Review article

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Childhood obesity and eating behaviour

Abstract: The prevalence of childhood obesity has increased substantially in the recent decade as a result of the reduction in physical activity and the availability of high-fat and high-energy-density foods which the paediatric population faces daily. Although children are highly exposed to these foods, there is a wide variation in body weight, suggesting the presence of different patterns of response to an "obesogenic" environment. This wide variability from the point of view of eating behaviour involves a number of social issues (e.g., food availability, cost) as well as genuine behavioural traits such as the response to satiety, energy compensation, eating rate, responsiveness to food, food reward and dietary preferences. This article reviews the main physiological variables related to energy intake affecting eating behaviour in the paediatric population.

Keywords: childhood obesity; children eating behaviour; food preference.

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Introduction

Obesity is an adverse health condition characterised by an excessive increase in body fat caused by a sustained positive energy balance over time (1). This state is associated with the subsequent development of dyslipidemia, hypertension and insulin resistance, which favour the development of diabetes mellitus, arteriosclerosis and cardiovascular disease (2).

Obesity has increased significantly in all age groups in both developed and developing nations (3–5). In Chile, the prevalence of childhood obesity rose from 7% to 17% between 1987 and 2003 amongst school children in the first grade (6). In preschool children attending schools under the National Board of Kindergartens, the prevalence of overweight (22%) was noted to have remained steady between 1995 and 2000, whereas that of obesity increased from 8.6% to 10.6% (7). According to data from December 2010, in Chile, there are 9.6% obese children (weight/height relation above 2 standard deviations of the WHO reference) and an additional 22.6% with overweight.

This dramatic increase is mainly the result of environmental changes (sedentary lifestyle and increased energy intake due to the large availability of foods high in fat and sugar and high-energy-density foods).

Although substantial environmental and social changes have occurred during the last decades, excess of body weight does not occur in all individuals, showing a great variability seen in the body mass index (BMI) in the general population (8). This extensive intra-individual variability suggests subjects have different interaction patterns when exposed to an "obesogenic" environment, a feature that is most evident in children in relation to the adult population (8). In this context, it is worth noting that twin studies have demonstrated the contribution of genetic factors in both childhood eating behaviour (9) and childhood obesity (10). A recent study in Chilean children using the Child Eating Behaviour Questionnaire showed strong and graded association between eating behaviour scores ("slowness in eating", "emotional overeating", "food responsiveness" and "enjoyment of food") with childhood obesity (11).

It is known that the origin of excess body fat is the result of an imbalance between energy intake and expenditure. In this sense, scientific literature indicates that the greatest contribution to this variation would be given mainly by food intake and eating behaviour, rather than energy expenditure. This article reviews evidence of association studies among adiposity, appetite and satiety focussed mainly on paediatric patients, describing factors involved in eating behaviour that affect food intake in childhood.

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Methods

An electronic search was conduct to identify articles published in PubMed and SCIELO with the following keywords: "energy compensation", "eating behaviour obesity", "eating in the absence of hunger" and "food reinforcement". Limits added were "humans", "children", "clinical trial" and "randomize control trail". The inclusion criteria for this review were published in English or Spanish conducted in populations of children.

Factors associated to eating behaviour in children

Satiety response The regulation of energy intake is dependent on the effects of satiety and appetite. Satiety is defined as an operational concept used to study the effects of food intake at a mealtime in relation to intake at the following mealtime. Moreover, the term "appetite" is defined as the process that mediates the initiation of a meal, activated primarily by stimuli that are initiated in response to exposure to food. Factors related to appetite and satiety can be analysed from several perspectives (12).

Energy compensation Energy compensation is understood as the ability to respond to the reduction of energy intake at a mealtime (e.g., dinner) in response to a previous preloading of a high-calorie food. This variable can be evaluated by the rate of compensation (COMPX). A COMPX score of 100% reflects a perfect compensation. It has been observed that younger children have a good compensation rate in response to high-energy snacks and may have COMPX scores of up to 80%. However, older children compensate less effectively in response to high-energy snacks, reaching up to 20% (13).

