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Gene-Environment Interplay in Child Eating Behaviors: What the Role of “Nature” Means for the Effects of “Nurture”

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Abstract

Purpose of Review: This narrative review describes the evidence for both genetic and environmental influences on child appetitive traits, and suggests ways of thinking about how these interact and correlate to influence how a child eats.

Recent Findings: Emerging evidence from social network analysis, and from longitudinal studies questioning the direction of association between parent feeding behaviors and child obesity risk, suggest that children’s genes may shape the environmental risk for obesity that they are exposed to.

Summary: There is strong evidence that child appetitive traits are both heritable, and shaped by the environment. Instead of thinking about how genetic and environmental factors operate independently on each appetitive trait, research needs to expand the current paradigm to examine how genes and environments interact and shape each other.

Keywords

BMI, Child, Dynamic Genetics, Eating behaviors, Environment, FTO; Genes, Gene-by-environment correlation; Gene-by-environment interaction; Heritability, Molecular Genetics, Nature, Nurture, Obesity, Obesogenic Environment, Parenting, Pediatrics; Review; Twin study

Introduction

The prevalence of pediatric overweight and obesity remains high in the United States.¹ The potential associations of pediatric obesity with interpersonal difficulties in childhood,² psychosocial difficulties³, and chronic disease later in life,³ underscores the importance of understanding what predisposes a child to developing obesity. In addition, economic and ethnic differences in the prevalence of pediatric obesity in the United States^{1,4} suggests that more effective prevention and treatment could reduce health disparities. Weight status arises

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Conflict of Interest

Alexis C. Wood declares that she has no conflict of interest.

Compliance with Ethics Guidelines

Human and Animal Rights and Informed Consent

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from a complex interplay between genomic, metabolic, biological, behavioral, and psychosocial factors, categories which are not mutually exclusive (e.g., biology can be genetic) and that interact. At its heart, pediatric obesity arises from a situation where more energy is expended than consumed; and that first part of the equation (energy intake) can be described by several child eating behaviors. Thus, studying the etiology of child eating behaviors may help reduce pediatric obesity. As for body mass index (BMI) and obesity, both genetic and environmental factors need to be considered. Traditionally, research has focused on how genes and environments work together, but independently, to affect child eating behaviors. It is not uncommon to hear descriptions of traits as “X% heritable, with the remaining variance due to environmental factors”, which may be read as implying each acts independently. However, genes and environments interact in many complex ways. After briefly reviewing the literature on genetic and environmental effects on child eating behaviors, this narrative review will then discuss some of the ways in which genes and environments may shape each other in the hope that this spurs new ways of thinking about old questions, which in turn will help us design studies that get us ever closer to understanding why some children develop obesity, while others do not.

Key Concepts

A Historical Perspective on Nature vs. Nurture

The notion that behavior may be influenced by our genetic code, as well as our environment, is often traced back to Francis Galton who in 1869 observed that “eminence” ran in families.⁵ Perhaps because of a later association between Galton and eugenics debates surrounding the relative roles of genes and environment in influencing behavior fell out of favor until 1960s.⁶ In the interim, the primary influence on child behaviors, such as eating behaviors, was often considered to be the home, and particularly the mother’s parenting behaviors, with some notion that the school environment and, in older children, peer groups could also have an influence. Some have noted that this still, to an extent, continues today - especially in the field of child obesity.⁷ However, a marked shift occurred in 1960 when the publication of the seminal book “*Behavior Genetics*” by John Fuller and William Thompson formalized the field of behavior genetics, which applies genetic research methodologies (in their broadest sense) to understanding individual differences in behavior, and ignited debated on the relative contribution of genes and the environment to shaping behavior.⁸ From that point in time, the field of behavior genetics has continued to make advance after advance at an almost unprecedented rate proving that behavior is more than a reflection of our upbringing. It is perhaps not hyperbolic to state that there is almost no aspect of child behavior which has not been shown to have some genetic influence.

