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Eating behaviors of children in the context of their family environment

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Abstract

Both a family history of obesity and early childhood obesity have been identified as strong predictors of adult obesity risk. The finding that parental obesity, maternal obesity in particular, increases a child's risk for developing obesity suggests that either shared genes, or environment, or likely a combination of both may promote overeating and excessive weight gain in children. Parents not only create food environments for children's early experiences with food and eating, but they also influence their children's eating by modeling their own eating behaviors, taste preferences, and food choices. Thus, it is important to identify intermediary behavioral eating traits which promote overeating and obesity in children and to determine the extent to which associations between eating traits and excessive weight gain in children may be influenced by genetic factors, environmental factors, or both. Behavioral genetic methods can be used to help partition genetic and environmental sources of variability in behavioral traits. The focus of this paper is to review and discuss findings from both short-term experimental and prospective cohort studies on eating behaviors of children at various stages in their lives. Select child eating traits and parent-child resemblances in eating will be further examined in the context of children's home environment and their familial predisposition to obesity.

Keywords

Eating behaviors; children; parents; familial predisposition; obesity

1. Introduction

Since the 1970s, the prevalence of obesity, defined as a body mass index (BMI; kg/m^2) at or above the 95th percentile for children of the same sex and age, has more than doubled for preschool children (ages 2–5 years) and adolescents (ages 12–19 years), and it has more than tripled for children ages 6–11 years¹. The increase in childhood obesity is alarming not only because obese children exhibit risk factors for chronic diseases such as hyperlipidemia, hyperinsulinemia, hypertension, and reduced high-density lipoprotein cholesterol^{2–6}, but also because both obesity and risk factors for chronic diseases can track from childhood into adulthood and thereby have lasting adverse health effects⁷. Data from a retrospective cohort study indicate that after six years of age, the risk for obesity in adulthood exceeded 50 percent for obese children, as compared with about 10 percent for nonobese children⁸.

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A family history of obesity has been identified as a strong risk factor for adult obesity. It is estimated that parental obesity more than doubles the risk of adult obesity among both obese and nonobese children under 10 years of age⁸. To better prevent childhood and adult obesity, it is crucial to identify early behavioral risk factors that predispose children to excessive weight gain during childhood. The aim of this review is to discuss select eating traits and parent-child resemblances in eating in the context of children's home environment and familial predisposition to obesity.

2. Obesity 'runs in families'

Parental obesity, maternal obesity in particular, is a significant predictor of obesity in the offspring. A prospective cohort study by Strauss and Knight⁹ showed that children of obese mothers were at a 3-fold increased risk for childhood obesity compared to children of nonobese mothers. Parental obesity confers its risk for obesity in the offspring through both shared genes and environmental factors. A growing body of research has examined direct genetic links between parent and child weight status^{10–13}; however, relatively little is known about behavioral factors which may mediate family resemblances in weight status.

Children's early experiences with food and eating occur in the home food environment. Parents not only create food environments for their children, but they also influence their children's eating by modeling eating behaviors and food choices¹⁴. Therefore, it is conceivable that parental behaviors and child feeding practices interact with genetic predispositions to promote the development of problematic eating behaviors or less nutritious food choices in children.

To design effective prevention and treatment strategies for childhood obesity, it is important to first identify intermediary behavioral eating traits which promote this increased energy intake and weight gain in children. Additionally, it is crucial to determine the extent to which the association between eating traits and excessive weight gain in children may be influenced by genetic factors, environmental factors, or both. Behavioral genetic methods can be used to elucidate specific environmental variables and intermediary behaviors associated with the development and maintenance of obesity. In this review, genetic influences will be discussed primarily in the context of a familial predisposition to obesity (i.e., obesity risk conferred to children by parental obesity). Environmental influences discussed in this review will focus on parental modeling of eating behaviors and food choices, child feeding practices, and the role of the home food environment (e.g., availability of healthy foods in the home).

