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# Satiety and the self-regulation of food take in children: A potential role for gene-environment interplay

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## Abstract

Child eating self-regulation refers to behaviors that enable children to start and stop eating in a manner consistent with maintaining energy balance. Perturbations in these behaviors, manifesting as poorer child eating self-regulation, are associated with higher child weight status. Initial research into child eating self-regulation focused on the role of parent feeding styles and behaviors. However, we argue that child eating self-regulation is better understood as arising from a complex interplay between the child and their feeding environment, and highlight newer research into the heritable child characteristics, such as cognitive ability, that play an important role in this dynamic. Therefore, child eating self-regulation arises from gene-environment interactions. Identifying the genes and environmental influences contributing to these will help us tailor our parental feeding advice to the unique nature of the child. In this way, we will devise more effective advice for preventing childhood obesity.

### Keywords

children; eating; satiety; self-regulation; gene-environment interplay

## Introduction

Child eating self-regulation refers to the ability, both inborn and socialized, to eat and stop eating in response to internal cues of hunger and fullness [1]. Two distinct processes are involved in child eating self-regulation – satiation and satiety. Satiation refers to the signals

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**Compliance with Ethics Guidelines**

**Conflict of Interest**

Sheryl O. Hughes and Alexis C. Frazier-Wood declare that they have no conflict of interest.

**Human and Animal Rights and Informed Consent**

With regard to the author's research cited in this paper, all procedures were followed in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2000 and 2008.

and processes that occur during the course of a meal that bring the meal to an end whereas satiety refers to the signals and processes that, following the end of a meal, inhibit eating before hunger returns [2]. Satiation and satiety are part of a regulated system within children's bodies that are responsive to the energy content of foods. The goals of this narrative review are to show that current research into the etiology of child eating self-regulation has focused on non-genetic influences, and to argue that this research would be extended by placing these findings in the context of innate predispositions towards child self-regulation abilities.

## Measures

A number of different assessments have been developed to measure satiation and satiety in children. Satiation has been measured through compensation trials by examining the extent to which children compensate for energy consumed in a preload during a subsequent *ad libitum* meal [3,4]. Traditionally, children are seen on two separate occasions only differing in the energy content of a first course preload that children are required to consume in full. Preloads across the two occasions are equal in volume and have similar sensory characteristics, but differ by about 100 calories per serving. Children are then allowed to self-select their intake at a meal served 25 minutes after the preload was consumed. Differences in energy intake at the meal are calculated across conditions [3,4].

The eating in the absence of hunger (EAH) task has also been used to evaluate satiety. In the EAH task children's intake of palatable foods is measured following a meal eaten to satiation [5]. Prior to the task, children are provided with a complete meal and a subjective measure of hunger is used to determine fullness once the children have finished the meal. Sweet and savory snacks such as potato chips, Skittles, pretzels, sherbet, ice-cream, Hershey bars and chocolate chip cookies are then offered to the child along with age appropriate toys. The child is left by him/herself in a room with the food and toys for a specified period of time (e.g., 10 minutes). The total number of calories eaten in the assumed absence of hunger is calculated for each child.

Satiation has also been measured by examining the microstructure of the meal such as measuring the number of mouthfuls eaten by the child per minute [6]. Faster average eating rate is thought to lead to excess child intake because fast eating does not allow internal satiation signals to take effect in sufficient time. External validation of this theory has been suggested by research showing that mouthfuls of food eaten per minute has previously predicted child weight at a later age [7].

More recently, satiation and satiety have been assessed using parent-report measures of these constructs. The Children's Eating Behavior Questionnaire (CEBQ) [8] assesses child eating self-regulation through subscales of slowness in eating (satiation) and satiety responsiveness (satiety). In addition, other aspects of child appetite regulation are measured in the CEBQ such as child food responsiveness, emotional overeating, and enjoyment of food [8]. Food responsiveness refers to how responsive the child is to food and eating (e.g., "My child is always asking for food") [8]. Emotional overeating refers to eating when experiencing negative emotions or boredom (e.g., "My child eats more when worried") [8]. Enjoyment of food refers to how responsive a child is to external food cues (e.g., "My child

