Influences on Child Eating and Weight Development from a Behavioral Genetics Perspective

Tanja V. E. Kral,1 PhD, and Myles S. Faith,1,2 PhD
1University of Pennsylvania and 2The Children’s Hospital of Philadelphia

Childhood obesity is a strong risk factor for associated comorbidities such as type 2 diabetes, cardiovascular disease, and orthopedic abnormalities in youth and its increasing incidents thus represents a major public health concern. The following review provides evidence for a familial association between parental and child weight status, eating behaviors, and food preferences. It further draws the link between environmental influences, such as parent feeding practices, and the development of child eating behaviors and thereby elucidates how genetic and nongenetic influences can contribute to the familial transmission of obesity. We use eating in the absence of hunger, an eating trait which refers to children’s susceptibility to eating in response to the presence of palatable foods in the absence of hunger, as an example to illustrate these associations. The review concludes with an outlook on possibilities for future research efforts in the field.

Key words behavior genetics; childhood obesity; eating in the absence of hunger; eating traits.

Introduction

The prevalence of overweight among children has more than tripled since the early 1960s (Ogden, Flegal, Carroll, & Johnson, 2002; Ogden et al., 1997; Strauss & Pollack, 2001). The most recent data from the National Health and Nutrition Examination Survey (NHANES) (2003–2004) indicate that approximately 14% of children 2–5 years and 19% of children 6–11 years are overweight, which represents a 25–35% increase since the beginning of this decade alone (Ogden et al., 2006). Children from certain ethnic subgroups are disproportionately affected by this increase in body weight. For example, among Mexican American and non-Hispanic Black youth the prevalence of overweight ranges from 10% to as high as 27% across different age and gender subgroups (Ogden et al., 2006).

Childhood overweight is a critical risk factor for associated comorbidities such as type 2 diabetes (T2D), cardiovascular disease, and orthopedic abnormalities (Berenson et al., 1998; Daniels et al., 1995; Hardy, Harrell, & Bell, 2004; Rosenbloom, Joe, Young, & Winter, 1999). There is currently great concern about the development of T2D in youth, which also is increasing in prevalence (Bloomgarden, 2004; Fagot-Campagna, 2000). As Cruz and colleagues noted, obesity and family history of T2D are two of the strongest risk factors for the development of the disease in youth (Cruz et al., 2005). Dietary factors have been shown to affect insulin dynamics and β-cell (i.e., cells which produce and release insulin) function in children. For example, in overweight Latino children, ages 10–17 years, total sugar intake (g/day) was found to be positively correlated with children’s body mass index (BMI), BMI z-scores and total fat mass and negatively correlated with insulin sensitivity and disposition index (i.e., index of β-cell function) (Davis et al., 2007). These associations underscore the relationship between diet and disease risk and the need for dietary interventions to promote healthy eating habits in children, especially those who are at high risk for the development of T2D. The question of how children develop eating patterns, food preferences, and the ability to regulate energy intake during growth is fundamental to understanding the development of childhood obesity and its complications, including T2D.

Obesity-promoting dietary environments are thought to encourage excessive energy intake among children by offering convenient access to large portions of palatable, energy-dense foods. At the same time, genetic predispositions determine how individuals will respond to the...
environment. One behavioral eating trait which has been shown to be influenced by both genetic and environmental factors is eating in the absence of hunger (EAH). EAH, which refers to children’s susceptibility to eating in response to the presence of palatable foods in the absence of hunger, has been shown to be a stable eating trait in young girls (Birch & Fisher, 2000; Birch, Fisher, & Davison, 2003; Fisher & Birch, 2002) and to be correlated with children’s weight status during middle childhood (Birch & Fisher, 2000; Faith et al., 2006).

