Familial aggregation of energy intake in children¹⁻³

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ABSTRACT

Background: Uncompensated overnutrition promotes obesity, but the controls of children’s eating behavior are poorly understood. Insights may be achieved by testing whether the eating patterns of children are associated with demographic variables or whether they aggregate among family members.

Objective: We tested whether children’s total energy intake and macronutrient intake and their ability to compensate for earlier energy intake were associated with sociodemographic variables and anthropometric indexes. We also tested whether these behavioral traits aggregate among siblings.

Design: Thirty-two sibling pairs aged 3–7 y consumed a multi-item lunch preceded by a low-energy (12.55 kJ) or high-energy (627.60 kJ) preload drink. Mixed-models regression tested the associations between children’s energy intake, demographic variables, and anthropometric measures. An intraclass correlation coefficient quantified the family correlation of the measures of children’s eating.

Results: Children consumed significantly more total energy after consuming the low-energy preload (5 ± SD: 2237.39 ± 1176.45 kJ) than after consuming the high-energy preload (1601.18 ± 930.65 kJ). Compensation ability was unrelated to the children’s age, sex, or ethnicity. Total energy and macronutrient intake, but not compensation propensity, were associated among siblings.

Conclusions: The familial association of total energy and macronutrient intakes, independent of anthropometric measures, suggests genetic or home environmental influences specific to these behaviors. Short-term energy compensation, although very accurate within this sample, showed no significant familial correlation. Am J Clin Nutr 2004;79:844–50.

KEY WORDS Feeding behavior, eating, childhood obesity, behavior genetics, energy compensation

INTRODUCTION

Childhood overweight is an increasingly prevalent condition that results from energy intakes that are greater than energy expenditures. Indeed, a daily energy imbalance between intake and expenditure as subtle as 2%, sustained over time, can promote overweight in growing children (1). Doubly labeled water studies further confirm that obese children consume more total energy than do their nonobese counterparts, although the 2 groups may not differ with respect to intake of high-energy snack foods (2). These data underscore the importance of identifying the controls of children’s energy intake and the use of sensitive methods to quantify their eating behavior.

The average child will adjust energy intake at multi-item ad libitum meals in proportion to the amount of energy consumed 30 min earlier; this phenomenon is called “compensation” (3). However, there is considerable interchild variation in compensation ability: certain children are more sensitive to the foods that they consume than are others (4). Poorer energy compensation has been associated with elevated body weight in girls (4), and other studies have established associations between total energy intake, fat intake, and overweight among children (5, 6). Understanding the sources of individual differences in children’s eating patterns might guide novel interventions for healthier eating and might lead to better weight management (7).

One potential source of interchild differences in eating behavior is genetic variation. Suggestive evidence comes from the literature on animal studies (8), although less research has been conducted in humans. Most twin studies have been conducted among adults whose food intakes were measured by dietary records, and these studies support a genetic influence on food intake (9, 10). Using an adult twin design, we found that genetic variation accounted for ≈33% of the variation in participants’ total ad libitum energy intakes in the laboratory (11). These collective findings lead us to suspect that genetic variations may contribute to differences in children’s eating behavior. This question has not yet been addressed through the study of related children.

The sibling design of the present study can address one of the most basic questions concerning variation in children’s eating behavior—namely, its family correlation, or “familiality.” Estimating familiality is traditionally the first step in documenting potential genetic bases for a trait among humans, and, in classic

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genetic doctrine, it precedes the formal partitioning of genetic and environmental influences on traits (12). We report here the results of a study of the familiality of children’s eating behavior.

The goal of this study was to elucidate the sources of variation in young children’s energy compensation tendency, total energy intake, and macronutrient intake. Our specific aims were to test the compensation phenomenon among an ethnically diverse sample of young siblings, to test whether variations in child eating measures were associated with sociodemographic variables and anthropometric indexes, and to test the familiality of energy compensation, total energy intake, and macronutrient intake. We predicted that children would eat more after the low-energy preload than after the high-energy preload, that poorer compensation and increased total energy intake would be associated with elevated body fat indexes, and that eating measures would correlate among siblings. We did not have a priori hypotheses concerning the sociodemographic variables or their associations with children’s eating outcomes.

