CARBOHYDRATES AND SATIETY

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1. BACKGROUND INFORMATION

Our body requires energy and nutrients for its biological functions; due to specific physiological signals of deprivation, such as empty stomach and/or blood depletion of nutrients, the sensation of "hunger" arises and creates the need for searching for and eating food. Satisfaction of hunger culminates with satiety.

Satiety is the state in which the organism has satisfied its energetic needs by the amount of food eaten.

Satiety must not be confused with "satiation", which refers to the processes involved in the termination of a meal, rather than to the effects of a food or a meal after the eating has ended, as is the case of "satiety" (Roll 1995).

"Hunger" must not be confused with "appetite", which is induced by signals of sensorial stimulation, such as view of food, smell and taste, or the recall of previous pleasant experiences with food.

2. SATIETY AND TASTE

Satiety seems to have an influence on the taste of food. In effect, it has been observed that a taste stimulus can be perceived as pleasant or unpleasant according to the internal satiety state of the subjects.

Several studies confirm that the hedonics of food change with satiety. Particularly, it has been shown that after eating a food to satiety, the pleasantness of taste decreases more than from other foods tested but not eaten. This phenomenon, known as "sensory-specific satiety", limits consumption of one type of food and helps to ensure that a variety of foods is consumed. (Rolls)

Thus, to a hungry individual a sweet food will be rated as extremely pleasant in taste, but as consumption proceeds this rating of pleasantness declines.

While satiety can influence taste, taste does not influence satiety. In fact, Rogers and Blundell (1989) demonstrated that a meal sweetened with saccharin does not possess the same satiating capacity as a meal sweetened with glucose or sucrose.

The artificial sweet taste, therefore, may influence appetite through the signals to central nervous system but not satiety.

3. CARBOHYDRATES AND SATIETY

Satiety is regulated by the central nervous system through neurotransmitters' activity. One of the most important neurotransmitters is serotonin, which has tryptophan as precursor.

The higher the serotonin secretion, the lower is the hunger stimulus.

Serotonin-releasing brain neurones are unique in that the amount of neurotransmitter they release is normally controlled by food intake. Serotonin release, however, is not the same with all foods, but is largely related to the specific food eaten: carbohydrates consumption, acting via insulin secretion and through the plasma tryptophan, increases serotonin release. Protein intake does not provide this effect.

It is well established that the ingestion of different types of carbohydrates (simple sugars and complex carbohydrates) provides signals to regulate food intake through the sensation of hunger and/or appetite.

Carbohydrates ingestion has a powerful effect on satiety – it is greater than the effect of fat (Roger 1988, Roger 1989, Van Amelsvoort 1989, Cotton 1994), but lower than the effect of proteins. However, the mechanisms by which carbohydrates influence satiety are not yet fully understood.

To support the cited role of carbohydrates on appetite control, another study (Johnstone 1996) shows the high satiating power of carbohydrates in the short-term (10-15 minutes). According to the findings of this study, high-protein diets proved to be more satiating than isoenergetically-dense high-carbohydrates or high-fat diets on the day they were eaten; the high-carbohydrate diet was transiently more satiating than the high-fat diet after each meal. A recent study (Werterterp Plantenga 1999) further shows that high-protein/high-carbohydrate diets increase both satiety and diet-induced thermogenesis, to a larger extent than high-fat diets.

To fully understand the role of carbohydrates on appetite control, it is also important to analyse the effects of different types of carbohydrates (monosaccharides, disaccharides, oligosaccharides, more complex carbohydrates), since it is possible that appetite signals arising from these products are different between carbohydrates and their perceived sweetness (Anderson 1995, Stubbs 1999).

Post-absorptive rates were considered for a long time the most important mechanisms responsible. The theory is based on the assumption that hypotalamic receptors (in the brain) for glucose respond to the level of glucose in blood: a low blood glucose level induces hunger, while an increase of glycemic level stimulates hypothalamic satiety and reduces eating desire (Mayer 1953).
Twenty years later, Russek (1971) suggested that signals of satiety be transmitted to the brain from the liver by means of hepatic glucoreceptors.

