Daily food intake in relation to dietary energy density in the free-living environment: a prospective analysis of children born at different risk of obesity\textsuperscript{1–3}

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ABSTRACT

Background: Young children adjust their short-term intake in response to variations in energy density (ED; kcal/g) from preloads in laboratory studies. It remains unknown whether this compensation also occurs under free-living conditions.

Objective: The aims of the study were to test whether children aged 3–6 y regulate their habitual daily food (g) and energy (kcal) intakes in relation to ED and whether compensation differs for children born at different risk of obesity.

Design: Participants were children born at high risk (n = 22) or low risk (n = 27) of obesity on the basis of maternal prepregnancy body mass index (BMI; in kg/m\textsuperscript{2}). Daily ED, food intake, and energy intake were assessed from 3-d food records that either included or excluded beverages. Intake regulation was explored by relating children’s daily food and energy intakes to ED and, more importantly, by examining residual scores derived by regressing daily food intake on ED.

Results: For both risk groups, daily food intake was inversely correlated with ED (P < 0.05), whereas daily energy intake was not significantly correlated with ED at most ages (P > 0.05). In analyses that excluded beverages, mean residual scores significantly increased from 3 to 6 y of age in high-risk children, which indicates relative overconsumption, but decreased in low-risk children, which indicates relative underconsumption (risk group × time interaction, P = 0.005).


KEY WORDS Energy density, daily energy intake, daily food intake, obesity risk status, children, caloric compensation

INTRODUCTION

The prevalence of childhood obesity is on the rise worldwide (1–10), and the dietary patterns contributing to this increase are poorly understood. One dietary factor that has received less attention in the study of pediatric obesity is dietary energy density (ED), which is defined as calories per weight of food (kcal/g). Controlled laboratory-based feeding studies found that experimentally increasing the ED of foods significantly increases short-term energy intake in adults (11–14). Specifically, across conditions of ED, subjects ate a similar weight of food, thereby increasing their energy intake during a single meal. This suggests that under laboratory conditions, as well as in the free-living environment (15), adults regulate the amount of food they consume (by weight or volume) to a greater extent than the calories they consume.

Studies of children that experimentally altered the ED of foods tested children’s ability to compensate for energy (16–23). Energy compensation refers to the adjustment of food intake during an ad libitum meal in response to variations in the ED from a preload (ie, a fixed amount of food or liquid consumed before the meal). As reviewed by Birch and Fisher (24), most laboratory studies have shown that young children compensate for energy from a preload, although compensation is usually partial and shows considerable interindividual variability. In addition, the effect of compensation ability on longer-term intake regulation (ie, beyond a single laboratory meal) remains poorly understood.

Laboratory protocols have the advantage of assessing children’s food intake objectively. However, they generally limit the food choices, which, in turn, may create conditions that alter the subjects’ habitual eating behavior. Children may compensate reasonably well for energy during a single meal under controlled conditions but not necessarily outside of the laboratory. It is important to assess children’s eating behavior in relation to dietary ED under free-living conditions when they have access to an ample array of foods that vary in ED. It is of particular interest to further elucidate the nature of children’s intake regulation. Specifically, do children regulate the number of calories they consume on a daily basis (25), or do they regulate the amount or the volume of food they ingest, as the data in adults suggest (26–28)? Also, are there differences in intake regulation between children who are born with a different predisposition to obesity and, if so, how does this affect their ability to compensate? It is possible that obesity-promoting genes or family environments influence compensation ability in children. It has also been
suggested that calories consumed from beverages are less well regulated than are those from solid foods (29, 30). Thus, it is possible that the results for children’s intake regulation may differ when liquids (ie, beverages) are included or excluded from the analyses.

A first aim of this study was to examine the interrelation between daily ED (kcal/g), daily food intake (g), and daily energy intake (kcal) among children born at high or low risk of obesity. We predicted that children would reduce their daily food intake as a function of the ED of their diet, thereby regulating daily energy intake. A second aim was to examine children’s daily food intake in relation to their predicted daily food intake for a given level of daily energy density and to assess potential changes in children’s compensation ability over time (from ages 3 to 6 y). We predicted that children’s daily food intakes, relative to their predicted ones, would differ as a function of their obesity risk status. We also predicted that compensation ability would deteriorate over time in both risk groups. All analyses were completed including or excluding all beverages.