The aim of this review is to illustrate how factors relating to children’s familial predisposition to obesity and to their home environment can act together to influence children’s eating and weight development. Specifically, the first two sections of the paper highlight the extent to which parental weight status and (eating) behaviors and food preferences can impact children’s weight status and their eating behaviors and food preferences. Next, using EAH as an example of an eating trait which promotes increased caloric intake in susceptible children, we illustrate how genetic and nongenetic factors can shape this trait. We present competing conceptual models which can be used to test these influences and their impact on EAH. We conclude the current review with a discussion of possible gaps in the literature on the study of child eating phenotypes and present an outlook on possibilities for future collaborations in the field. We note that this review focuses on “unmeasured genotype” studies (also referred to as behavioral genetics or quantitative genetics studies) that did not test specific genes, rather than molecular genetics studies that test specific genes. These latter studies will be critically important to future developments in the field, and merit their own review.

Parental Obesity Status Predicts Child Overweight Status: The Familial Association

“Obesity in one or both parents probably influences the risk of obesity in their offspring because of shared genes or environmental factors within families” (Whitaker, Wright, Pepe, Seidel, & Dietz, 1997). The notion that “obesity runs in families” has been supported by cross sectional and longitudinal studies (Gibson et al., 2007; Maffeis, Talamini, & Tato, 1998; Schaefer-Graf et al., 2005; Wang, Patterson, & Hills, 2002; Whitaker et al., 1997). These studies provided evidence that parental overweight (defined as a BMI ≥25 kg/m²) and obesity (defined as a BMI ≥30 kg/m²) are significant risk factors for overweight in their offspring. Data from two cross sectional studies showed that parental overweight or obesity was an independent risk factor for childhood obesity (Wang et al., 2002) and that maternal BMI, in particular, was a significant predictor of BMI z-scores in children ages 6–13 years (Gibson et al., 2007). Data from several longitudinal studies confirmed these findings and showed that children of obese parents were at increased risk for developing obesity later in their life (Maffeis et al., 1998; Schaefer-Graf et al., 2005; Whitaker et al., 1997). For example, Whitaker and colleagues (1997) showed that after 6 years of age, the probability of a child becoming an obese adult exceeded 50% for obese children, as compared with ~10% for nonobese children. In addition, results showed that the risk of adult obesity was significantly greater if either the child’s mother or father was obese (Table I). That is,

<table>
<thead>
<tr>
<th>No. of subjects obese as adults/Total No. (%)</th>
<th>Odds ratio (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject’s age and mother’s obesity status</td>
<td></td>
</tr>
<tr>
<td>1–2 years</td>
<td></td>
</tr>
<tr>
<td>Mother not obese</td>
<td>73/575 (13)</td>
</tr>
<tr>
<td>Mother obese</td>
<td>29/85 (34)</td>
</tr>
<tr>
<td>3–5 years</td>
<td></td>
</tr>
<tr>
<td>Mother not obese</td>
<td>77/597 (13)</td>
</tr>
<tr>
<td>Mother obese</td>
<td>36/104 (35)</td>
</tr>
<tr>
<td>6–9 years</td>
<td></td>
</tr>
<tr>
<td>Mother not obese</td>
<td>76/611 (12)</td>
</tr>
<tr>
<td>Mother obese</td>
<td>42/131 (32)</td>
</tr>
<tr>
<td>10–14 years</td>
<td></td>
</tr>
<tr>
<td>Mother not obese</td>
<td>73/594 (12)</td>
</tr>
<tr>
<td>Mother obese</td>
<td>53/177 (30)</td>
</tr>
<tr>
<td>15–17 years</td>
<td></td>
</tr>
<tr>
<td>Mother not obese</td>
<td>65/549 (12)</td>
</tr>
<tr>
<td>Mother obese</td>
<td>64/233 (27)</td>
</tr>
<tr>
<td>Subject’s age and father’s obesity status</td>
<td></td>
</tr>
<tr>
<td>1–2 years</td>
<td></td>
</tr>
<tr>
<td>Father not obese</td>
<td>55/435 (12)</td>
</tr>
<tr>
<td>Father obese</td>
<td>28/98 (29)</td>
</tr>
<tr>
<td>3–5 years</td>
<td></td>
</tr>
<tr>
<td>Father not obese</td>
<td>56/475 (12)</td>
</tr>
<tr>
<td>Father obese</td>
<td>34/123 (28)</td>
</tr>
<tr>
<td>6–9 years</td>
<td></td>
</tr>
<tr>
<td>Father not obese</td>
<td>60/497 (12)</td>
</tr>
<tr>
<td>Father obese</td>
<td>40/149 (27)</td>
</tr>
<tr>
<td>10–14 years</td>
<td></td>
</tr>
<tr>
<td>Father not obese</td>
<td>62/505 (12)</td>
</tr>
<tr>
<td>Father obese</td>
<td>46/184 (25)</td>
</tr>
<tr>
<td>15–17 years</td>
<td></td>
</tr>
<tr>
<td>Father not obese</td>
<td>54/491 (11)</td>
</tr>
<tr>
<td>Father obese</td>
<td>56/221 (25)</td>
</tr>
</tbody>
</table>

