Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity\textsuperscript{1–3}

Susan Carnell and Jane Wardle

ABSTRACT

Background: Pressures from the “obesogenic” environment are driving up obesity rates, but adiposity still varies widely within the population. Appetitive characteristics could underlie differences in susceptibility to the environment.

Objective: We examined associations between adiposity and 2 appetitive traits: satiety responsiveness and food cue responsiveness in children.

Design: Parents of 2 groups of children, 8–11-y-olds (n = 10 364) from a population-based twin cohort and 3–5-y-olds (n = 572) from a community sample, completed the Child Eating Behavior Questionnaire. Adiposity was indexed with body mass index (BMI; in kg/m\textsuperscript{2}) SD scores. For the 8–11-y-olds, waist circumference was also recorded and used to derive waist SD scores.

Results: In both samples, higher BMI SD scores were associated with lower satiety responsiveness (8–11-y-olds: r = −0.22; 3–5-y-olds: r = −0.19; P < 0.001) and higher food cue responsiveness (r = 0.18 and 0.18; P < 0.001). In the twin sample, waist SD scores were associated with satiety responsiveness (r = −0.23, P < 0.001) and food cue responsiveness (r = 0.20, P < 0.001). By analyzing the data by weight categories, children in higher weight and waist categories had lower satiety responsiveness and higher responsiveness to food cues in both samples (8–11-y-olds: both P < 0.001; 3–5-y-olds: both P < 0.05), but the effect was more strongly linear in the older children. All associations remained significant, controlling for child age and sex and parental education and BMI.


INTRODUCTION

Obesogenic modern environments have caused marked increases in mean population weights over the past few decades (1, 2), but there is still tremendous variability in weight within populations. There is a significant genetic component to variability in weight (3, 4), but the mechanisms that cause some persons to eat more than their metabolic requirement and gain weight, while others stay slim, remain poorly understood.

A strong body of research indicates that obese adults differ from normal-weight adults in appetite-related characteristics. In a series of studies in the 1960s, obese adults were shown to have less effective down-regulation of appetite after food consumption (5), lower sensitivity to gastric motility (6), and they over-consumed when misled into believing that it was their usual time for a meal (7). Obese adults also exhibited stronger up-regulation of intake in response to palatability than did normal-weight controls (8). More recent research has shown slower declines in salivary response with repeated presentations of a palatable food (9) and higher scores on psychometric measures of perceived hunger and disinhibited eating (10, 11).

Similar findings were reported in children. Obese children show poorer caloric compensation after a preload (12, 13), increase their food intake more than normal-weight controls after exposure to food cues (12), have higher levels of snack consumption in the absence of hunger (14), and score higher on psychometrically assessed “external eating” (15). They also fail to show the “normal” pattern of deceleration of eating during a meal (16, 17).

With the use of the traditional “obese case” compared with “normal-weight control” design, those studies indicate that obese persons have distinctive appetitive profiles both in terms of responsiveness to internal satiety cues and responsiveness to external environmental cues to eat (18, 19). Although informative, this methodologic approach to obesity is now under pressure as it becomes clear that many people who are obese today would not have been obese if they had lived 30 y ago, and persons often change their “case” status throughout life, moving from normal-weight through overweight to obesity (20). Adiposity is also a quantitative trait with an approximately normal distribution. Examining the correlates of variation in adiposity across the population may therefore be a useful approach to the study of common obesity in contemporary environments.

We hypothesized that quantitatively distributed appetitive characteristics would be significantly associated with variation in adiposity. We tested this theory in 2 large samples of children with the use of validated, parent-reported measures of satiety

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sensitivity and responsiveness to food cues. The use of psychometric measures allowed us to gather large amounts of data and to tap enduring appetite traits, studying children meant that eating behaviors were less influenced by dietary restraint, and using 2 age groups allowed us to test the generalizability of the theory across childhood. Ethical approval for the study was granted by the University College London and King’s College London Ethics Committees.

SUBJECTS AND METHODS

Study 1 (8–11-y-olds)

Participants and procedure

Parents and their 8–11-y-old twins were drawn from the Twins Early Development Study, a population-based cohort of twins followed from birth (21). Packs containing questionnaires, reply-paid envelopes, and 2-m tape measures were mailed to a total of 12,212 families, of whom 8978 (73.5%) were considered active and 3234 (26.5%) inactive in terms of current participation in other aspects of the Twins Early Development Study.

