Eating rate is a heritable phenotype related to weight in children 1–3

Clare H Llewellyn, Cornelia HM van Jaarsveld, David Boniface, Susan Carnell, and Jane Wardle

ABSTRACT

Background: There is growing interest in the heritability of behavioral phenotypes related to adiposity. One potential candidate is the speed of eating, although existing evidence for an association with weight is mixed.

Objective: We aimed to assess the speed of eating in a sample of 10–12-y-old children to test the hypotheses that higher eating rate is related to greater adiposity and that eating rate is a heritable characteristic.

Design: Video data of 254 twin children eating a standard meal at home were used to record eating rate (bites/min) and changes in eating rate across the 4 quarters of the meal. Adiposity was indexed with body mass index SD scores relative to British 1990 norms; for some analyses, children were categorized into groups of overweight or obese and into 2 subgroups of normal-weight (lower normal-weight or higher normal-weight) for comparison of the eating rate within the normal range as well as between clinical and nonclinical groups. All analyses controlled for clustering in twin pairs. Heritability of eating rate was modeled by using standard twin methods.

Results: There was a significant linear association across the 3 weight groups for eating rate (P = 0.010), and regression analyses showed that eating rate increased by 0.18 bites/min for each 1-unit increase in body mass index SD score (P = 0.005). The heritability of eating rate was high (0.62; 95% CI: 0.45, 0.74). There was no association between weight group and a change (ie, deceleration) in eating rate over the mealtime.

Conclusion: Faster eating appears to be a heritable behavioral phenotype related to higher weight. Am J Clin Nutr 2008;88:1560–6.

INTRODUCTION

The idea that eating behaviors contribute to the development of obesity has a long history (1). Eating rate has attracted attention as an indicator of appetite avidity and satiety sensitivity (2); a higher eating rate is thought to indicate greater motivation to eat, and more rapid deceleration over the course of the meal is thought to indicate a stronger response to internal satiety signals. Experimental studies that manipulated eating rate in a controlled setting confirmed that faster eating is associated with greater food intake (3, 4).

The most widely used method of assessing eating rate is a microstructural analysis of ingestive patterns. Eating behavior is analyzed in terms of smaller structures (eg, bites/min) or stages (eg, mealtime quarters) to make within-meal and between-subject comparisons of eating speed and deceleration (2).

Comparisons between normal-weight and overweight or obese adults by using this methodology have produced mixed findings: some studies reported faster eating or less deceleration over the meal in overweight or obese groups (5, 6), but others found no difference (7, 8). Methodologic heterogeneity may account for some of these inconsistencies, but the widespread cultural belief that eating slowly aids weight loss could also lead overweight adults to eat more slowly during observed meals.

Pediatric studies, in which issues of self-presentation may be less salient, have generally supported the hypothesis that faster or nondecelerated eating characterizes overweight or obese groups (9, 10). Furthermore, eating rate in early infancy predicts adiposity at 1, 2, and 3 y of age (11), and it differentiates persons at higher and lower risk of obesity [according to parental body mass index (BMI; in kg/m²)] at 3 mo of age (12). None of the existing studies, however, related eating rate to adiposity as a quantitative trait, although, given that weight is normally distributed, it is likely that underlying behavioral risk factors also will be continuous.

As evidence accumulates for the heritability of a number of appetitive traits (13, 14), and as genetic influences on endocrinologic factors are identified (15), investigation of the heritability of eating rate is warranted. Twin studies can be used to assess the relative contribution of genes and environment by comparing the resemblance between monozygotic (MZ) twins, who are genetically identical, and that between dizygotic (DZ) twins, who share, on average, 50% of their segregating genes (16).

The present study tested the predictions that eating rate is related to adiposity across the full range of weight and that eating rate is a heritable characteristic. Video data for children eating a light meal in their own home were obtained in a sample of preadolescent twins.

SUBJECTS AND METHODS

Participants

Participants were drawn from the Twins Early Development Study, a population-based sample of >15 000 twin pairs born in

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England and Wales between 1994 and 1996 (17, 18). A sub-sample of same-sex twins (214 pairs; 63 MZ pairs and 64 DZ pairs) was selected in 1999 for detailed investigations of genetic and environmental influences on appetite and growth. Families (n = 214) were selected on the basis of parental BMI to maximize the range of familial risk of obesity. Approximately one-half of the families had 2 obese or overweight parents (mother’s reported BMI ≥ 28.5; father’s reported BMI ≥ 25), and one-half had 2 normal-weight parents (both parents’ reported BMI < 25). Higher- and lower-risk families were matched for socioeconomic status (SES) by using the father’s occupation and the geographic location where the family resided.