More recently (1995), a study conduced by Lavin demonstrated that an increased contact of glucose with intestinal receptors plays an important role in the satiating effect of carbohydrates. Infact, in his study Lavin showed that satiety was increased and hunger decreased as intestinal absorption of glucose was slowed by the addition of guar gum, independently from the gastric emptying.

4. GLUCOSE AND SATIETY

Glucose is the major source of energy for the body, and it is considered as a major contributor to satiety.

It is known that both gastrin and cholecystokinin (CCK) promote glucose-induced release of insulin. Several studies showed that endogenous CCK did indeed mediate hunger and satiety in humans.

A mechanism by which glucose could influence food intake is hyperinsulinemia. Results of a well controlled study suggest that a sustained blood glucose level induces satiety in humans. This can be obtained through slow release carbohydrates. The effect seems not to be mediated by insulin, although an enforcing effect of endogenous insulin of the satiety due to high blood glucose levels cannot be excluded.

To investigate the associations between appetite sensations, on the one hand, and macronutrient intake and measures of glucose metabolism, on the other, it has been shown (Raben 1996) that total serving weight and carbohydrate content of meals, as well as postprandial glucose metabolism, seem to be involved in the changes of postprandial hunger and satiety sensations after a meal. Due to the covariation between the single variables it is not possible, however, to distinguish between the different factors involved.

Further investigations have been conducted on the role of glucose on satiety.

Holt and colleagues (1996) studied the effects of 38 common foods on postprandial glucose and insulin responses in young healthy volunteers. Total carbohydrate content appeared to be a stronger determinant of short-term satiety compared to food glycaemic impact. It is concluded that the total amount of carbohydrates consumed in a meal and subsequent insulinaemia may partly determine the degree of hunger arising within the subsequent 2 hours.

A recent study (Flint 1998) shows that the infusion of glucacon-like peptide 1 (GLP-1) affects insulin and blood glucose levels; it enhances satiety and fullness, reducing consequently energy intake.

5. PHYSIOLOGICAL CONDITIONS AND SATIETY

High-fat foods are readily overeaten. Fat is overeaten for various reasons: because it is highly palatable, because it provides a high level of energy in a given volume of food, and because it has a smaller satiety value when compared to carbohydrates.

Carbohydrates and fat, ingested in equal volumes, have similar effects on hunger and satiety; however, carbohydrates provide less than 50 per cent of energy (calories) provided by fats.

In obese and restrained subjects, preloads of high-carbohydrate yoghurts suppressed subsequent food intake more than high-fat yoghurts; this indicates a relative insensitivity to the satiety power of fat (Rolls 1995).

A dose-response preloading paradigm gives the most sensitive index of satiety. In one such test (Rolls 1995) it was found that the effects of yoghurts with different fat and carbohydrate content did not respond in normal-weight, unrestrained men. However, in obese individuals – or in those concerned with body weight – yoghurt fat content was less effective in reducing subsequent food intake when compared to carbohydrates.

In an other study Lawton (1995) investigated 12 obese women for two weeks. Subjects were provided lunch meals with fixed energy value rich in either carbohydrate or fat. The effect of these meals on satiety was assessed through a battery of tests: subjects felt less hungry after consuming the high-carbohydrate meal than after consuming the high-fat meal.

6. CONCLUSIONS

- On a short and medium-term, carbohydrates induce a higher degree of satiety if compared to fats.
- Carbohydrate-rich meals may help reduce the intake of energy (calories) more than fat-rich meals.
- A regular intake of carbohydrates, by reducing the total energy intake, may prevent obesity. This effect can be further strengthened by associating a regular intake of carbohydrates to a more active lifestyle.

- A food sweetened with saccharin does not possess the same satiating capacity as a food sweetened with glucose or sucrose. The artificial sweet taste may influence appetite but not satiety.