The review will illustrate how the familial transmission of eating traits can be studied using various behavior genetic methods. First, findings from a high-risk design of obesity will demonstrate how select eating traits and dietary choices can be compared in children with different familial predispositions to obesity. Second, findings from twin studies will provide heritability estimates for select food preferences and eating traits. Lastly, data from family correlation designs will give examples of parent-child resemblances for food choices and eating behaviors.

3. High-Risk Design of Obesity

In a high-risk design of obesity, children who are born at 'high-risk' are compared to children who are born at 'low-risk' for obesity on specific eating behaviors or food preferences. In this design, child risk status for obesity is commonly defined on the basis of parental, typically maternal, weight status¹⁵. Comparing eating behaviors of children whose parents differ in weight status (e.g., normal-weight versus overweight/obese) can be a useful strategy for testing whether familial vulnerability for obesity may express itself through specific eating traits. Further, data from a prospective cohort study can be used to test whether specific eating traits mediate the relationship between obesity risk status and subsequent adiposity gain. The design

is not suited for discriminating between genetic and environmental influences on children's eating behavior and weight development.

The 'Infant Growth Study' (IGS) is an example of a high-risk design of obesity. It is a prospective cohort study which assesses the growth and development of children born with different familial predispositions to obesity. Child participants' obesity risk status was based on maternal pre-pregnancy body mass index (BMI; kg/m²). Children who were born to mothers with a BMI of less than the 33rd percentile (mean maternal BMI 19.5 ± 1.1 kg/m²) were classified as at 'low-risk' for obesity, while children who were born to mothers with a BMI greater than the 66th percentile (mean maternal BMI 30.3 ± 4.2 kg/m²) were classified as at 'high-risk' for obesity. Details of parental characteristics and study design were reported previously^{16–18}.

Children's weight and adiposity were assessed every 3 months during the first year, every 6 months through year 4, and yearly thereafter. These assessments showed that by age 4, high-risk children were significantly heavier than low-risk children as indicated by a significantly greater weight, BMI, lean body mass, and waist circumference¹⁹. By age 6, high-risk children also showed a significantly greater fat mass and percentage body fat than low-risk children. Given these early differences in the weight development and adiposity, it is important to identify behavioral mediators related to children's eating or physical activity patterns that may underlie the diverging growth patterns in the two obesity risk groups. The energy density of food is one dietary component that has been identified as an important predictor of energy intake in both children and adults.

3.1. Dietary energy density

Energy density is the amount of energy provided in a particular weight of food (kcal/g). The energy density of a food is influenced by its water content and macronutrient composition. Water has the greatest impact on energy density because it adds weight but no calories. Of the macronutrients, fat is the most energy dense, providing 9 kcal/g, followed by carbohydrates and protein which both provide 4 kcal/g. In experimental studies, the energy density of foods has been identified as an important predictor of short-term energy intake in both adults^{20–27} and children^{28–31}. To date, few randomized behavioral treatment studies with children and their parents have tested the effects of reducing dietary energy density on longer-term energy intake and weight development^{32,33}. Data from cross-sectional studies showed that dietary energy density was independently and significantly associated with higher BMI³⁴, elevated fasting insulin levels, and the metabolic syndrome in U.S. adults³⁵. Similarly, data from prospective studies in children indicated that higher dietary energy density was associated with excess adiposity during childhood^{36–38}.

To date, little is known about the genetic and learned familial influences on the relationship between dietary energy density and energy intake in children. There is evidence for a genetic contribution to daily and meal-specific dietary energy density³⁹. Therefore, it is conceivable that children may seek energy-dense foods by way of their genetic predisposition to the taste or higher energy content of those foods.

