The Role of Snacking in Energy Balance: a Biobehavioral Approach\textsuperscript{1,2}

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Abstract

Snacking is often presumed to contribute to obesity, but to date, studies have not demonstrated such a causal relationship, probably because a clear definition of snacking is still elusive. The usual one, i.e. any intake between traditional meals, has no physiological basis. Moreover, because some evidence suggests that frequent meals may prevent overweight, any confusion between snacks and meals may mask the deleterious effect of snacks on energy balance. Therefore, we developed a biobehavioral approach to assess whether objective criteria for eating a meal and snacking could be determined. Our main findings were that regardless of the time of consumption or macronutrient composition, snacks exerted a weak satiety effect, with those higher in protein having the strongest. The energy content of snacks was never compensated for at the next meal and led consistently to a positive energy balance compared with no-snack conditions. Biologically, the snack-induced insulin secretion suppressed the late increase in plasma FFA, which may have contributed to the inhibition of satiety. Lastly, snacking was not preceded by the glucose and insulin profile observed prior to a spontaneously requested meal. In conclusion, further studies on the role of snacking in energy balance should include criteria other than nutrient composition or consumption between meals for defining these eating occasions as snacks. J. Nutr. 141: 158S–162S, 2011.

Introduction

Snacking is often considered to contribute to the epidemic of obesity via a disruption of the homeostatic control of eating behavior leading to overeating. Unfortunately, epidemiological studies have not provided much argument in support of this claim. Several possible explanations of the failure to demonstrate a deleterious role of snacking in energy balance have been listed elsewhere (1). However, one, which is always underrecognized, is that no clear difference between a high meal frequency and snacking is possible with the current definitions. Snacking is often defined as any eating occasion between meals, but the question then becomes: what is a meal? Usually, it is considered to be breakfast, lunch, and dinner, but this is more a historical and sociological definition than a physiological one. For example, during childhood, the dietary pattern consists of 4–8 meals/d, each one preceded by a clear endogenous motivation to eat, at least during the first few years of life. The fact that some of them are considered as snacks and not meals has no scientific justification; rather, the labeling comes from terminology commonly used. An accurate distinction between meals and snacks is important, because they are hypothesized to have opposite effects on energy balance. Specifically, a high meal frequency may prevent fat mass deposition, yet snacking may contribute to it (2). Moreover, the absence of a clear definition of meals precludes disseminating this information on a wide scale, because the risk is that it will encourage snacking, even if in one study, snacking did not impair weight loss during an obesity treatment (3). There is therefore a need for distinguishing between meals and snacks. This is all the more urgent that even defined as extra-meal intakes, the contribution of snacks to the daily energy intake is increasing in most countries. In Irish adolescents (4), 30\% of daily energy intake was derived from snacks in 1997 and \textgreater32\% in 2005. In the US, snacking (defined as intake of foods over a 15-min period and that were not included at a meal) was reported to have increased in prevalence from 71 to 97\% between 1977–1978 and 2003–2006, with a contribution to daily energy intake increasing from 18 to 24\% over the same time period (5). In 2005, among a population of American university students, 20\% reported eating a morning snack, 54\% an afternoon snack, and 73\% an evening/late-night snack, with an increase of these frequencies in upper- compared with lower-level students (6).

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Snacking and overweight

One important review about the role of snacking in energy balance was published in 1996 by Drummond et al. (9). The analysis of the literature was drawn from a definition of a snack as “...any food taken outside a regular mealtime (breakfast, lunch and dinner) or snack item taken in place of such meal.” It was therefore a dual definition, because snacks could be food items or eating occasions. On that basis, the authors’ conclusion was that there was “...evidence that snacking may not predispose to overweight and may have positive advantages in terms of body weight control.” Until recently, this conclusion had not received a clear rebuttal.

