THE MISUSE OF BIOLOGY IN DEMOGRAPHIC RESEARCH ON RACIAL/ETHNIC DIFFERENCES: A REPLY TO VAN DEN OORD AND ROWE*

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In an article in the August 2000 issue of Demography *titled "Racial Differences in Birth Health Risk: A Quantitative Genetic Approach," van den Oord and Rowe attempted to study the genetic and environmental factors contributing to the black/white gap in infant birth weight. Their findings indicate that this difference may be explained by shared environmental influences rather than by fetal genes. Yet the authors insisted in their conclusions that a strong genetic component still must play a role in determining the racial gap in birth weight, if only through maternal effects. The incompatibility between the authors' findings and their conclusions is due largely to a weakness in their conceptualization of the relationship between race and biology. Their insistence that racial groups represent discrete genetic entities, coupled with a failure to account for interactions between biological and environmental processes, illustrates the methodological and ethical problems that threaten future interdisciplinary research on racial/ethnic disparities in health.*

Demography is a multidisciplinary field whose course is determined in part by which disciplines are emphasized (Crimmins 1993). In the past decade a relatively new perspective in demographic research, which certainly will play an increasingly important role in the future of demography, involves contributions from the biological sciences. The program at the 2000 annual meetings of the Population Association of America, for example, included sessions titled "Biological Data to Complement Demographic Approach" and "Biosocial Perspectives on Demographic Behavior." In a 1997 paper published in *Demography*, James R. Carey discussed how "demographers can benefit from knowledge of new developments in biology and biomedicine" and argued that studies of biology of death, mortality, longevity, and life are all informed by biological processes that demographers cannot afford to ignore (Carey 1997:26).

This cross-pollination of disciplines has been fruitful in that it has increased the depth of demographic research with respect to processes such as aging, health, and mortality. Yet it is also plagued by a conceptual imprecision regarding the relationship between race and biology. In his 1999 presidential address to the Population Association of America, Andrew Cherlin (1999) alluded to this potential problem: he noted social scientists' reluctance to incorporate biological research, specifically genetics, into their work, and cited political concerns involving the use of genetic arguments in the past to deny equal rights and opportunities to women and minorities.[1] This concern is especially relevant for demographic research in which race/ethnicity remains a key variable of interest. Particularly for social demographers, the explanation of racial/ethnic disparities across a variety of demographic outcomes is a primary research focus (Hummer, Rogers, and Eberstein 1998). For these reasons, as demography moves toward an increasingly interdisciplinary relationship with the biological sciences, demographers must be clear on the relationship between race and biology, which has long been misunderstood (Brace 1982; Cooper 1984; Cooper and David 1986; Hirschman, Alba, and Farley 2000; Williams, Lavizzo-Mourey, and Warren 1994).

The tensions inherent in incorporating biological insights into demographic research were expressed most recently in the area of racial/ethnic disparities in infant health outcomes. According to the preliminary 1999 vital statistics estimates, African American infants are more than twice as likely as non-Hispanic white infants to be born at low weights (< 2,500 grams) and almost three times as likely to be born at very low weights (< 1,500 grams) (Curtin and Martin 2000). In turn, slightly more than two-thirds of the racial/ethnic difference in neonatal mortality (which accounts for two-thirds of the difference in black and white infants' mortality rates) is explained by the elevated rate of black infants with birth weights of less than 1,500 grams (Wise 1993). The strong association between birth weight and infant mortality, and the racial/ethnic differences involved in this association, have led to intense scrutiny of the causes and consequences of racial differentials in birth weight.

Social demographers typically argue that these differences are due to structural inequalities and historical legacies of discrimination, which have formed intractable disparities across a wide array of health outcomes. They maintain that more causally distant factors, such as socioeconomic status and discriminatory histories, affect ultimate health outcomes through an array of proximate determinants such as access to health care and behavioral characteristics (Hummer et al. 1998). Income, education, marital status, and receipt of financial assistance are among the numerous socioeconomic factors that have been implicated in contributing to the racial gap in birth weight (Cramer 1995).

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1. J. Richard Udry made a similar statement in his 1994 PAA presidential address, in which he argued that one reason why social scientists have been reluctant to consider a biological gender theory is that it is considered "politically incorrect; some call it 'sexist' and 'ideological'" (Udry 1994:563).

