Nature + nurture > 100%: genetic and environmental influences on child obesity

Dear Sir:

In a recent article in the Journal describing the genetic influence of child adiposity, Wardle et al (1) provide an important confirmation of the genetic contribution to child obesity and waist circumference. Readers should be cautious, however, to avoid 2 common misconceptions when interpreting such studies.

The atomistic or individualist fallacy occurs when population inferences are made based on individual-level data (2). For example, imagine a hypothetical sample of children from 2 classrooms. Classroom A is instructed by an incompetent teacher in a very resource-poor setting. Classroom B is taught by a highly competent teacher in a resource-rich setting. Researchers interested in the relation between intelligence quotient (IQ) and academic achievement over time conduct assessments on all of the children from both classrooms over several years. Low and behold, they discover that IQ strongly predicts academic achievement within each of these classrooms over time; in fact, it accounts for 80% of the variance in math and reading performance within each group of children. However, researchers fail to note the escalating overall academic achievement of Classroom B over time contrasted with the declining performance of Classroom A. A dangerous conclusion is potentially drawn. Because IQ predicts 80% of the variance in achievement within each classroom, then across all classrooms everywhere, academic success must be largely attributed to IQ. Therefore, we need not waste time and money on improving educational environments in resource-poor settings, because we could only hope to influence the remaining 20% of variance in academic achievement.

The problems with these conclusions are 2-fold. First, caution should be taken in making inferences for a population based on observed relations at the individual level, as noted in the atomistic fallacy. Individual differences in IQ may not explain much of the variance in the differences between Classroom A and B in achievement or classroom A’s decreased performance over time. Second, the fraction of achievement that can be accounted for by IQ and the fraction that can be accounted for by environmental factors need not sum to one. It is a common misconception that all of the causal components leading to certain diseases or health outcomes must sum to 100%. For instance, Wardle et al stated that, (p 403) “strongly genetic conditions—notably, phenylketonuria (PKU)—have proved to be entirely treatable by environmental interventions.” One could feasibly argue that, because the condition can be treated entirely through changes in environmental conditions, mental retardation related to PKU is wholly environmentally determined. Similarly, one could also argue that PKU is a strongly genetic condition and thus is wholly genetically determined. The fact is that both conditions are necessary, but neither is sufficient to cause PKU-related mental retardation. In this example, the genetic and environmental determinants completely overlap. Failing to account for such gene-environment interactions or overlaps may result in an inaccurate estimation of the individual effects of each of these causal components. A parallel can certainly be drawn to obesity and numerous other health outcomes.

Wardle et al appropriately noted that (p 403), “the epidemic of obesity is attributed squarely to changes in the environment, whereas individual differences are attributed to genetic differences between individual persons.” However, the title of the article, “Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment” is a bit of a misnomer. Authors did not assess the influence of the obesogenic environment on child weight status, except by exclusion. That is to say, whatever variance in individual body weight unaccounted for by genetics was taken to be the sum total environmental influence, thus ignoring the potential gene-environment interaction. As noted above, nature and nurture need not be exclusionary and need not sum to one. Additionally, despite the authors’ distinction between the root cause of population shifts toward higher body weights and the determinants of individual differences in body weights, readers could easily fall prey to the atomistic fallacy in interpreting such studies. For example, one could conclude that well-documented racial-ethnic or socioeconomic disparities in obesity might be attributable to genetic differences. Such misinterpretations may tragically undermine public health efforts toward reducing disparities and ameliorating the obesity epidemic across populations.

The author had no conflicts of interest.

Lauren M Gibbons

1703 East West Highway
#203
Silver Spring, MD 20910
E-mail: lgibbons@jhsph.edu

REFERENCES