SUBJECTS AND METHODS

Subjects

The subjects in this report were part of an ongoing longitudinal study of growth and development in early life that has been conducted at the University of Pennsylvania and the Children’s Hospital of Philadelphia. The children in this study were born at either low or high risk of obesity (31). The children’s obesity risk status was based on maternal prepregnancy body mass index (BMI; in kg/m²). Infants born to mothers with a prepregnancy BMI less than the 33rd percentile (mean BMI of 19.5 ± 1.1) were classified as being at low risk of obesity (n = 37). Children born to mothers with a prepregnancy BMI greater than the 66th percentile (mean BMI of 30.3 ± 4.2) were classified as being at high risk of obesity (n = 35). Children were enrolled in the study at the age of 3 mo, and their growth and development were followed up to year 12. All children in the study were white. Further details of parental and subject characteristics and the study design were reported previously (31–35). The present report is based on a subsample of this cohort (low-risk: n = 27; high-risk: n = 22) for whom 3-d weighed-food records were available at 3, 4, 5, and 6 y of age. Written informed consent was obtained from the parents. The protocol was approved by the institutional review boards of the University of Pennsylvania and the Children’s Hospital of Philadelphia.

Dietary assessment

Each year, within 2 wk of each child’s birthday, the primary caretakers of children in both risk groups were asked to complete 3-d weighed-food records (2 weekdays, 1 weekend day). Caretakers were provided electronic food scales to preweigh all foods and beverages (except water) consumed by the child and to weigh all leftovers. Food records were analyzed by research nutritionists at the General Clinical Research Center at the Children’s Hospital of Philadelphia by using the Food Processor Nutrition Analysis software (ESHA Research, Salem, OR). Only those food records that were completed for ≥2 d were included in the present analyses. Two- and three-day food records were available for the following numbers of children at ages 3, 4, 5, and 6 y: 45 (40 three-day records and 5 two-day records); 48 (45 three-day records and 3 two-day records); 42 (38 three-day records and 4 two-day records); and 42 (39 three-day records and 3 two-day records), respectively. One three-day food record was not included in the analyses because the child was fed infant formula almost exclusively. Likewise, 2 one-day records were not included because the children were either sick or fell asleep before dinner.

Dietary outcome measures

The following 3 dietary outcome measures were computed from the 3-d weighed-food records: daily ED (kcal/g), daily food intake (g), and daily energy intake (kcal). All 3 outcome variables were computed either to include or exclude all beverages. The daily dietary ED was computed by dividing the total daily calories by the total amount of food and beverages (ie, weight in grams) consumed (36). Daily food and energy intakes were computed as the sums of the individual weights and calories consumed from foods and beverages on a given day. All dietary outcome measures were averaged across the total number of days of completed records included in the analyses. A detailed description of the children’s daily energy density and daily energy intake is provided elsewhere (37).

Statistical analysis

Descriptive statistics are presented as means ± SEMs. To test aim one, a Pearson correlation analysis examined relations between daily ED and daily energy and food intakes. Dietary ED was correlated first with daily food intake and second with daily energy intake within each risk group for each year. These correlation analyses were completed by using intakes that included or excluded all beverages.

Because the correlations tested in aim one could have been due in part to the functional relation between these variables (38), we proceeded by using an analysis of residuals to further examine children’s intake regulation. Thus, to test the second aim of the study, an analysis of residuals was conducted in which, for each age, daily food intake was regressed onto daily ED and the residual scores were saved. The residual is that part of daily food intake that is uncorrelated with daily ED. Residuals are the differences between an observed value of the response variable and the value predicted by the model. In our model, the residuals represent the vertical difference between the regression line (or the predicted value) and the actual (or observed) data. The residual represents the difference between an individual’s actual food intake and what their food intake would be expected to be for a given level of ED based on this sample. Thus, the residuals allow us to break down the sample into children who eat more or less than would be expected. Positive residuals represent greater food consumption than would be expected (ie, overconsumption), whereas negative residuals represent less food consumption than expected (ie, underconsumption). A residual of zero represents what the model would predict food intake to be for a given ED value on the basis of this sample; thus, the deviation from the perfect prediction is zero.