Yet despite the substantial portion of the gap in infant birth weight that is explained by sociodemographic factors, large racial differences remain unexplained. The failure of statistical models to account for these differences have led many researchers to question the quality of existing measurements, controls, and data (Kaufman, Cooper, and McGee 1997; LaVeist 1996; McDermott 1998). These critics cite a failure to adequately measure other dimensions of economic status, such as duration of poverty and assets of wealth, as well as a reluctance to consider the importance of race and racism apart from economic status (Williams 1997; Williams and Collins 1995). In addition, we lack data on contextual effects and have insufficient information on specific behavioral and biological pathways through which sociodemographic differences are realized. As a result we are limited in our ability to explain many of these racial/ethnic differentials (Collins and David 1997; LeClere et al. 1997).

In an alternative explanation for statistical models' failure to explain racial/ethnic differences in birth weight, some researchers claim that genetic factors are responsible: that is, genetic differences between racial groups result in differential distributions of infant birth weight (e.g., Wilcox and Russell 1989). This latter argument was articulated most recently in the August 2000 issue of *Demography*, in a paper titled "Racial Differences in Birth Health Risk: A Quantitative Genetic Approach" (van den Oord and Rowe 2000). There the authors attempt to study the genetic and environmental factors contributing to the black/white gap in birth weight. Citing the failure of previous analyses to explain this differential, they directed their attention toward genetic explanations. They argued that it is reasonable to expect that genetic differences may partially explain the gap because racial groups are "both genetic and social categories" (p. 286). The authors argued that races constitute genetic entities because "generations of 'reproductive isolation' have led to differences in gene frequency across racial groups" and because, "[d]uring prehistoric times, the physical barrier of the Sahara Desert reduced gene flow between Europeans and sub-Saharan Africans" (van den Oord and Rowe 2000:286).[2]

In past literature, this conceptualization of race was labeled "a biological concept of race" in that racial taxonomies are viewed as meaningful classifications of genetic differences between human population groups (Cooper 1984; Williams 1997; Williams et al. 1994; Witzig 1996). With respect to health outcomes, it follows that as races have evolved separately, they have come to possess different gene pools with different gene frequencies, which in turn result in differential health outcomes across racial groups (Templeton 1999).

The authors used this conceptualization of race and genetics as the cornerstone of their analysis, even though it runs afoul of the available scientific evidence showing that racial categories cannot appropriately capture biological distinctiveness because human biological variation is not racially patterned.[3] Because racial groups do not meet any scientific criteria (Brace et al. 1993), the biological nature of a widely distributed population cannot be understood sufficiently if racial categories are used as the starting point. For this reason anthropologists, in whose discipline the concept of race most naturally belongs, have argued that races do not exist (Brace 1964, 1982, 1996; Livingstone 1964; Montagu 1942, 1964). This means, at the very best, that in health research, the use of racial categories as genetic markers is equivalent to basing fine-grained statistical analyses on an imprecise, constantly changing, contextually loaded categorization (Goodman 1997; Marks 1994, 1995). At worst, the use of racial groups as rough proxies for biological variation impinges on empirical design, restricts exploration of additional causal factors, and reinforces the arguments of a small number of researchers who argue that genetic racial differences actually connote hierarchical differences between racial groups. The article by van den Oord and Rowe illustrates all three of these attendant dangers.

Using data from the National Longitudinal Survey of Youth (NLSY), the authors examine six separate survey years in which they divide the children into groups of full and half-siblings. Then they decompose the variation in their outcome variable, "birth health risk," into three theoretical components of variation: heredity, shared environment, and nonshared environmental influences.[4] The shared environment component involves the influences that are identical for siblings and are estimated from the resemblance of siblings' birth health risks. The nonshared environmental influences refer to environmental effects that are uncorrelated across pairs of biological relatives and are estimated according to siblings' dissimilarity in a trait. Heredity or genetic similarity is estimated by comparing biological half-siblings with biological full siblings according to whether correlations in birth health risk occupy the same rank order as the biological relatedness of the pairs.

Relevant to the present discussion is that the authors constrained their causal model so that "the genetic and environmental factors do not correlate or interact" (p. 287). This assumption contradicts the fact that traits are influenced by many factors, typically several or many genes acting together with environmental factors (Hartl and Clark 1997:41). In this sense, most polygenic traits are considered multifactorial: that is, they are influenced by several genes acting in conjunction with environmental effects. For example, as Cartmill (1999) noted, "[E]verything in principle is 100% limited by both heredity and environment…with the environment determining to the extent to which a given trait is influenced by genetic factors" (p. 657).

