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COMMENTARY

Striking a balance: Orexigenic and energy-consuming effects of energy expenditure on body weight

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Energy balance (EB) is often depicted as a set of kitchen scales in which the size of two quantities (energy intake [EI] and energy expenditure [EE]) determines the existence of a positive or negative EB and a gain or loss of weight. This gives rise to the slogan urging people living with obesity to "move more and eat less." This request is perceived as an unhelpful health message and is, moreover, based on an assumption of a static EB system. In reality, EB is a complex biobehavioral system in which EI influences EE and EE modulates EI. The effect of EI on EE is represented by the thermic effect of food, but the effects of EE on EI remain underappreciated (1). The insightful study by Piaggi et al. in the current issue of *Obesity* (2) aimed to quantify the orexigenic effect of EE on body weight alongside its more commonly acknowledged "energy-consuming" effect. Partitioning these effects into independent pathways is a statistical rather than biological reflection of EB regulation, but this approach highlights the multiple pathways through which EE directly or indirectly influences body weight. Approaches that quantify these counteracting effects, and the factors that alter their partitioning, may provide new insight into why and when compensatory changes in EB are seen following EE perturbation.

Partitioning the effects of EE on body weight between orexigenic and energy-consuming pathways is likely to be individually subtle and influenced by a range of biological (e.g., age, sex, body composition, genetics) and behavioral factors (e.g., diet selection, physical activity). An important question arising from Piaggi et al.'s study is how malleable this balance between counteracting effects is and whether this partitioning is altered under differing states of EB. Indeed, of practical relevance is how increases in EE induced via exercise or physical activity alter this balance. Whereas chronic exercise training can lead to an increase in EI, sedentariness does not downregulate EI (3). Although this might initially suggest that chronic exercise leads to greater orexigenic partitioning, under conditions of high energy flux (achieved via elevated physical activity rather than body weight), physiological adaptations may occur that favor the maintenance of a "healthy" body weight, including better control of appetite (4). The effects of EE on body weight and its partitioning between orexigenic and energy-consuming pathways will likely reflect the physiological and experimental environment in which EE is characterized, and therefore these effects should not be expected to be constant within or between individuals.

When considering the effects of EE on EI, it is also important to distinguish between absolute changes in EI and overconsumption (i.e., EI in excess of energy requirements). Although EE was not experimentally manipulated, Piaggi et al. suggest that "increased EE may drive overeating, thereby promoting future weight gain." This implies that EE is tightly coupled with EI, and that increases in EE will stimulate EI in excess of energy requirements. However, studies have suggested a loose coupling between EE and EI (3), and, although logical that the body's demand for energy may exert influence on day-to-day EI (1), whether increases in EE are a causal driver of overconsumption per se is debatable. Indeed, the "net effect" of the orexigenic and energy-consuming effects reported by Piaggi et al. favored a negative EB, but body weight increased over the follow-up period (3.4 [7.5] kg over ~1.7 years). This suggests that factors other than EE-induced increases in orexigenic drive may promote overeating and weight gain. A workable hypothesis is that EE represents a drive to eat but will lead to overconsumption only when coupled with a high-energy-dense diet. Food choice, and the energy density of foods selected, have been shown to

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be key in promoting passive overconsumption as a risk factor for weight gain (5). Importantly, food choice is not deeply rooted in homeostatic principles but is embodied in cultural and social issues. Therefore, it is important to recognize that inappropriate food intake and overconsumption are much more than the result of biological dysregulation.

CONFLICT OF INTEREST

The authors declared no conflict of interest.

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