The Role of Carbohydrate on Appetite Regulation

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Introduction
The physiological regulation of human appetite involves a complex interplay between signals originating from the gastrointestinal (GI) tract, liver and adipose tissue, and signals generated from the central nervous system (CNS). A great amount of studies have been conducted on this topic, and evidence has shown that the existence of carbohydrate in the GI tract plays an important role in the moderation of signaling and the regulation of appetite [1].

Carbohydrates are essential source of energy for the body, and they are also vital in the production of DNA, RNA and glycoproteins. The term carbohydrate refers to a broad range of molecules with various structures, from shorter chain carbohydrates to unavailable complex carbohydrates. When digested in the body, carbohydrates are usually absorbed as simple monosaccharides or disaccharides and consumed or stored as glucose [2].

Carbohydrates mainly regulate food intake through appetite modulation via gut hormones and the CNS [3,4]. Important appetite modulators include leptin, ghrelin, cholecystokinin (CCK), pancreatic polypeptide (PP), glucagon-like peptide (GLP)-1, and oxyntomodulin (OXM). For instance, leptin, a hormone produced by the adipose tissue 5, has been shown to reduce food intake and body weight and increase energy consumption, and therapies with leptin have been used against obesity-related metabolic disturbances [3,5]. Take the satiety signal GLP-1 as another example - it plays an important role in increasing insulin secretion and suppressing glucagon secretion after food intake, serving as an incretin [6]. In response to intraduodenal glucose, there is an increase in GLP-1 concentration, which is associated with decreases in appetite and subsequent food intake [6].

In the CNS, the hypothalamus is the key region involved in the regulation of appetite [7]. When carbohydrates are digested, short-term signals such as neural signals and humoral signals are released from the brain and the gut respectively. The receipt and integration of such signals then take place in the hypothalamus, and are mainly regulated by the hypothalamic arcuate nucleus (ARC) [4]. Within the ARC, there are two categories of neurons responsible for appetite regulation, one group being the proopiomelanocortin (POMC) appetite-inhibiting neurons and the other group being the neuropeptide Y (NPY) and agouti-related peptide (AgRP) appetite stimulating co-expressing neurons [8,9]. Signals from the periphery then lead to releases of neuropeptides from the neurons, which will subsequently change appetite and feeding behavior.

Lavin et al. studied the different influence on appetite between intravenous glucose and intraduodenal glucose [10]. The result suggested that intravenous injection of glucose did not influence hunger or satiety, while intraduodenal glucose infusion suppressed hunger, enhanced satiety ratings, resulting in a decreased appetite and energy intake. Research has confirmed that the regulatory impacts of intestinal glucose on appetite are controlled via small intestinal stimulation of release of either insulin or intestinal incretins, rather than an increase in blood glucose [10].

It is generally accepted that high carbohydrate diet is associated with obesity and weight gain. There is evidence showing that some carbohydrates can elevate energy intake via both nutritional and sensory mechanisms [11]. In addition, a two-year randomized controlled study demonstrated that food or calorie restriction (e.g. a low-carbohydrate diet) is an effective strategy to reduce food cravings and appetite [12]. More recent research has shown that the effects of dietary carbohydrate on human appetite vary depending on their site and structure [2]. For instance, shorter chain carbohydrates are believed to have more of an intake-promoting effect while unavailable complex carbohydrates (UCC) are more likely to have an intake-restraining impact [11].

UCC intake at one meal was shown to deduce hunger and energy intake in the next meal; and increased ingestion of UCC has been associated with decreased body weight, although the effect is modest [11]. In addition to chain length, a vast amount of factors need to be considered when evaluating the sensory and physiological effects of carbohydrates, such as the ratio of α- to β-links and the branching of chains. For example, some carbohydrates are likely to suppress appetite and food intake because they limit digestibility of foods although they do not elevate satiety [11].

As a result, it is particularly important to understand how carbohydrates of different structures and properties to process regulate the appetite and how the potentially beneficial effects of carbohydrate structure can be used to prevent obesity and weight gain.

Reference