Home environmental factors, such as availability and easy accessibility of specific foods, have also been shown to influence the formation of food preferences and consumption patterns in children^{40,41}. At the same time, parental feeding practices may enhance innate predispositions for the liking of foods higher in energy density, consequently promoting eating habits that favor an increased consumption of energy-dense foods. For example, repeated exposure to foods has been associated with increased liking and consumption of those foods^{42–44}. On the other hand, it is also possible that parents of obesity-prone children restrict access to more energy-dense foods in an effort to moderate their children's energy intake and weight gain. The use of overly

restrictive feeding practices, however, has been associated with greater energy intake and increased child weight status^{45–47}. It is important to note that several previous studies were either conducted in relatively narrowly defined study samples, namely Caucasian children⁴⁶, or have focused only on girls⁴⁷. The lack of diversity in these study samples limits generalizability of the findings to children as a whole. It thus would be desirable to examine child feeding practices in families from more diverse racial and ethnic backgrounds and to also include boys.

No prospective study had assessed developmental changes in daily energy density in young children, while also taking into account children's familial predisposition to obesity. It is possible that obesity-promoting genes exert their influence through consumption of more energy-dense diets. If true, children born at high risk for obesity would be expected to consume diets of higher energy density compared to children born at low risk for obesity. Hence, as part of the IGS, we characterized 4-year trajectories in dietary energy density across both high-risk and low-risk children at ages 3–6 years; we also sought to examine the relationship between dietary energy density and child weight status within each year. Dietary energy density (with and without beverages) was computed from 3-day weighed food records which parents completed at each of the respective years⁴⁸.

Figure 1A depicts the mean daily dietary energy density, based on solid foods only (all beverages excluded), consumed by low-risk and high-risk children at ages 3, 4, 5, and 6 years. The findings from this study indicated that dietary energy density increased as children grew older, but did not significantly differ for low-risk or high-risk children at any of the years. The lack of a significant difference in dietary energy density between high-risk and low-risk children suggests that the types of foods consumed by children at an early age were similar. Because young children often have a more limited exposure to different types of foods than do older children, it is possible that differences in dietary energy density between obesity risk groups may start to emerge later in life when children have access to a wider array of foods both inside and outside the home.

With respect to daily energy intake (calories consumed from foods only, all beverages excluded), a significant risk group-by-time interaction was found, indicating that high-risk children consumed significantly more energy from food at age 6 than did low-risk children (Figure 1B). The finding of a significant difference in energy intake from foods at age 6 in the absence of a significant difference in dietary energy density suggests that children in the two risk groups may have consumed differential quantities of foods at that age. Child BMI z-scores, waist circumference, and percent body fat were not significantly correlated with dietary energy density in any of the 4 years.

When the IGS subjects were 12 years of age, they were invited to participate in a dietary assessment as part of their annual study visit. The aim of this assessment was to determine if, when given a choice, the dietary energy density and energy intake at a single, self-selected meal differed for adolescents in this study as a function of their familial predisposition to obesity and their sex⁴⁹. For lunch, subjects were served a buffet meal consisting of a variety of foods and beverages ranging in energy density. The meal provided approximately 5200 calories, and subjects could freely choose the types and amounts of foods and beverages they wanted to consume. The energy density of the meal was based on subjects' intake and was computed by dividing the total calories consumed by the total weight of food consumed excluding all beverages. High-risk subjects consumed a significantly more energy-dense meal than low-risk subjects (1.84 vs. 1.42 kcal/g). This difference remained significant when adjusting for subjects' current BMI z-score. There was a trend ($p = 0.16$) for energy intake, when expressed as a percentage of subjects' daily estimated energy requirement, to be different between high-risk ($42\% \pm 4$) compared to low-risk ($33\% \pm 4$) subjects. The susceptibility of

high-risk subjects to consume a more energy-dense diet, if sustained, may predispose this youth to negative long-term health consequences. Data from a cross-sectional, nationally representative survey in adults indicated that diets that are higher in energy density tend to be of lower diet quality⁵⁰, which in the long term, can lead to adverse health consequences, such as the metabolic syndrome³⁵.