is interested in food") [9]. *Developmental trajectories* It has been demonstrated that children have the ability to self-regulate their eating in response to covert changes in the energy content of a preload [3,4]. However, large individual differences in eating self-regulation have been observed in children, with poorer eating self-regulation being associated with higher child weight. In several studies using compensation trials to measure eating self-regulation, children with higher levels of body fat showed poorer eating self-regulation by failing to adjust energy intake at the subsequent meal [10–12]. Moreover, in other studies, older children have been found to show poorer compensation compared to younger children [13,14], leading to the notion that individuals may be born with an ability to self-regulate their energy intake, but that this ability becomes compromised or decreased in some individuals as they age. Similarly, in studies using the EAH task to measure eating self-regulation, heavier children were observed to consume greater amounts of food in the absence of hunger [5,15–20]. As with studies using compensation trials to measure eating self-regulation, older children ate more in the absence of hunger than younger children [12,16,17]. Parent-report of child appetitive traits based on the CEBQ (food responsiveness, emotional overeating, and enjoyment of food) have been positively associated with child weight; in contrast, satiety responsiveness was negatively associated with child weight [9,21–24].

When using a mixed-methods approach to examining child eating self-regulation in samples of young children (parent-report; observation trials) a few studies have identified small correlations between various measures of eating self-regulation [25,26]. The results of these studies support the notion that child eating self-regulation is a complex process and different measures may access relatively independent aspects of the construct. So, when identifying the etiology of child eating regulation we are likely to be looking for the additive and interactive effects of many etiological factors.

## Environmental influences on child eating self-regulation

Many factors may influence child eating self-regulation. One such factor is parent feeding. For example, Johnson and Birch [11] reported *higher parental control* in feeding to be associated with more imprecise caloric compensation in children (measured by compensation trials). These findings suggest that greater parental control in feeding may lessen children's focus on internal cues of fullness thus reducing self-regulation of eating. Other studies examining parent-reported feeding practices ( goal directed behaviors used by parents to get children to do something specific ) found high maternal restriction to be associated with lessened child eating self-regulation (measured by the EAH task) [5,27]. Studies using parent-reported measures of child eating self-regulation found that higher scores on parent modeling and monitoring (autonomy promoting feeding practices) were associated with better child eating self-regulation [28–31].

Other studies suggest that parental feeding may be more complex than originally thought. More recent research examining feeding styles – a more global parent feeding measure than practices involving broad parental attitudes regarding feeding – has shown less controlling feeding attitudes to be related to higher child weight status. Specifically using the Caregiver's Feeding Styles Questionnaire (Hughes et al., 2005) to measure styles, the

indulgent feeding (a style in which parents are very responsive to their child during eating episodes but show little structure and boundaries around feeding) has been associated with higher child weight across multiple studies of children ages 3 to 12, see El-Behadli et al. [32] for a review. It is posited that *lower parental control* in feeding may also lessen children's focus on internal cues of fullness thus reducing self-regulation of eating as well.

This research has started to reveal parental feeding styles or behaviors that may influence the dysregulation of the innate ability children have to self-regulate their eating behaviors. This is clearly an important piece of the puzzle as to why, by preschool, some children are already showing a lesser ability than their peers; however, it does not address questions such as: are children born with differing capacities to self-regulate their eating, and do children respond to these external pressures on their self-regulatory ability differently?