The Definition of Nature

For the purposes of this narrative review, *nature* refers the association of differences in the base pair sequence that makes up our DNA (deoxyribonucleic acid), in that ~12% of our genetic code where genetic variation exists between humans,⁹ with individual differences in a trait. Something that is often overlooked or misperceived is that while our DNA sequence is, to all intents and practical purposes, unchangeable, and although the expression or the activity, of our DNA can vary, the pattern of base pairs remains stable and beyond our

control, this does not imply that traits under genetic control cannot be changed, or that the portion of a trait attributable to genetic differences is immutable; statements which are simply not true. The most famous counter-example to this error is phenylketonuria (PKU): PKU is a single disorder where mutations in the phenylalanine hydroxylase (*PAH*) gene cause severe intellectual disabilities.¹⁰ However, this behavioral or psychological outcome can be completed or mitigated by removing phenylalanine from the diet.¹¹ A less clear cut but still compelling example is that of childhood obesity. The heritability of pediatric BMI has been robustly demonstrated; after around 5 months of age genetic influences consistently account for at least 50% individual differences in child BMI, and around middle childhood (7 to 9 years of age) this increases to around 75%.¹² Yet we know that changing children's diet and habitual physical activity (themselves potentially heritable child traits^{13,14}) can, for many, reduce their BMI.¹²

The Definition of Nurture

Here, *nurture* refers to anything non-genetic which associates with individual differences in a trait, and so can be thought to influence, or shape that trait. The appeal of concluding that “nurture” underlies a trait is the implication that this trait is then more under our control; if we change the environment we change the trait. However, this must be seen as an over simplification; altering obesity-associated environmental factors at either the individual- (e.g., dietary intake), or societal- (e.g., aspects of the built environment such as walkability¹⁵) may not be so easily under our control and responses of body weight to such changes may differ between individuals - something much harder to predict.

Defining Appetitive Traits

“Child eating behaviors” refers to both “what” a child eats, such as their fruit and vegetable consumptions, and “how” a child eats, such as their tendency to eat when not hungry. For this review, the descriptors of “how” children eat will be termed “appetitive” traits and distinguished from the “what”. As a relatively new field in child psychology, these definitions are constantly being expanded and refined, and even subdivided into, for example, “food approach” versus “food avoidant” behaviors.¹⁶ However, there are some commonly used and broadly accepted appetitive traits, and their definitions are outlined in Table 1.¹⁷

Internal Cues Driving Eating Behaviors

Notably, the association of most appetitive traits with BMI and / or obesity at the theoretical or empirical level, demonstrates their intrinsic relationship to a construct known as the “self-regulation of eating behaviors”. The self-regulation of eating behaviors was defined by Baumeister and Vohs as “the ability, both inborn and socialized, to eat and stop eating in response to internal cues of hunger and fullness”.¹⁸ These internal cues center concern those of hunger, satiation and satiety. Hunger, satiation, and satiety arise from a complex, and not fully delineated, deluge of interdependent and interacting hormones, neurotransmitters, and polypeptides termed “the satiety cascade” in the late 80s by Blundell, Rogers and Hill.¹⁹ The most well-known contributors to these cues are ghrelin (the “hunger hormone”) and leptin (“the satiety hormone”). Hunger is the cue that is expected to drive meal initiation. Hunger is perhaps the least understood of the three internal cues driving eating (or the cessation of