The families were followed up in 2006, at which time 173 families (346 children) were successfully recontacted, and they agreed to take part (81% of the original sample). A small number of families (n = 13) provided questionnaire data but declined the home visit. A total of 160 families (320 children) were visited at home by researchers; at that time, the twins were videotaped while eating standard meals. As a result of technical errors, video data were unavailable for 42 twins. To increase the homogeneity of the sample, data from only the white participants were included (93% of the sample). Four extreme statistical outliers on eating rate, total bites, and total minutes were excluded from all the analyses (≥ 4 SD score (SDS)). Each participant (and his or her co-twin) whose age- and sex-standardized scores for eating rate exceeded 2 SDS above or below the mean (n = 12) also were excluded from the heritability analyses (19). In the final sample, data from 254 children were used in the multilevel analyses of covariance (ANCOVA) and regression analyses, and data from 242 children were used in the heritability analyses.

Parental informed consent was obtained verbally on the telephone before the home visit and in writing at the beginning of the home visit, before the test meal was initiated. The University College London Committee for the Ethics of non-National Health Service Human Research granted ethical approval for the study.

Measures

Eating rate and deceleration

The twins were filmed while eating a meal at home, and eating behavior in the video was coded by independent observers to quantify eating rate and deceleration. Children were given a meal of sandwich and fruit, and the choice of sandwich was based on information on each child’s food preferences, food allergies, or special dietary requirements obtained from the child’s mother a few days before the home visit. Mothers were asked not to feed the children immediately before the task, and the task took place either before lunch, between 1100 and 1330 (n = 67), or immediately after school, between 1500 and 1800 (n = 187).

During the home visit, the test meal was set up in the twins’ usual eating environment. A small video camera was placed in a fixed position in the room where the meal was set up, so that both children could be observed, while the camera remained unobtrusive. This set-up ensured that the researchers did not need to be present to film the children while they were eating. Twenty-four sandwich quarters were presented on 2 plates and placed on the table along with 2 previously prepared mixed and chopped fruit salads. This was more food than the children could eat, because in all cases sandwiches were left at the end of the meal. Sandwiches and fruit were chosen as conventional foods for a light meal of medium palatability that could be tailored to suit children’s preferences. Children were told they could eat as much or as little as they wished for as long as they wanted and that they could eat from either plate. They were asked to tell the researcher when they had finished. They were told that the video camera had been set up to remind the researchers of what had happened during the home visit and that they should try to ignore the camera. The twins were left together for the meal without researchers or other family members present. In no cases were children watching television, but in most cases they talked to each other during the meal.

Video data analysis

Five researchers viewed 2–3 videos independently to devise codes for various eating behaviors. Guidelines included codes for tallying fruit bites and sandwich bites, typical bite size, and the time the child spent eating. Fruit bites were counted when the child bit half a piece of fruit or more but were not counted if he or she ate less than half a piece of fruit or merely sucked the fruit. A sandwich bite was counted if a child was clearly seen to take a bite from a sandwich quarter. A bite was discounted if the child subsequently spat the piece of food out. A series of tiny rapid nibbles (eg, taking several nibbles to eat the corner of a sandwich quarter) was counted as one bite, but an isolated nibble was not counted. Fruit and sandwich bites were tallied for every minute the child spent eating.

Because this was an ecological study, children occasionally moved out of camera view during the mealtime. Minutes during which the child was out of camera view were discounted so that the minute before the child’s movement out of camera view and the minute that the child came back into view were counted as consecutive. Most of the children (n = 212) were in full camera view for 100% of the mealtime; a small number of the children (n = 42) were out of camera view for part of the time (range: 4–30% of the time), but the percentage of time that children spent out of camera view did not influence the results of the analyses.

Each child’s typical “bite size” was based on an estimate of the number of bites taken to eat a sandwich quarter, and the size was categorized as small (≥7 bites/sandwich), average (4–6 bites/sandwich), or large (1–3 bites/sandwich). Sandwich bite size was used to estimate typical bite size for both sandwiches and fruit; this value offered a more standardized quantification of bite size than did fruit bite size, because all sandwich quarters were the same size, but the size of the pieces of fruit varied with the type of fruit. All coding for the eating behaviors was conducted by one researcher.

Each child was given a score representing his or her average bite size during the meal. Total number of bites was calculated by tallying the total number of fruit and sandwich bites taken over the entire mealtime. The percentages of total bites taken from sandwiches and from fruit were calculated to ensure that this variable did not differ across the 3 weight groups. The total time each child spent eating was recorded. Eating rate was calculated by dividing the total number of bites by the total number of minutes spent eating to compute bites/min.