SUBJECTS AND METHODS

Procedures

Newspaper advertisements and fliers solicited families with young children to participate in a children’s nutrition study. This study was advertised to test the determinants of children’s food preferences and to assess whether eating habits run in families. We recruited 3–7-y-old sibling pairs (in which the siblings were ≤3 y apart in age) who were free of food allergies that would preclude participation. No restrictions were made with respect to child or parent weight status. Each laboratory visit lasted ≈90 min and occurred around the lunch hour (ie, ≈1130–1300). We instructed families not to eat for at least 2 h before coming to the laboratory.

As in previous research in children (4), families made 2 visits to the laboratory to consume a low-energy preload beverage and a high-energy preload beverage, respectively. The preload beverages were presented in a randomized order, such that children were assigned to one of 3 conditions. In the low/low and high/high condition, both siblings received low-energy preloads on visit 1 and high-energy preloads on visit 2; in the high/high and low/low condition, both siblings received high-energy preloads on visit 1 and low-energy preloads on visit 2; and in the low/high and high/low condition, one member of a sibling pair received a low-energy preload and the other sibling received a high-energy preload on visit 1, and the order of the preloads was switched for visit 2. This study received full approval from the Institutional Review Board of St Luke’s–Roosevelt Hospital.

Visit 1

Families were greeted by staff and acclimated to the feeding laboratory. After completing the informed consent form, mothers were provided a questionnaire to be completed while children consumed the liquid preload and lunch test meals. Mothers sat within 5 ft of their children in the same room. The questionnaire packet included demographic items and items concerning maternal eating attitudes and feeding styles.

Approximately 15 min after arrival, siblings were seated at a round table where they were served either a low-energy or high-energy preload fruit drink. Recipes for low- and high-energy preloads were adapted from Johnson and Birch (4). These cherry-flavored carbohydrate liquid preloads were matched for volume, mass (173 g), and sensory properties and were served in clear plastic cups with straws through the lids. All drinks were served at room temperature. Children were verbally encouraged to finish the entire preload drink within a few minutes, drinking until they heard a “slurpy sound” in the cup when they sucked on the straw. The fundamental difference between the 2 preloads was the total energy content, which was achieved by serving either standard cherry Kool-Aid (Kraft General Foods, White Plains, NY) that we supplemented with maltodextran (high-energy preload) or Kool-Aid manufactured with aspartame artificial sweetener (low-energy preload). The high- and low-energy preloads contained 628 kJ (150 kcal) and 12.5 kJ (3 kcal), respectively.

After consuming the preload, siblings were allowed a 25-min interim play period, during which the staff postweighed the plastic cups for determination of preload consumption (ie, weight of the cup plus drink before consumption minus weight of the cup plus drink after consumption). Children were then invited to sit at the table for the ad libitum lunch. They were served a multi-item lunch that included macaroni and cheese, canned string beans, string cheese, graham crackers, green grapes, baby carrots, and whole milk (4). These items, which provided 3353 kJ of energy (≈800 kcal), are summarized in Table 1. This corresponds to ≈50–65% of the total daily energy requirements for 4- to 7-y-old children (13). Research assistants read stories to the children while they ate lunch. Children were free to consume the foods they wished and could ask for additional servings. Research assistants were instructed to ask children if they wanted more of a particular food once they finished the amount on their plate. Siblings were instructed not to share their foods with one another and could not eat off each other’s plates. We let siblings sit together to enhance their comfort and to simulate the more typical conditions under which they eat at home. Mothers sat off to the side during these lunch meals, and interactions with their children were discouraged.

After lunch, children had additional play time in the lab, received prizes, and had their pictures taken while staff postweighed the food containers for determination of test meal intake by using the same procedure as for postweighing of plastic cups. Families then departed.

Visit 2

Visit 2 was identical to the first visit with a few exceptions. First, parents were not given questionnaires to complete, but they had access to a variety of magazines to read. Second, each child received the preload drink that they had not consumed on the first

<table>
<thead>
<tr>
<th>Food</th>
<th>Percentage as fat</th>
<th>Percentage as carbohydrate</th>
<th>Percentage as protein</th>
<th>Energy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macaroni and cheese, 133 g</td>
<td>52</td>
<td>35</td>
<td>13</td>
<td>820</td>
</tr>
<tr>
<td>String cheese, 30 g</td>
<td>69</td>
<td>3</td>
<td>27</td>
<td>353</td>
</tr>
<tr>
<td>Carrots, 35 g</td>
<td>0</td>
<td>91</td>
<td>9</td>
<td>63</td>
</tr>
<tr>
<td>Grapes, 113 g</td>
<td>0</td>
<td>100</td>
<td>0</td>
<td>317</td>
</tr>
<tr>
<td>Green beans, 57 g</td>
<td>0</td>
<td>90</td>
<td>10</td>
<td>48</td>
</tr>
<tr>
<td>Graham crackers, 25 g</td>
<td>21</td>
<td>72</td>
<td>7</td>
<td>443</td>
</tr>
<tr>
<td>Whole milk, 513 g</td>
<td>49</td>
<td>30</td>
<td>21</td>
<td>1309</td>
</tr>
</tbody>
</table>

1 Children could request additional servings of food.
visit. Children’s weight, height, and waist circumference measures were taken on this visit.