Adapted from Whitaker et al., 1997; Copyright © 1997 Massachusetts Medical Society. All rights reserved.
parental obesity more than doubled the risk for children, obese or nonobese, to become obese adults. This risk especially applied to children who were under 10 years of age. The authors note that among nonobese 1- and 2-year-olds, those with at least one obese parent had a greater chance of being obese as adults compared to those without an obese parent (28% vs. 10%). Among obese 3- to 5-year-olds, the chance of developing adult obesity increased from 24% if neither parent was obese to 62% if at least one parent was obese (Whitaker et al., 1997).

**Parent (Eating) Behaviors and Food Preferences Predict Child Eating Behaviors and Food Preferences: The Familial Association**

The home food environment also plays a crucial role in forming children’s food preferences and eating traits. Parents can influence the development of food acceptance patterns by structuring children’s early eating environments (Birch, 2002). They decide the types and amounts of food that are served to their children, determine the timing of children’s meals and snacks, and provide the social context in which eating occurs.

Parents influence their children’s eating by exerting feeding practices which may either be conducive or hindering to the development of healthy eating and growth patterns. For example, data have shown that those children whose parents exert much control over their eating or restrict certain desired foods from them, tend to show a weaker intake regulation (Birch, McPhee, Shoba, Steinberg, & Krebs, 1987) and more pronounced EAH (Birch et al., 2003; Francis & Birch, 2005), an eating trait which will be discussed further below. It should be noted that these feeding relationships are often bidirectional in that parents also respond to children’s requests for particular foods. On the other hand, restrictive feeding practices may be the result rather than the cause of increased BMI in children.

Parental feeding practices can also modify genetic predispositions for likes and dislikes of foods and help shape food preferences in children. Data from a recent study (Breen, Plomin, & Wardle, 2006) with monozygotic (MZ) and dizygotic (DZ) twins suggest that variations in food preferences are heritable when aggregated across certain groupings of foods. Specifically, the investigators found a modest heritability for fruits (heritability, $h^2 = 51\%$), vegetables (37%), and desserts (20%) and a high heritability for liking of protein foods such as meat and fish (78%). There also exist data on family correlations which provide further evidence for a familial transmission of food preferences. Across studies, the relationship between child and parent food preferences appears to be statistically significant but small in magnitude (Faith, 2005). A meta-analysis by Borah-Giddens and Falciglia (1993) indicated mean parent–child correlations of $r = .19$ for mother–child pairings and $r = .14$ for father–child pairings.

Genetic predispositions for food preferences can be modified by home environmental factors such as parent feeding practices. For example, children are born with a genetic predisposition to neophobia which refers to the hesitancy to eat novel foods (Barnett, 1963). Neophobia can impede children’s acceptance of new foods such as fruits and vegetables. Studies have shown that repeated exposure to novel foods can significantly reduce neophobia in children (Birch & Marlin, 1982; Sullivan & Birch, 1994). In support of these findings are results from studies which have shown that simply making certain types of foods available and easily accessible to children can impact their eating habits. For example, Cullen and colleagues (Cullen et al., 2003) 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. Thus, early exposure to foods can shape children’s liking of these foods not only for ones that parents desire their children to consume (e.g., fruits and vegetables), but also for energy-dense snack foods, which may facilitate the overconsumption of calories.