A 2 (risk group) × 4 (age) mixed linear model analysis of variance (ANOVA) tested for (linear) differences in the mean residual food scores as a function of risk group and child age.
Risk group was a between-subjects variable with 2 levels (low-risk or high-risk), and child age was a within-subjects variable with 4 levels (3, 4, 5, or 6 y). A main effect of risk group would imply that the tendency to overconsume food differs for high-risk and low-risk children. Because of the repeated-measures structure of the data, the model used restricted maximum likelihood estimates and a compound symmetry error structure. Significant main effects or interactions were followed up by pair-wise comparisons, as well as contrasts, to test for potential linear effects of time across all 4 ages and linear time by risk group interactions. Again, these analyses were completed by using intakes that included or excluded all beverages.

The ANOVA model to test aim 2 was run with and without child BMI $z$ score as a covariate to ensure that any risk group differences in residual scores were not merely due to differences in children’s weight status. We identified 2 children in the low-risk group who, at age 4 y, qualified as statistical outliers per Tukey’s criteria (39) with regard to BMI $z$ score. We comment in the Results section that the differences in residual scores were not merely due to differences in BMI $z$ score within their respective risk group. The mean ($\pm$ SEM) BMI $z$ scores for high-risk and low-risk children were $-0.4 \pm 0.3, 0.4 \pm 0.3, 0.5 \pm 0.4$, and $0.2 \pm 0.3$, and those for low-risk children were $-0.4 \pm 0.2, 0.1 \pm 0.1, 0.1 \pm 0.2$, and $-0.3 \pm 0.2$ at ages 3, 4, 5, and 6 y, respectively. Student $t$ tests showed that, at each of the years, none of these BMI $z$ scores were significantly different between risk groups. More detailed analyses of anthropometric measures for this subsample (37) as well as the full cohort (34) are provided elsewhere.

### RESULTS

#### Child characteristics

The number of low-risk children included in the present study at ages 3, 4, 5, and 6 y were 23 (13 boys, 10 girls), 27 (14 boys, 13 girls), 23 (11 boys, 12 girls), and 22 (8 boys, 14 girls); the number of high-risk children were 22 (12 boys, 10 girls), 21 (9 boys, 12 girls), 19 (10 boys, 9 girls), and 20 (11 boys, 9 girls), respectively. The mean ($\pm$SEM) BMI $z$ scores for high-risk children were $-0.4 \pm 0.3, 0.4 \pm 0.3, 0.5 \pm 0.4$, and $0.2 \pm 0.3$, and those for low-risk children were $-0.4 \pm 0.2, 0.1 \pm 0.1, 0.1 \pm 0.2$, and $-0.3 \pm 0.2$ at ages 3, 4, 5, and 6 y, respectively. Student $t$ tests showed that, at each of the years, none of these BMI $z$ scores were significantly different between risk groups. More detailed analyses of anthropometric measures for this subsample (37) as well as the full cohort (34) are provided elsewhere.

### Daily food intake, energy intake, and energy density

The mean daily food intake (g), daily energy intake (kcal), and daily energy density (kcal/g) for children in both risk groups at ages 3, 4, 5, and 6 y are shown in Table 1. These data are displayed for descriptive purposes only. A detailed discussion of these results can be found in a previously published paper by Kral et al (37) that investigated the changes in dietary energy density in children in this cohort over time.

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**Table 1**

<table>
<thead>
<tr>
<th>Daily intake and risk group</th>
<th>Age 3 y</th>
<th>Age 4 y</th>
<th>Age 5 y</th>
<th>Age 6 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake, food only (kcal)$^1$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>898 ± 47</td>
<td>939 ± 55</td>
<td>1064 ± 52</td>
<td>1159 ± 45</td>
</tr>
<tr>
<td>High risk</td>
<td>847 ± 51</td>
<td>965 ± 57</td>
<td>1177 ± 70</td>
<td>1393 ± 67$^1$</td>
</tr>
<tr>
<td>Energy intake, food and beverages (kcal)$^2$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>1145 ± 49</td>
<td>1219 ± 62</td>
<td>1311 ± 58</td>
<td>1435 ± 43</td>
</tr>
<tr>
<td>High risk</td>
<td>1189 ± 59</td>
<td>1253 ± 61</td>
<td>1493 ± 84</td>
<td>1687 ± 69</td>
</tr>
<tr>
<td>Food intake, food only (g)$^3$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>443 ± 25</td>
<td>451 ± 30</td>
<td>466 ± 25</td>
<td>525 ± 31</td>
</tr>
<tr>
<td>High risk</td>
<td>395 ± 24</td>
<td>447 ± 24</td>
<td>526 ± 34</td>
<td>620 ± 36</td>
</tr>
<tr>
<td>Food intake, food and beverages (g)$^4$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>948 ± 45</td>
<td>1018 ± 58</td>
<td>980 ± 54</td>
<td>1104 ± 42</td>
</tr>
<tr>
<td>High risk</td>
<td>1062 ± 74</td>
<td>1097 ± 58</td>
<td>1210 ± 90</td>
<td>1262 ± 51</td>
</tr>
<tr>
<td>Energy density, food only (kcal/g)$^5$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>2.14 ± 0.10</td>
<td>2.23 ± 0.08</td>
<td>2.37 ± 0.09</td>
<td>2.31 ± 0.08</td>
</tr>
<tr>
<td>High risk</td>
<td>2.23 ± 0.09</td>
<td>2.24 ± 0.11</td>
<td>2.36 ± 0.11</td>
<td>2.34 ± 0.10</td>
</tr>
<tr>
<td>Energy density, food and beverages (kcal/g)$^6$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>1.26 ± 0.06</td>
<td>1.27 ± 0.05</td>
<td>1.39 ± 0.06</td>
<td>1.35 ± 0.06</td>
</tr>
<tr>
<td>High risk</td>
<td>1.21 ± 0.07</td>
<td>1.19 ± 0.05</td>
<td>1.30 ± 0.07</td>
<td>1.39 ± 0.09</td>
</tr>
</tbody>
</table>