Measurements

Energy compensation tendency

Children’s energy compensation tendency was quantified by a compensation index called COMPX (4), which measures the difference in energy intake between the 2 multi-item test meals divided by the difference in preload consumption:

\[
COMPX = \left[ \frac{(Meal_{low} - Meal_{high})}{(Preload_{high} - Preload_{low})} \right] 
\times 100 \quad (1)
\]

where Meal\textsubscript{low} = total energy intake at the test meal after the low-energy preload, Meal\textsubscript{high} = total energy intake at the test meal after the high-energy preload, Preload\textsubscript{high} = total intake of energy from the high-energy preload, and Preload\textsubscript{low} = total intake of energy from the low-energy preload. Thus, the index essentially compares ad libitum intake after consuming a 150-kcal preload to that after consuming a negligible-energy preload control. The COMPX is a continuous scale such that that 100\% equals “perfect” compensation. Lower scores (undercompensation) reflect a progressively greater tendency to overeat after consuming the 150-kcal preload than after consuming the 3-kcal preload. Higher scores (overcompensation) reflect a progressively greater tendency to undereat after consuming the 150-kcal preload than after consuming the 3-kcal preload.

Total energy intake

We computed the mean total energy intake across the 2 test meal lunches, by using the manufacturer’s information and food labels. Energy intake from the preloads was not included in this calculation. We used a similar calculation in a previous preloading study with adults (11).

Macronutrient intake

We computed the mean percentage intake of fat, carbohydrate, and protein across the 2 test meal lunches, by using the manufacturers’ information and food labels.

Anthropometric indexes

Children’s weight and height were measured with the use of a digital scale and a stadiometer, respectively, and converted to body mass index (BMI; in kg/m\(^2\)). BMI values, in turn, were converted to BMI z scores and percentiles according to the Centers for Disease Control and Prevention growth charts (14). Child waist circumference was measured in the standing position from midway between the last rib and the iliac crest (15). Because of their young ages, a number of children would not allow us to collect these measures. These children either were intimidated by this particular aspect of the protocol or were tired, or they equated the equipment with a doctor’s office. We honored these requests. BMI and waist circumference were calculated on 50 and 34 children, respectively, representing 78\% and 53\% of the total sample.

Mothers were asked to self-report their weights and heights, which we converted to BMI. BMI was calculated for the 38 mothers (59\% of the sample) who reported this information.

Sociodemographic measures

The following information was obtained from the mothers: their educational level (≤high school, college, or graduate or professional school), marital status (never married, married, or separated), and current employment status (employed or unemployed). Child sex was coded as 0 = boys and 1 = girls.

Statistical analysis

Descriptive data are presented as means ± SDs. Associations among child eating measures, demographic variables, child anthropometric indexes, and maternal BMI were tested by mixed-models regression. These analyses tested the associations among variables while adjusting for the dependency in the data structure due to family membership (16, 17). Specifically, we regressed the given outcome variable onto individual predictors at the same time as we controlled for family membership—ie, the random-effects variable.

Familial aggregation among eating measures was estimated by an intraclass correlation coefficient (p; 18), which quantifies variation between families relative to total variation. Higher p scores (approaching 1) imply a stronger familial resemblance. For 5 eating outcomes (COMPX, total energy intake, percentage fat intake, percentage carbohydrate intake, and percentage protein intake), we computed p according to 3 categories of statistical adjustment. First, we analyzed unadjusted food intake scores. Second, we adjusted scores for child age, sex, and ethnicity (dummy-coded 0 = whites and 1 = nonwhites). Third, we adjusted scores for child age, sex, ethnicity, and BMI. Statistical adjustments were achieved through multiple regression (19) to ensure that any familial association for eating measures was not simply due to common sibling demographics or anthropometric indexes. Data were analyzed by using SPSS software (version 11; SPSS Inc, Chicago).