Studies have shown that not only the types of foods that are available in the home, but also the amount of food that is being served to children significantly impacts how much children eat. For example, data from nationally representative cross sectional studies revealed positive associations of food portion sizes consumed and daily energy intake in children ranging from 6 months to 5 years (McConahy, Smiciklas-Wright, Birch, Mitchell, & Picciano, 2002; McConahy, Smiciklas-Wright, Mitchell, & Picciano, 2004). Specifically, data from the Continuing Survey of Food Intakes by Individuals (1994–1996, 1998) showed that portion sizes for the 10 most frequently consumed foods in the diets of 2- to 5-year-old children accounted for 17–19% of the variability in children’s energy intakes (McConahy et al., 2004). Similarly, experimental studies showed that increases in the portion size of a main entrée resulted in significant increases in intake among young children (Fisher, 2007; Fisher, Liu, Birch, & Rolls, 2007; Orlet Fisher, Rolls, & Birch, 2003; Rolls, Engell, & Birch, 2000). Thus, food environments which offer children convenient access to large portions of palatable, energy-dense
foods may contribute to excessive energy intake and weight gain.

In summary, the early home environment in which eating occurs is believed to influence, in important ways, children’s eating regulation and the development of food preferences. Genes exert their influence through eating behaviors which in turn are directly affected by the immediate food environment and feeding styles. Therefore, early experiences with food and eating have the potential to not only establish healthy eating habits but also to modify genetic predispositions (e.g., neophobia) which in turn may lead to better food acceptance patterns.

**EAH: A Behavioral Eating Trait**

Perhaps the best example of how genetic and environmental factors act in concert to influence a specific eating trait linked to obesity is the study of “eating in the absence of hunger” (EAH). EAH refers to children’s tendency to eat in response to the presence of palatable foods in the absence of hunger. The trait has been extensively studied both from a genetic and environmental perspective and is associated both with hyperphagia (overeating) and increased weight gain in children.

EAH in children is operationally assessed by the “free access procedure” which was developed by Birch and colleagues (Birch et al., 2003; Fisher & Birch, 1999ab). Following the consumption of a self-selected standard meal, each child rates his/her perceived hunger and fullness to ensure they are fully satiated. Each child is then asked to participate in a rank-order preference assessment of a variety of snacks with different sensory properties (e.g., potato chips, chocolate bars, and frozen yogurt) to provide him/her with the opportunity to taste each experimental snack food. Following the preference assessment each child is given toys and asked to play by him/herself. During this “play session” the child is given access to an array of sweet and savory snack foods and told that they could eat any of these foods and as much as they want. EAH refers to the number of calories the child consumes from the snack foods despite being fully satiated.

EAH in children shares behavioral characteristics with the trait of disinhibited eating (or “disinhibition”) in adults. Disinhibition is the loss of self-imposed cognitive control over eating in response to external or emotional stimuli, and is the eating pattern that most consistently differentiates obese and nonobese adults (Lindroos et al., 1997). There is evidence that obese individuals show greater disinhibition than do nonobese individuals (Carmody, Brunner, & St Jeor, 1995; Westenhoefer, Stunkard, & Pudel, 1999) and that the degree of disinhibition is strongly associated with energy intake (Lawson et al., 1995; Lindroos et al., 1997), weight status and weight gain (McGuire, Wing, Klem, Lang, & Hill, 1999; Williamson et al., 1995), weight fluctuations (Carmody et al., 1995), binge eating (Howard & Porzelius, 1999), and body fat (Provencher, Drapeau, Tremblay, Despres, & Lemieux, 2003). Interestingly, maternal disinhibition has been shown to predict daughters’ overweight and, when both maternal disinhibition and daughters’ EAH were used to predict daughters’ overweight, mothers’ disinhibition showed an independent prediction (Cutting, Fisher, Grimm-Thomas, & Birch, 1999).

