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Commentary to Accompany

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Physical Activity and the Missing Calories

The obesity epidemic is widely thought to result from sustained high food intake combined with limited physical activity, which is further exacerbated by our maladaptation to these conditions. According to geneticist James Neel's "Thrifty Gene Hypothesis," the human genome evolved 'thrifty genes' via natural selection to help individuals fatten more quickly during periods of feasting and survive better during times of famine or when exposed to predators (2,6). While the thrifty genotype conferred a survival advantage to our hunter-gatherer ancestors, these genes are detrimental in our modern society where food is perpetually abundant and there are fewer demands for physical activity. This evolutionary hypothesis is widely endorsed as a plausible explanation for the high prevalence of obesity and type-two diabetes in modern society.

In this issue of the journal, Pontzer proposes a similar evolutionary hypothesis that natural selection has shaped our physiology to conserve energy (3). He begins by stating that scientists typically assume energy expenditure to be additive (factorial); total daily energy expenditure is the sum of resting metabolic rate (energy necessary for maintenance and repair), the thermic effect of feeding (energy needed to digest, absorb, and store the ingested food), and the energy cost of structured and spontaneous physical activity. According to the additive model, an increase in physical activity always increases energy expenditure.

In contrast to the additive model, Pontzer postulates that daily total energy expenditure is rather stable, despite rather large changes in physical activity. Invoking evolutionary principles, Pontzer proposes a "constrained energy expenditure" model, where energy balance is homeostatically regulated. In Pontzer's model, total daily energy expenditure (adjusted for body weight) does not increase in proportion to physical activity, but instead is constrained within a rather surprising narrow range that favors evolutionary survival. In support of this model, Pontzer presents data from both humans and many animal species showing that total daily energy expenditure is not influenced much by physical activity. Pontzer then provides ecological reasons why high levels of physical activity do not alter energy balance as much as expected, thus explaining why physical activity often has little impact on weight-loss strategies.

Although it is known that extra calories burned through exercise often do not translate into weight loss, the lack of exercise efficacy—those "missing calories"—has been attributed to compensation through increased food intake. However, Pontzer argues that the missing calories can be explained by a decrease in non-activity energy expenditure, particularly resting energy expenditure, rather than an increase in food intake. In support of this constrained energy expenditure model, Pontzer cites evidence of no difference in energy expenditure between wild and captive primates. He also compares populations in different settings in which they have high food availability with low physical activity (Westernized societies) versus high physical activity with limited food availability, such as in the Hadza, a modern-day hunter-gatherer population in Africa.

However, there are compelling data from humans showing that energy intake is indeed increased in response to exercise (e.g., (1)). Unfortunately, the evidence presented by Pontzer does not include data on energy balance, and it is possible that these constraints on energy expenditure only operate under certain conditions of ill-favored negative energy balance. Another possibility is that energy expenditure does not increase with structured physical activity due to decreases in spontaneous physical activity (e.g., fidgeting) or reductions in the energy cost of physical activity (e.g., improved muscle efficiency), rather than from reductions in resting metabolic rate. Indeed, spontaneous physical activity can account for a large amount of caloric expenditure even in sedentary conditions and may represent a buffer against changes in structured physical activity (5). Moreover, as Pontzer admits, athletes and subsistence farmers present a clear challenge to his model because they exceed the proposed limits on energy expenditure, even after correcting for body size (3). And even in the case of the Hadza who have comparable energy expenditure to Westerners once adjusted for fat-free mass, the Hadza expend more energy in physical activity and are leaner than their Western counterparts (4). Therefore, despite a possible upper limit on energy expenditure, physical activity can reduce adiposity.

Despite some limitations with his model, Pontzer makes an important contribution to convincingly show that increases in physical activity often do not translate into increases in total energy expenditure. Moreover, he demonstrates that models of energy expenditure and physical activity must accommodate both evolutionary and ecologic pressures in the context of maintaining energy balance.

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References

1. Charlot K, Chapelot D. Energy compensation after an aerobic exercise session in high-fat/low-fit and low-fat/high-fit young male subjects. Br J Nutr. 2013; 110(6):1133–42. Epub 2013/03/20. 10.1017/S0007114513000044 [PubMed: 23506960]
2. Neel JV, Weder AB, Julius S. Type II diabetes, essential hypertension, and obesity as “syndromes of impaired genetic homeostasis”: The “thrifty genotype” hypothesis enters the 21st century. Perspectives in Biology and Medicine. 1998; 42(1):44–74. [PubMed: 9894356]

3. Pontzer H. Constrained Total Energy Expenditure and the Evolutionary Biology of Energy Balance. Exercise and Sport Sciences Reviews. 2015

4. Pontzer H, Raichlen DA, Wood BM, Mabulla AZ, Racette SB, Marlowe FW. Hunter-gatherer energetics and human obesity. PLoS One. 2012; 7(7):e40503. Epub 2012/08/01. 10.1371/journal.pone.0040503 [PubMed: 22848382]

5. Ravussin E, Lillioja S, Anderson TE, Christin L, Bogardus C. Determinants of 24-Hour Energy-Expenditure in Man - Methods and Results Using a Respiratory Chamber. Journal of Clinical Investigation. 1986; 78(6):1568–78.10.1172/Jci112749 [PubMed: 3782471]

6. Speakman JR. A nonadaptive scenario explaining the genetic predisposition to obesity: the “predation release” hypothesis. Cell Metab. 2007; 6(1):5–12.10.1016/j.cmet.2007.06.004 [PubMed: 17618852]