Anthropometric measures

Parents were asked to weigh and measure their children and to record the weights to the nearest pound or tenth of a kilogram and to record the heights to the nearest centimeter. They were asked to use the tape measure to measure the child’s waist circumference directly over the skin at 4 cm above the navel, after a gentle exhalation (22). We tested the correspondence between parent and researcher measurements in a subsample of 228 families participating in a more intensive study of weight and eating and activity behaviors and found correlations of 0.90, 0.83, and 0.92 for height, weight, and waist circumference, respectively (4).

Questionnaire measures

Parents completed 2 scales from the Child Eating Behavior Questionnaire (CEBQ) for each child. The CEBQ was developed to assess a range of eating behavior traits in children and has good internal consistency, test-retest reliability, and stability over time (23, 24). A shortened version of the combined Satiety Responsiveness-Slowness in Eating (SR-SE) scale (6 items) was used to measure satiety responsiveness, and the full Enjoyment of Food (EF) scale (4 items) was used to measure responsiveness to food cues. The SR-SE scale assesses responsiveness to internal satiety cues (eg, my child cannot eat a meal if he or she has had a snack just before; my child eats more and more slowly during the course of a meal), and the EF scale assesses the child’s general responsiveness to food and interest in eating (eg, my child loves food). Both scales show good validity against objective behavioral measures, including eating rate, intake in the absence of hunger, and caloric compensation (25). Responses to all items were on 1–5 Likert scales labeled never, rarely, sometimes, mostly, and always. Parents were also asked to report their own height and weight.

Statistical analysis

CEBQ scale scores were calculated by generating item means. Adiposity was indexed with body mass index (BMI; in kg/m²) SD scores, derived with the use of the lmsGrowth macro (from http://homepage.mac.com/tjcole). BMI SD scores represent the number of SDs the child’s BMI value is from the 1990 UK reference data for children of the same age and sex (26). IOTF (International Obesity Task Force) weight categories, which use age- and sex-specific values of BMI centiles associated with BMIs of 25 (adult overweight) and 30 (adult obesity) at 18 y of age (27), were derived with the use of the same program. To examine the appetite-adiposity relation in detail, normal-weight children were further divided into those who were ≤50th centile (low-normal-weight) and those >50th centile but not meeting IOTF criteria for overweight or obesity (mid-normal-weight).

Waist SD scores based on 1990 data (28) were also generated with the use of lmsGrowth and were used to derive waist centiles. Because no cutoffs are established to indicate clinical risk of waist for children, for categorical analyses we used the 91st to 97th centiles (high waist) and ≥98th (very high waist), corresponding to cutoffs for BMI based on the 1990 data (26). The normal waist size category was further divided into low-normal waist (0th–50th centile) and mid-normal waist (>50th but <91st centile) groups, as for the BMI categories. Waist analyses were repeated with the use of 85th and 95th centile cutoffs to correspond approximately to BMI centiles representing overweight and obesity (29), but results were the same and are not reported here.

Pearson’s correlations were used to assess associations between CEBQ scales and BMI SD scores and to check for associations with child age and parental education. Univariate analyses of variance with polynomial contrasts were used to test for differences in CEBQ subscale scores by weight categories and to test the significance of the linear trends across weight categories. Bonferroni-adjusted post hoc tests were used to test the significance of differences between weight categories. These analyses were repeated with child age and sex and with parental education and BMI as covariates, although, because of missing parent data, the sample size for the analysis of covariance (ANCOVA) was reduced. Multivariate analyses with both appetite variables included as potential predictors of BMI SD scores were conducted within a General Linear Model (GLM). All analyses used the GLM FOR COMPLEX SAMPLES in SPSS (SPSS Inc, Chicago, IL), which allows adjustment for clustering within twin pairs.

Study 2 (3–5-y-olds)

Participants and procedure

Parents or primary caregivers (henceforth referred to collectively as parents) and their 3–5-y-old children were recruited through preschool (nursery) classes in 16 primary schools in London, England. Schools were chosen to represent a range of socioeconomic deprivation as indexed by the proportion of pupils eligible for free school meals (a UK government benefit available to lower-income families). Parents (n = 1082) were sent letters that described the study and invited to participate. The CEBQ (23) was enclosed with a reply-paid return envelope. Nonrespondents were sent one reminder after 2 wk.

