Nutrition 30 (2014) 252–256

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Review

Neurobiologic basis of craving for carbohydrates

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ARTICLE INFO

Article history:
Received 23 January 2013
Accepted 28 June 2013

Keywords:
Dietary carbohydrates
Addictive behavior
Biological evolution
Anxiety
Eating behavior
Neurobiology

ABSTRACT

Objectives: There is a relationship between emotional disorders, obesity, and craving for carbohydrates. This relationship complicates the success of treatments aimed at combatting obesity, which is considered to be the epidemic of the twenty-first century. We conducted a review of the neurobiologic basis for carbohydrate craving, with the hope that this understanding will enable the design of more efficient therapeutic strategies.
Method: We conducted a non-systematic literature search in PubMed using MeSH.
Results: Research on the basis of carbohydrate craving is varied, but may be grouped into five main areas: the serotonergic system, palatability and hedonic response, the motivational system, stress response systems, and gene–environment interaction.
Conclusions: The models that integrate motivational systems with palatability and hedonic response studies are the ones that we believe can best explain both craving for carbohydrates and related addictive phenomena. Research has contributed to a greater understanding of the neurobiologic basis of carbohydrate craving. The latter, in turn, contributes to an understanding of the implications, challenges, and possible therapies that might be put in place to cope with this phenomenon.

Introduction

The growing global obesity epidemic and its associated pathologies [1] have focused research on the variables associated with overeating and the barriers to achieving changes in nutritional habits [2]. Research has ranged from social and cultural variables, such as the availability of food or the effects of advertising in childhood [2,3], to attempts at elucidating the mechanisms associated with the tendency toward excessive consumption of fats and carbohydrates. Such research also has suggested the possibility that there may be an addictive pattern to the consumption of these nutrients under certain circumstances [4–6].

We know that emotional factors have become increasingly important based on associations found among obesity, anxiety, and mood disorders [5]. Carbohydrates are exceptionally involved in these associations, which has led to further research into their patterns and roles. Sugar addiction, carbohydrate craving, and emotional eating have been correlated with obesity and being overweight. This has led to the establishment of a strong correlation between obesity, anxiety, and mood disorders [4–10], all of which have important public health implications.

The scientific literature supports the existence of carbohydrate-craving syndrome, in which carbohydrate intake medicates a dysphoric mood state [8]. This syndrome is defined as a food and mood disorder, characterized by an irresistible urge to consume sweets or starchy foods in response to negative moods [9]. The criteria listed in Table 1 have been proposed to operationalize this phenomenon [8]. Carbohydrate intake appears to temporarily improve mood in carbohydrate cravers, whereas individuals who have no craving for carbohydrates usually complain of fatigue after eating these foods [9], thus revealing a different relationship with this nutrient.

However, despite the important role of these relationships, the underlying neurobiologic mechanisms remain unclear and have not been directly related to emotional factors. Understanding these phenomena potentially could be important for several reasons. On the one hand, this would allow public health specialists and clinicians to understand realistically the behavioral changes of individual patients and the general population, considering how our biology works. On the other hand, this knowledge would allow specific pharmacologic strategies to be developed that may lead to more successful outcomes when dealing with obesity and its consequences.

Given the importance of understanding the neurobiologic mechanisms underlying the phenomenon of carbohydrate craving, we aim to review the main neurobiologic hypotheses

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http://dx.doi.org/10.1016/j.nut.2013.06.010

Table 1
Carbohydrate*-craving syndrome criteria [8]

- Cravings during the afternoon or at night to eat sweets or starchy foods at least four times a week.
- Eating carbohydrates in the afternoon or night at least four times a week, supported by records.
- Carbohydrate craving that occurs in conjunction with dysphoria, which is relieved by eating these foods.

* A carbohydrate is defined as a food consumed between meals that has a carbohydrate to protein ratio of ≥ 6:1.

that explain carbohydrate craving, and link carbohydrate cravings with related phenomena, such as emotional eating and obesity, from a neurobiologic perspective.

Methods

A focused review of the available literature was conducted by using the database www.pubmed.org. The MeSH tool was used and the search matrix included the terms *stress, carbohydrates, dietary carbohydrates, food, emotional eating, addictive behavior*, and *evolution*. By combining these terms, 2187 articles were found, of which some studies were selected according to the objectives of this review.