Case-control studies have shown that obese children are more vulnerable to triggering an overeating behaviour. Jansen et al. (14) evaluated the energy compensation in response to three interventions in children aged 8–12 years: (a) after a snack of 146 kcal (610 kJ) (preload condition); (b) after exposure to strong odours of foods considered tasty (10 min); and (c) after a control situation. In this study, it was observed that obese children did not reduce ad libitum intake after food preload, and when they are exposed to different odours, they consumed more energy compared with normal weight children (14). These data support the low energy regulation and lower responses to satiety that obese children have. These results are consistent with those of Johnson and Birch (15), who showed that a greater degree of adiposity decreased the ability to regulate energy intake, further establishing that the control exercised by parents in relation to their children feeding would be an indicator of energy compensation in later life. In this area, other research has shown that preschool children present a high sensitivity to the energy density (kcal/g or mL) of snacks delivered. It has been reported that in response to different energy density preloads, younger children tend to compensate energy more effectively in relation to older ones (16). Johnson and Taylor-Holloway (17) described an incomplete energy compensation (mean COMPX score of 48%) in children of 5–12 years, with older children having lower scores. Hetherington et al. (18) evaluated in children aged 2–5 years and 7–10 years the energy compensation in response to a high (73 kcal/68.8 kJ) vs. low calorie preload (6 kcal/25.1 kJ). Using the compensation index (COMPX), it was shown that younger children had a better capacity to compensate (88.1%±22.4% and 21.5%±21.9%, respectively) (18), hence establishing a better regulation of energy in early age.

Recently, in another study conducted in siblings aged 5–12 years, Kral and colleagues (19, 20) compared caloric compensation in a dinner in response to different energy density preload. The authors described that overweight/obese siblings undercompensated energy in comparison with normal-weight siblings (%COMPX, −48.8±56.3 and 101.3±51.9, respectively). One of the factors described to influence energy compensations in children is television viewing. In this field, Francis and colleagues (21, 22) have described that children who reported to see more hours of TV daily ate more lunch when they are exposed to a TV condition (a 22-min video on five occasions), and that parental reports of the frequency of children eating during TV viewing at home were linked to higher energy intake. These results suggest that eating in front of the TV promoted higher energy intakes at lunch, affecting energy compensation.

Eating rate The speed of eating, or "eating rate", has been proposed as a determinant of obesity, having been recognised as an indicator of lower response to satiety. It has been established that increased eating speed through a mealtime (e.g., lunch) indicates higher motivation for food consumption, and conversely, its reduction over the course of the same mentioned establishes a strong and progressive activation of satiety signals (23). In this context, studies in 6-year-old children have shown that obese individuals eat and chew faster (bites per minute), compared with normal weight children (24, 25). Additionally, it has been established that the eating rate during childhood predicts adiposity in early life (26). Evidence in adults show that obese subjects eat faster compared with non-obese individuals (27), and some authors suggest a possible failure of a slowdown in a mealtime. Studies in the Japanese adult population have shown that BMI is positively correlated with eating rate, when they are classified in the categories "very slow", "relatively slow", "medium", "relatively fast" and "very fast" (28, 29). Interestingly, obese Chilean children showed significantly lower scores of "slowness in eating" compared with normal-weight children, which may indicate that the mastication process itself and/or the higher motivation for eating are related to excess body weight (11). Taken together, these findings support the hypothesis that increased eating rate favours the development of obesity.

Response to food The evidence of greater response to foods is derived from behavioural studies that have shown increased consumption of highly palatable foods.

The study of salivary flow from children and parents has been widely accepted as a noninvasively procedure to assess response to food. In this regard, Epstein et al. (30) evaluated the response of salivation in obese and normal-weight individuals exposed to 10 different presentations of yoghurt. The results showed a significant difference in the response of salivation pattern, exhibiting a reduced response to decreased salivation in obese subjects. Furthermore, a behaviour in children that has been described to assess the response to food is the consumption of food in the absence of hunger (eating in the absence of hunger, EAH). This behaviour has been used to assess whether exposure to a highly palatable food modifies internal feelings of satiety. It was first evaluated in preschool children aged 5–7 years, determining the ad libitum intake after consumption of a standard lunch and having reported feelings of satiation (31). Using this approach, it has been reported that obese children have the ability to consume, on average, 216±14 kcal (895±58 kJ) after informing a

fullness sensation without feeling hungry. Thus, it has been demonstrated that obese children are less responsive to internal satiety signals (32). Moens and colleagues (32, 33) have shown similar results, suggesting that obese children ingest twice the energy intake in the absence of hunger, compared with normal weight subjects. These results are consistent with those observed in 1348 children aged 7–9 years, in which the consumption of highly palatable snacks after lunch was evaluated. Along with this, a positive association between BMI and energy intake in the absence of hunger was observed (19). Butte et al. (34) showed that the amount of the energy consumed in the absence of hunger was associated with weight gain.

Similarly, Hill and colleagues (35, 36) has established that the EAH is a non-exclusive phenotypic behaviour of obese children and is also associated with adiposity in children. Birch et al. (37) have described that EAH increase significantly from 5 to 9 years of age and that higher levels of maternal restriction at 5 years predicted higher scores of EAH at 9 years.