Thus, to the extent that EAH in childhood is a behavioral precursor to disinhibited eating in adulthood, these findings further support the importance of EAH as an early behavioral marker or risk factor for obesity onset. Indeed, there is evidence that EAH is relatively stable overtime in childhood (Birch et al., 2003), which suggests that the trait could extend into adulthood.

**Genetic Influences on EAH**

“Heritability” (h²) refers to the extent to which variability in a trait is influenced by genetic variations within a population (Maes, Neale, & Eaves, 1997), such that a trait with h² = 0% is completely influenced by environmental factors while a trait with h² = 100% is entirely genetically influenced. Most traits are at least partially genetically influenced, and so heritability is useful for understanding the relative influence of genes, or the overall “genetic loading” of a trait. Heritability is most commonly estimated from twin studies that compare the phenotypic similarity of a trait in MZ twins compared to DZ twins, using “biometrical” statistical methods for heritability estimation (for details, see Neale & Cardon, 1992). There are a number of methodological issues, caveats, and limitations to twin designs. One example is the “equal environments assumption” (i.e., the assumption that the extent to which MZ twin pairs are exposed to similar environments is not different from the extent to which DZ twin pairs are exposed to similar environments). These methodological issues have been reviewed extensively by Maes and colleagues (Maes et al., 1997) and Guo (2001).

Numerous studies show that child weight status is highly genetically influenced. In a sample of 66 pairs of 3- to 17-year-old twins residing in the New York metropolitan area, the heritability of BMI and percent body fat was estimated to be 86% and 76%, respectively (Faith et al., 1999). Analyses of a population-based sample of 608 MZ and 1210 DZ twin pairs from the UK estimated the heritability of weight (corrected for height) to be 64%
for boys and 61% for girls (Koeppen-Schomerus, Spinath, & Plomin, 2003). In a large national sample of adolescents from the United States, Jacobson and Rowe found that total genetic influences on BMI were 67%, 45%, and 81% for Black females, White females, and all males (i.e., all ethnic groups combined), respectively (Jacobson & Rowe, 1998). High heritability estimates for child overweight from twin studies have been shown in the literature, even in recent publications from relatively current cohorts and despite the increasing prevalence of pediatric obesity (Musani, Erickson, & Allison, 2008). Thus, despite secular changes in childhood obesity prevalence in recent decades, the prominent role of genes cannot be dismissed.

By contrast, the genetics of child eating patterns is a relatively understudied field (Faith & Keller, 2004). Studies which have examined EAH point to the importance of genetic factors on the trait. Faith and colleagues (Faith et al., 2006) found that EAH in boys who were born at high risk for obesity on the basis of maternal prepregnancy body weight was more than twice that of boys who were born at low risk for obesity. This investigation, however, could not tease apart genetic from environmental influences on the trait. Findings from a study with 801 children from 300 Hispanic families who were part of the Viva la Familia Study (Fisher, Cai et al., 2007) indicated that EAH was a highly heritable eating trait (51%).

The notion that EAH is heritable is consistent with findings from prior studies establishing the heritability of disinhibition in adults. For example, Steinle and colleagues (Steinle et al., 1985), Steinle et al. (1985), disinherit showed the highest heritability ($h^2 = 40\%$) and the strongest association with obesity traits. Findings from the Quebec Family Study indicated a heritability estimate for disinhibition of 17.5% as well as significant parent–offspring and spousal correlations for disinhibited eating (Provencher et al., 2005).

### Environmental Influences on EAH

Parental feeding practices have been identified as one environmental factor which can considerably influence children’s susceptibility to EAH. Specifically, dietary restriction, or the tendency of parents to restrict children’s access to desired, palatable foods (such as snack foods), has been found to be a significant predictor of EAH. EAH was predicted by mothers’ reports of using restrictive feeding practices at age 5 (Birch et al., 2003; Fisher & Birch, 2002; Francis & Birch, 2005), and was associated with increased risk of overweight in children (Fisher & Birch, 2002). Interestingly, the association between restrictive feeding patterns and child EAH depended upon maternal overweight status. That is, among daughters of overweight mothers only, greater use of restrictive feeding patterns when the children were 5 years of age predicted greater EAH by daughters when they were 7 (Birch et al., 2003; Fisher & Birch, 2002) and 9 years of age (Birch et al., 2003; Francis & Birch, 2005). However, daughters of normal-weight mothers showed significantly greater EAH only at age 5, but not at ages 7 and 9 years (Francis & Birch, 2005). In addition, those girls who were overweight at age 5 and subject to higher levels of restriction showed the greatest EAH at age 9 (Birch et al., 2003; Fisher & Birch, 2002).