However, over the past several years, some studies have provided some support to the hypothesis of a detrimental role of snacking, mostly defined as between-meal eating occasions, in overweight and obesity. Among them was the XENDOS study, showing that obese individuals were more frequent snackers than nonoverweight participants and that eating snacks was positively correlated to energy intake, irrespective of physical activity (10). Then, in assessing whether snacking was associated with meal skipping, Avinge et al. (11) began to introduce the necessary distinction between these 2 eating occasions. They actually found that adolescents who snacked were more likely to skip meals (considered here as breakfast, lunch, and dinner). In the study by Howarth et al. (12), the categorization of snacks was improved even further, because participants self-reported the type of eating occasion according to a 6-category list: breakfast, brunch, lunch, supper, dinner, and snacks. Brunch was recoded as breakfast or lunch, and supper recoded as lunch or dinner and implausible energy intake reporters (defined as within ± 22% of predicted energy requirements) were excluded from the analysis. Results showed that younger overweight and obese individuals and older obese individuals had higher snack intake than their normal weight counterparts. However, similar results were found for breakfast, lunch, and dinner. Thus, in that study, snacking was associated with overweight and obesity but was not an isolated risk factor. In France, among overweight adolescents enrolled in an obesity treatment, more snacking was one of the significant factors explaining why they failed to maintain the weight loss (13). In Colombia, among 3075 children between 5 and 12 y of age, a snacking pattern was the only behavioral factor associated with overweight (14). None of those studies support the conclusion of Drummond et al. (9).

Interestingly, genetic determinants could also contribute to the snacking behavior. Thus, in French obese children, Swiss obese adults, and the Finnish general population, 1 allele (C) of the gene rs17782313, situated near the melanocortin receptor MC4R, was associated with a greater prevalence of snacking (15). This was not the case for the fat- and obesity-associated gene FTO (rs1421085), another gene that seems to influence food intake and whose A allele has been shown to reduce satiety (16). It must be noted that in those studies, snacks were not defined differently than in previous ones and therefore the snack effect seems strong enough to be found even if an unknown proportion is misclassified as meals.

Biobehavioral consequences of a snack

These results do not, however, shed light on the mechanisms underlying the effects of snacking on energy balance. Some authors (9) have argued that part of the effect of eating pattern on body weight may be mediated by their effects on physical activity. Because high eating frequency and snacking have opposite effects on body weight, this would suggest that high meal frequency is associated with higher physical activity and snacking with being more sedentary. That notion is supported by some studies for meal frequency (17,18), but the causal link and the direction of this putative causal link (snacking leading to or caused by low physical activity, eating frequency leading to or caused by higher physical activity) is impossible to determine from those studies. Recently, it was shown that increasing high carbohydrate and caffeine snacking in firefighters was associated with an increase of spontaneous activity during work (19), but the conditions in that study were too specific and preclude any extrapolation to the snacking behavior of sedentary individuals.

Some years ago, we developed an experimental approach that we called biobehavioral to try to understand how snacking may impair the physiological control of food intake such that its energy content contributes poorly to eating behavior and metabolism. The conceptual basis of our approach was that physiologically, satiety is primarily modulated by the time interval until the next meal, either in animals (20) or in humans (21). Social constraints counteract this spontaneous pattern, leading to a transition from this postprandial correlation to a preprandial one, with meal size being dependant on the interval since the previous meal (22). Our hypothesis was that recreating a spontaneous “physiological” eating pattern may help to reveal subjective and objective differences between meals and snacks. For this purpose, our participants were time-blinded, i.e. deprived of time cues, and had to request their meals spontaneously. To evaluate intake and not food choice, these meals consisted of 1 main course, with participants being encouraged to eat until satiation. To detect discrete variations in hormones and substrates involved in eating behavior, blood was drawn continuously using a specially designed double lumen catheter.

In our first study (23), we assessed the consequences of a snack according to the time of its consumption. To mimic a snacking situation, this intake had to occur during a usual satiety period. Therefore, a basal session was planned during which participants ate lunch until satiation and were asked to request their dinner meal when they felt the need to eat. Importantly, these participants never or very rarely ate snack in their everyday life. The next days, glucose concentrations were measured, and on the 3 further sessions, a snack (1045 kJ, 51% CHO, 17% protein, and 32% fat) was provided in a random order, either 5 min before or 40 min after the glucose peak measured in this basal session or 2 h before the dinner request in this basal session. All these periods corresponded to a different postprandial metabolic situation discussed elsewhere (23). Results showed that whatever the time of its consumption, the snack failed to significantly delay the dinner request, did not lower
hunger ratings, and did not reduce energy intake at dinner, leading to a higher total energy intake over the whole session. Thus, energy from the snack had no effect on any dimension of satiety: duration, perception, or consumption. Biologically, the snack only marginally increased plasma glucose but induced an increase in insulin during the next 2 h and dramatically suppressed the free-fatty acids (FFA) FFA increase observed in the late part of the inter-meal interval.