$^1$ All values are $\bar{x} \pm$ SEM. These data were part of a previous publication by Kral et al (37).

$^2$ 2 × 4 ANOVA indicated a significant (linear) risk group × time interaction ($P < 0.02$).

$^3$ Significantly different from the low-risk group, $P < 0.05$.

$^4$ 2 × 4 ANOVA indicated a significant main and linear effect of time ($P < 0.0001$) but no significant risk group × time interaction ($P = 0.19$).

$^5$ 2 × 4 ANOVA indicated a significant risk group × time interaction ($P = 0.02$).

$^6$ 2 × 4 ANOVA indicated a significant linear effect of time ($P < 0.0001$) but no risk group × time interaction ($P = 0.40$).

$^7$ 2 × 4 ANOVA indicated a significant linear effect of time ($P = 0.03$) but no risk group × time interaction ($P = 0.98$).

$^8$ 2 × 4 ANOVA indicated a significant linear effect of time ($P = 0.002$) but no risk group × time interaction ($P = 0.58$).
TABLE 2
Pearson correlation coefficients summarizing the relation between dietary energy density and daily food and energy intakes (excluding beverages) by risk group and child age

<table>
<thead>
<tr>
<th>Risk group</th>
<th>Daily intake</th>
<th>Age 3 y</th>
<th>Age 4 y</th>
<th>Age 5 y</th>
<th>Age 6 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk</td>
<td>Food intake, food only (g)</td>
<td>-0.55†</td>
<td>-0.49‡</td>
<td>-0.50§</td>
<td>-0.70*</td>
</tr>
<tr>
<td>High risk</td>
<td>Food intake, food only (g)</td>
<td>-0.40†</td>
<td>-0.23</td>
<td>-0.65*</td>
<td>-0.51*</td>
</tr>
<tr>
<td>Low risk</td>
<td>Energy intake, food only (kcal)</td>
<td>0.31</td>
<td>0.10</td>
<td>0.30</td>
<td>-0.01</td>
</tr>
<tr>
<td>High risk</td>
<td>Energy intake, food only (kcal)</td>
<td>0.21</td>
<td>0.50‡</td>
<td>0.13</td>
<td>0.29</td>
</tr>
</tbody>
</table>

*With the use of Fisher r-to-z transformations and a 2-tailed significance test, none of the correlation coefficients was significantly different within each age group or across age groups.
† Correlation significant at 0.01 level.
‡ Correlation significant at 0.05 level.
§ P < 0.006 (Bonferroni correction, α = 0.05/8).
* P = 0.06.

Relation between daily food intake and energy density

And between daily energy intake and energy density

All beverages excluded

Daily food intake was significantly inversely correlated with daily ED for the low-risk group at all ages and for the high-risk group at ages 5 and 6 y (P < 0.05; Table 2). The inverse relation was borderline significant in the high-risk group at 3 y of age (P = 0.06). This indicates that children who consumed a diet that was relatively higher in ED tended to eat less food on a daily basis than did children who consumed a diet that was relatively lower in ED. Two of these 8 associations remained significant, and 2 were borderline significant, when applying a Bonferroni-adjusted α (0.05/8 = 0.006).