RESULTS

Participant characteristics

Thirty-two sibling pairs participated in the study (Table 2). The ethnic breakdown was 25\% white, 40\% African American, 25\% Hispanic, and 10\% other or unspecified. The mean child age was 62 ± 2.0 mo. Fifty-six percent of the participating children were female. Children tended to be heavier than sex- and age-matched children in the overall US population, and their mean BMI corresponded to the 59th percentile on Centers for Disease Control and Prevention growth charts. Twenty percent of the children in whom we measured BMI were overweight.

Participating mothers were 34.45 ± 6.8 y old and had a mean BMI of 30.3 ± 10.4. Most mothers had at least a college education (78.5\%), were currently married (67.7\%), and were currently employed (70.0%: Table 3).

Evidence for energy compensation

Test meal intakes after the low- and high-energy preloads were 2237 ± 1176 kJ (ie, 534 ± 281 kcal) and 1601 ± 930 kJ (ie, 382 ± 222 kcal), respectively (Figure 1). The mean difference of 636 kJ (ie, 152 kcal) was significantly different from zero (P = 0.0001). The mean COMPX score of 103.6 ± 106.5% suggests that, on average, the children’s energy compensation tendency was very accurate. A one-sample t test confirmed that the mean
Correlates of children’s eating measures

COMPX scores were unrelated to child age (P = 0.26), sex (P = 0.72), ethnicity (P = 0.60), BMI (P = 0.74), BMI z score (P = 0.91), and waist circumference (P = 0.20) or to maternal age (P = 0.80) and maternal BMI (P = 0.87) in mixed-models regression analyses. Total energy intake was positively associated with child age (P < 0.001) and waist circumference (P = 0.02). There was a significant sex difference (P = 0.009): boys consumed more total energy after lunch than did girls (2146 ± 1100 and 1743 ± 906 kJ, respectively). Total energy intake was not significantly associated with the children’s ethnicity (P = 0.12), BMI (P = 0.07), or BMI z score (P = 0.29) or with maternal age (P = 0.99) or maternal BMI (P = 0.92).

Associations between maternal BMI and children’s anthropometric indexes

Maternal BMI was not associated with children’s BMI (P = 0.62), BMI z score (P = 0.88), or waist circumference (P = 0.46) when tested by mixed-models regression.

Familiality of children’s eating measures

There was significant familial aggregation for total energy intake (r = 0.39, P < 0.05), percentage fat intake (r = 0.66, P < 0.001), percentage carbohydrate intake (r = 0.67, P < 0.001), and percentage protein intake (r = 0.61, P < 0.001), but not for COMPX scores (r = 0.11, P = 0.29; Table 4). The similarity of within-family and between-families total energy intakes is depicted in Figure 2. These findings remained the same when eating measures were adjusted for children’s age, sex, ethnicity, and BMI (Table 4). Thus, the familial resemblance for food intake was not entirely attributable to similarities in child anthropometric indexes.

DISCUSSION

The present study, to our knowledge, is among the first to use laboratory measures to establish the familial resemblance of children’s eating behavior. Although the familial aggregation of pediatric body composition per se is well established, the clustering of dietary practices has not been. Given the link between total energy intake and body fat, the familial resemblance with respect to children’s weight may derive in part from common eating styles. That a common set of genes or environmental factors might influence both children’s eating and body fat is suggested from previous studies in adults (11). At the same time, the familial correlations for total energy intake and percentage

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**TABLE 2**

Characteristics of the participating children

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mo)</td>
<td>62 ± 2.0^2</td>
</tr>
<tr>
<td>Sex (%)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>56.3</td>
</tr>
<tr>
<td>Male</td>
<td>43.7</td>
</tr>
<tr>
<td>Ethnicity (%)</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>25</td>
</tr>
<tr>
<td>African American</td>
<td>40</td>
</tr>
<tr>
<td>Hispanic</td>
<td>25</td>
</tr>
<tr>
<td>Other or missing</td>
<td>10</td>
</tr>
<tr>
<td>COMPX (%)</td>
<td>103.6 ± 106.5</td>
</tr>
<tr>
<td>Intake</td>
<td></td>
</tr>
<tr>
<td>Total energy (kJ)</td>
<td>1919.9 ± 1007.9</td>
</tr>
<tr>
<td>Fat (% of energy)</td>
<td>41.6 ± 11.5</td>
</tr>
<tr>
<td>Carbohydrate (% of energy)</td>
<td>45.2 ± 15.3</td>
</tr>
<tr>
<td>Protein (% of energy)</td>
<td>13.1 ± 4.0</td>
</tr>
<tr>
<td>BMI (kg/m^2)</td>
<td>17.3 ± 4.3</td>
</tr>
<tr>
<td>Percentile</td>
<td>58.7 ± 33.5</td>
</tr>
<tr>
<td>z score</td>
<td>0.4 ± 1.4</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>55.1 ± 10.0</td>
</tr>
</tbody>
</table>

^1 Valid responses for children’s measures: age, n = 62; sex, n = 64; ethnicity, n = 64; COMPX, n = 64; total energy intake, n = 64; percentage macronutrient intake, n = 64; BMI (kg/m^2), percentile, and z score, n = 50; waist circumference, n = 34.

