Carbohydrates and appetite control

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Abstract Many studies have shown that consumption of high carbohydrate foods can give rise to a clear modulation of the expression of human appetite. The potency and time course of the effects of various carbohydrates on satiety vary with the amount consumed and the chemical structure. There is evidence that this biological cascade can modulate the temporal profile of hunger and the eating pattern of meals and snacks. One important issue is the action of carbohydrate foods on satiation (within meals) and satiety (after meals). These effects can be contrasted with the relatively weaker effects of high fat foods. The physiological mechanisms through which carbohydrates exert an action on appetite include plasma glucose levels, glucose receptors, hepatic glucose metabolism and glycogen stores. Experimental evidence indicates that the encouragement to eat high carbohydrate (low fat) snacks or high carbohydrate breakfasts can significantly reduce daily fat intake, limit energy intake, prevent weight gain and even induce weight loss. It is therefore possible to design high carbohydrate diets that provide good nutrition with adequate control over appetite and a beneficial effect on body weight. (Aust J Nutr Diet 2001;58 Suppl 1:S13–S18)

Appetite control and the satiety cascade

The control of human appetite is based on the balance between excitatory processes, which initiate eating, and inhibitory processes, which arise from food consumption (and other metabolic events). The biological drive to eat can be linked with the satiating power of food. ‘Satiating power’ or ‘satiating efficiency’ is the term applied to the capacity of any consumed food to suppress hunger and to inhibit the onset of a further period of eating (1,2). Food brings about this effect by way of certain mediating processes that can be roughly classified as sensory, cognitive, post-ingestive and post-absorptive (see Figure 1). The operation of these processes is generated by the impact of food on physiological and biochemical mechanisms.

Collectively these processes have been referred to as the ‘satiety cascade’ (3). The way in which food is sensed and processed by the biological system generates signals (neural and humoural) that are utilised for the control of appetite. It follows that any self-imposed or externally applied reduction in the food supply (creating an energy deficit) will weaken the satiating power of food. One consequence of this will be the failure of food to adequately suppress hunger (the biological drive). The satiety cascade appears to operate as efficiently in obese people as in lean individuals. Hence a normal appetite response to a reduced energy intake is evident in obese subjects.

Technically, satiety can be defined as that inhibition of hunger and eating that arises as a consequence of food consumption. It can be distinguished from satiation which is the process that brings a period of eating to a halt. Consequently, satiation and satiety act conjointly to determine the pattern of eating behaviour and the accompanying profile of motivation. The conscious sensation of hunger is one index of motivation and reflects the strength of satiation and satiety. It is worth keeping in mind that hunger is a biologically useful sensation. It is a nagging, irritating feeling that prompts thoughts of food and reminds us that the body needs energy. Moreover, there is good evidence that in most cases hunger is related positively to food intake and is a valid marker of the motivation to eat (4). For example, after tracking diurnal rhythms of hunger and eating it was noted that:

- the correlation using hunger ratings and intake during the same hour of the day was $r = 0.5$ ($P < 0.02$). That is, hunger ratings at the start of each hour were correlated with reported intake in the hour following each hunger rating (5).

Other researchers measuring spontaneous feeding in humans have concluded that ‘subjective hunger represents an intermediary step in the cause-effect sequence between gut filling and cessation of meal ingestion’ (6). Therefore, the monitoring of hunger, together with energy intake, constitutes an important feature of the action of carbohydrates on appetite in humans.

Mechanisms of action of carbohydrates on appetite

Human appetite is controlled by a complex matrix of factors within a psychobiological system that represents the interaction between physiological processes in the body and brain, and the social and physical environments (7,8). The nutritional composition of food is one significant aspect of the physical environment and there is plenty of evidence that different types of foods exert differing actions on the mechanisms responsible for the expression of appetite (9). Indeed, the demonstration that foods varying in nutrient composition exert differing tendencies to induce body weight gain (10) implies that nutrients are significant factors in the regulation (or dysregulation) of appetite. However, which nutrients are measured, and how? There are strong logical reasons why glucose should

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Figure 1. The ‘satiety cascade’, a conceptualisation for understanding the impact of foods on appetite control and energy intake

