Commentary: Should I blame mom or dad? Identifying the relative contribution of each parent’s body size to that of their offspring

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There is increasing agreement that preventing obesity in childhood is a sensible long-term approach to halting the obesity epidemic. However, there is little consensus about how early in life to start interventions to prevent obesity, what these interventions should be and whether these interventions should focus on all children or only on some children. Despite interest in beginning interventions early in life, we still don’t understand the aetiologies of the many conditions that are lumped together under the diagnosis of obesity. Further, obesity is a diagnosis that most newborns will not experience in their lifetime, and many who do experience it, will not be impaired by it. Nonetheless, the prevalence of childhood obesity continues to increase with forecasts of enormous societal costs and reduced life expectancy for today’s children. It appears as urgent to act as it does to avoid harm.

How can the field of epidemiology help address this dilemma? Before embarking on an epidemiological investigation in childhood obesity, researchers might benefit from asking if their results (usually their hypothesized results) can help address either of two broad goals. One goal would be to provide evidence, beyond what already exists, that a particular risk factor is, in fact, a cause of obesity. Such evidence might make that risk factor a candidate for: (i) an intervention to alter the risk factor or (ii) an experimental study to understand if the risk factor can be altered and to more definitively identify the risk factor as a causal one. A second goal might be to identify a factor associated with obesity in order to stimulate further investigation, often in conjunction with other disciplines, into the mechanism by which the factor might cause obesity.

The article by Griffiths et al.1 in this issue of the International Journal of Epidemiology is one that is intended to stimulate understanding of mechanism rather than to inform intervention. Although the authors use the word obesity only once, obesity is the most relevant application of their findings. The authors propose a mechanism to statistically test the difference in the relative contribution of the size of each parent to a child’s birth weight and rate of weight gain during infancy—two factors associated with the development of obesity.2,3 The primary findings are that the size (height and weight) of each parent contributes equally to their child’s weight gain during infancy, but that the child’s birth weight is more influenced by the mother’s weight than the father’s. Where mechanism is concerned, these findings are most consistent with the mother’s weight and related metabolic status having influence over the intrauterine environment and fetal growth in a way that the father’s weight cannot. However, it is sobering to note that the proportion of the variance in birth weight or infant weight gain explained by parent size is quite small and that birth weight and infant weight gain, in turn, explain only a small proportion of the variability in later weight.

Even as the study’s findings encourage continued investigation into the mechanisms by which the intrauterine environment contributes to obesity risk in the offspring, some care is needed in applying the study’s methodological innovation. At the clinical level, advancing our understanding of the mechanisms by which nature and nurture interact to produce health and disease in children inevitably leads one to ask which parent is making the greater contribution to a child’s outcomes. In any well-intentioned effort to parse the relative contribution of each parent, we must remain aware that the methods to do so could be used by some to assign credit or blame to one parent. For example, such methods could be used to test the differential contribution of each parent to a child’s IQ, another number, like the body mass index, which is packed with emotional meaning and which is the result of the complex interplay between genes and environment.

Parents in many countries now face the challenge of rearing their children to have a healthy weight and body image in an environment that fights against both. Epidemiological research that assigns a relative contribution of each parent to a child’s weight poses the risk that some might use this information to split parents over an issue on which they need to be united in support of their child. Many of the collective decisions our society has made to advance itself, from how we build our neighbourhoods to how we transport ourselves to how we obtain our food, appear to have contributed to a harm we never intended—the obesity epidemic. As we make advances in epidemiological research—in the questions we ask, in the methods we apply and in the way we interpret our findings—we should also remain vigilant about unintended harms.

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