In contrast to the findings from earlier years⁴⁸, this study showed that, during a single multi-item meal, high-risk subjects self-selected a more energy-dense meal compared to low-risk subjects when they were 12 years of age. It is possible that differences in the methodologies used to assess subjects' intake (maternal self-report vs. laboratory measure) may have contributed to the difference in findings with respect to dietary energy density. It is also conceivable that differentiations in children's dietary patterns may develop over time, thus explaining why differences in dietary energy density between low-risk and high-risk children did not yet appear at a younger age. Home food environments, which help shape eating behaviors and food preferences in children¹⁴, may change over time or may exert their influence at critical developmental periods. For example, in households with a family history of obesity, adolescents may have had easier access to more energy-dense foods during their upbringing, which in turn may have helped shape their long-term preferences for those foods. Parents also may have used different feeding strategies at different stages of their children's lives. It is possible that, out of concern for their children's increasing adiposity, parents of high-risk youth may have restricted access to energy-dense foods more than parents of low-risk youth. In turn, this restriction may have enhanced high-risk adolescents' preference for more energy-dense foods. Lastly, parents, mothers in particular, serve as important role models for eating behavior and food choices for their children^{15,51–55}. It is conceivable that mothers, through many years of role modeling, may have passed on to their children some of their own food preferences.

Although the types of foods consumed are important determinants of dietary energy density and energy intake in children, daily energy intake can also be affected by children's eating behaviors. Eating phenotypes such as the rate of eating or children's responsiveness to portion size changes can significantly affect the number of calories consumed at a meal. Identifying eating behaviors that distinguish normal-weight from overweight and obese children and that may make overweight and obese children more prone to overeating are important first steps in the prevention and treatment of childhood obesity.

3.2. Sucking and eating rate

When examining the microstructure of eating behavior, the rate of eating has been identified as an indicator of appetitive avidity⁵⁶. Specifically, it has been suggested that a higher eating rate signals a greater motivation to eat. Findings from studies comparing the eating rates of normal-weight adults to those of overweight or obese adults have been mixed; while some studies showed a faster eating rate^{57–59} or a less pronounced deceleration in the rate of eating over the course of a meal⁶⁰ in obese individuals, others found no differences between weight groups^{61–64}. Findings from studies in children are more consistent and indicate that obese children exhibited a higher eating rate during meals and a less rapid deceleration of intake towards the end of the meal compared to normal-weight children^{65,66}. More recently, eating rate has been identified as a highly heritable behavioral phenotype (heritability, $h^2 = 62\%$)⁶⁷. Together, these data suggest that differences in eating rate, which seem to be in part genetically determined, could be implicated in the development of childhood obesity. Longitudinal studies are needed to examine causal relationships between eating rate and child adiposity.

When IGS participants were 3 months of age, their sucking behavior was measured in the laboratory using an automated nutritive sucking apparatus¹⁷. In this experiment, infants were

fed either expressed breast milk or their customary formula milk with their customary nipple type. High-risk infants, compared to low-risk infants, showed a more vigorous sucking behavior as indicated by a significantly higher total number of sucks (920 ± 89 vs. 620 ± 47 sucks), a higher sucking rate (0.75 ± 0.01 vs. 0.59 ± 0.04 sucks/second), and a larger amount of milk consumed (150 ± 9 vs. 123 ± 8 g), but no difference in feeding time. It remains unknown if energy intake differed between risk groups during the sucking behavior assessment. A prospective analysis showed that the total number of sucks at 3 months of age was a significant predictor of weight and weight-for-length at 12 months of age, accounting for approximately 9% of the variance¹⁷.