Results

We found dispersed information from different lines of research on carbohydrate craving. We then categorized these findings into five groups that had similar research lines. The serotonergic hypothesis is presented first because it is the first hypothesis to emerge and the one that has received the most attention in the literature. The hypotheses that follow are presented in terms of the degree to which they focus on physical consumption variables, thus beginning with palatability and ending with environmental aspects. A summary of these hypotheses is given in Table 2.

Serotonergic hypothesis

Brain serotonin is an important regulator of appetite, mood, and preference for certain macronutrients, among its other functions. Low levels of serotonin stimulate appetite and a preference for carbohydrates in the diet [11]. Furthermore, increases in brain serotonin improve mood levels in individuals who are vulnerable to stress [11].

The serotonergic hypothesis proposes that carbohydrate intake associated with states of anxiety is an attempt at self-treatment, given the increase in brain serotonin that occurs after carbohydrate intake [11]. The behavioral expression of this hypothesis is the craving for carbohydrates that an individual experiences within an episode of anxiety. After this macronutrient is consumed, the individual's emotional state improves.

It has been observed that during acute stress there is an increase in serotonergic neuronal activity due to increased plasma levels of cortisol and beta-endorphins [12]. An increase in serotonin regulates the hypothalamic–pituitary–adrenal (HPA) axis, which participates in stress adaptation [13]. If this continues over time, the use of serotonin exceeds its synthesis capacity, which has been associated with the depletion of tryptophan (Trp), the precursor of serotonin synthesis, and with depressive mood levels [12]. Changes in brain serotonin are mainly due to the availability of Trp in the brain. Serotonin synthesis is known to increase due to carbohydrates and to decrease or remain unaltered from proteins in the diet [10]. In fact, it has been observed that Trp levels after a meal rich in carbohydrates and low in protein tend to increase along with serotonin, due to a 42% rise in brain Trp compared with diets rich in protein and low in carbohydrates [14].

Based on the serotonergic hypothesis, it is inferred that other means of increasing brain serotonin levels may constitute potential treatments for obesity in individuals with anxiety. The use of serotonin, including fluoxetine, in rats with a history of food restriction under stress, was shown to decrease compulsive eating (binging). However, the effect was not the same in rats with a history of food restriction that were not under stress [5].

Controversy exists regarding the serotonergic hypothesis because some studies show conflicting results as to whether increased brain serotonin induced by a diet rich in carbohydrates effectively translates into a change of affective state and appetite [11,15,16]. Moreover, the positive effects on mood of increased brain serotonin induced by a carbohydrate-rich diet occur in patients suffering from anxiety due to carbohydrate craving, late

Table 2
Summary of the main hypotheses that explain carbohydrate craving

Neurobiologic hypothesis	Highlights
Serotonergic	• Increased brain serotonin improves mood [11]. • Brain serotonin levels depend on the availability of its Trp precursor [14]. • Dietary carbohydrates increase the passage of Trp through the blood–brain barrier, unlike proteins, which alter LNAA [14]. • Faced with anxiety, an individual eats carbohydrates, which increase brain serotonin, thus improving mood [11].
Palatability and hedonic response	• The pleasurable experience of eating food with high palatability immediately improves mood [17]. • This occurs in individuals with greater genetic sensitivity to sweet taste through the activation of the endogenous opioid system [19]. • Faced with anxiety, an individual eats a food with high palatability, activating the hedonic mechanism, which improves mood [16].
Motivational system	• Carbohydrates act in the motivational system in the same manner as abused substances [6]. • This increases dopamine and endogenous opioids, which are associated with a known pleasurable effect, [4] improving mood. • If this behavior is repeated over time, structural changes in the brain are produced that generate dependence on highly palatable foods [4,6,23].
Stress response	• Faced with anxiety associated with stress, the HPA axis activates [30]. • Highly palatable foods activate the motivational system and reduce the HPA axis, thus regulating the stress system [30]. • Therefore, when faced with anxiety, highly palatable food produces a hedonic reward as well as reducing the state of anxiety [30].
Gene–environment	• Eating is a coping tool to relieve negative emotions. • The behavior is learned through inadequate parenting and environment [9]. • It also stems from an inability to distinguish hunger from other aversive internal states [9]. • There is greater susceptibility in carriers of the A1 allele of the *DRD2* dopamine receptor and carriers of the short allele of the serotonin transporter gene [33,34].

HPA, hypothalamic–pituitary–adrenal; LNAA, large neutral amino acids; Trp, tryptophan