2. Reproductive continuity and gene flow across the Sahara have been sufficient to ensure that differences were never a product of isolation by distance or genetic drift, as the authors implied.

3. In an alternative conceptualization set forth in the anthropology literature, human biological variation is captured with a cline and cluster approach: clines represent geographic gradients of particular biological traits, and clusters involve those trivial biological traits that are not connected to any particular selective force but instead are perpetuated by heredity. This approach and its application in anthropology have been discussed extensively elsewhere (see Brace 1964, 1996; Brace and Hunt 1990).

4. The authors used three variables to measure birth health risk: birth weight, length of gestation, and birth length.

Health outcomes are created by various physiologic processes that are preceded by an elaborate schema involving interactions between biological and social factors. One must be capable of integrating the complex of interactions that may intervene between gene products and their ultimate phenotypic expression (Roberts 1995; Stern and González 2000).[5] The failure of van den Oord and Rowe to allow for these interactions, coupled with their insistence that racial groups constitute appropriate categories with which to understand human biological variation, culminates in their analysis of the findings.

To evaluate the relative importance of the various components in explaining the racial gap in "birth health risk," the authors specified an independent pathway model. For each variable in the model, they computed the total genetic and environmental contribution to the racial differences in means by multiplying in each group, for each given component, the path coefficient and the deviation from the overall mean. The authors then compared the contributions to the mean difference in order to determine which components most fully explain the racial differential in birth weight. The main finding from the model is that although shared environmental influences may account for this difference, fetal genes do not appear to contribute to racial differences in birth weight.

Yet despite their finding, or lack thereof, regarding a genetic contribution to the racial gap in birth weight, the authors insisted that a strong genetic component must play a role. When their analysis failed to provide the genetic contribution in the form of fetal genes, they cast around for an alternative genetic explanation and settled on maternal genetic effects, which are not measured explicitly in their analyses. Because shared environmental effects must be stable across pregnancies, the authors argued that maternal genetic effects are the source of this shared influence, reasoning that these effects are unlikely to change substantially between births. Again, they stated that these maternal genetic effects can reasonably be expected to differ systematically across racial groups; they did so by invoking their assumption that racial groups are reliable categories with which to capture human genetic variation.

Williams and Collins (1995) maintained that fundamental assumptions about what race is will shape the research questions on racial disparities in health and will influence the interpretation of findings. In the analysis by van den Oord and Rowe, the underlying assumption is that, "in addition to the obvious environmental differences, there are demonstrable genetic differences consisting of variations in gene frequencies across racial groups" (p. 297). Furthermore, the genetic component in their model is immutable in that the environmental and the genetic contributions to determining infant birth weight are not permitted to interact. These assumptions are the most obvious explanation for why their conclusions so blatantly contradict their own findings. It is also likely that these assumptions explain why the authors ignored other well-known and widely disseminated research examining how physiological pathways for growth are influenced by environmental and social factors, and in turn affect the uterine environment across pregnancies.

In human biology research, extensive literature examines the ways in which physiologic pathways for growth are changed in direct response to environmental and social influences, such as access to health care, better nutrition, education, and positive health practices (Bogin 1995; Pritchard 1995). These indices vary systematically by racial/ethnic group and are much more likely to contribute to between-group differences in birth weight through interpregnancy physiologic influences on the uterine environment. In a review of research on racial/ethnic differences in diabetes prevalence, McDermott (1998) observed that the overriding concern with racial genetic effects has come at the cost of further investigation into more mutable causes of diabetes, such as inherited (or metabolically adapted) and therefore changeable susceptibility. Indeed, the importance of evaluating these more mutable causes of racial/ethnic disparities in health, as opposed to racial genetic effects, is underscored by Lasker, who stated,

> [T]oday there is resurgent nationalism based on racism (the false notion that all of what one can do is predominantly imbedded in genes derived from a circumscribed biological race). Therefore a full understanding of the role and extent of biological plasticity by which the messages of DNA are modified in the anatomy and behavior of people deserves wide dissemination. (Lasker 1995:113)

An even more egregious set of ethical violations complements the methodological errors committed when racial groups are conceptualized as discrete genetic entities. Goodman (1997) argued that researchers fall into a number of groups with regard to understanding race and biology. The most malignant are the "true believers," who subscribe to the typological distinctions that imply hierarchical rankings of worth across different races. Although this group remains small, the members' work is often widely publicized and well known (e.g., Herrnstein and Murray 1994; Rushton 1991). At the opposite extreme are those who believe that biologically distinct races do not exist and that "race-as-bad-biology has nothing to do with race-as-lived-experience" (Goodman 1997:22).