Anthropometric measures

Trained researchers weighed and measured children at school following standard protocols, with the use of calibrated Tanita digital scales (Tanita Corp, Tokyo, Japan) and Leicester height measures (Seca, Birmingham, United Kingdom). Height was recorded to the nearest millimeter and weight to the nearest tenth of a kilogram.
Questionnaire measures

The questionnaire measures were the same as for study 1.

Statistical analysis

CEBQ scale scores were based on item means, and BMI SD scores and weight status were generated as for those in study 1. All analyses were conducted in SPSS (version 14; SPSS Inc). Pearson’s correlations were used to assess associations between CEBQ scales and BMI SD scores and to check for associations with child age and parental education. As with the 8–11-y-olds, we subdivided the normal-weight group at the 50th centile. Univariate analyses of variance with polynomial contrasts were used to test for differences in CEBQ subscale scores by weight categories and for the presence of a linear trend, and Bonferroni-adjusted post hoc tests were used to test for differences between the weight categories. Prediction of BMI SD scores controlling for parental BMI and education and child age and weight were conducted within a GLM.

RESULTS

Study 1 (8–11-y-olds)

Response rates

Of the 8978 active families, 5543 (61.7%) parents returned complete questionnaires, and of the 3234 inactive families, 359 (11.1%) parents responded, giving a total sample of 5902 (48.3%) of the United Kingdom in terms of ethnicity (93% white), but parents were mothers of the child. The sample was representative of the whole UK population (Health Survey for England 2003). Mean BMI among parents was 25.4 ± 5.19, with 26% classified as overweight and 14% as obese. Characteristics for parents and children for each sample are given in Table 1.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Sample characteristics</th>
<th>3–5-y-olds</th>
<th>8–11-y-olds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children</td>
<td>n</td>
<td>572</td>
<td>10 364</td>
</tr>
<tr>
<td>Age (y)</td>
<td>4.4 ± 0.622</td>
<td>9.9 ± 0.86</td>
<td></td>
</tr>
<tr>
<td>Girls [%]</td>
<td>268 (46.9)</td>
<td>5340 (51.5)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>16.6 ± 1.77</td>
<td>17.2 ± 2.94</td>
<td></td>
</tr>
<tr>
<td>BMI SD score</td>
<td>0.56 ± 1.12</td>
<td>0.02 ± 1.17</td>
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<tr>
<td>BMI centile</td>
<td>64.3 ± 28.1</td>
<td>49.7 ± 31.0</td>
<td></td>
</tr>
<tr>
<td>Weight status [%]</td>
<td>Normal-weight</td>
<td>424 (74.1)</td>
<td>8880 (85.7)</td>
</tr>
<tr>
<td>Of whom were low-normal-weight</td>
<td>175 (30.6)</td>
<td>5392 (52.0)</td>
<td></td>
</tr>
<tr>
<td>Of whom were mid-normal-weight</td>
<td>249 (43.5)</td>
<td>3488 (33.7)</td>
<td></td>
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<tr>
<td>Overweight</td>
<td>108 (18.9)</td>
<td>1183 (11.4)</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>40 (7.0)</td>
<td>301 (2.9)</td>
<td></td>
</tr>
<tr>
<td>Waist SD score</td>
<td>—</td>
<td>62.4 ± 7.10</td>
<td></td>
</tr>
<tr>
<td>Waist SD centile</td>
<td>—</td>
<td>0.80 ± 1.00</td>
<td></td>
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<tr>
<td>Waist centile</td>
<td>—</td>
<td>71.1 ± 24.6</td>
<td></td>
</tr>
<tr>
<td>Weight status [%]</td>
<td>Normal waist</td>
<td>—</td>
<td>7457 (72.0)</td>
</tr>
<tr>
<td>Of whom have low-normal-waist</td>
<td>—</td>
<td>2208 (21.3)</td>
<td></td>
</tr>
<tr>
<td>Of whom have mid-normal-waist</td>
<td>—</td>
<td>5249 (50.6)</td>
<td></td>
</tr>
<tr>
<td>High waist</td>
<td>—</td>
<td>1672 (16.1)</td>
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<tr>
<td>Very high waist</td>
<td>—</td>
<td>1235 (11.9)</td>
<td></td>
</tr>
<tr>
<td>Parents</td>
<td>n</td>
<td>572</td>
<td>5182</td>
</tr>
<tr>
<td>Age (y)</td>
<td>34.9 ± 5.823</td>
<td>41.4 ± 6.47</td>
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<tr>
<td>Ethnicity [%]</td>
<td>White</td>
<td>402 (68.2)</td>
<td>4796 (92.6)</td>
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<tr>
<td>Nonwhite</td>
<td>151 (28.5)</td>
<td>281 (5.4)</td>
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<tr>
<td>Ethnicity missing</td>
<td>19 (3.3)</td>
<td>105 (2.0)</td>
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<td>Parental education [%]</td>
<td>No qualifications</td>
<td>59 (10.3)</td>
<td>271 (5.2)</td>
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<tr>
<td>GCSEs or equivalent</td>
<td>219 (38.3)</td>
<td>2526 (48.8)</td>
<td></td>
</tr>
<tr>
<td>A-levels or equivalent</td>
<td>141 (24.7)</td>
<td>1082 (20.9)</td>
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<td>Degree or higher</td>
<td>142 (24.8)</td>
<td>1074 (20.7)</td>
<td></td>
</tr>
<tr>
<td>Education missing</td>
<td>11 (1.9)</td>
<td>229 (4.4)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>24.4 ± 4.30</td>
<td>25.4 ± 5.19</td>
<td></td>
</tr>
<tr>
<td>Weight status [%]</td>
<td>Normal-weight</td>
<td>324 (56.6)</td>
<td>2701 (52.1)</td>
</tr>
<tr>
<td>Overweight</td>
<td>137 (24.0)</td>
<td>1368 (26.4)</td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td>45 (7.9)</td>
<td>740 (14.3)</td>
<td></td>
</tr>
<tr>
<td>Missing data</td>
<td>66 (11.5)</td>
<td>373 (7.2)</td>
<td></td>
</tr>
</tbody>
</table>