Eating rate deceleration was calculated by dividing the total time spent eating by each child into quarters to create a standardized mealtime structure that both accounted for intersubject variation in total time spent eating and allowed for repeated-measures and between-participant comparisons. The mean
eating rate for each mealtime quarter was then calculated for each child.

**Anthropometric measures**

During the home visit in 2006, trained researchers measured the twins’ heights and weights. Height was measured to the nearest 1.0 mm by using a portable stadiometer (Leicester Portable Height Measure, Birmingham, United Kingdom), and weight was measured to the nearest 0.1 kg by using a digital scale (Tanita Corporation of America Inc, Arlington Heights, IL). BMI centiles relative to 1990 British norms (20) were calculated. Children with a BMI SDS of ≥91st centile were classified as overweight or obese, in line with the 1990 British normative data. The overweight and obese groups were combined because of the small number of obese children in the sample (n = 29). To determine whether there was an association between eating rate and adiposity across the full weight range, normal-weight children were divided into those with a BMI SDS of ≤50th centile (classified as lower normal-weight) and those with a BMI SDS of >50th and <91st centile (classified as higher normal-weight).

**Sociodemographic data**

Parents reported the twins’ ethnic origin from one of 4 possible groups: Asian, black, mixed race, or white. Family SES was indexed from the father’s occupation by using the Registrar General’s Classification of Occupations (21). Children’s ages were calculated for the day of the home visit. Information about paternal height and weight; twins’ zygosity, sex, and ethnicity; and family SES was obtained from data collected in 1999. The mothers’ heights and weights were measured by researchers in 2006. DNA analysis was used to confirm the zygosity of ≈5% of pairs for whom it was uncertain.

**Statistical analysis**

A multilevel mixed ANCOVA was used to assess differences between the 3 weight groups in eating rate and eating deceleration over the meal after control for age, sex, the time of day when the meal was consumed, and clustering of the twins in families. A polynomial contrast test estimated the linearity of eating rate across the 3 weight groups after adjustment for age, sex, the time of day when the meal was consumed, and family clustering. Multilevel linear regression was used to assess the relation between eating rate and BMI SDS after adjustment for age, sex, the time of day when the meal was consumed, and family clustering. Multilevel between-group ANCOVAs were used to assess differences between the 3 weight groups in total bites taken during the meal, the percentage of bites taken from sandwiches (rather than fruit), and the typical bite size, are controlled for in the analyses (Table 2).

**RESULTS**

**Sample characteristics**

No significant differences were found between the weight groups for father’s occupation or child sex or zygosity, but there was a significant (although small) difference by age (Table 1). By design, all adiposity measures differed significantly across the 3 groups (Table 1).

There were no group differences for typical bite size [chi-square test (df = 2; n = 251) = 4.828; P = 0.143] or bite percentage scores (P = 0.471), so neither of these variables was controlled for in the analyses (Table 2).

**Eating rate, eating deceleration, and adiposity**

The adjusted means and SEs for eating rate, as well as the total number of bites taken during the meal, the total number of minutes taken to complete the meal, the percentage of bites taken from sandwiches (rather than fruit), and the typical bite size, are shown in Table 2. The overweight or obese group ate the fastest, at 4.3 bites/min; the higher-normal-weight group ate on average 4.1 bites/min; and the lower-normal-weight group ate the slowest, at 3.8 bites/min. Multilevel ANCOVA confirmed a significant difference between groups for eating rate (P = 0.034), and the polynomial contrast test showed a significant positive linear effect (P = 0.010), which indicated that the eating rate increased with higher weight.

Scores for total minutes and total bites indicated that the lower normal-weight group ate significantly less than did the overweight or obese group and that the lower normal-weight group spent significantly more time eating than did the higher-normal weight group. The higher normal-weight group spent significantly less time eating than did the lower normal-weight group or the overweight or obese group. The overweight or obese group ate significantly more bites than did either the lower normal-weight or the higher-normal weight group, and the overweight or obese group spent significantly more time eating than did the higher-normal weight group. There was a significant positive association between total bites and eating speed (n = 254; r = 0.502, P < 0.001, 2-tailed), which indicated that a faster eating speed was associated with a greater consumption of food.