Daily energy intake was not significantly correlated with daily ED for the low-risk group at all ages and for the high-risk group at ages 3, 5, and 6 y (Table 2). This indicates that across a range of dietary ED, daily caloric intake did not vary significantly as a function of ED during these years.

All beverages included

Daily food intake was significantly inversely correlated with daily ED for both the low-risk and the high-risk group at all ages (P < 0.05; Table 3). Six of these 8 associations remained significant when applying a Bonferroni-adjusted α (0.05/8 = 0.006). Daily energy intake, however, was not significantly correlated with daily ED for either group at any age (Table 3).

TABLE 3
Pearson correlation coefficients summarizing the relations between dietary energy density and daily food and energy intakes (including beverages) by risk group and child age

<table>
<thead>
<tr>
<th>Risk group</th>
<th>Daily intake</th>
<th>Age 3 y</th>
<th>Age 4 y</th>
<th>Age 5 y</th>
<th>Age 6 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk</td>
<td>Food intake, food and beverages (g)</td>
<td>-0.58**</td>
<td>-0.45‡</td>
<td>-0.64*</td>
<td>-0.78*</td>
</tr>
<tr>
<td>High risk</td>
<td>Food intake, food and beverages (g)</td>
<td>-0.69**</td>
<td>-0.49‡</td>
<td>-0.62*</td>
<td>-0.61*</td>
</tr>
<tr>
<td>Low risk</td>
<td>Energy intake, food and beverages (kcal)</td>
<td>0.28</td>
<td>0.13</td>
<td>-0.05</td>
<td>0.26</td>
</tr>
<tr>
<td>High risk</td>
<td>Energy intake, food and beverages (kcal)</td>
<td>-0.002</td>
<td>0.40</td>
<td>0.13</td>
<td>0.42</td>
</tr>
</tbody>
</table>

*With the use of Fisher r-to-z transformations and a 2-tailed significance test, none of the correlation coefficients was significantly different within each age group or across age groups.
** Correlation significant at 0.01 level.
‡ Correlation significant at 0.05 level.
P < 0.006 (Bonferroni correction, α = 0.05/8).
Correlation significant at 0.05 level.
There was a main effect of BMI $z$ score when added to the model as a covariate ($P < 0.0001$), whereas the main effect of risk group ($P = 0.21$) and the risk group $\times$ age interaction ($P = 0.76$) were not significant. After removal of the 2 outliers, the main effect of BMI $z$ score remained significant ($P = 0.0001$); the main effect of risk group remained nonsignificant ($P = 0.19$), as did the risk group $\times$ age interaction ($P = 0.72$).

**DISCUSSION**

The main finding of the present study was that across a range of ED, children born at high or low risk of obesity adjusted the daily amount of food they consumed on the basis of the ED of their diet. Children who consumed a diet that was higher in ED consumed less food daily than did children whose diet was lower in ED. These findings held true regardless of whether beverages were included or excluded. This is the first longitudinal study to show that children aged 3–6 y also adjust their daily intake under free-living conditions. These findings thus confirm and extend previous research conducted under laboratory conditions.

The prospective nature of this design also allowed us to test for changes in energy regulation ability over time. It has been suggested that developmental differences in energy compensation ability may exist in children (24). Preschool children, on average, show compensation (24); however, Anderson et al (41) found no effect on food intake when varying the ED of preload in 9–10-y-old children. These findings indirectly suggest developmental changes in compensation ability; however, this phenomenon has never been documented in long-term prospective studies that track children during growth.

With the use of a novel approach of residual score analysis, this present study assessed changes in compensation ability over the course of 4 y. The results indicated different patterns of residuals between high- and low-risk children, which suggests that familial predisposition to obesity may partially operate through errors in compensation. This supports theories and experimental studies regarding energy compensation ability and the development of pediatric obesity (22–24, 42). However, it should be pointed out that the results from the residual analysis are sample-dependent and therefore may not apply to other cohorts of children. The main finding of the residual analysis was that high-risk children tended to overconsume, whereas low-risk children tended to underconsume, relative to their predicted intake, although the results differed depending on whether beverages were included in the analyses. For analyses that excluded beverages, the risk group differences gradually emerged over time, with high-risk children not showing a tendency to overconsume until after 4 y of age. This is consistent with the notion that compensation ability gradually deteriorates during early childhood, possibly starting during the years corresponding to adiposity rebound (43), rather than being compromised consistently and as early as 3 y of age. This gradual decline in compensation ability was not seen when beverages were included in the analyses (see below). Another key finding was that the risk group differences remained significant even when the analyses were controlled for BMI $z$ score, which suggests that the errors in compensation cannot be fully explained by differences in weight status. Again, this was not the case when beverages were included in the analyses (see below).