^2 ± SD (all such values).

^3 COMPX, a compensation index representing a child’s propensity for adjusting energy intake at a meal in relation to energy consumed 25 min earlier as a preload. It is calculated as the difference in energy intake between the 2 lunches divided by the difference in preload consumption times 100.

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**TABLE 3**

Characteristics of the maternal subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>34.5 ± 6.8^2</td>
</tr>
<tr>
<td>Education level (%)</td>
<td></td>
</tr>
<tr>
<td>≤ High school</td>
<td>21.5</td>
</tr>
<tr>
<td>College</td>
<td>64.2</td>
</tr>
<tr>
<td>Graduate or professional school</td>
<td>14.3</td>
</tr>
<tr>
<td>Marital status (%)</td>
<td></td>
</tr>
<tr>
<td>Never married</td>
<td>19.4</td>
</tr>
<tr>
<td>Married</td>
<td>67.7</td>
</tr>
<tr>
<td>Separated</td>
<td>12.9</td>
</tr>
<tr>
<td>Currently employed (%)</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>70.0</td>
</tr>
<tr>
<td>No</td>
<td>30.0</td>
</tr>
</tbody>
</table>

^1 Valid responses for maternal measures: age, n = 29; education level, n = 28; marital status, n = 31; currently employed, n = 30.

^2 ± SD.
macronutrient intake in the current study remained significant after control for BMI and demographic variables, which suggests the presence of genetic or environmental influences that are unique to these traits. The identification of these specific genetic or environmental influences would elucidate the development of eating patterns.

That genetic variations may partially underlie the familial correlation of child energy intake is consistent with twin studies that suggest a heritable component to the intake of specific foods and the preference for specific tastes. There is evidence for genetic influences on the reported intake of spicy foods (20), sweet foods (21), salty foods (22), fruit and fruit juices (21), vegetables (23), and meats and dairy products (23). Indeed, positional cloning of the genes for taste sensitivity to phenylthiocarbamide (PTC) was recently reported (24). Reviews of this literature are provided elsewhere (25, 26). Also noteworthy are studies documenting genetic bases for energy intakes and preferences for sweet foods in animals (8, 27). At the same time, research by Birch and Davison (28), Rozin (29), and others suggests the importance of sociocultural and familial influences on food preferences and aversions. These publications set the backdrop for future studies that will test genetic and environmental influences on children’s eating behavior.

Increased total energy intake was associated with increased waist circumference. This association may have important health implications, given the metabolic health risks associated with abdominal body fat (30, 31). A growing literature has linked obesity to certain metabolic complications in children, and waist circumference cutoffs have been established to delineate elevated cardiovascular disease risk factors in young children (32). The role of dietary practices in the development of this relation remains unknown, but it may be important to an understanding of the development of metabolic syndrome during growth and to the prevention of obesity-related comorbidities (33).

With respect to energy compensation, the participating children were very sensitive to the preload manipulation, compensating on average for 104% of the energy difference. However, individual variations in this trait were quite high. This finding replicates past studies with children (3, 4) and also provides evidence for short-term energy compensation in an ethnically mixed sample. COMPX scores were unrelated to age, sex, or ethnicity, which suggests that an energy compensation ability