The assessment of eating rate was repeated when IGS participants were 4 years of age⁶⁸. This time, children consumed a multi-item test meal in the laboratory in the presence of a parent. Eating rate was computed as calories and mouthfuls consumed per minute. Results showed a non-significant trend ($p = 0.10$) for eating rate to be greater among high-risk (2.60 ± 1.08 mouthfuls/min) compared to low-risk (2.16 ± 0.96 mouthfuls/min) children. Further, energy intake per minute was significantly correlated with meal energy intake and daily energy intake (≥ 0.40). When controlling for maternal BMI, a prospective analysis of eating rate at 4 years of age, expressed as either mouthfuls per minute or calories consumed per minute, was significantly associated with an increased probability of a child being overweight or obese at age 6. These data suggest that an accelerated eating rate may present a behavioral risk factor for increased energy intake and excessive weight gain in children. Interventions designed to slow the eating rate in obesity-prone children may be a promising strategy to moderate their energy intake. For example, Epstein and colleagues⁶⁹ tested the effects on eating rate and food intake of instructing 7-year-old children to place their utensils on the table after every ingested bite. The results of the study indicated that both eating rate and the amount of food consumed significantly decreased for both obese and non-obese children as a function of putting eating utensils down between bites combined with praise for compliance.

The microstructure of children's eating has also been shown to be affected by environmental factors such as the portion size of foods. When the portion size of an entrée was doubled, 2- to 9-year-old children consumed 29% more of the entrée and 13% more energy at the meal than when served a standard entrée portion⁷⁰. The increase in entrée intake was in part attributable to an increase in children's bite size. Further, children who increased their intake when served the large portion entrée showed increases in both bite size and the total number of bites taken. These data suggest that interventions which combine slowing children's rate of eating with modifying the amounts and also types of foods served may be a successful strategy to moderate energy intake in children who are prone to excessive weight gain.

Identifying eating traits which promote increased energy intake in children is crucial for the design of successful interventions to prevent and treat childhood obesity. At the same time, however, it is important to study the home environment in which children have early experiences with food and eating. Additionally, because parental behaviors surrounding eating and child feeding are known to also play a crucial role in the formation of food preferences and eating behaviors in young children, the familial transmission of taste preferences, food selections, and eating behaviors must also be studied closely.

4. Twin Studies and Family Correlation Designs

4.1. Familial transmission of taste preferences and food selections

Parents influence the development of children's taste preferences and food choices in multiple ways. One, parents pass their own food preferences and taste perceptions on to their children through genetic transmission. Twin designs can be used to provide estimates of genetic and environmental influences on child eating traits and food preferences¹⁵. For example, when

assessing food preferences in 4- to 5-year-old monozygotic and dizygotic twins using maternal report, modest heritability for food preferences was found for dessert foods ($h^2 = 20\%$), vegetables ($h^2 = 37\%$), and fruits ($h^2 = 51\%$); high heritability was found for protein foods such as meat and fish ($h^2 = 78\%$)⁷¹. In addition to passing on preferences for specific foods groups, parents also transmit eating traits that may limit food selections in the offspring. For example, sensitivity to the bitter tastant 6-*n*-propylthiouracil (PROP), which can influence liking ratings and food selection⁷², has also shown to be highly heritable ($h^2 = 72\%$). Food neophobia, or the avoidance of new foods, is an eating trait which is associated with lower diet quality or variety^{73,74} and has been shown to be highly heritable ($h^2 = 66\text{--}78\%$)^{75,76}.

Family correlation designs can be used to examine resemblances among members of the same family (e.g., parent-child; siblings) for food selections, food preferences, or eating traits¹⁵. For example, many studies have found significant parent-child similarities in food preferences⁷⁷ or neophobia^{78–80}; however, the magnitude of the associations is often small. These examples of a familial transmission of taste preferences and food selection point to a biological component that predisposes children to like or dislike certain foods or food groups.

Genetic predispositions for food preferences or eating behaviors can be modified by environmental factors such as parent feeding practices, parent modeling of eating behaviors and food selections, and availability and accessibility of foods in the home. Exposure to different food flavors and flavor learning begins in utero (prenatal effects) where flavor compounds are transmitted from mothers' diet to amniotic fluid⁸¹. Flavor learning then continues after birth through exposure of the nursing infant to breast milk or formula milk^{82, 83}. During early childhood, parents influence the development of food acceptance patterns and eating behaviors by structuring children's early eating environments⁸⁴. They not only decide the types and amounts of foods that are served to children, but they also provide the social context in which eating occurs.