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The idea that the metabolism of glucose in the body is related to the existence of hunger and eating is represented in the glucostatic hypothesis formulated by Mayer (12,13). It was postulated that the short-term articulation of energy intake with energy needs is under glucostatic control (14). In turn, this implies that dietary carbohydrates clearly are involved in the short-term control of energy intake. Similar views are embodied in the energostatic hypothesis (15), metabolic control of food intake (16), the glycogenostatic hypothesis (17) and the energetic concept of appetite (18). Although the most obvious mechanism is that hunger is directly related to fluctuations in the concentration of blood glucose, the relationship between energy intake and glucose could be mediated via arteriovenous glucose differences, the rate of glucose utilisation in the liver, or the glycogen stores. Evidence for differences, the rate of glucose utilisation in the liver, or the glucose could be mediated via arteriovenous glucose differences, the rate of glucose utilisation in the liver, or the glycogen stores. Evidence for the role of glucose in the initiation of eating comes from the detection of transient declines in blood glucose being related to meal initiation (19) and the observation that increases in energy intake follow injections of 2-deoxy-d-glucose which stimulate glucose deprivation at glucose-sensitive neurons (20). Conversely, glucose infusions (particularly into the hepatic portal system) produce an inhibition of the tendency to eat (21). After the consumption of a carbohydrate food, the glucose produced could be monitored early by glucose-detecting interoreceptors in the upper gastrointestinal tract linked to visceral afferent fibres in the vagus nerve (22). In addition, Oomura (23), has described glucose-sensitive neurons (which decrease their activity in response to applied glucose) and glucose-depriving interoreceptors (24) which are functionally coupled to eating behaviour and food selection. The evidence for all the stages in this biobehavioural loop is equivocal (25), but the ingestion of pure carbohydrate certainly alters the ratio of plasma amino acids (26). The mediator of this alteration is the glucose-induced release of insulin.

Apart from these direct links between the detection of glucose and neural transmission, an indirect mechanism has been postulated through which carbohydrate in the diet could be related to neurochemical activity. This mechanism relates the proportion of carbohydrate to protein in the diet to the ratio of tryptophan to other large neutral amino acids in plasma, which determines the uptake of tryptophan into the brain (24). In turn, it is postulated that this leads to an activation of serotoninergic neurons, which are functionally coupled to eating behaviour and food selection. The evidence for all the stages in this biobehavioural loop is equivocal (25), but the ingestion of pure carbohydrate certainly alters the ratio of plasma amino acids (26). The mediator of this alteration is the glucose-induced release of insulin.

It follows that many mechanisms exist to monitor the activity of glucose released by ingested carbohydrates. Therefore, it should be possible to demonstrate a relationship between consumed carbohydrates and a modulation of the expression of appetite. Although sweet carbohydrates induce some positive feedback for eating through the induction of oral afferent stimulation by sweet receptors, this should be countered by the potent inhibitory action via post-ingestive and post-absorptive mechanisms.

Carbohydrates and appetite—experimental evidence

On the basis of studies on rats it was argued some years ago that if the cumulative inhibitory effects of carbohydrate on feeding are indeed energostatic...then any substance that can readily be used by the animal to provide energy should produce an appropriate food intake compensation over a period of several hours after loading (15). Studies have shown that this is also the case for humans. A variety of carbohydrates, including glucose, fructose, sucrose, maltodextrins and polysaccharides, exert measurable effects when given in a preload or an experimental meal, i.e. they suppress later intake by an amount roughly equivalent to their energy value, although the time course of the suppressive action may vary according to the rate at which the carbohydrates are metabolised.

The preload paradigm is a sensitive procedure for disclosing the effects of glucose and maltodextrin (glucose polymer) on appetite when these carbohydrates are delivered in a natural food product. For example, in a study using the uncoupling design, the actions of sweetness and energy content of a yoghurt preload were experimentally separated by using judicious combinations of glucose, maltodextrins, high intensity sweeteners, and the yoghurt base (27). After consumption, feelings of hunger were significantly suppressed by the high energy preloads (glucose and maltodextrins) to a greater extent than after the lower energy loads. Reduction of energy intake in the test meal was proportional to the energy difference (678 kJ) between the preloads. The accuracy of this adjustment confirms the frequent, though not universal, finding that human subjects adjust their voluntary intake in response to covert manipulations of the carbohydrate content of a food. In a more recent study, a comparison was made between preloads containing maltodextrin or sucrose compared with lower energy loads with similar taste characteristics. At one hour after consumption, both high energy preloads had suppressed intake compared with their low energy counterparts.

Evidence from meal manipulations

A good deal of evidence for an action of carbohydrates on appetite control is provided by the preload design using experimentally manipulated food substances. However, perhaps the most convincing evidence comes from studies in which manipulations have been made within a natural eating pattern. A number of these studies have compared the effects of carbohydrate and fat.
For example, a group of healthy male subjects consumed a standard breakfast of 1.84 MJ (440 kcal) or the same breakfast supplemented with either fat or carbohydrate. The standard breakfast consisted of orange juice, scones, and fruit yoghurt and was chosen to disguise well the nutrient manipulations. After consuming the various breakfasts (on separate occasions one week apart) hunger was assessed using rating scales for a period of four hours. The effects were particularly noticeable for a period of one to three hours after eating. The carbohydrate supplement suppressed hunger and desire to eat but increased fullness, whereas the fat supplement failed to produce such effects (28,29). The effects of the supplements were apparent during a ‘post-ingestive window’ during which time the physiological processing of the carbohydrate was taking place and generating physiological satiety signals.