eating) but we currently think arises, in part, from an increase in ghrelin in the blood given the well-known ghrelin peak before meal onset,²⁰ even though mice without ghrelin do not reduce their food intake.²¹ The effect of ghrelin in initiating meal intake is augmented when the hypothalamus registers the decline of plasma glucose.^{22–25} The end of a meal is called satiation; the feeling of satiation is a response to reduced ghrelin, sometimes enabled through the release of cholecystokinin (CKK)^{26,27}. Once eating has stopped, a state known as “satiety” should prevent the initiating of eating until hunger returns; hence a strong and appropriate behavioral response to satiety, the appetitive trait “satiety responsiveness” is associated with the better self-regulation of eating behaviors, and a reduction in obesity risk.^{28–31} The hormone leptin is the most famous satiety factor, which has been shown to cross the blood-brain barrier and bind to receptors in brain regions which contribute to feeding control. Leptin is a ghrelin agonist, and in this sense, the state of “satiety” can also be seen as the “absence of hunger”. “Eating in the absence of hunger” is thus eating without internal cues to initiate a meal, perhaps in response to emotions (“emotional overeating”) or to the sight and smell of food (“food responsiveness” a form of “external eating”). Thus, eating in the absence of hunger indicates poorer self-regulation of eating behaviors and may increase obesity risk in children.^{32–37} In the broadest sense, the prevailing theory dominating the field of pediatric obesity is that children are born with the ability to self-regulate their eating behaviors and maintain a healthy BMI, however, this ability becomes compromised as the child ages and their appetitive traits become socialized to reflect external not internal cues.

Evidence for Nature in Child Appetitive Traits

The Heritability of Appetitive Traits in Children

Twin studies have been the mainstay for substantiating arguments that child appetitive traits are heritable.^{36,38,39,40–43} Through comparing twin pair correlations between members of genetically identical (monozygotic; MZ) and members of fraternal (non-identical, dizygotic; DZ) twin pairs, twin studies have broken down the variance in child appetitive traits into genetic, shared environmental (those environmental factors which make members of a twin pair in the same way), and child-specific environmental (those environmental factors which contribute to differences in eating behaviors between all twin pairs, and subsume measurement error) influences. Twin studies almost universally show a heritable component to child appetitive traits,^{36,38,39,40–43} with most studies estimating that genetics can explain somewhere between ~50% to 75% of the individual differences in in appetitive traits^{36,38,40–43} (Figure 1; Table 2). While a small number of studies do show smaller heritabilities^{39,43} (Figure 1; Table 2), it is not clear why; there is no compelling evidence that the appetitive trait under investigation, the way it is measured (e.g. questionnaire vs. direct observation) nor the age of the population under study is a reason for a lower than expected heritability, although one study noted a shift in puberty which could explain some differences.^{42,43} However, the weight of the current evidence is in favor of a moderate-to-strong heritable component to child appetitive traits.

Molecular Genetic Associations with Child Appetitive Traits

The Fat Mass and Obesity Associated (*FTO*) gene, expressed in the hypothalamus,⁴⁴ is known for its role in behaviors that affect energy homeostasis and is the gene where

variation is most robustly associated with child eating behaviors. Supported by numerous murine studies, small scale candidate gene studies have associated *FTO* variation with greater food intake during a test meal, and less caloric compensation after a milkshake preload in 76 children,⁴⁵ and more eating in the absence of hunger increases in preschoolers (ages 4–5 years).⁴⁶ Larger scale candidate gene approaches have also associated *FTO* variants with, increased energy intake from 3-day weighted food records in a large scale investigation of 3,641 children ages 10–11,⁴⁷ with food responsiveness, but not enjoyment of food, emotional overeating nor satiety responsiveness in 1,718 children,^{48,49} and again with satiety responsiveness in the largest questionnaire-based investigation (N=3337)⁴⁸ with all these data being collected in the United Kingdom.

Evidence for Nurture in Child Appetitive Traits

Environmental Effects on Child Appetitive Traits

Despite the robust heritability to child appetitive traits, evidence suggests that environmental factors also have an important influence. Certainly the environment has many levels through which it can exert influence; Storey, Neumark-Sztainer and French described three such levels of influence: the social environmental or interpersonal level (such as parent feeding behaviors); the physical environmental or community level (including many aspects of the built environment such as the availability of food); and finally the macrosystem or societal level (which includes cultural norms and laws affecting influences such as advertising and mass media).⁵⁰

