Understanding ethnic differences in energy balance: can we get there from here?¹,²

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The striking excess of obesity in US black women has been observed for decades (1) and continues to be reconfirmed (2). When consideration of obesity prevalence is expanded to include men and other ethnic minority populations, a similar finding of excess obesity is noted in men and women in other minority groups, but not in black men (3). Increased prevalence of obesity in US black children suggests that, as in some other minority populations, there is a heightened predisposition to obesity in black females throughout the life span (4).

There are numerous behavioral or environmentally determined candidates for the causes of excess obesity in minority populations. These include factors leading to energy intake in excess of physiologic needs, to energy expenditure levels too low to offset energy intake, or to factors limiting intentional weight loss. Theoretically, a population group with a higher-than-average predisposition to obesity must have more individuals who have or who are hypersensitive to such variables when present. Given the striking differences in obesity prevalence, it seems straightforward to compare these variables between ethnic groups to both pinpoint the nature of the ethnic differences and derive clues to the causes of obesity itself. In reality, however, the ability to reap the benefits of such ethnic comparisons is fraught with both conceptual and methodologic problems, and it is not clear that this approach is taking us where we want to go.

The article in this issue by Weyer et al (5) is a case in point with respect to the potential for ethnic comparisons to raise at least as many questions as they answer. Based primarily on the findings of prior studies in Pima Indians, the hypothesis of the study was that a low resting energy expenditure and high respiratory quotient (indicative of a low rate of fat oxidation) are markers of the predisposition to obesity. Weyer et al used a cross-sectional design to compare measurements of black and white women and men taken over a 24-h period under tightly controlled conditions in a respiratory chamber. A main finding of the study was lower resting energy expenditure (in this case, sleeping metabolic rate) in black than in white women but no such difference between black and white men. This is at least consistent with the findings of a substantial number of studies reporting lower resting energy expenditure in black than in white females of various ages and weights and with the epidemiologic finding that black men are not more obese than white men. The other main finding suggested that black men are at excess risk of obesity compared with white men because of a higher respiratory quotient, a finding that has been generally less common.

These sex differences underscore the importance of including both men and women in such studies as well as the potential added complexity of doing so. Other interesting findings of sex differences include a report of differential sensitivity of black men and women to the effects of insulin on lipid and glucose metabolism (6) as well as possible sex-relevant effects of fat distribution (amount of abdominal fat) on energy metabolism (7).

Attempts to use ethnic comparisons to understand potential behavioral and psychosocial influences on obesity have been relatively successful. Although many links are yet to be made, findings consistent with an excess of obesity determinants in black compared with white women have been reported frequently, eg, lower physical activity, a more obesity-tolerant body image, greater energy intake, greater weight gain during pregnancy, greater difficulty losing weight, shorter duration of weight loss attempts, less smoking, and lower alcohol intakes (1–4). To some extent it has been possible to reconcile the behavioral findings with the expectation that black women are more frequently obese than black men (eg, men are not affected by pregnancy-related weight gain). To the extent that all of these variables are influenced by social and cultural factors, ethnic comparisons make conceptual sense. The problems of interpretation relate primarily to methodologic variables, such as the ability to separate socioeconomic factors (eg, low income, which predisposes both black and white women to excess obesity) from other ethnicity-related factors.

The failure of social and behavioral variables to explain the black-white differences in female obesity (8), as well as the tendency to persist in viewing ethnicity as a biological variable (9), motivates the search for other risk factors. In this context, it is certainly reasonable to pursue the theory of “thrifty genes” even though the US black population is at a different stage of the epidemiologic transition than are populations such as the Pima Indians. However, studies of variables such as energy metabolism, which are also strongly influenced by environmental or adaptive influences, are extremely complex. As in the study by Weyer et al (5), such comparisons often involve attempts to standardize

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the ethnicity of the participants within each group (eg, selection for self-reported US black or white ancestry in both parents and grandparents) as well as painstaking efforts to control for the known influences on energy metabolism or on the measurement of energy metabolism (eg, age, sex, hormonal status, prior energy intake and physical activity, usual diet and activity patterns, smoking, alcohol intake, body size and body composition, and weight stability), particularly because these variables often vary by ethnicity. Even when these factors are rigorously controlled, however, what is left are phenotypes (“races”) that, although often recognizable by physical characteristics, are not at all genetically homogeneous. The somewhat vague “ethnic” or “racial” comparison that results does not necessarily lead to any particular hypothesis or next step, even when a positive result is found. In the absence of genotyping for genes known or thought to be related to energy regulation, the chances of establishing that an ethnic difference is not either sociobehavioral or methodologic in origin might be viewed as very small.

The finding of Weyer et al in women has the most support elsewhere in the literature and one can argue that the repeated finding in different samples of lower resting energy expenditure in black than white women is probable evidence of an underlying biological (although not necessarily genetic) truth. One could also argue that the similarity in obesity predisposition across a variety of ethnic groups of presumably different genetic admixture and, indeed, among women of European descent with very low socioeconomic status, is probably evidence of a common environmental factor that overwhelms any genetic contribution.

Along with the critical conceptual problem of what is actually being compared come the considerable methodologic issues involved in obtaining any valid group-level comparison of energy-metabolism variables. The more rigorous and detailed the study, the smaller the number of subjects might be or the fewer and farther between the repeated measures that might support a fuller understanding of the mechanisms of weight gain. The study conditions themselves tend to be artificial, eg, there are extreme constraints on physical activity in a metabolic chamber such that the observation of total energy expenditure is distorted with respect to free-living conditions. The number of factors to potentially be controlled for is large, and no one study, including that of Weyer et al, seems to be able to control for all of them. For example, there was no standardization or measurement of menstrual cycle phase and only age was used as a crude proxy for menopausal status. Thus, one must decide, on scientific grounds if possible (but arbitrarily if the literature does not provide a consistent answer), whether the uncontrolled factors could have made a difference. For example, are race-specific assumptions needed when fat-free mass is adjusted for?

For the moment, it is unclear how the Weyer et al findings relate to black-white differences in obesity. I note that cross-sectional comparisons of putative thrifty gene variables are often null, including a study comparing Pima Indians in Mexico and Arizona with different rates of obesity and different environments (10). Again, this emphasizes the importance of asking how this issue should be studied. If the issue is predisposition to obesity, and especially if becoming obese changes the entire metabolic picture, then studies within an ethnic group living in the same environment but followed over time should provide more useful answers. Factors found not to differ significantly between blacks and whites might still differ between black women who gain excessive weight over time and those who do not. Identification of such within-population factors would lead more directly to an answer about how to address the issue of obesity in a high-risk group.

REFERENCES