In a later study when a test snack was given to subjects during the post-ingestive window, the carbohydrate supplement significantly suppressed the snack food intake (29) (see Figure 2).

In a further series of breakfast manipulations subjects consumed four realistic isoenergetic (2035 kJ) breakfasts varying in macronutrient content (two fat-rich, two carbohydrate-rich—low fibre and high fibre) in a random order on separate mornings. Ratings of appetite and measures of food consumption were continued for the rest of the day. The results indicated that the low energy-dense, high carbohydrate breakfasts were strongly satiating compared with the equi-energetic high fat meals. Figure 3 shows the change in perceived fullness upon the different breakfasts. Both fat-rich breakfasts were more palatable but less satiating than the carbohydrate-rich meals and were followed by greater food intake during the morning (30).

A further interesting study has used a meal design to illustrate the impact of a high carbohydrate meal on appetite control. In this study a comparison was made between two methods of inducing an acute energy deficit (exercise and breakfast omission). Subjects consumed a 2085 kJ (500 kcal) breakfast (70% carbohydrate) or a low energy breakfast (267 kJ, 64 kcal) in association with a 40-minute period of exercise (70% VO2 max) or a rest period. The exercise session or the breakfast omission was designed to produce a similar energy deficit of approximately 1668 kJ (400 kcal). The results showed that the absence of the high carbohydrate breakfast produced a rise in hunger, which was preserved for the whole morning period (Figure 4) and gave rise to a larger energy intake at lunch. Interestingly the energy deficit produced by exercise did not increase hunger and did not increase the lunchtime food consumption (31). This study indicates that a high carbohydrate intake at breakfast can suppress hunger and control appetite at a mid day lunch meal.

In a study based on the operations of the satiety cascade (see Figure 1) the effects of high carbohydrate foods on both satiation and satiety were assessed in a group of obese women with a mean body mass index (BMI) of 42 kg/m2. The experimental design was superimposed on a natural meal pattern of lunch, dinner and evening snacks (32). The obligatory consumption of a high or low energy lunch gave rise to high or low levels of hunger during the afternoon. At the dinner meal subjects were allowed to eat to a level of ‘comfortable fullness’ from either a selection of high carbohydrate foods or (on a separate occasion) a range of high fat foods. The data in Figure 5 shows that high carbohydrate foods restricted energy intake at the dinner meal to approximately 2502 kJ (600 kcal) and therefore exerted a pronounced control over satiation. On the other hand the high fat foods permitted an intake of approximately 542 kJ (1300 kcal) and therefore had a very weak effect on satiation. Of course these effects arise primarily from the differing energy densities of high fat and high carbohydrate foods. However, Figure 5 also shows that the much smaller intake of carbohydrate foods had an equally strong effect on post-ingestive satiety as did the huge amount of fat. This study indicates that high carbohydrate foods can exert a potent control over appetite both during the eating process itself (satiety) and in the post-prandial period (satiety).

Figure 2. Temporal profile of hunger ratings over four hours following consumption of test breakfasts. Significant suppression of hunger brought about by the carbohydrate but not by the fat supplement. Adapted from Cotton et al. (29)

Figure 3. Changes in perceived fullness after equi-energetic breakfasts until lunch. From Holt et al. (30)(a)
Carbohydrate and the satiety index

Almost 20 years ago it was suggested that ‘it could be of great value to have tables showing the energy-satiety ratio of all the common foods to indicate their potential for causing over nutrition’ (33). Subsequently, the term ‘satiating efficacy’ was proposed ‘as an index to compare the satiating potency of different foods’ (2).

A further measure explored by Holt et al. (34) relates the satiety response following a food, to the satiety response following a reference food (white bread). Isoenergetic preloads (1000 kJ) of 38 foods were consumed, following which subjective ratings of motivation to eat were completed over two hours. Significant differences in the satiating effects of the foods were seen, indicating different foods differ in their satiating capacities. This novel approach quantifies the effects of a food on satiety and was based on the idea of developing a satiety index analogous to the glycaemic index. These studies showed that high carbohydrate foods had a high satiety index measured by the area under the satiety curve.

