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PERSPECTIVE

What is obesity?

Obesity Musings

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What is the definition of obesity, what is obesity, and what causes or contributes to obesity, are three similar sounding questions with very different answers. Obesity is defined using body mass index (BMI) cutoffs to reflect the associated health risk. Various cutoffs for BMI or the amount or distribution of body fat serve as an indicator of overweight and obesity in descriptive statistics. The BMI cutoff for adult obesity was around 27 kg/m^2, which later changed to current cutoff of 30 kg/m^2 in the Western world. It continues to be challenging to establish one cutoff for the Asian population [1]. Thus, the definition of obesity seems labile. Then, what is obesity?

Human body is evolved to store energy surplus as fat for use during periods of energy deficit. Physiological controls appear to be in place to restrict this fat accumulation in a certain range [2]. Yet, certain individuals exceed that range while others do not. Obesity is excessive accumulation of body fat, that results from the impairment in energy balance mechanisms [3, 4]. Example of the condition edema can illustrate this point. Edema is not a result of drinking excess water. In health, water balance is exquisitely controlled, but is disrupted in some diseases, which leads to excess water accumulation. Similarly, several centrally and peripherally acting mechanisms influence long- and short-term energy intake and expenditure, including hunger and satiety hormones, various adipokines, cytokines, hormones, and thermogenic adipose tissue. As elaborated previously [3, 4], the disruption in one or more of these regulatory mechanisms of energy balance can lead to excess energy storage, and may eventually result in obesity. Thus, obesity is a consequence of impaired energy balance.

This leads to the third question about the causes and contributors of obesity. It is posited that most "causes" of obesity are intrinsic whereas "contributors" are extrinsic to the human body. Causes could be considered as impairment in energy balance regulation due to defects in physiology or behavior which predispose an individual to excessive energy storage. Considering that genes can influence brain function and behavior [5, 6], genes may have a hitherto under-recognized role to play in obesity development. Whereas, "contributors" are the factors such as energy dense or palatable foods, or low levels of physical activity, which make it easier to achieve positive energy balance in presence of "causes". For example, active thermogenic adipose tissue is more in individuals who are lean and less in obesity [7]. While this is not established as a causation, a scenario could be conjectured. Overfeeding increases total daily energy expenditure (TDEE), and the increase in TDEE shows considerable interindividual variation [8]. When the energy intake is excessive, an adequately functioning thermogenic adipose tissue may dissipate excess energy and help maintain a stable body weight, whereas, an inadequate thermogenic response may lead to weight gain. While the underlying reasons are unclear, widely different responses in weight gain to identical positive energy balance have been well documented [9]. It seems that causes and contributors are both needed to express obesity, or at least one of them needs to be present in overwhelming proportion. For example, mechanisms exist in an individual to resist excessive weight gain or weight loss [2, 10]. Yet, those mechanisms could be overwhelmed by excessive positive or negative energy balance to alter body weight.

These concepts have practical significance in realistic obesity management and prevention. For example, in obesity research, it should no longer be adequate to conclude that obesity is due to overeating. It is important to understand which upstream defect in physiology leads to that overeating. A true prevention of obesity would entail preventing the intrinsic causes of obesity such as leptin deficiency or a delayed onset of satiety hormones. Until medical science learns to regulate these causative factors, we are left with controlling the contributory factors such as energy intake and activity. These will not "prevent" obesity—the defect in energy balance mechanism, but may help attenuate its expression.

Nikhil V. Dhurandhar [1]✉
[1]*Department of Nutritional Sciences, Texas Tech University, Lubbock, TX 79409, USA.* ✉*email: Nikhil.Dhurandhar@TTU.EDU*

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COMPETING INTERESTS

The author declares no competing interests.

ADDITIONAL INFORMATION

Correspondence and requests for materials should be addressed to Nikhil V. Dhurandhar.