## Characteristics of the child underlying eating self-regulation

An increasing understanding of the role of child characteristics in child eating self-regulation is complementing research into parental influences. Phenotypes measuring cognitive ability – particularly those abilities covered under the umbrella term "executive functions" - have been shown to be related to child body mass index (BMI) providing the potential that executive functions and child eating self-regulation are linked [33–38]. Executive functioning reflects a number of cognitive functions that are processed by the prefrontal cortex and required for such activities as carrying out plans, obeying social rules, and adapting to changing environmental circumstances [39]. Core domains of executive functions for children are inhibitory control (the ability to stop a prepotent response), cognitive flexibility (being able to think about two concepts simultaneously and switch between the concepts as needed), and working memory (the ability to control attention to maintain information in an active, quickly retrievable state) [40]. Although we are unaware of published research directly associating executive functioning with child eating self-regulation, a growing body of literature in adults shows associations between executive function domains and other food behaviors, or provides theoretical links between the two constructs. When adults complete tasks which require more intensive cognitive self-regulatory abilities, such as increased inhibition or more intensive memory recruitment, higher food consumption is seen than with lower demand tasks [41] [42]. This suggests that both cognitive self-regulation and eating self-regulation compete for the same brain processes [41]. Similarly, among female adults with a given goal of not consuming sweets, those with lower working memory act more in line with their automatic affective reactions toward food and consume more sweets than those with higher working memory [42]. This suggests that there may be shared pathways underlying working memory and eating self-regulation and so working memory ability may correlate with eating self-regulation. Similarly, when eating breakfast is a targeted goal, achieving this goal was partially dependent on planning ability [43]. Additionally, in healthy adolescents lower executive ability (termed "dysexecutive traits") was correlated with disinhibited eating and with cognitive restraint of eating [44].

The link between executive function and child eating self-regulation is still somewhat controversial because there is a lack of empirical associations between the two and the

direction of association is unclear. However, we argue that evidence suggests that individual differences in child eating self-regulation associate with individual differences in executive functioning, such that child eating self-regulation may be, in part, one behavioral manifestation of a broader general self-regulatory ability. This theory is appealing, since self-regulatory abilities such as effortful control do not start to manifest until late infancy at the earliest, and continue to develop into executive functions by middle to late childhood. As general self-regulation develops into executive functions, we start to see different developmental trajectories [45], just as we see different developmental trajectories for child eating self-regulation. A key research direction is whether these two trajectories track together–which would provide some evidence that they reflect the same underlying processes. Research is therefore on the cusp of recognizing the role children's characteristics have in shaping their eating self-regulation in addition to the external factors discussed above. Thus, the importance of parent-child interplay in the development of child eating self-regulation comes to the foreground, and this raises the potential for integrating the innate predisposition of the child into this paradigm.

## Genetic influences on child eating self-regulation

### Genes, BMI and a behavioral link between the two

Genetics are the starting point for understanding how innate biology influences the parent-child interplay underlying child eating self-regulation. To support the idea that genetics plays a role in the etiology of child eating self-regulation, it is well established that BMI is highly heritable (40–80%) [46–49]. Further, in the most recent large-scale meta-analysis of adult BMI, over 75% of the newly identified BMI-influencing variants (representing ~30% of all BMI-influencing variants identified and validated thus far) were expressed in the brain, with enrichment in sites of appetite regulation [50]. This suggests eating behaviors are a mediator between genes and BMI.

### Insights from monogenic obesity

Monogenic forms of obesity are those where the excess adiposity is attributable it a single gene mutation. In single-gene forms of obesity, the gene-obesity association arises in part from dysregulated eating behaviors. For example, deficiencies in the *Leptin* (*LEP*) gene which lead to a complete lack of circulating leptin, although extremely rare in humans [51], are known to give rise to patients whose early-onset obesity arises in part from hyperphagia (alongside additional endocrine abnormalities; [52]. Similarly, *melocortin 4 receptor* (*MC4R)* mutations seen in just over 0.05% of the population [53] are the primary cause of obesity in 1–6% of obese individuals. *MC4R* mutations present different phenotypes across the lifespan. Before puberty the manifestation is largely behavioral when the extent of functional impairment in *MC4R* signaling correlates positively with adiposity and hyperphagic behaviors. This association disappears post-puberty, and as the hyperphagia reduces, feelings of satiety increase and a reduction in obesity is seen [53]. Initially it was hoped that understanding monogenic forms of obesity would give valuable insights into common obesity, where it was assumed that mutations at different loci in the same genes would give rise to much milder differences in eating behaviors, and thus be associated with lower incremental increases in BMI. However, this does not seem to be the case and more

fruitful research has screened the genome to uncover genetic causes of common obesity without *a priori* hypotheses based on the etiology of monogenic forms of obesity.