During early childhood, parents serve as important role models for their children^{52,85–88}. Modeling of eating behaviors and food selections has been shown to play an important role in the establishment of food acceptance patterns in children⁸⁹. Watching a model (e.g., mother) eat an initially disliked^{90,91} or novel⁹² food can significantly enhance taste preferences in children. A recent cross-sectional study⁹³ with mothers and their 5- to 6-year-old children found that children's daily intake of 13 different fruits and 21 different vegetables was significantly associated with their mothers' daily intake of those fruits and vegetables when assessed by maternal report. The mother-child resemblances in fruit and vegetable intake found in this and other studies^{94–97} suggest that family influences can play an important role in food choices. The cross-sectional nature of many studies, however, limits inferences regarding causality, and it should be noted that mother-child feeding relationships are often bi-directional. While mothers can influence their children's food choices and eating behaviors by modeling food consumption or providing certain foods, it is also conceivable that children's innate taste preferences influence what mothers are feeding. For example, children may request particular foods that mothers consequently provide.

Besides modeling healthy food selections, child feeding practices can also impact food acceptance patterns in children. For example, repeated exposure (8–15 taste exposures) to novel foods has been shown to significantly reduce neophobia in children^{42,44}. Also, making healthy foods available and easily accessible to children can influence their intake behaviors. Cullen and colleagues⁹⁸ showed that the availability and accessibility of fruits and vegetables in the home accounted for 35% of the variance in reported consumption of those foods.

In sum, food preferences in children can be modified by factors in the home environment which include, but are not limited to, parental modeling of taste preferences and food selections and access to certain types and amounts of foods.

4.2. Familial transmission of eating behaviors

Parent-child similarities not only have been found for food preferences and types of foods consumed, but resemblances have also been identified for specific eating behaviors such as dietary restraint and disinhibition. Dietary restraint refers to the tendency of individuals to consciously restrict food intake to control body weight or promote weight loss⁹⁹. Disinhibition, on the other hand, is defined as the loss of self-imposed cognitive control of eating in response to external or emotional stimuli¹⁰⁰. Both of these eating behaviors are heritable. Heritability estimates for disinhibition range between 18 and 45%^{101–103}, and those for cognitive restraint range between 0 and 59%^{101–104}. Environmental factors, such as parental modeling of behavior or child feeding practices, have also been shown to influence the regulation of energy intake in children. For example, Johnson¹⁰⁵ examined whether parents' eating behaviors were related to their 3- to 4-year-old children's ability to self-regulate energy intake in a laboratory setting. The findings from this study showed that mothers' disinhibition was inversely associated with their children's ability to regulate energy intake. Mothers who practiced greater cognitive control over their own eating had daughters who showed poorer self-regulation in their energy intake. These findings suggest that mothers who have difficulty controlling their own food intake had daughters who had greater difficulty in self-regulating their intake. Interestingly, after teaching children to focus on internal cues of hunger and satiety, the association between maternal disinhibition and daughters' ability to self-regulate energy intake was no longer significant¹⁰⁵. This finding suggests that learning plays a vital role in the development of children's eating behaviors and that parents can serve as important role models in this process.

Early child feeding and parenting practices further influence the development of children's food preferences and eating behaviors. A study with 4- to 6-year-old girls showed that daughters' dietary restraint and emotional disinhibition were related to their perceptions of parents to pressure them to eat more¹⁰⁶. Girls' external disinhibition, on the other hand, was related to their perceptions of having restrictions placed on their eating by their parents. A retrospective study in women who were asked to recall parent feeding practices during early childhood indicated that women who were encouraged to clean their plate as a child (pressure to eat) and whose parents used food as a reward showed significantly higher scores on disinhibition and dietary restraint, respectively¹⁰⁷. These findings suggest that restrictive or overly controlling feeding practices may contribute to the development of dietary restraint and disinhibition in young girls, ultimately impeding their ability to self-regulate their eating.