Parent Feeding Behaviors

Most research into environmental influences on child appetitive traits at the interpersonal level has centered around the role of caregiver feeding behaviors. Although there have been associations between parent feeding behaviors in infancy with child appetitive traits, such as some unreplicated associations between breastfeeding and caloric intake in late infancy,⁵¹ and between breastfeeding and better satiety responsiveness in toddlerhood,⁵² the mainstay of research has been around behaviors the caregiver exhibits when after weaning, when the child is self-feeding solid foods. Practices such as restriction, where parents try to restrict children's access to and / or consumption of what they deem to be 'unhealthy' foods, has been associated with eating more calories in the absence of hunger,^{53–55} higher eating disinhibition,^{53–55} and great food responsiveness^{56,57} in children, although longitudinal studies do not always substantiate this link or support a causal relationship.^{58,59} On the other side, pressure to eat, where parents try to promote and increase the consumption of foods perceived as "healthy", has shown associations with better child satiety responsiveness,^{56,57} but has also been associated with a greater likelihood that children will eat in response to external cues in study,⁶⁰ and show food fussiness.⁵⁷ Finally, *monitoring* - just the awareness parents have of their children's snack food consumption - has also shown associations with child eating behaviors, but with inconsistent directions of association.⁵³ When these feeding practices are coalesced into feeding styles, this latter of which reflect the parent's overall attitude and emotional climate of meals, little research has examined the associations with child appetitive traits, but given some robust associations between an authoritative feeding style, which is defined as a feeding style which sets clear boundaries and so is high on

demandingness, but is also highly responsive to the child,⁶¹ with lower child weight status,⁶² and lower child consumption of energy dense snacks⁶³ is a promising avenue for associations with child appetitive traits. Overall, the prevailing message at the group level is that parent feeding behaviors (whether particular practices or overarching styles) should avoid influencing how much children eat, even with the best of intentions - for example, eating more healthy foods. Observational studies suggesting that the average request a parent makes for a child to eat past satiation is “three more bites”⁶⁴ and this can compromise the abilities of children to self-regulate their eating; current advice is that parents should set boundaries within which children can maintain a natural ability to self-regulate their eating by providing healthful foods (the “what” of eating), but leave how much to eat (the “how” of eating) up to the child.

The Community and Society as Influences on Child Eating Behaviors Parents.

Caregivers, and their feeding behaviors, operate within the physical environment, their community and the broader society. Macro-level shifts have occurred in child-care patterns as mothers (still considered the primary caregiver at a population level) increasingly working longer hours out of the home,⁶⁵ within communities with easy availability of “convenience” food. Alongside a concomitant time trend of increasing portion size,⁶⁶ these aspects of the so-called “obesogenic environment” have been considered to act in concert with interpersonal influences such as parent feeding behaviors to increase obesity risk. However, I am not aware of any studies associating variables at this level with child appetitive traits per se, although associations exist with “what” aspect of child eating behaviors, for example between reduced availability of fruits and vegetables in schools with children’s overall consumption of fruits and vegetables,⁶⁷ and positive associations between portion size and child energy intake have been taken as an example of an environmental factor which can reduce children’s eating self-regulation.

The Nurture of Nature

Epigenetics

The field of epigenetics has raised old debates on the logic behind (or lack of), and ease with which we can define an influence on child behavior as purely nature or nurture. Epigenetics refers to “changes in structure of DNA which do not change the sequence of the DNA but may have phenotypic consequences”.⁶⁸ Often described as turning genes on and off like a light switch,⁶⁹ the effect of epigenetic marks on gene expression are better thought of like a dimmer; across a given genetic region epigenetic marks, such as the addition of a methyl group (methylation), cause an increase or decrease (even to zero in some cases) in the amount of functional product synthesized by a gene, and therefore on the potential biological effect of the gene. Epigenetic marks need to be considered in understanding the etiology of child appetitive traits because results from the Dutch Famine Study,⁷⁰ alongside adiposity differences in genetically identical mice reared together,⁷¹ have earmarked epigenetics as a potential contributor to pediatric obesity.