Between these two extremes falls a third group, by far the largest. Goodman called this the "confused category," in which racial biology is perceived as an issue of political correctness, and in which race continues to be applied in a quasi-genetic manner. As Hoffman (1994) observed, "[I]n certain respects scientists are as confused as the rest of us about how race matters" (p. 4). Goodman concluded that the "soft" use of racial categories is omnipresent and that its continued use in health research only reinforces the "hard" conceptualization of race as advocated by a small group of researchers. In a discussion of the controversial book *The Bell Curve*, in which Herrnstein and Murray (1994) argued that racial differences in intelligence exist, Muntaner, Nieto, and O'Campo (1996) asserted that a parallel exists between

5. For an excellent diagram and summation of the multifaceted role of race in determining health outcomes, see Williams (1997).

those authors' epistemological stance and mainstream health research. They stated that the main conclusion in *The Bell Curve* is that the variable "race" has a fundamentally biological interpretation, and that research in public health has done nothing to reject this understanding but rather everything to support it (p. 531).

The point here is not that a biological conceptualization of race should be avoided for fear of supporting a racist agenda. Rather, such a conceptualization should not be used in health research because human biological variation is not racially patterned. The use of racial categories as a starting point for understanding genetic variation represents shoddy and imprecise research, leading to the misspecification of models and the misinterpretation of findings. In addition to these methodological flaws, researchers should be aware that the tacit support of a biological conceptualization of race sets a dangerous precedent. It gives credence to the discredited findings of disreputable researchers: those who argue explicitly that there are many measurable genetic differences between racial groups, and implicitly that these racial differences connote hierarchical differences in worth.

In their conclusion, van den Oord and Rowe asserted that "to produce a comprehensive picture, the possible role of genetic factors should be evaluated empirically, or at least considered" (p. 297). I argue, however, that before demographers attempt to account empirically for the role of genetic factors in contributing to racial/ethnic disparities in health outcomes, they should be conceptually clear on the relationship between race and biology. This includes the understanding that race is not a valid biological category but a second-rate, contextually loaded proxy for an infinitely complex pattern of human biological variation.[6]

In conducting analyses based on the assumption that races connote discrete biological entities, perhaps the most troublesome consequence is a lack of attention to the ways in which biological processes and social categories in fact interact to produce differential health outcomes. With reference to the relationship between racial categories and health status, Hayward et al. (2000) observed that "(b)iology's major role is to define the array of possible physiologic responses to the social, economic, and environmental conditions...these conditions influence the expression of genetic susceptibility although the specific relationships are not well documented" (p. 913).

In regard to racial disparities in birth weight, future research need not avoid contributions from the biological sciences. Yet these contributions must not be forced to fit an outdated conceptual framework that posits discrete genetic differences between racial groups. Instead, more attention should be given to deciphering the biological pathways through which sociodemographic differences are realized. A growing appreciation for gene-environment interactions, coupled with increased scrutiny of the measurements, controls, and data currently available, will allow demographers to push past anachronistic conceptualizations of race in their attempt to explain the persistent racial gap in birth weight.

The trend toward an increasing collaboration between biological and demographic researchers has belatedly exposed demographers to complications that other disciplines have grappled with for a long time. Yet the recency of our exposure to these problems is also advantageous in that it gives demographers the opportunity to avoid making those mistakes that are so deeply entrenched in the research of other disciplines. Writing with regard to epidemiological research, Bhopal (1999) warned that those in the twenty-first century may view much of the present-day research on race no more favorably than we view the work of race scientists of the nineteenth century. As demographers we are in a position to take advantage of the cross-disciplinary nature of our field and to welcome a new era of research—one that is not based on an outdated conceptualization of race but instead acknowledges the social, environmental, and biological complexity underlying the existence and persistence of racial/ethnic disparities.

6. In a paper that appeared in the same issue of *Demography* as van den Oord and Rowe's article, Hirschman et al. (2000) favored recognizing "the artificial nature of the concept of race and the increasing patchiness of the schema of measurement" (p. 390).

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