Our interpretation was that in suppressing the concentration of FFA in the latter part of the inter-meal interval, the snacks precluded them to improve satiety. One hypothesis is that FFA may delay meal initiation in spared glucose during the inter-meal interval via the glucose-fatty acids cycle (24). The more FFA available for oxidation in the inter-meal interval, the more glucose oxidation will be spared and the longer the glucose disposal for brain cells involved in eating behavior will be increased. Actually, in animals (25) and in humans (26), a plasma glucose decline is observed before a spontaneous eating occasion is initiated. This glucose shortage stimulates cells of the central nervous system involved in eating behavior and in particular in triggering intake (27). In a second experiment using a similar procedure (28) but without blood measurements, we assessed the consequences of a snack on satiety according to its macronutrient composition. The snack was consumed 4 h after lunch, had the same energy content as in the previous study but contained 84% CHO, 58% fat, or 77% protein. It must be noted that the food items comprising the snacks differed and did not match in sensory characteristics. Results showed that dinner request was significantly delayed after the 3 macronutrient versions of the snacks but that the high-protein snacks induced a longer satiety than the high-CHO and high-fat ones (Fig. 1A). However, no change in dinner intake was observed across conditions and therefore, similar to the previous study, total energy intake was higher in all snack conditions than in the basal condition (Fig. 1B). So, the satiety power was only effective for the duration dimension of satiety and was more strongly so after the high-protein snack.

In the next experiment (29), we evaluated whether the high-protein snack delayed the dinner request more than the high-CHO snack by inducing less insulin and therefore lowering the reduction in FFA blood levels and oxidation in the late part of the inter-meal interval. Results showed that a high insulin secretion in fact followed the high-CHO but not the high-protein snack. The duration of satiety (indicated by the amount of time passing until dinner request) was not changed by the high-protein snack, whereas dinner request was significantly delayed by 38 min after the high-protein snack, supporting the robustness of the satiety power of protein. As expected, the late increase in plasma FFA concentrations was almost completely blunted after the high-CHO snack, whereas it was suppressed, having a smaller magnitude, after the high-protein snack. Substrate oxidation confirmed our hypothesis, with a high glucose oxidation and a smaller plasma FFA concentrations was almost completely blunted after the high-protein snack, supporting the robustness of the satiety power of protein. One hypothesis is that FFA may delay meal initiation in spared glucose during the inter-meal interval via the glucose-fatty acids cycle (24). The more FFA available for oxidation in the inter-meal interval, the more glucose oxidation will be spared and the longer the glucose disposal for brain cells involved in eating behavior will be increased. Actually, in animals (25) and in humans (26), a plasma glucose decline is observed before a spontaneous eating occasion is initiated. This glucose shortage stimulates cells of the central nervous system involved in eating behavior and in particular in triggering intake (27). In a second experiment using a similar procedure (28) but without blood measurements, we assessed the consequences of a snack on satiety according to its macronutrient composition. The snack was consumed 4 h after lunch, had the same energy content as in the previous study but contained 84% CHO, 58% fat, or 77% protein. It must be noted that the food items comprising the snacks differed and did not match in sensory characteristics. Results showed that dinner request was significantly delayed after the 3 macronutrient versions of the snacks but that the high-protein snacks induced a longer satiety than the high-CHO and high-fat ones (Fig. 1A). However, no change in dinner intake was observed across conditions and therefore, similar to the previous study, total energy intake was higher in all snack conditions than in the basal condition (Fig. 1B). So, the satiety power was only effective for the duration dimension of satiety and was more strongly so after the high-protein snack.