Blurring the lines

Epigenetic marks are a strange case in the nature / nature debate. Epigenetic changes can sometimes (but not always) be transient; and in the case of methylation, the most well-known and researched candidate epigenetic mark for influencing child adiposity risk, the extent of methyl groups in the genome, as well as, the placement of each methyl group can be influenced by both genetic and environmental influences across the lifespan from embryogenesis⁷² through the neonate period,⁷³ through adolescence⁷⁴ and into older adulthood.⁷⁴ For example, the mother's nutritional state at the time of conception influences methylation in the offspring,⁷⁵ and stress,⁶⁰ particularly trauma,^{66,67,76} associates with patterns of methylation showing how the environment can influence, if not the DNA code itself, then the expression of our DNA. In addition, the notion that environmentally-induced epigenetics marks are only somatically and not transgenerationally heritable (i.e. inherited across generations of cells, but not passed between parent and offspring) is now being challenged, showing how the effects of "nurture" may be transgenerationally heritable.^{77,78}

Dynamic Genetics

Traditionally, genetic and environmental effects were thought to act in an additive fashion, and as applied to child appetitive traits this would mean that the variance in, for example, satiety responsiveness could be portioned into separate genetic and environmental influences which worked together, but did not affect each other. Thinking has moved on since then. Building on the idea that the heritability of a trait does not change the extent to which the environment can alter that trait for individuals, Haworth and colleagues coined the term "dynamic genetics" to describe the observation that within a population, the variance in a trait attributable to genetic influences can change with age, context, or interventions;⁷⁶ for example, along with many other child behaviors, the heritability of appetitive traits appears to increase across childhood (Table 2; Figure 1). Thus, we can immediately start to think of how genes and environment can interact such that shifting the environment can alter the proportion of individual differences in trait that can attributable to genetic differences.

Gene-Environment Interactions

Dynamic genetic refers, in part, to gene-environment interactions (as well as other interactions, such as gene-age interactions). Gene-environment interactions refer to the situation where the genes underlying a trait and / or the extent of genetic influence underlying the trait are dependent on the environment. There is very little research on gene-environment interactions with child appetitive traits, with only one group showing that the "hormonal environment" (i.e. pubertal status, which I acknowledge is also a heritable trait⁷⁹) alters the heritability of "disordered eating" (measured as total score on the Minnesota Eating Behavior Study) with the proportion of variance in individual differences in disordered eating increasing with advancing pubertal development.^{42,43,80}

The Nature of Nurture

More on Gene-Environment Interactions

We saw that genetic effects associated with individual differences in traits can be dependent on the environment. However, the effect of the environment can also be dependent on genetic background. Genetic risk for obesity (a risk score of all known obesity-risk alleles at the time) moderated the association between parent monitoring with child BMI⁸¹ - an association that is thought, in part, to arise from alterations in child appetitive traits suggesting that genes may interact with parent feeding practices.

Gene-Environment Correlation

Genes can also shape environmental exposure for children, through gene-environment correlations. *Active* (or selective) gene-environment correlations refer to situations where children select their own environments based on their genes, for example having obesity is associated with an increased likelihood of having peers with obesity (presumably self-selected to an extent).² Christiakis and Fowler have argued that social networks ‘spread’ obesity through exposure to obesity risk behaviors,⁸² and although this has been challenged, it opens the possibility that obesity risk alleles operate, in part, by increasing the likelihood that a child has a peer group with high obesity prevalence, and therefore increased exposure to obesity risk behaviors. In other words, the environmental risk for obesity (peers with obesity risk behaviors) may be actively sought out due to the child’s genetic predisposition. *Evocative* (or reactive) gene-environment interactions refer to the shaping of the environment due to reactions to a child’s genetic predisposition, which are arguably beyond the child’s control. Although not discussed in terms of gene-environment interaction, this has started to enter the field of child appetitive traits as the field has questioned the direction of association between child appetitive traits and parent feeding behaviors. Children with poor eating self-regulation are more likely to have obesity, and evidence that having obesity elicits more monitoring from caregivers^{83,84} (although there is evidence to the contrary⁸⁵) could be taken as evidence for an evocative gene-environment correlation.