When beverages were included in the residual score analyses, the results indicated a trend ($P = 0.07$) for high-risk children to...
overconsume and low-risk children to underconsume across all 4 y. That is, errors in compensation did not appear to gradually emerge over time but may have been present as early as 3 y of age. This interpretation, if true, would suggest that errors in compensation ability for (food and) beverages are established by 3 y of age and, therefore, that any deterioration in compensation ability may have occurred before the age of 3 y. This finding raises the possibility that infancy and the first 2 y of life may be critical periods for the development of compensation ability for food and beverages (20, 21, 44, 45) and therefore may be important periods for early-life interventions. To more convincingly identify critical periods of time when children’s ability to compensate may start to deteriorate, however, studies are needed that examine (or track) the stability of compensation indexes. Findings from the current study are also consistent with studies reporting that excess beverage consumption (especially from fruit juice) is associated with excess weight gain in preschool-age children (46, 47). Hunger and thirst mechanisms are believed to be distinct entities, because the consumption of beverages primarily acts on thirst and not hunger mechanisms (48). It is possible that liquids, which are thought of as having a generally lower satiating efficiency (49), may thus further weaken children’s compensation ability.

Another unique finding of the residual score analyses that included beverages was that the risk group differences no longer approached statistical significance when BMI z score was added as a covariate. BMI z score was consistently associated with greater residual scores across analyses, which suggests that errors in compensation ability for beverages (and food) may be affected by child weight status and the determinants thereof. Heavier children may be more likely to exceed their energy needs through consumption of beverages (46). Despite these findings, it should be noted that beverages, because of their low energy density, add weight but fewer calories to the daily total, and, thus, by computation, disproportionately influence dietary ED. Hence, residual analyses that include beverages may need to be interpreted with more caution.

The result of this investigation agrees with findings from 2 recent experimental studies (22, 23) that showed that energy compensation in children I) is often incomplete and shows great interindividual variability and 2) tends to decline with age. It is possible that children, especially those who are genetically predisposed to obesity, may become more responsive to environmental influences (such as the portion size of foods) as they become older. A study conducted by Rolls et al (50) showed that when presented with larger portions of food, 5-y-old children, but not 3-y-old children, significantly increased their intakes relative to when they were presented with smaller portions of food. The authors suggested that as children age they become increasingly responsive to environmental, social, and cultural factors that all are likely to affect their intake. To what extent these factors increase children’s vulnerability to overeat may depend on parental obesity, a line of research that has yet to be further explored.

The strengths of the present study include 1) the unique sample of children born at different risk of obesity and who were prospectively studied and 2) the use of extensive training and electronic food scales to measure the weights of the foods that the children consumed. The study might well be extended in several ways. First, it would be desirable to examine ability to adjust food intake in relation to dietary ED in a larger cohort of children who are ethnically diverse and who show greater variability in their weight status and age than did the children in the current cohort. Thus, the results from this study are limited to the relatively narrow study sample, namely, healthy, full-term, white children, and may not be generalizable to children as a whole. Second, it would be desirable to obtain intake data over a longer period of time at each age to assess children’s habitual diet. Third, the validity of the data may be increased by complementing self-reported intakes, which may be subject to measurement or reporting errors by mothers, with measured intake data in the feeding laboratory. Fourth, future research should also collect data of energy expenditure to be able to relate children’s intake data to their energy needs. Last, the present design, unlike a classic twin design or other more genetically sensitive designs, could not formally test the respective influence of genetic and environmental influences on compensation ability.

In conclusion, the results of the present study suggest that children between the ages of 3 and 6 y have the ability to adjust their daily food intake on the basis of the ED of their diet to maintain a certain daily energy intake in the free-living environment. Energy compensation ability, however, may differentiate children who were born at high-risk or at low-risk of obesity. Future research is needed that separates the effects of food and beverages on children’s compensation ability.

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REFERENCES

12. Bell EA, Rolls BJ. Energy density of foods affects energy intake across