7. IOCCC POSITION

Plenty of scientific evidence show that carbohydrates have a high medium-term satiating capacity.

Consequently, it can be suggested that several small meals based on carbohydrates may reduce the overall daily intake of calories.

A diet based on a regular carbohydrates consumption, especially if associated with an active lifestyle, can help prevent obesity.

BIBLIOGRAPHY

1. Anderson GH. Sugars, sweetness, and food intake. Am. J. Clin. Nutr. 62(Suppl): 195S-202S, 1995

2. Cotton JR, Burley VJ, Weststrate JA and Blundell JE. Dietary fat and appetite: similarities and differences in the satiating effect of meals supplemented with either fat or carbohydrate. J. Hum. Nutr. Diet. 7:11-24, 1994

3. Flint A, Raben A, Astrup A, Holst JJ. Glucacon-like peptide 1 promotes satiety and suppress energy intake in humans. J. Clin. Invest. 101:515-520, 1998

4. Gielkens HA, Verkijk M, Lam WF, Lamers CB, Masclee AA. Effectes of hyperglycemia and hyperinsulinemia on satiety in humans. Metabolism 47: 321-324, 1998

5. Holt SH, Brand Miller JC, Petocz P. Interrelationships among postprandial satiety, glucose and insulin responses and changes in subsequent food intake. Eur. J. Clin. Nutr. 50: 788-797, 1996

6. Johnstone AM; Stubbs RJ; Harbron CG . Effect of overfeeding macronutrients on day-to-day food intake in man. Eur. J. Clin. Nutr. 50(7); 418-30, 1996

7. Lavin JH, and Read NW. The effect on hunger and satiety of slowing the absorption of glucose: relationship with gastric emptying and post prandial bllod glucose and insulin responses. Appetite 25: 89-96, 1995

8. Lawton CL, Wales JK, Hill AJ, Blundell JE. Serotoninergic manipulation, meal-induced satiety and eating of fluoxetine in obese female subjects. Obes. Res. 3: 345-356, 1995

9. Mayer J. Glucostatic mechanism of regulation of food intake. N. Engl. J. Med. 249:13-16, 1953

10. Raben A; Holst JJ; Christensen NJ; Astrup A. Determinants of postprandial appetite sensations: macronutrient intake and glucose metabolism. Int. J. Obes. Relat. Metab. Disord. 20(2); 161-9, 1996

11. Reid M, Hetherington M. Relative effects of carbohydrates and protein on satiety- a review of metodology. Neurosci. Biobehav. Rev. 21(3): 295-308. 1997

12. Roger PJ, and Blundell JE. Separating the actions of sweetness and calories. Effects of saccarin and carbohydrates on hunger and food intake in human subjects. Physiol. Behav. 45:1093-1099, 1989

13. Rogers PJ, Carlyle J, Hill AJ, Blundell JE. Uncoupling sweet taste and calories: comparison of the effects of glucose and three intense sweeteners on hunger and food intake. Physiol. Behav. 43:547-552, 1988.

14. Rolls JB, Dimeo KA, Shide DJ. Age-related impairments in the regulation of food intake. Am. J.Clin. Nutr. 62: 923-931, 1995

15. Rolls JB, Hammer VA. Fat, carbohydrate, and the regulation of energy intake. Am. J.Clin. Nutr. 62(Suppl): 195S-202S, 1995

16. Rolls JB, Hammer VA. Fat, carbohydrate, and the regulation of energy intake. Am. J.Clin. Nutr. 62(Suppl): 1086S-1095S, 1995a

17. Van Amelsvoort JMM, Van Stratum P, Kraal JH, Lussemburg RN, and Houtsmuller UMT. Effects of varying the carbohydrate ratio in a hot lunch on postprandial variables in male volunteers. Br. J. Nutr. 61:267-283, 1989

18. Wurtman RJ, Wurtman JJ. Brain serotonin, carbohydrate-craving, obesity and depression. Obes. Res. 3(Suppl. 4): 477S-480S, 1995