## The heritability of eating behaviors

Like BMI, eating behaviors are consistently heritable, albeit with more of a moderate heritability estimate (40–75% across a variety of child age groups and ethnicities [54–56]. As yet, there are few associations between genetic variants and child eating self-regulation. Those that have been identified have arisen from candidate gene approaches and there is a lack of genome wide approaches (the one genome-wide approach taken to identifying loci associated with eating behaviors did not produce associations at genome-wide levels of significance) [55]. With candidate gene approaches, select sets of genes are examined for association with child eating self-regulation and an immediate challenge is selecting genes for inclusion in such analyses.

## BMI raising alleles and eating behaviors

Since heritable eating behaviors are thought to be one route through which the genetic predisposition to obesity manifests, a natural set of genes to examine for associations with eating behaviors are those that are associated with BMI. *The Fat Mass and Associated* (*FTO*) gene is one of the genes most consistently associated with BMI, with variants showing the largest effect size of all known BMI-raising alleles to date [50]. Using data on 3,337 UK children, a variant in *FTO* was associated with reduced satiety responsiveness scores on the CEBQ [57]. Further, the association of this variant with increased adiposity in this sample was explained in part through effects on satiety responsiveness, showing that child eating self-regulation lies on the pathways from genes to BMI. A smaller study on 139 normal-weight and 238 obese Chilean children (ages 6–12 y) reported similar results with a variant in *MC4R* where the allele seen more frequently in the obese group was also associated with lower CEBQ satiety responsiveness scores (as well as higher CEBQ enjoyment of eating scores; [58]. This study was unusual for a genetic study in that direct observations of child eating self-regulation were also examined for genetic associations; those with the BMI-raising allele in *MC4R* showed a trend to eat more appetizing snack foods after a preload in the EAH paradigm [58].

Of course, as with all complex phenotypes, not all genetic investigations have turned up positive associations. Across 229 children with obesity, no associations were found between variants in *Melanocortin-3 receptor* gene [*MC3R*] or *LEP* / *LEPR* and CEBQ-measured child eating self-regulation. There were associations reported with the emotional eating subscale and the enjoyment of food subscale for *MC3R* [59] and with “slowness in eating, emotional eating” and enjoyment of food” with certain polymorphisms in *LEP* / *LEPR* [60].

## The dopamine-serotonin gene system and eating behaviors

Domains of executive functioning (or their antecedents in younger children, known as general self-regulation) are associated with BMI. There is evidence that this relationship is mediated in part by food behaviors. Therefore, genes underlying executive function theoretically may underlie child eating self-regulation. While few genes have been validated as associating with executive function measures, especially in children, dopamine and

serotonin levels are associated with a cognitive-behavioral profile characterized by heritable components such as a difficulty in inhibiting responses [61,62], the preference for small immediate rewards over larger delayed rewards [62,63], and choice impulsivity [62]. Child eating self-regulation assessed with the CEBQ measures several of the same constructs (difficulty in inhibiting responses [61,62], the preference for small immediate rewards over larger delayed rewards and choice impulsivity) but as applied to food behaviors [64]. Dopamine and serotonin genes have been found to associate with BMI in adults [50], as well as underlie total food intake, eating disorders and preference for sweet, or rewarding foods [65,66]. Their associations with child eating self-regulation remain to be investigated.

## Conclusions and the potential for gene-environment interactions in child eating self-regulation

Child eating self-regulation develops across the lifespan from early childhood. Individual differences emerge and continue to develop, and associate with weight status. It has long been recognized that parent feeding styles and practices influence the developmental trajectory and ultimately, associate with whether a child is a good self-regulator during eating. We also recognize that heritable child phenotypes may associate with the development of child eating self-regulation, and thus it is not surprising that a flurry of recent research has tried to uncover the genetic variants which may account for differences in child eating self-regulation. This work needs to be furthered through large scale genome-wide approaches, and through candidate gene approaches which include diverse gene lists from the literature. It is clear that the biology of the child needs to be integrated into our understanding of child eating self-regulation. This will lay the ground work for better identifying how parent-child interplay shapes child eating self-regulation. It will also inform an understanding of why the associations between parent feeding styles and practices and child eating self-regulation differ among children. For example, although an indulgent feeding style is associated with poorer child eating self-regulation, some children whose parents display an indulgent style remain good eating self-regulators. This research will pave the way for tailoring parent feeding advice to factors such as child temperament and cognitive ability.

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