1 BMI and waist SD scores andcentiles were calculated with reference to 1990 UK data. Overweight and obese categories were determined with criteria from the International Obesity Task Force (IOTF); low-normal category includes children with a BMI centile score of <50; mid-normal category includes children >50th centile according to 1990 UK reference data but not meeting IOTF criteria for overweight or obesity. High and very high waist categories correspond to the 91st and 98th centiles according to 1990 UK reference data; low-normal category includes children with a waist centile score of <50; mid-normal category includes children between the 50th and the 90.99th waist centiles. GCSE, General Certificate of Secondary Education.

2 ± SD (all such values).

3 Because of rounding, these 2 figures do not add up to the 72.0% in the normal waist category.
Internal consistency was high for both scales (SR-SE: α = 0.81; EF: α = 0.87). Mean SR-SE scores were 2.60 ± 0.71 in boys and 2.72 ± 0.69 in girls (P < 0.001), and mean EF scores were 4.11 ± 0.73 in boys and 4.12 ± 0.76 in girls (P = 0.300). The SR-SE scale was approximately normally distributed, whereas the EF scale showed negative skewness, but skewness statistics did not exceed one in either case. The SR-SE and EF scales were negatively correlated (r = −0.53, P < 0.001). Child age was negatively correlated with the SR-SE scores (r = −0.09, P < 0.001) but unassociated with the EF scores (r = 0.00, P = 0.450). The SR scores showed a small negative association with maternal education (r = −0.03, P = 0.036), but the EF scores showed no association (r = 0.00, P = 0.671).

**CEBQ subscales and child adiposity**

Descriptive statistics for the CEBQ subscales in both samples are presented in Table 2. Pearson’s correlations showed significant negative associations between the SR-SE score and both BMI SD (r = −0.22, P < 0.001) and waist SD (r = −0.23, P < 0.001) scores. The EF score was significantly positively correlated with both indexes (BMI SD: r = 0.18; waist SD: r = 0.20; both P < 0.001) (Table 3). All correlations were similar in boys and girls.

