Editorial

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Variations in energy intake: it is more complicated than we think

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Weight gain and the resulting obesity is fundamentally the consequence of chronic energy imbalance, with energy intake exceeding energy expenditure, which then leads to excessive energy (fat) storage. Since the rise of the obesity epidemic, decades of research effort have gone into understanding the biological, cognitive, psychosocial, behavioral, and environmental factors that drive food intake and energy expenditure. What went wrong that made us more prone to store energy? Are we eating more, exercising less, or do our bodies just tend to hold on to body fat? Although a lot of work has been done on what contributes physiologically and psychologically to the 2 sides of the energy balance equation (energy in and energy out), it is less clear how these 2 sides work with each other to modulate energy homeostasis. The article by McNeil et al. (1) in this issue of the Journal is a secondary analysis that uses compiled data from 7 studies with a comprehensive set of objective measures of energy requirement, energy intake, appetite sensations, and eating behavior traits. In individuals who are weight stable and presumably in (or close to) energy balance, the authors showed that resting metabolic rate (RMR) and fat-free mass (FFM) are the major determinants of energy intake. Appetite and eating behavior ratings were only associated with daily, but not acute (single-meal), energy intake, with fasting prospective food consumption ratings accounting for approximately one-third of the remaining variance in daily energy intake that is unexplained by basal metabolic needs. This study uses one of the largest data sets in the field to elucidate the relation between energy intake and expenditure and provides new quantitative insights into the interactions between biological and behavioral variables that affect how much we eat.

A key finding of this study—that RMR and FFM are the strongest determinants of energy intake—largely confirms the positive correlation between RMR and FFM and energy intake reported elsewhere (2, 3). Such a relation is not surprising given that the first law of thermodynamics predicts that all energy input will be spent in physiologic processes under conditions of energy balance (i.e., energy in = energy out). Our studies and others have consistently shown that RMR and FFM, when considered independently, both account for ~70% of sedentary 24-h energy expenditure in weight-stable individuals, irrespective of metabolic phenotype (4). The almost interchangeable role of RMR and FFM in energy expenditure is likely to reflect the energy requirement of metabolically active components of the human body, including muscle tissues and the vital organs (heart, liver, gastrointestinal tract, and brain). These data argue for feeding behaviors being at least partly driven by the physiologic demand of energy to maintain metabolic mass. Another key component of the energy balance equation that was not addressed in the article by McNeil et al. (1) is activity-related energy expenditure (AREE), which accounts for 15–50% of total daily energy expenditure depending on the level of physical activity (5). From an energy equilibrium perspective, we could easily see AREE together with RMR as the key biological drivers for perceived need for food. Being the most variable component of energy expenditure, we hypothesize that AREE is a key factor that probably contributes to fasting prospective food-consumption ratings, which the authors identified as a major determinant of variations in energy intake.

The notion of food intake as a function of energy requirement was proposed as early as the 1950s (6), but surprisingly, there is very little biological evidence of causality between energy expenditure (requirement) and intake, let alone evidence of the underlying molecular mechanisms. It is tempting to hypothesize that a signal or signals from metabolically active tissues regulate feeding behavior, just as leptin inhibits food intake (white adipose tissue deposition increases leptin secretion, which negatively affects appetite signaling pathways to reduce energy intake) (7). The search for such "energy-demanding" signals, however, is no easy task. Skeletal muscle, heart, liver, brain, and kidney are the major organs that contribute to RMR (8) and each of them may produce different signals that are most likely to converge in a downstream signaling pathway to exert collective effects on food intake driven by biological needs. Identifying such molecular events may be critical for understanding metabolic adaptation, a largely unresolved phenomenon of energy expenditure dissociated from metabolic mass during dynamic weight changes in response to caloric restriction or overfeeding.

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Abbreviations used: AREE, activity-related energy expenditure; FFM, fat-free mass; RMR, resting metabolic rate.

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Finally, the amount of food that we choose to eat is undoubtedly not as simple as fulfilling our energy requirement. Cognitive, psychosocial, and emotional factors, even the gut microbiota, have all been implicated in appetite regulation. There are also current hypotheses that depict a role of diet composition, or quality of diet, to affect food intake, irrespective of energy requirement (9, 10). With obesity still being perceived by many as a problem of "overeating," understanding how and why energy intakes differ across individuals is fundamentally important. For this we need to distinguish "true variations" from "measurement errors." From the good-old food diary and questionnaire to image-assisted dietary assessment and fMRI, we are moving toward more objective (and presumably more accurate and precise) measures of energy intake and eating behaviors. We are now able to take more reliable energy intake measurements in the laboratory setting, but acquiring high-quality data under free-living conditions remains a huge challenge. The use of a combination of doubly labeled water and precise measures of body composition [known as the intake-balance method (11)] is perhaps the most accurate and precise way to measure long-term energy intake, but this method is technically demanding and details of specific food consumption (e.g., composition and meal times) are not captured. In an ideal world, we need novel methods that are easy to use, that cause minimal interference with everyday life, and at the same time provide a wide array of accurate and precise real-time measurements of food intake. We are still in need of developing a device, almost like a "collar around the neck," that would precisely count the ingested calories from protein, carbohydrate, and fat.

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REFERENCES

1. McNeil J, Lamothe G, Cameron JD, Riou M-È, Cadieux S, Lafrenière J, Goldfield G, Willbond S, Prud'homme D, Doucet É. Investigating predictors of eating: is resting metabolic rate really the strongest proxy of energy intake? Am J Clin Nutr 2017;106:1206–12.
2. Caudwell P, Finlayson G, Gibbons C, Hopkins M, King N, Naslund E, Blundell JE. Resting metabolic rate is associated with hunger, self-determined meal size, and daily energy intake and may represent a marker for appetite. Am J Clin Nutr 2013;97:7–14.
3. Blundell JE, Caudwell P, Gibbons C, Hopkins M, Naslund E, King NA, Finlayson G. Body composition and appetite: fat-free mass (but not fat mass or BMI) is positively associated with self-determined meal size and daily energy intake in humans. Br J Nutr 2012;107:445–9.
4. Ravussin E, Burnand B, Schutz Y, Jequier E. Twenty-four-hour energy expenditure and resting metabolic rate in obese, moderately obese, and control subjects. Am J Clin Nutr 1982;35:566–73.
5. Levine JA. Non-exercise activity thermogenesis (NEAT). Nutr Rev 2004;62:S82–97.
6. Edholm OG, Fletcher JG, Widdowson EM, McCance RA. The energy expenditure and food intake of individual men. Br J Nutr 1955; 9:286–300.
7. Könner AC, Klockener T, Bruning JC. Control of energy homeostasis by insulin and leptin: targeting the arcuate nucleus and beyond. Physiol Behav 2009;97:632–8.
8. Wang Z, Ying Z, Bosy-Westphal A, Zhang J, Schautz B, Later W, Heymsfield SB, Muller MJ. Specific metabolic rates of major organs and tissues across adulthood: evaluation by mechanistic model of resting energy expenditure. Am J Clin Nutr 2010;92:1369–77.
9. Ludwig DS, Friedman MI. Increasing adiposity: consequence or cause of overeating? JAMA 2014;311:2167–8.
10. Simpson SJ, Raubenheimer D. Obesity: the protein leverage hypothesis. Obes Rev 2005;6:133–42.
11. de Jonge L, DeLany JP, Nguyen T, Howard J, Hadley EC, Redman LM, Ravussin E. Validation study of energy expenditure and intake during calorie restriction using doubly labeled water and changes in body composition. Am J Clin Nutr 2007;85:73–9.