The adjusted means and SEs for eating rate over the 4 quarters of the mealtime for the 3 weight groups, including the deceleration patterns, are shown in Figure 1. There was a strong trend of eating deceleration over the meal for all weight groups; the fastest eating rate was seen in quarter 1 and the slowest in quarter 4. The multilevel mixed ANCOVA showed a significant main
TABLE 1
Anthropometric and demographic characteristics of the study sample

<table>
<thead>
<tr>
<th></th>
<th>Lower normal-weight (n = 99)</th>
<th>Higher normal-weight (n = 87)</th>
<th>Overweight or obese (n = 68)</th>
<th>Total sample (n = 254)</th>
<th>P for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father’s occupation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.137</td>
</tr>
<tr>
<td>Professional, managerial, or technical [n (%)]</td>
<td>42 (56.0)</td>
<td>39 (50.0)</td>
<td>23 (36.5)</td>
<td>104 (48.1)</td>
<td></td>
</tr>
<tr>
<td>Skilled manual or nonmanual [n (%)]</td>
<td>23 (30.7)</td>
<td>32 (41.0)</td>
<td>32 (50.8)</td>
<td>87 (40.3)</td>
<td></td>
</tr>
<tr>
<td>Unskilled or partly skilled [n (%)]</td>
<td>10 (13.3)</td>
<td>7 (9.0)</td>
<td>8 (12.6)</td>
<td>25 (11.6)</td>
<td></td>
</tr>
<tr>
<td>Child sex [n (%)]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.157</td>
</tr>
<tr>
<td>Girl</td>
<td>66 (66.7)</td>
<td>46 (52.9)</td>
<td>40 (58.8)</td>
<td>152 (59.8)</td>
<td></td>
</tr>
<tr>
<td>Boy</td>
<td>33 (33.3)</td>
<td>41 (47.1)</td>
<td>28 (41.2)</td>
<td>102 (40.2)</td>
<td></td>
</tr>
<tr>
<td>Zygosity [n (%)]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.930</td>
</tr>
<tr>
<td>Monozygotic</td>
<td>48 (48.5)</td>
<td>43 (49.4)</td>
<td>35 (51.5)</td>
<td>126 (49.6)</td>
<td></td>
</tr>
<tr>
<td>Dizygotic</td>
<td>51 (51.5)</td>
<td>44 (50.6)</td>
<td>33 (48.5)</td>
<td>128 (50.4)</td>
<td></td>
</tr>
<tr>
<td>Age (y) [n]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.036</td>
</tr>
<tr>
<td>11.2 ± 0.52</td>
<td>11.3 ± 0.53</td>
<td>11.0 ± 0.58</td>
<td>11.2 ± 0.55</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm) [n]</td>
<td>143.5 ± 7.86</td>
<td>147.3 ± 6.43</td>
<td>149.7 ± 7.18</td>
<td>146.4 ± 7.62</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight (kg) [n]</td>
<td>33.6 ± 3.43</td>
<td>41.4 ± 4.34</td>
<td>54.3 ± 10.05</td>
<td>41.8 ± 10.44</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight (kg/m²) [n]</td>
<td>16.3 ± 0.96</td>
<td>19.1 ± 1.05</td>
<td>24.1 ± 3.37</td>
<td>19.3 ± 3.69</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI centile [n]</td>
<td>29.5 ± 13.88</td>
<td>74.3 ± 11.63</td>
<td>96.7 ± 2.75</td>
<td>62.9 ± 30.15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI SDS [n]</td>
<td>-0.6 ± 0.53</td>
<td>0.7 ± 0.38</td>
<td>2.1 ± 0.58</td>
<td>0.6 ± 1.19</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

1 Weight groups were defined by age- and sex-specific BMI SD score (SDS) centile: lower normal-weight: <50th centile; higher normal-weight: >50th and <91st centile; and overweight or obese: ≥91st centile.

2 Group differences were assessed by using a chi-square test.

3 Group differences were assessed by using ANOVA.

4 Group differences were assessed by using ANOVA.

5 Mean ± SD (all such values).

Effect for meal quarter (P < 0.001), which indicated that, for all weight groups, eating rate decreased significantly over the meal. The repeated-measures polynomial contrast test for linearity was significant (P < 0.001), which indicated a linear deceleration in eating rate. There was no significant interaction with weight group (P = 0.632), which indicated that the change in eating rate over time was similar for all 3 weight groups.

Multilevel regression analyses with BMI SDS entered as a continuous predictor variable was used to support the findings from the group comparisons. There was no significant interaction between BMI SDS and mealtime quarter for eating rates, which indicated no differences in deceleration. BMI SDS, age, time of visit, and mealtime quarter all showed significant relations with eating rate (P = 0.005, P = 0.009, P = 0.006, and P < 0.001, respectively). Specifically, for each 1-unit increase in BMI SDS, the eating rate increased by 0.18 (95% CI: 0.06, 0.31) bites/min, the older children (≥11.2 y old) ate 0.45 (95% CI: 0.12, 0.79) bites/min faster than did the younger children (<11.2 y old), those who consumed the meal at midday ate 0.55 (95% CI: 0.16, 0.94) bites/min faster than did those who consumed the meal in the afternoon, and eating rate decreased by 0.62 (95% CI: 0.55, 0.69) bites/min with each mealtime quarter.