The mean CEBQ subscale scores by BMI and waist circumference categories are shown in Figure 1A–D. Univariate analysis of variance showed significant differences between the 4 weight categories for the SR-SE scores (n = 10 359; P < 0.001), and polynomial contrasts showed a significant linear trend, with increasingly low SR-SE scores at higher BMIs (P < 0.001). Controlling for child age and sex and parent education and BMI in the model (ANCOVA) did not change the results (SR-SE score: P < 0.001; EF score: P < 0.001) and no attenuation when including parental BMI and education and child age and sex. Each group differed significantly from the others on both scales (SR-SE: P < 0.001; EF: P < 0.001). Entering the SR-SE and EF scores simultaneously in GLM showed that each scale made independent contributions to variance in BMI SD (SR-SE score: P < 0.001; EF score: P < 0.001) and waist SD (SR-SE score: P < 0.001; EF score: P < 0.001).

### Study 2 (3–5-y-olds)

**Response rates**

From the 1082 questionnaires that were distributed, 573 (53%) were returned with completed CEBQ scales. Response rates varied between schools from 16% to 80%, with lower rates among more socioeconomically deprived and ethnically diverse schools. One child with an outlying BMI value of 31 was omitted from further analyses, leaving a sample of 572. No significant weight differences were observed between children whose parents returned the questionnaire and those whose parents did not.

**Sample characteristics**

Children’s ages ranged from 3 to 6 y, with a mean of 4 y. Mean (±SD) BMIs were 16.7 ± 1.87 in boys and 16.5 ± 1.63 in girls (P = 0.049), with BMI SD scores of 0.64 ± 1.21 in boys and 0.47 ± 1.01 in girls (P = 0.064). BMI indexes were unrelated to either child age or the educational qualification of the parent. On the basis of the IOTF criteria, 19% of children were overweight (19% of boys, 19% of girls) and an additional 7% were obese (8% of boys, 6% of girls). The average age of parents was 35 y, and 94% were the mother of the child. Average parental BMI was 24.4 ± 4.28, and 24% were overweight and 8% obese. Only 68% of the sample was white, which is below the UK population average of ≈91% and reflects the ethnic diversity of the participating urban schools. Parents who responded were slightly better educated than in the United Kingdom as a whole, with 50% having achieved A-levels or higher qualifications (see Table 1 for sample characteristics).

### CEBQ subscales

Descriptive statistics for the CEBQ subscales are included in Table 2. Cronbach’s α was acceptable for the SR-SE score (0.73) and high for the EF score (0.88). Mean scores were 3.07 ± 0.61 (0.001) but not from the overweight group (4.33 ± 0.02) (P < 1.000); differences between the low-normal-weight, mid-normal-weight, and overweight groups were all significant (all P < 0.001). Analyses by waist circumference group showed even stronger gradients, with a significant linear trend (SR-SE score: P < 0.001; EF score: P < 0.001) and no attenuation when including parental BMI and education and child age and sex. Each group differed significantly from the others on both scales (SR-SE: P < 0.001; EF: P < 0.001). Entering the SR-SE and EF scores simultaneously in GLM showed that each scale made independent contributions to variance in BMI SD (SR-SE score: P < 0.001; EF score: P < 0.001) and waist SD (SR-SE score: P < 0.001; EF score: P < 0.001).

### Table 2

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Child Eating Behavior Questionnaire subscale scores $^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3–5-y-olds (n = 572)</td>
</tr>
<tr>
<td>Satiety Responsiveness</td>
<td>3.07 ± 0.61</td>
</tr>
<tr>
<td>Enjoyment of Food</td>
<td>3.46 ± 0.81</td>
</tr>
</tbody>
</table>

$^1$ All values are $\bar{x}$ ± SD.

### Table 3

Unadjusted Pearson’s correlations between Child Eating Behavior Questionnaire subscales and child adiposity $^1$

<table>
<thead>
<tr>
<th>BMI SD score</th>
<th>Waist SD score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Satiety Responsiveness</td>
<td>-0.19</td>
</tr>
<tr>
<td>Enjoyment of Food</td>
<td>0.18</td>
</tr>
</tbody>
</table>

$^1$ P = 0.001 for all.
for the SR-SE score and 3.46 ± 0.81 for the EF score and did not differ significantly by sex. Both scales were approximately normally distributed and were negatively correlated with one another (r = −0.62, P < 0.001). Neither scale was significantly associated with parental education or child age.