### Table 4

<table>
<thead>
<tr>
<th>Measure</th>
<th>Unadjusted</th>
<th>Adjusted</th>
<th>Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td>COMPX (%)</td>
<td>0.10 (−0.25, 0.43)</td>
<td>−0.13 (−0.49, 0.26)</td>
<td>−0.26 (−0.64, 0.22)</td>
</tr>
<tr>
<td>Intake</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total energy (kJ)</td>
<td>0.39 (0.05, 0.65)</td>
<td>0.70 (0.43, 0.85)</td>
<td>0.49 (0.05, 0.77)</td>
</tr>
<tr>
<td>Fat (% of energy)</td>
<td>0.66 (0.42, 0.82)</td>
<td>0.66 (0.38, 0.84)</td>
<td>0.53 (0.10, 0.79)</td>
</tr>
<tr>
<td>Carbohydrate (% of energy)</td>
<td>0.67 (0.42, 0.82)</td>
<td>0.66 (0.39, 0.84)</td>
<td>0.57 (0.16, 0.82)</td>
</tr>
<tr>
<td>Protein (% of energy)</td>
<td>0.61 (0.33, 0.79)</td>
<td>0.58 (0.25, 0.79)</td>
<td>0.66 (0.29, 0.89)</td>
</tr>
</tbody>
</table>

1 n = 32 sibling pairs.
2 Adjusted for age, sex, and ethnicity, n = 26 sibling pairs.
3 Adjusted for age, sex, ethnicity, and BMI, n = 18 sibling pairs.
4 COMPX, a compensation index representing a child’s propensity for adjusting energy intake at a meal in relation to energy consumed 25 min earlier as a preload. It is calculated as the difference in energy intake between the 2 lunches divided by the difference in preload consumption times 100.
5 P ≤ 0.05.
6 P ≤ 0.001.
may not be unique to one demographic subgroup. That this protocol was implemented among siblings further suggests the feasibility of this trait as a focus of behavior genetics studies.

Curiously, COMPX scores were unrelated to children’s anthropometric indexes. This null finding may be due to our assessment of short-term rather than long-term energy compensation (34) or to our sample size. The sample size issue is noteworthy, because our power to detect small associations was limited. For example, to detect a familial correlation as small as \( p = 0.20 \) (95% CI: 0.05, 0.35) in the population, 267 families would be necessary to have 80% power at \( \alpha = 0.05 \) (35). The present sample size was not powered to detect such effects.

Another reason for the lack of familial correlation for COMPX might be model misspecification—that is, our model may not have included other pertinent variables that were necessary for the expected association to be observed. Birch and Fisher (36) found that poorer compensation was predictive of elevated body weight in girls, but only as part of a larger model that included maternal perceptions of daughter weight, feeding restriction, and other variables. Finally, the finding may reflect a genuine null association, given the paucity of data on this topic. The relation between short- and long-term energy compensation and children’s weight status is ideally tested via longitudinal design.

COMPX scores showed no significant familial association, which suggests that energy compensation may be influenced primarily by nonshared or random environmental influences. The exact identity of such influences is unknown, but the literature may provide material for speculation about several. Two putative factors are parental feeding control (4) and the intake of high-sugar beverages (37, 38), which may disrupt children’s energy compensation ability and thereby promote obesity.

Maternal BMI was unrelated to children’s anthropometric measurements in the present sample, a finding that is inconsistent with prior research (39). This null finding most likely relates to our relatively small sample size for these analyses. It is noteworthy that maternal BMI was marginally associated with childhood BMI (\( P = 0.07 \)) and children’s BMI z scores (\( P = 0.10 \)) in ordinary regression analyses that did not adjust for family membership (unreported analyses).

These results must be interpreted in light of study limitations. First, our sample size was insufficient to conduct in-depth ethnicity- and sex-specific analyses. Second, this study was not designed to disentangle genetic and environmental influences on eating traits, an issue that we are currently investigating (40). Third, we did not test specific environmental variables that may be correlated with measures of children’s energy intake. Future studies will address this possibility. Fourth, the findings cannot be generalized beyond the specific compensation protocol, preloads, and lunch provisions used here. We chose the specific carbohydrate liquid preload on the basis of previous research with children (4). Fifth, the present study examined only the regulation of short-term energy intake, and therefore findings may not be generalizable to longer-term regulation (34). Sixth, we did not ascertain body-composition measures in this sample. Seventh, the study examined familial correlations only among siblings. Although these data constitute an acceptable test of familiality (12), it would be more informative to have data on parent-child associations as well. Such analyses have been conducted for reported food intake (41). Eighth, this study did not include measures of habitual physical activity levels (42), which may introduce variation in short-term regulation.

In sum, variations in children’s ad libitum total and macronutrient intakes can be explained in part by family membership. The extent to which genes or shared environmental factors induce this familial association should be tested. A burgeoning literature has begun to document the molecular bases of human eating behavior (43, 44), and other investigators are exploring the unfolding nature of feeding dynamics in parents and their children (45). The merger of laboratory feeding protocols with genetics designs may provide new insights into the development of children’s eating practices and childhood obesity.

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