

Snacking and Energy Balance

Richard D. Mattes, MPH, PhD, RD

The high prevalence and severe consequences of overweight and obesity are now well established. The less well-understood issue is what is driving this energy imbalance. Though some have argued decreased physical activity is largely responsible, findings from the gold standard method to measure energy expenditure—doubly-labeled water—clearly indicate this is not the case. There has been no substantive change of energy expenditure over the past forty years, and absolute expenditure matches predictions based on cross-species analyses.¹ The decreased expenditure due to lower physical activity is offset by the increased energy required to support the increasing fat mass. Thus, attention is focused on energy intake. Intake is determined by how much energy is consumed in each eating event and how frequently eating occurs. Theoretically, there is reciprocity between the two actions so that it is possible to eat large portions infrequently or small portions frequently, and maintain body weight equally well under each scenario. However, the rising prevalence of overweight/obesity documents that such reciprocity is not precise. Much attention has centered on increasing portion sizes and insensitivity and/or disregard for physiological cues to moderate intake. Considerable evidence supports this view. However, the role of increased eating frequency has not received comparable consideration and may be the larger problem.² Snacking is not synonymous with eating frequency as meals may be skipped, but it is a very close proxy. This review will focus on the health effects of snacking.

The first issue related to the study of snacking is to define the term. This has proven to be highly problematic. Common definitions are based on when an eating event occurs (eg, mid-morning, mid-afternoon, evening), type of food consumed (eg, salty and sweet single-serve convenience items), energy content (eg, researcher defined—fixed amount, less than a meal), some combination of these, or consumer defined (eg, consumer identifies an eating event as a “snack”). The latter is used most commonly in epidemiological studies. No definition is both tightly defined and ecologically valid. Snacks can provide more energy than meals (also not well defined), occur at any time of day and at any interval relative to “meals,” and may be comprised of any type of food. So, clinical interventionists tend to define snacks by energy content and timing of consumption with known questionable ecological validity while epidemiologists base decisions on consumer self-report, with poor precision as a consequence. Nevertheless, there is unequivocal evidence that by any definition, the prevalence of snack and eating frequency has been increasing. It is now estimated that there is a mean of approximately 2.25 snacking events per day per person in the US population.³

The energy contributed by snacks is less well characterized. National Health and Nutrition Examination Survey (NHANES) data indicate snacks contribute approximately 600 kcal/d for males and 450 kcal/d for females, and the trend dating back to the 1971-1974 survey is steadily increasing for both sexes. Indeed, the increment in snacking energy has outpaced the energy contributed by meals (Fig 1).³ So, in summary, snacking is prevalent, increasing in frequency and accounts for approximately 20-25% of daily energy intake.

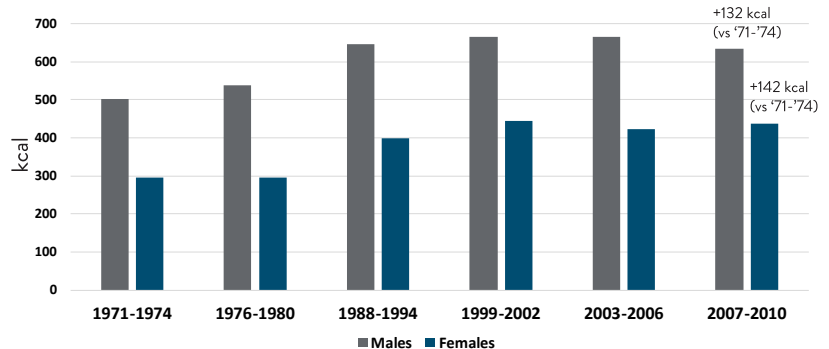


Fig 1. Trends in snack energy intake among US adults. National Health and Nutrition Examination Survey (NHANES) data.³ NHANES energy from snacks (among all population average; 1971-2010, P trend all <0.0001)

Given this high contribution of energy, it would be expected that snacking would be clearly associated with weight gain and overweight/obesity. There are data demonstrating this,⁴ but NHANES data are equivocal. How can this be reconciled? Under-reporting is a well-known shortcoming of collecting dietary intake data in free-living individuals. In NHANES data, the lack of association between snacking and body weight reported with the full sample shifts to a strong positive association when implausible reporters are excluded.⁵ This holds for males and females across the life cycle. The clinical trials are similarly not straight-forward. However, in a recent narrative review, it was noted that in 16 of the 18 published clinical trials, the study duration was less than or equal to only 8 weeks.⁶ This is an inadequate timeframe to measure body weight as an outcome. Additionally, in randomized clinical trials exploring the association between eating frequency and appetite, none had a sample size greater than 20 and for 9/12 trials, the duration was less than one day.⁶ For an outcome of known high variability (appetite), this again is an inadequate basis to draw conclusions. So, in summary, the epidemiologic evidence supports an association between eating frequency and body weight when plausible reports are assessed and the clinical literature is not sufficiently robust to evaluate this association.