**CEBQ subscales and child adiposity**

Correlation analyses showed that the SR-SE score was negatively associated with BMI SD score (r = −0.19, P = <0.001), whereas the EF score was positively associated (r = 0.18, P = <0.001) (Table 3). Correlations were similar in boys and girls.

The mean CEBQ subscale scores by BMI category with 95% CIs are shown in Figure 2A–B. Univariate analysis of variance for the SR-SE score showed a significant overall group difference (n = 549; P < 0.001), and polynomial contrasts showed a significant linear trend, with higher BMI associated with a lower score (P < 0.001). Neither scale was significantly associated with parental education or child age.

The partial eta squared was 0.033. Post hoc tests with Bonferroni’s corrections showed that the SR-SE scores for the low-normal-weight children (3.23 ± 0.63) were significantly higher than for the mid-normal weight and overweight categories (3.02 ± 0.60; 2.95 ± 0.58; both P <0.01), although the difference from the obese group (2.99 ± 0.61) did not reach significance because of the smaller number of children in that category (n = 39; P < 0.130). The EF scores also differed between groups (n = 550; P = 0.001), with a significant linear trend across weight categories such that higher EF scores were associated with higher BMI (P = 0.002). Controlling for other parent and child variables did not change the results. The partial eta squared was 0.031. Post hoc tests showed significant differences emerging for the low-normal-weight (3.26 ± 0.76) than for the mid-normal-weight (3.54 ± 0.83) and obese (3.73 ± 0.83) groups (all P < 0.01), although the difference with the overweight group (3.49 ± 0.08) did not reach significance (P < 0.123). Entering the SR-SE and EF scores simultaneously in the GLM showed that each scale made independent contributions to variance in BMI SD scores (SR-SE score: P = 0.029; EF score: P = 0.027).

**DISCUSSION**

These results show that appetitive traits do not show a simple pattern of abnormality in obese or overweight children, but rather a graded relation, such that lower satiety responsiveness and higher food cue responsiveness are associated with progressively higher adiposity. The linear pattern was stronger for waist circumference than for BMI, which could be partly attributable to the more equal size of the categories, but it may also reflect the fact that waist circumference is a more direct measure of adiposity. The linear pattern also appeared to be stronger for the older
children than for the younger children. The observed associations were largely unaffected by controlling for other known predictors such as parental adiposity and education. The same broad relations held across different recruitment methods (cohort study and community daycare sample), different indexes of adiposity (BMI SD score and waist circumference SD score), and parent-measured compared with researcher-measured data.

The 2 samples differed in the results of the post hoc tests comparing weight groups. In the younger children, the largest differences for both scales (and the only significant post hoc results) were between the low-normal-weight group and the others, whereas for the older children the post hoc tests for differences were all significant (at least for waist circumference) and the effect was more strongly linear, although the largest difference appeared to be between the overweight or obese children and the others. This could mean that high satiety responsiveness and low food cue responsiveness act to keep weight low in early childhood, whereas by school age low satiety responsiveness and high food cue responsiveness have promoted greater weight gain. However, interpretation of the differences in the patterning across the 2 ages should be cautious because the correlations, effect sizes, and statistics for the linear trend were not significantly different in the 2 age groups, and the relatively small size of the obese samples limits our power to detect significant differences.

The magnitude of the association between adiposity and appetite was modest, but this was expected given that appetite is only one of many influences on weight. Nonetheless, the effect sizes exceeded the usual genetic effect sizes for BMI (30), and they were comparable to those associated with other recognized influences on obesity such as television viewing (31, 32).

One important issue is whether the 2 appetitive traits we measured, satiety responsiveness and food cue responsiveness, are independent characteristics or effectively 2 sides of the same coin. They were significantly correlated in both samples ($r = -0.53$ and $r = -0.62$), but this does not show whether they are imperfect measures of the same phenomenon (one positively phrased, one negatively phrased) or related because each affects the other. If a child has weak satiety responsiveness, he or she will feel less sated after eating and therefore better able to respond to new food cues. Likewise, if the child is responsive to food cues, this could make it easier to override satiety signals. Thus, even if the 2 underlying traits had different origins, they would likely be correlated phenotypically. In the present study we examined whether they had independent associations with adiposity and found that the SR-SE and EF scores each contributed independently to predicting adiposity, tentatively supporting the idea that they are distinct but related traits. Future studies that address the biological basis of each characteristic, eg, in terms of genetic or endocrinologic correlates, will help to shed light on this issue.