Parental disinhibition and dietary restraint also seem to mediate the development of increased adiposity in children. A prospective study with 3- to 5-year-old children showed that children whose parents both scored high on disinhibition and dietary restraint had greater increases in body fat over a 6-year period than children with only one parent or neither parent scoring high on those eating behaviors¹⁰⁸. Another study indicated that higher maternal disinhibition was associated with greater eating in the absence of hunger in daughters and maternal disinhibition also mediated the relationship between mothers' BMI and daughters' overweight⁵². These findings suggest that children who are raised by parents who display high levels of disinhibited eating in conjunction with high dietary restraint may be at an increased risk for developing excess body fat.

Parent-child similarities in some eating behaviors may be sex-specific and may diminish as children grow older. A study with 14- to 22-year-old adolescents and their parents examined parent-child resemblances in cognitive restraint, uncontrolled eating, and emotional eating

¹⁰⁹. In sons, uncontrolled eating was positively associated with fathers' cognitive restraint, but not with fathers' uncontrolled eating nor with mothers' eating behavior. Daughters' cognitive restraint was positively related to mothers' uncontrolled eating, and daughters' uncontrolled and emotional eating were positively associated with the same eating domains in mothers. None of the daughters' eating behaviors were associated with fathers' eating behaviors. These data suggest that adolescents' eating behavior more closely resembles that of their same-sex parent than that of their opposite-sex parent. The aim of a longitudinal study with adolescents between 13 and 16 years of age was to examine family resemblances and reciprocal parent-child influences on restrained eating ¹¹⁰. Cross-sectional analyses showed significant parent-child associations in restrained eating. Over time, however, parents' restrained eating did not systematically predict their children's restrained eating, or vice versa. These data suggest that as children grow older and more independent, familial influences or resemblances on eating behavior may diminish; in turn, other factors, such as peer influences, may become more influential. Other studies ^{111,112} with older children (> 17 years of age) also found no significant parent-child association in cognitive restraint.

In sum, the inter-generational transmission of eating behaviors is facilitated by genetic and environmental factors that involve parent modeling of eating behaviors and use of certain child feeding practices. Parent-child similarities in certain eating behaviors seem to diminish as children grow older.

Conclusion

The purpose of this review was to illustrate the role of familial predispositions and early influences in the home environment in the intergenerational transmission of food preferences and eating behaviors from parents to their children. More studies are needed to examine early-life risk factors for overeating and excessive weight gain in children from more diverse racial, ethnic, and socioeconomic backgrounds. It is essential to identify children who are at greatest risk for excessive weight gain and to also determine critical periods for when children may be most responsive to dietary interventions that help them acquire taste preferences for healthy foods, such as fruits and vegetables, while moderating their intake of less healthful foods. These critical periods for dietary interventions may be earlier than previously thought and may include the earliest stages of life such as pregnancy and infancy ¹¹³.

The study of behavioral mediators of family resemblances in weight status is expected to provide critical information for the prevention and clinical management of childhood obesity. Familial predispositions to obesity, which are in part mediated through specific eating traits and food preferences, can be modified by parental behaviors (e.g., modeling, child feeding behaviors) and the home environment (e.g., accessibility to healthy foods). These findings suggest that efforts to increase healthy eating while moderating energy intake in at-risk children should be extended beyond the level of the individual child to parents and families. Interventions targeting healthy eating in children may have a greater impact if healthful foods are made available and easily accessible in the home and if these healthful foods are also consumed by mothers or other family members in the household.

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Figure 1. Mean ($\pm$ SE) energy density (Panel A) and energy intake (Panel B) consumed by low-risk and high-risk children from ages 3 to 6 years