Conclusions.

A complex interplay between Genes and Environment Mechanisms

explaining possible gene-environment interactions are not well understood, but for the purposes of thinking about future research opportunities, a prevailing theory is an individual’s genes set their potential, or propensity, but for this to be fulfilled the environment has to foster trait development. We now need to build in how genes and environments interact and shape each other in the etiology of child appetitive traits. For example, noting that factors in the satiety cascade have a genetic component, it is reasonable to question whether there are individual differences in children’s cues of hunger, satiation and satiety and so some children may have a reduced ability to self-regulate their eating. Based on this, it has previously been suggested that some children (those with a less strong ability to self-regulate their eating) may benefit from more parental control, such as monitoring and restriction⁸⁶ - a potential example for how genes could interact with the environment.⁸⁶

As Sandra Scarr said in her Presidential address to the Society for Research in Child Development: the reason we consider questions of nature and nurture, is to allow us to ask “why do people differ?” and then to ask: “how can we use this information to not to understand human behavior, but improve health?”.⁸⁷ This underscores the importance of refining our understanding of how gene-environment interplay influences child appetitive behaviors, as does fears that not thinking about these aspects in a sensitive and contemporary manner leads to “caregiver guilt” in the face of a child suffering from obesity. Thus it is worth emphasizing several supported notions: (1) child appetitive traits show heritability; (2) there are environmental associations with child appetitive traits; (3) we know both that genes and environments interact such that one prevention or intervention strategy may not have the same effect on all children’s behavior; and (4) that genes shape our environments. Given the recently noted lack of effective interventions targeting child appetitive traits through parent feeding behaviors;⁷ it may be time for behavioral scientists and geneticists alike to expand their studies to incorporate the multitude of interactions and interdependencies between the two influences on child obesity risk.

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Figure 1:
Heritability estimates of Child Appetitive Traits by Age

Table 1:**Key Appetitive Traits**

Eating Behavior	Description
<i>Eating Behaviors Theorized to be “Obesogenic”</i>	
Eating in the absence of hunger	Eating appetizing snack food soon after satiation
Eating rate	How many bites are taken per minute, as well as changes in this across a meal
Enjoyment of food	The extent to which food liked by a child provoke the consumption
Emotional Eating / Overeating	Increased consumption of food in the presence of negative emotions
External eating	Eating in response to external, not internal cues
Desire to drink	The inclination to carry and consume drinks
Food fussiness	Rejecting multiple different types of foods
Food responsiveness	Eating in response to food cues (such as the sight or smell of food)
<i>Eating Behaviors Thought to Protect Against Obesity Risk</i>	
Emotional undereating	When negative emotions trigger lower food consumption
Restrained eating	Employing strategies to reduce food consumption
Satiety responsiveness	Not eating after satiation until hunger returns
Slowness in eating	Fewer bites per minutes, or a reduction in bites per meal during a the meal

Table 2:

Heritability estimates for child appetitive traits

Appetitive Trait	Estimated Heritability
<i>Infancy (0–1 years)</i>	
Slowness in eating	0.74 ³⁸
Satiety responsiveness	0.56 ³⁸
Appetite size	0.49 ³⁸
<i>Early and Middle Childhood (3–12 years)</i>	
% compensation after a preload	0.00 ³⁹
Satiety Responsiveness/Slowness in eating	0.63 ⁴⁰
Enjoyment of Food	0.75 ⁴⁰
Eating rate	0.62 ⁴¹
Disordered Eating	0.02 ⁴²
<i>Post-pubertal</i>	
Disordered Eating	54 ⁴³
<i>Late Childhood (15–17 years)</i>	
Disordered Eating	0.60 ⁴²
<i>Childhood (5–18 years)</i>	
Eating In the Absence of Hunger	0.51 ^{36a}