A full understanding of the contribution of snacking to health requires identification of mechanisms that could account for such a relationship. A large number of options have been tested. First, it is uncontested that the food industry has made the food supply highly palatable, convenient, reasonably priced and readily available. Moreover, social norms have shifted making it acceptable to eat in diverse locations and at almost any time of day. Thus, snacking has been enabled. Second, snacks may paradoxically augment hunger. Eating small amounts in the absence of hunger can increase the conscious desire to eat for any number of reasons (eg, boredom, sensory stimulation) and the generally smaller energy load of snacks fails to exert a strong effect on hunger. Third, the timing of snacks leads to short-term shifts in appetite that are no longer relevant when planned eating events occur.⁷ Thus, snacks tend to add to the energy content of the diet rather than displace other energy sources (eg, smaller meals). Fourth, it is possible that by increasing eating frequency through snacking, the normal endocrine signaling systems that modulate intake are desensitized.⁸ That is, they fail to show clear increases and decreases and this flux may be the functional dimension of the signaling system. Some have argued that snacking later in the evening is especially problematic for weight gain, but this has not proven robust. If this were the case, one might expect European nations with a cuisine that entails late night eating (eg, Spain) should have the highest prevalence of overweight/obesity, but this is not the case.⁹ Fifth, though the literature is mixed, multiple trials have demonstrated that eating fewer times per day is associated with reduced postprandial thermogenesis (energy dissipation as heat). Thus, more energy is available for storage. This is especially true with irregular eating patterns.¹⁰

Snacks do not necessarily result in positive energy balance and weight gain. Some are energy neutral. Nuts are a good example. Because they are highly satiating, their energy is not efficiently extracted and they may lead to elevated energy expenditure when consumed chronically. The literature shows nuts can be incorporated in a healthful diet without posing a risk for weight gain.¹¹ Other foods may behave similarly, but this must be documented.

Having argued snacks pose a threat for weight gain and increased health risk, it should be acknowledged that sub-groups of the population (eg, patients with cancer, some elderly) would benefit from this outcome. For these individuals, snacks comprised largely of beverages are likely to be the most effective and they lead to the weakest compensatory response. High protein foods may hold some greater satiety value than the other macronutrients, but the differences between them are small and of short duration.

The issue of snacking and energy balance aside, snacks may present opportunities to improve nutrient intake and overall health. The nutrient density and effects on other outcomes such as postprandial glycemia or lipemia should be evaluated before judging the impact of snacks on health.

References:

1. Westerterp KR, Speakman JR. Physical activity energy expenditure has not declined since the 1980s and matches energy expenditure of wild mammals. *Intl J Obes (Lond)*. 2008;32(8):1256-1263.
2. Mattes R. Energy intake and obesity: ingestive frequency outweighs portion size. *Physiol Behav*. 2014;134:110-118.
3. Kant AK, Graubard BI. 40-year trends in meal and snack eating behaviors of American adults. *J Acad Nutr Diet*. 2015;115(1):50-63.
4. Kahleova H, Lloren JI, Mashchak A, Hill M, Fraser GE. Meal frequency and timing are associated with changes in body mass index in Adventist Health Study 2. *J Nutr*. 2017;147(9):1722-1728.
5. Murakami K, Livingstone MB. Eating frequency is positively associated with overweight and central obesity in US Adults. *J Nutr*. 2015;145(12):2715-2724.
6. Hutchison AT, Heilbronn LK. Metabolic impacts of altering meal frequency and timing – Does when we eat matter? *Biochimie*. 2016;124:187-197.
7. Porrini M, Santangelo A, Crovetti R, Riso P, Testolin G, Blundell JE. Weight, protein, fat, and timing of preloads affect food intake. *Physiol Behav*. 1997;62(3):563-570.
8. Solomon TP, Chambers ES, Jeukendrup AE, Toogood AA, Blannin AK. The effect of feeding frequency on insulin and ghrelin responses in human subjects. *Br J Nutr*. 2008;100(4):810-819.
9. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2014;384(9945):766-781.
10. Farshchi HR, Taylor MA, Macdonald IA. Beneficial metabolic effects of regular meal frequency on dietary thermogenesis, insulin sensitivity, and fasting lipid profiles in healthy obese women. *Am J Clin Nutr*. 2005;81(1):16-24.
11. Tan SY, Dhillon J, Mattes RD. A review of the effects of nuts on appetite, food intake, metabolism, and body weight. *Am J Clin Nutr*. 2014;100(suppl 1):412S-422S.

Disclaimer: This summary does not imply any endorsement of Abbott or Abbott products.