As many writers of news headlines are all too keen to tell us, obesity is an epidemic that has an enormous impact on health care systems throughout the developed world, as it increases the risk for type 2 diabetes, cardiovascular disease, and various other ailments. Media reports on obesity are often alarming, so much so that one recent headline, drawn from statements made by a public health expert in the United Kingdom, equated the threat of obesity with that of terrorism (1). The “nature versus nurture” debate has been central to the discussion about the rising incidence of obesity. Some consider obesity to be the result of overconsumption of food and limited exercise. Logically, this problem can be fixed by eating less and exercising more. However, recent studies have indicated that things might not be quite as straightforward as this for everyone. Indeed, we now know that many factors contribute to whether a person is likely to become obese if their calorie intake is excessive and that these factors can have very subtle and complex effects. One factor that influences whether a person is at risk of becoming obese is genetic makeup. During the past two decades, many genes that control appetite, energy expenditure, and fat metabolism have been discovered. In some cases, mutations in single genes can lead to […]
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The “nature versus nurture” debate has been central to the discussion about the rising incidence of obesity. Some consider obesity to be the result of overconsumption of food and limited exercise. Logically, this problem can be fixed by eating less and exercising more. However, recent studies have indicated that things might be many such genes, each exerting a modest effect and thereby reduce their risk of becoming obese. Earlier this year, a study reported in the *Journal of Clinical Investigation* (JCI) that the fact that fat cell numbers do not decrease following significant weight loss (5). The researchers found that weight loss was instead associated with a decrease in the size of the fat cells. Kirsty Spalding, the lead author of the study, told the JCI that the fact that fat cell numbers do not decrease following significant weight loss might help explain why many individuals who are obese struggle to keep weight off after losing it—they are not losing the fat cells, but simply reducing their size.

Despite new research identifying factors beyond the control of an individual as influencing that person’s risk of becoming obese, the debate surrounding the causation of obesity and what to do about it is likely to continue. For those who favor an environmental/behavioral etiology, the rational explanation is the first law of thermodynamics, whereby energy intake equals energy expenditure; obesity results when energy intake exceeds expenditure. However, as Rexford Ahima (an expert in CNS regulation of body weight and energy balance at the University of Pennsylvania) summed up nicely for the JCI, “The notion that ‘a calorie is a calorie’ is attractive but fails to address the complexity of energy metabolism and how excess fat storage leads to disease. Given that most people consume more calories than they expend, it is remarkable that some manage to stay thin. Beyond the headlines, the fact remains that obesity is a complex trait, and progress on this front will require greater understanding of gene-environment interactions.”

Karen Honey