In another study, Faith et al. (Faith et al., 2004) showed that maternal use of restrictive feeding patterns was associated with excess weight gain in children, but only among children who were born at high risk for obesity based on elevated maternal prepregnancy body weight. There was no association between maternal use of restrictive feeding and excess weight gain among children born at low risk for obesity. These findings suggest that the use of restrictive feeding practices by parents may interact with a child’s genetic predisposition for obesity to promote excess weight gain, which may occur through greater EAH. Although Faith and colleagues did not measure EAH per se, the findings are consistent with those generated by Francis and Birch (Francis & Birch, 2005).

That being said, maternal obesity as a risk factor for overweight in their offspring may not necessarily be conferred by ways of maternal feeding practices. A study by Wardle and colleagues (Wardle, Sanderson, Guthrie, Rapoport, & Plomin, 2002) found that obese mothers did not differ from normal-weight mothers in the extent to which they offered food to deal with emotional distress, used food as a form of reward, or encouraged children to eat more than they wanted. With respect to exerting control over their child’s eating, the results of this study showed that obese mothers reported significantly less control over children’s intake than did normal-weight mothers (Wardle et al., 2002).

Table I depicts key findings from select investigations which further illustrate EAH’s role as a behavioral eating trait in the development of childhood obesity.

### Competing Conceptual Models of Factors Influencing EAH

An important area for future research will be to determine the extent to which the association between EAH and excess weight gain is mediated by genetic factors,
environmental factors, or both. That is, research studies should attempt to discern if EAH and weight gain have genetic factors in common, environmental factors in common, or both genetic and environmental factors in common. Figure 1 illustrates three competing models that could be tested. Model 1 posits that the relationship between EAH and excess weight gain is entirely due to genes that influence both traits, while Model 2 posits that the association is entirely environmentally mediated. Model 3 posits that both genetic and environmental factors are influential. A properly designed study could test these competing models, as illustrated in a recent study by Fisher and colleagues (Fisher, Cai et al., 2007).

It will be important to know the extent to which response to childhood obesity treatment and prevention initiatives is determined by genetic and environmental factors. Behavioral genetics research is expected to contribute to future research endeavors by, first, continuing to identify the heritability of child eating phenotypes that are associated with excess body weight, as well as by estimating genetic and environmental influences on treatment response. A range of behavioral genetics studies including twin designs, adoption designs, and (extended) family designs can address these questions (see Neale & Cardon, 1992, for details). However, it is implausible that, for example, the classic twin design, by itself, will be able to address the full range of research questions concerning the familial transmission of child eating traits. The various behavioral genetics designs have their own respective strengths and limitations that make certain designs more desirable than others for certain research questions. A more detailed discussion of these designs is provided elsewhere (Plomin, DeFries, McClearn, & McGuffin, 2000).

The value of such studies rests in their potential to resolve the extent to which the association between EAH and weight gain is due to genetic and environmental factors, respectively. If the association is primarily environmentally mediated, this would provide greater support for the potential of environmental interventions that could foster healthier eating patterns and weight trajectories among children. However, because child weight status is highly heritable, it is conceivable that obesity-promoting genes also drive increased EAH. Genetically informative designs are very powerful for resolving these questions.

