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

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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, Avie et al. (11) began to introduce the necessary distinction between these 2 eating occasions. They actually found that adolescents who snacked more 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 than 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 sparing 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-CHO 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. Substrate oxidation 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.

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 goûter. 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 goûter. We expected that in goûter eaters carefully selected, the goûter would be preceded by biological character-

FIGURE 1 Consequences of the macronutrient composition of the snack consumed 240 min after lunch on the delay of dinner request (A) and on energy intake (B). All values are means ± SEM, n = 10. *Different from basal, P < 0.05. Adapted with permission from (28).
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 pregouˆter 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 gouˆter 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