Another approach has been developed by Bruce et al. (38), who examined the motivation for food consumption in a group of obese adolescents, matched for age, gender and educational level. Activation in different areas of the brain was studied using magnetic resonance images under a condition of hunger (pre-prandial) and immediately after the intake of a standard food preload. Both groups showed activation in brain areas (prefrontal cortex and orbitofrontal cortex) related to the limbic system in response to food images. However, the obese group showed significantly greater activation in the pre- and post-prandial stages compared with normal weight subjects. Furthermore, obese adolescents showed a smaller post-prandial reduction in the activation of regions of the brain such as the prefrontal cortex and nucleus accumbens, limbic system areas related to reinforcement and reward (38).

Reinforcement of food Subjective reinforcement experienced after consuming a highly palatable food is a powerful intake motivating factor (8). In general, bland-tasting food is not eaten in excess, whereas palatable food is often consumed even after energy requirements have been covered. Evidence in rodents and humans supports the theory that the consumption of highly palatable foods and the use of drugs converge on common mechanisms to mediate motivational behaviour (39–41).

Indeed, obtaining the pleasurable effects of palatable food is a powerful motivating force that in certain individuals can override homeostatic signals (42, 43). In this sense, animal model studies have shown that when presented with a choice, rats prefer to consume a sweet saccharin-free calorie solution rather than a self-intravenous cocaine infusion (44). Research in this field has focussed on the mesolimbic system because it has been observed that food and drugs increase the dopamine signal from neurons originating from the ventral tegmental area to the nucleus accumbens (also called the ventral striatum) (45). Studies have shown that a potent release of dopamine in the nucleus accumbens is induced in response to the presentation of highly palatable foods (46, 47).

One of the factors associated with positive energy balance is the different motivation for eating experienced by individuals. It has been established that normal neuronal activity patterns associated with motivation for food begins in childhood (48). An index used to evaluate this aspect is the "relative reinforcing value of food" (RRV). RRV is defined as the effort an individual is willing to make for a particular food. RRV can be evaluated in the laboratory, requesting individuals to perform a given activity for obtaining a particular tasty food that they consider reinforcing (e.g., pressing a key on a computer keyboard to get a slice of pizza). In this sense, those individuals who show more effort to acquire a food are considered reinforced individuals and, under this assumption, will spend more time and effort to eat than those individuals who do not view food as a reinforcement.

Using this model, it has been shown that obese individuals consider food more reinforcing than normal weight and that the degree of motivation for food is correlated with BMI z-score and energy intake, acting as a predictor (49, 50). Furthermore, it has been shown that obese adolescent girls have an increased activation of the insula and other cortical brain regions in response to palatable food (chocolate milkshake) or food cues, compared with lean control subject (51). Evidence in obese individuals shows the existence of a greater sensibility to reinforcement, reflecting the activity of dopamine receptors (52). In children, the results are consistent in showing that in obese children, the reinforcing value of food is higher than in normal-weight children and that predicts the change in adiposity in the short term (49, 53).

The feeling of reward or pleasure for intake of food associated with the limbic system (45, 54–56) is one of the signals for the initiation of feeding and belongs to the non-homeostatic intake mechanisms. This may be influenced by temporary (time of day or season), emotional and cognitive factors (learning, memory, social cues) amongst others (57). In this way, the existence of two mechanisms has been proposed: (a) "homeostatic mechanisms", which are involved in the regulation of energy balance (leptin, ghrelin and others), and (b) "non-homeostatic mechanisms", which are known as the hedonic signals of intake regulation. In this new approach, the following are proposed: (i) different brain systems may mediate different motivations for eating; (ii) different signals considered relevant to homeostatic control (leptin, insulin, ghrelin, amongst others) may impact on neural circuits associated with non-homeostatic control; and (iii) neuronal activation of non-homeostatic systems could eventually overtake the controls arising from homeostatic systems. Studies in adolescents conducted by Farooqi et al. (58) using magnetic resonance imaging have elucidated that the adipokine leptin (a principally homeostatic signal) would act in neural circuits that regulate food intake, diminishing the perception of reward of a specific food. These novel results indicate that leptin may also act through non-homeostatic mechanisms. Likewise, other authors suggest the idea that hormones recognised for their effects on the food intake regulation would also exert actions on the motivation to obtain food through the dopaminergic system. In this sense, leptin would act to decrease the basal secretion of dopamine and the hormone ghrelin would stimulate dopamine release in the striatum (57, 59) (Figure 1).

Food preferences Another way in which obese individuals differ from those of normal weight is in food preferences. As omnivores, humans have adapted the dentition and digestive system by eating a variety of foods, an adaptation that has been considered an advantage for survival.