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The next step was to assess whether biology may help to differentiate eating driven by a physiological need at the end of satiety and eating triggered by the mere exposure to palatable foods during the normal satiety period (30). We chose to study a mid-afternoon eating occasion popularly called the gouˆter. In France, lunch is usually consumed between 1200 and 1300 h and dinner between 1900 and 2000 h. All children, most adolescents, but <30% of adults take a small meal between 16 and 17 h called the gouˆter. We expected that in gouˆter eaters carefully selected, the gouˆter would be preceded by biological character-istics that would not be present before snacking, defined here as an intake during a period of satiety. Moreover, in that study, we wanted snacks to be completely realistic so that participants initiated their consumption voluntarily. Thus, nongouˆter eaters, who usually never eat anything in the afternoon were either left without food until they requested their dinner, or at 210 min after lunch and for 30 min were presented the same food items (biscuits, orange juice, and fruits) as were gouˆter eaters. They were free to eat or not, but all consumed at least some foods. Results showed that at the presentation of the snacks, participants had low hunger ratings, whereas hunger ratings were high in gouˆter eaters when they asked for their gouˆter. Moreover, the snack did not delay significantly the dinner request, whereas gouˆter eaters requested their meal later than did nongouˆter eaters. Ad libitum dinner intake did not differ between conditions, leading to a higher total energy intake in the snacking condition. Interestingly, dinner intake was lower in the gouˆter eaters than in the nongouˆter eaters, whether they snacked or not. Blood analyses showed that in gouˆter eaters, glucose and insulin levels declined sharply before the gouˆter request, whereas this preeating profile was not found prior to the snack consumption in the nongouˆter eaters (Fig. 2). As expected, the snack increased blood insulin concentrations and completely suppressed the FFA.
release in the late part of the inter-meal interval that occurred in nonsnackers. A biological distinction was therefore clear between eating because of food availability during a usual satiety period and eating because of an endogenous metabolic signal, the first being characteristic of snacks, the second of meals. Importantly, we verified that this biobehavioral pregouter profile was not a conditioned reflex based on time of day by adding 2.8 MJ fat in lunch (D. Chapelot, C. Marmonier, and J. Louis-Sylvestre, unpublished data). Results showed that the goûter request was significantly delayed, suggesting that its main determinant was energy homeostasis and not conditioning.

**Some perspectives for the future**

Apparently, a biological difference between initiating a meal triggered by a homeostatic motivation to eat and consuming a snack triggered by a liking signal may seem of poor relevance for everyday life, because measuring glucose is presently not easily accessible. However, the development of ambulatory continuous glucose monitoring systems could change this situation. It is very easy to insert and wear and was effective in detecting the preprandial glucose decline in nondiabetic participants eating in their natural environment (31). Moreover, it is striking that diabetic patients can be trained efficiently to evaluate accurately their blood glucose level from their hunger sensation and then to delay intake until this hunger level is reached (32). If the distinction between meals and snacks demonstrates its usefulness for preventing body weight gain, overweight individuals or those at risk of overweight showing difficulties in perceiving the endogenous nature of their motivation to eat could represent a first nondiabetic target population to benefit from this device.

However, even in the absence of definitive proof, it seems reasonable to apply the precautionary principle and try to reduce snacking in the diets of children and adolescents, in particular in developing countries (33). The question remains whether limiting access to snack items, especially those of poor nutritional quality, will be effective for preventing gain of excess fat tissue. Restricting snack availability at school has been associated with significantly higher frequency of fruit and vegetable consumption (34). Moreover, in schools following the advice presented in “guidance for healthy snacks,” consumption of beverages and salty and sweet snacks declined (35) and there was no compensation at home. The strategy of restricting the availability of low-nutrition snacks in schools on a broad scale could be quite effective and may provide a rapid answer to the putative negative health consequences of snacking.

In conclusion, according to the current definitions, recent data support the hypothesis that snacking contributes to overweight and obesity. Studies on the influence of snacking on health may benefit from a physiological definition that distinguishes it from eating a meal. The weak effect of snacking on satiety is primarily linked to their typically high-carbohydrate content and resulting increase in insulin secretion, which blunts the incorporation of FFA into metabolic pathways. Snacks of high nutritional quality and high-protein content may limit or reverse their deleterious effect on energy.

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**Literature Cited**


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**FIGURE 2** Plasma concentrations of glucose (A), insulin (B), and FFA (C) in goûter eaters (GE) and nongoûter eaters (NGE) until the presentation of the snack to one-half of the group, and in the GE, nongoûter snack eaters (NGSE) and nongoûter nonsnack eaters (NGNSE) until the spontaneous dinner request. The x-axis is in percentage of the inter-meal intervals. The first interval begins at the end of the lunch meal and ends at the goûter request in GE and at snack-time (210 min) in NGE. The second interval begins at the start of the goûter in GE and at 210 min in NGSE and NGNSE and ends at the dinner request. Values are means ± SEM. a, GE differs from NGSE and NGNSE, P < 0.05; b, GE and NGSE differ from NGNSE, P < 0.05. Adapted with permission from (30).