Table II. Genetic and Non-genetic Factors Affecting EAH

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Findings</th>
<th>Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heritability</td>
<td>• EAH shown to be a heritable eating trait</td>
<td>Fisher, Cai et al., 2007</td>
</tr>
<tr>
<td></td>
<td>• EAH shown to be more pronounced in boys born at high risk for obesity compared to those born at low risk for obesity</td>
<td>Faith et al., 2006</td>
</tr>
<tr>
<td></td>
<td>• EAH shown to share similarities to disinhibition in adults, a trait which has been found to be heritable</td>
<td>Cutting et al., 1999; Provencher et al., 2005; Steinle et al., 2002</td>
</tr>
<tr>
<td>Environment</td>
<td>• EAH shown to be exacerbated by restrictive feeding practices and monitoring</td>
<td>Birch et al., 2003; Fisher &amp; Birch, 1999a; Francis &amp; Birch, 2005</td>
</tr>
<tr>
<td>Stability</td>
<td>• EAH shown to be an eating trait which is stable over time</td>
<td>Fisher &amp; Birch, 2002; Francis &amp; Birch, 2005</td>
</tr>
<tr>
<td>Energy intake</td>
<td>• EAH shown to be associated with increased energy intake</td>
<td>Fisher &amp; Birch, 1999b</td>
</tr>
<tr>
<td>Body weight</td>
<td>• EAH shown to be predictive of increased weight gain among children</td>
<td>Birch et al., 2003; Fisher &amp; Birch, 2002; Shunk &amp; Birch, 2004</td>
</tr>
</tbody>
</table>

For all odds ratios, the nonobese group served as the reference group. The subject’s obesity status during childhood is based on the mean z-score for the child’s BMI during the age interval, as follows: not obese, \( z < 1.036 \) (corresponding to the 85th percentile for a normal population of the same age and sex); obese, \( z \geq 1.036 \) (85th percentile); and very obese, mean \( z \geq 1.645 \) (95th percentile).

Summary and Limitations

EAH is an eating trait which has been widely studied both from an environmental and a genetic perspective in children. Despite the many important insights these studies have generated so far, there still exist large gaps as to how children’s eating behavior and weight regulation can be ameliorated. For one, EAH has been studied primarily in white, middle class girls, although more recently the eating trait has also been examined in Hispanic children (Fisher, Cai et al., 2007). The fact that EAH has been studied primarily in Caucasian girls limits the generalizability of the findings to a broader population of children. It will be crucial to further expand these studies to children from different ethnic groups and of different socio-economic status (SES) in the future. Second, the mechanisms which underlie the development and maintenance of EAH in children remain poorly understood and need further investigation. Furthermore, the systematic study of children’s eating behavior should be widened to
other eating traits and properties of foods (e.g., energy density), which may affect children’s eating. These studies should encompass both short- and long-term investigations. It will also be important to investigate the effects of parental factors such as SES, education, and income on child eating behavior. Lastly, self-report instruments need to be developed for EAH and other eating traits because the assessment of these traits in the laboratory will not be feasible for large-scale genetics studies.

The purpose of the current review was to provide an overview of how genetic and nongenetic factors can affect children’s eating and weight development. This review primarily focused on behavior genetics and did not take into account molecular genetics studies which tested the effects of specific genes on child eating traits or body weight. The specific genes influencing EAH currently are unknown and, in principle, one could speculate about any of the hundreds of genes that have been associated with variations in human weight status or adiposity (see the “Human Obesity Gene Map”) (Rankinen et al., 2006). Future research is expected to address the specific genes influencing child EAH. This review also did not discuss the influence of physical activity on children’s eating and weight regulation.

Conclusions

In conclusion, the study of genetic and home environmental influences with respect to children’s eating and weight development is an important area of research which presents a great opportunity for collaboration among scientists from diverse disciplines. The findings from studies in this area likely will have important implications for the prevention and treatment of childhood obesity in both research and clinical settings. As detailed earlier, children’s response to dietary interventions may depend on their genotype. Once identified, it is conceivable that dietary regimens may be tailored to children’s genetic predisposition to promote healthy growth and eating patterns. Parents’ involvement in child feeding and the structuring of the early home food environment will be crucial components in these interventions.

Conflicts of interest: None declared.

Received December 13, 2007; revisions received and accepted March 19, 2008

References