Children have food preferences, which suggest the existence of an innate predisposition to certain tastes. For example, the preference of sweet flavours mediated by observation of facial expression is found to be present in neonates as well as the aversion to bitter and acidic flavours. This probably has an adaptive value because sweetness indicates the presence of sugars and calories, whereas the bitter taste could be a sign of the presence of toxins and/or bacteria (60). The preference for salty taste is not present at birth; this taste is

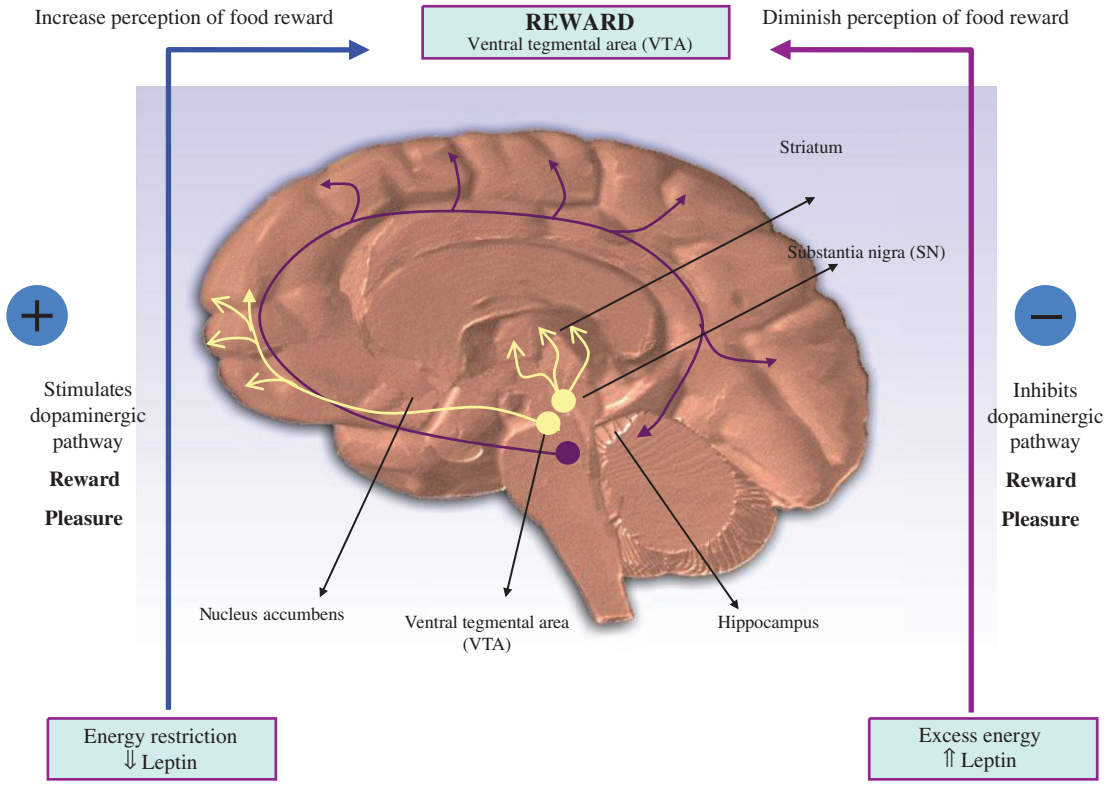

Figure 1 Mesolimbic system: cross-sectional image of the brain.
In conditions of excess energy and high concentration of circulating leptin, this hormone would act by inhibiting the circuits related to the reinforcement, thereby inhibiting the search for food. Under conditions of energy deficit, ghrelin would act by stimulating the pathway related to the reinforcement and induce the search of food.

being acquired at approximately 4 months of age (61). Nevertheless, the addition of monosodium glutamate, conferring umami taste, has been shown to increase the palatability of various foods (62). This suggests that, like the salty taste, umami must be taken in conjunction with other flavours, which classifies it as a flavour enhancer rather than a pleasant taste per se.

Additionally, there are responses to food features that are learned. Children tend to prefer foods with higher energy densities because they provide pleasure when are consumed (63). Along with this, one aspect that markedly influences the acceptability of a food is familiarity. Previous studies have shown familiarity accounts for 50% of the variance in preschoolers' preferences when selecting a food type such as a sandwich (64). For many children and adults, the unfamiliarity of a food is a reason for not choosing it. It is possible that familiar tastes confer a suspected safety in the food presented.

Finally, another less studied aspect that influences food preferences is called social facilitation, a phenomenon that, in other species, shows how the observation of another individual consuming a particular food increases the acceptance amongst peers (65, 66).

Conclusion

Scientific evidence shows that in addition to physiological systems traditionally known as regulators of intake (homeostatic system), eating behaviour in children is influenced by a number of factors that affect the variability in the responses of the paediatric population, such as response to satiety, energy compensation, rate of food consumption, responsiveness to food, food reward and food preferences. It is essential to consider these aspects in the design of the intervention strategy for a patient with obesity, as it is highly likely to influence the success or failure of the plan. All these aspects are relevant and have been associated with adiposity and obesity in paediatric populations.

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