). The problem lies in their conceptualization of race as a sufficient covariate for treatment assignment. Given their definition of race, it is not possible to imagine how the latent variables could take different values on the same unit, because there is no genetic knowledge about the existence of racial heredity. When race is used as a latent variable, it should not be treated as a cause.

CONCLUSIONS

Cramer defines race as a "social rather than genetic" entity (1995:235). This point is important and is consistent with his effort to understand the social and demographic processes that underlie the racial differences in birth weight. Cramer's study helps us understand how the social status of race affects differences in birth weight. His method of analysis employs standard regression equations and moves us a step closer to understanding this important problem.

The quantitative genetic approach used by van den Oord and Rowe is based on a false notion of heredity, and

in fact is governed by knowledge (or processes) at a different level. Their analysis is not based on genetic research regarding race. Such research suggests that race is not an appropriate basis for discussing biological differences (see Marks 1995). The results of van den Oord and Rowe's study refer to a statistical model and its system of equations, not to the "racial genetics" that they suggest are the empirical basis of their findings.

Nothing in van den Oord and Rowe's article depends on the statistical analysis because the causal inferences pertain to unobservables. The authors avoid the implications of their own statistical results in order to restate their theory, which is tied to unobservables that are not open to empirical testing. The conceptualization of race is fundamental to all subsequent uses of racial data; thus researchers must be cautious when using race as a variable in demographic research.

In recognition of the biological and social connections in demographic processes, demographers have integrated biological and social factors into their models of population variation (for examples of this research, see Adams et al. 1990; Martin and Soldo 1997; Presser 1978; Udry 1986; Wachter and Finch 1997; Weiss 1990). This new effort is an attempt to understand biological processes outside the political framework of eugenics, and has been most successful in the analysis of births and mortality. One potential benefit of this research is its use as a scientific alternative to race as a proxy for genetic variation. The use of race as a proxy subverts even the most positive attempts to understand the impact of genetic variation on demographic processes.

Research in this area has significant potential. In the future, more biological data will be collected in demographic surveys. This new information will create new opportunities to examine the relationship between biology and social and economic processes. Researchers, however, must be careful to not commit the errors of the past by misusing race as a causal variable.

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