The findings of this study are consistent with results from predominantly clinical populations that show obese-normal weight differences in constructs such as perceived hunger, disinhibited eating, and external eating (10, 11, 15). The findings add to recent literature that describes impaired intake regulation among overweight children with the use of behavioral methods of assessing appetite (12, 14, 17). They are also consistent with a previous study that showed moderate associations between satiety responsiveness, food cue responsiveness, and children’s food intake measured over 5 meals (20).

The findings have a number of implications, both theoretical and practical. The fact that satiety responsiveness and food cue responsiveness are related to adiposity as early as 3 y of age suggests that over time these traits could contribute significantly to lifetime risk of obesity. It is therefore possible that assessing these traits could identify persons with high-risk appetite characteristics who are at increased risk of becoming overweight in the future. For example, of those children with SR-SE scores in the lower half of the distribution, 18% were currently overweight but 82% were not. By adolescence more children will be overweight, and the CEBQ scores could constitute an independent indicator of risk. Population-based longitudinal data will be necessary to identify the cutoff for risk of later weight gain.

Significant strengths of the study were the large sample sizes, replication of the results in 2 different populations, and inclusion of waist circumference, which is strongly associated with cardiometabolic risk factors (33, 34). However, there were also limitations. Twins have lower birth weights than singletons and remain leaner in early childhood (35), and, as expected, obesity rates in our twin sample were low. However, the similarity of associations between adiposity and appetite in the 2 samples suggests that generalizability was not significantly compromised in the twins. In both samples, appetite assessments relied on parents’ reports, which may introduce additional measurement error. However, parents have privileged observational access to their children over a range of situations and may give more valid appraisals of their child’s appetite than are provided by performance in a one-off eating behavior task. We also cannot rule out the possibility of a social desirability basis in the parental reports,

![FIGURE 2. Mean Child Eating Behavior Questionnaire (CEBQ) Satiety Responsiveness and Slowness in Eating (SR-SE) (A) and Enjoyment of Food (EF) (B) scores by weight category in 3–5-y-olds. Univariate ANOVA (P < 0.001). Error bars represent 95% CIs.](image-url)
but elsewhere we have shown good correspondence with objective measures (24). Response rates were ≈50% for both studies, and, although reasonable for a postal survey, this nevertheless limits the generalizability of the results.

Another obvious limitation of the study is its cross-sectional nature, which precludes inferences about causation. For example, as early as 3 y of age, fatter children may have been exposed to a variety of environmental factors (eg, parental feeding style, high-fat diets, large portions) with the dual effect of increasing their weight and promoting appetitive responses. Longitudinal studies are essential to find out whether appetitive traits predict the development of obesity (eg, Stunkard et al (36)).

If the association between appetite and adiposity is causal, it raises the question of the origins and consequences of differences in appetite. We have elsewhere shown that these 2 appetitive traits are strongly heritable in the twin sample (S Carnell, CMA Haworth, R Plomin, and J Wardle, unpublished observations, 2008), which indicates a genetic cause but is not informative about the specific genetic mechanisms. Future studies could examine associations with particular genes, eg, the fat mass and obesity associated gene (37), to determine whether BMI-related genes in terms of consequences, the results raise the question of whether the link between a risky appetitive profile and becoming overweight can be broken. Persons respond well to personalized information on obesity risk (38), and mere awareness of appetitive traits are strongly heritable in the twin sample (S Carnell, CMA Haworth, R Plomin, and J Wardle, unpublished observations, 2008), which indicates a genetic cause but is not informative about the specific genetic mechanisms. Future studies could examine associations with particular genes, eg, the fat mass and obesity associated gene (37), to determine whether BMI-related genes in terms of consequences, the results raise the question of whether the link between a risky appetitive profile and becoming overweight can be broken. Persons respond well to personalized information on obesity risk (38), and mere awareness of appetitive traits may motivate individual action to control weight, although fatalistic attitudes must be guarded against. Another option is individualized training: one study in 3–4-y-olds


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