A further measure has been termed the ‘satiety quotient’ which permits a measure of the effect of a food on satiation (within an eating episode) and satiety (the effect following eating). This allows the determination of the degree of suppression of hunger which can be brought about per unit of food energy consumed (35) and permits the tracking of changes over time. Using data from a variety of foods freely consumed it was demonstrated that hunger was reduced more per kilojoule of carbohydrate food consumed than of high fat food—but the strength of this varied with time. This finding confirms the proposal that ‘joule for joule’ carbohydrate is more satiating than fat (36).

Evidence from nutritional interventions

The results from short-term studies show clearly that carbohydrates exert a marked influence over the expression of appetite. Real life intervention studies have demonstrated that this degree of appetite control has practical consequences. In one study, people identified as snackers were encouraged to eat 25% of their daily energy intake from high carbohydrate or high fat snacks during separate three-week periods. The subjects incorporated the snack foods into their normal eating repertoires and maintained a normal lifestyle. The study was very carefully monitored to ensure compliance to the experimental requirements and to assess food intake from the snack foods and from the rest of the eating pattern. Measures of total energy intake indicated that food consumption was physiologically valid and quite in keeping with body size and the level of physical activity. First, generous consumption of these palatable high carbohydrate foods did not generate abnormally high energy intakes. Indeed daily energy intake was an average of 362 kJ (87 kcal) less than with the high fat snacks—a value that could lead to a weight gain of approximately 3.6 kg over the course of a year. Second, the high carbohydrate foods significantly lowered the total daily fat intake down to a level recommended in the national dietary guidelines (37). Consequently this high carbohydrate intervention which was achieved easily exerted a notable control over appetite and the pattern of nutrient intake.

In a series of studies carried out in Scotland, subjects have been required to increase their consumption of high carbohydrate breakfast cereals by 60 g per day for twelve weeks (38). This intervention resulted in a 5.4% reduction in energy from fat and a 5.1% increase in energy from starch. In a second study, overweight men (BMI 29.4) were required to increase high carbohydrate breakfast cereal intake by 90 g per day (approximately three bowls) and an increase in carbohydrate consumption from 40 to 47% of total energy resulted (39). The positive changes in carbohydrate intake were accompanied by small weight losses.

Figure 4. Profiles of hunger across a nine-hour period following the exercise and breakfast manipulations. From Hubert et al. (31)(a)

Figure 5. Average energy intakes from the high fat and high carbohydrate dinner meals and subsequent energy intake in response to these meals measured for the remainder of the day and for the next 24 hours. ** P < .001, significantly different from carbohydrate. From Lawton et al. (32)(a)
Taken together these studies show that a high intake of carbohydrate foods can lower fat intake and does not compromise energy balance.

**Habitual high carbohydrate intake**

The evidence from short-term manipulations and medium-term interventions suggests that carbohydrate foods can exert a substantial effect over the pattern of eating via short-term mechanisms influencing appetite control. If this is the case then the habitual consumption of a high carbohydrate diet should exert a healthy control over energy balance. In particular the habitual intake of high carbohydrate foods should help to prevent the development of a positive energy balance and therefore protect against weight gain.

The analysis of different patterns of nutrient intake can be carried out using large scale surveys in national databases. However, in these analyses it is necessary to take account of the serious problem of misreporting, usually under-reporting, which is a consequence of the form of data collection (40). This was achieved in an analysis of one UK national database (41), by excluding all subjects whose reported intakes were physiologically inappropriate (42). Using the data from subjects whose food intake records were believable, a comparison was made of subjects (aged 18 to 54 years) who were habitual consumers of high carbohydrate foods or high fat foods. Figure 6 shows that among the high carbohydrate consumers, only one person reached a BMI of 30 which normally is regarded as a cut-off point for obesity. On the other hand, among the low carbohydrate, high fat consumers the level of obesity was 19 times higher.

**Figure 6. Frequency distribution of body mass index (BMI) among high carbohydrate, low fat consumers (A) and low carbohydrate, high fat consumers (B).** Adapted from Macdiarmid et al. (42)

**Overview**

Taken together, the experimental manipulations, nutritional interventions and dietary surveys indicate that carbohydrate foods markedly influence the expression of appetite and can control the pattern of eating. These foods can help to prevent people developing a positive energy balance and can therefore protect against weight gain. High carbohydrate foods can therefore function as protection factors, which oppose dietary risk factors and help to prevent the loss of appetite control. However, it should be recognised that this link between carbohydrate and appetite control does not constitute a biological inevitability. In certain cases an individual could over-ride physiological signals and reach a high energy intake on carbohydrate foods (or beverages). Many routes to weight gain are possible (43). However, carbohydrate over-eating is probably an exception and in general carbohydrate foods provide considerable protection against the possibility of over-eating, positive energy balance and weight gain.

**References**