Heritability of eating rate

Intraclass correlations for eating rate were higher for MZ twin pairs (0.59; 95% CI: 0.40, 0.73) than for DZ twin pairs (0.30; 95% CI: 0.05, 0.52), which is consistent with a strong genetic contribution. The results of model-fitting analyses are shown in Table 3: high heritability (0.62; 95% CI: 0.45, 0.74) for eating rate, with 95% CIs not spanning zero, which indicates that the estimate is

TABLE 2
Adjusted values for eating rate, total bites, total minutes, percentage of bites from sandwiches, and typical bite sizes for the 3 weight groups [n]

<table>
<thead>
<tr>
<th></th>
<th>Lower normal-weight (n = 99)</th>
<th>Higher normal-weight (n = 87)</th>
<th>Overweight or obese (n = 68)</th>
<th>P for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating rate (bites/min) [n]</td>
<td>3.8 ± 0.14 [a]</td>
<td>4.1 ± 0.14 [a, b]</td>
<td>4.3 ± 0.16 [b]</td>
<td>0.034</td>
</tr>
<tr>
<td>Total bites (n) [n]</td>
<td>49.0 ± 2.35 [a]</td>
<td>46.8 ± 2.31 [a]</td>
<td>56.3 ± 2.90 [b]</td>
<td>0.009</td>
</tr>
<tr>
<td>Total minutes (n) [n]</td>
<td>13.6 ± 0.66 [a]</td>
<td>11.6 ± 0.52 [b]</td>
<td>13.3 ± 0.79 [a]</td>
<td>0.005</td>
</tr>
<tr>
<td>Bites from sandwiches (%) [n]</td>
<td>71.8 (2.12)</td>
<td>73.9 (1.99)</td>
<td>75.6 (2.13)</td>
<td>0.361</td>
</tr>
<tr>
<td>Typical bite size [n (n)]/n</td>
<td>37 (59.4)</td>
<td>38 (43.7)</td>
<td>38 (55.9)</td>
<td>0.143</td>
</tr>
</tbody>
</table>

1 Weight groups were defined by age- and sex-specific BMI SD score (SDS) centile: lower normal-weight: <50th centile; higher normal-weight: >50th and <91st centile; and overweight or obese: ≥91st centile. Means sharing the same superscript letter do not differ significantly after adjustment for multiple comparisons, P > 0.05 (Bonferroni).

2 Group differences were assessed by using multilevel ANCOVAs after control for age, sex, the time of day when the meal was consumed, and clustering of the twins in families.

3 Group differences were assessed by using a chi-square test.

4 Group differences were assessed by using ANOVA.

5 n = 251.
significant. The best-fitting and most parsimonious model for eating rate was the AE submodel, because dropping the shared environment parameter resulted in no change in the chi-square test, which suggested that the fit of the AE model was no worse than that of the full ACE model.

**DISCUSSION**

A significant association between eating rate and adiposity was observed in the present study, which supports previous research that found that overweight infants or children eat more rapidly than do their normal-weight counterparts (9–12). The present study went beyond previous research in assessing eating rate across the full weight spectrum and also showed that thinner normal-weight children eat more slowly than do fatter normal-weight children; thus there is a quantitative association between eating rate and adiposity and not only an abnormality of eating rate among overweight or obese children.

Eating deceleration over the mealtime, however, did not differ between weight groups, a finding that contrasts with findings from previous studies in children (10, 23) but corresponds with findings from some studies in adults (8, 24). Methodologic heterogeneity between studies may account for the different findings. Previous studies were conducted in laboratories, whereas the present study was conducted at the children’s homes, and it is possible that overweight children respond differently in the 2 conditions. Furthermore, the sandwiches used in the present study may have had a lower palatability than the food used in some studies, a factor that is found to be predictive of nondecelerated eating in adults (2).

The genetic analyses in the present study provided new evidence that eating rate has a substantial heritable component. Evidence is accumulating for the heritability of a range of appetitive traits related to adiposity, including food preferences (14), food neophobia (25), palatability (26), and disinhibited eating (27, 28), and there is growing evidence for a genetic influence on endocrinologic mechanisms related to hunger and satiety (15). The heritability estimate of 0.62 for eating rate that the present study found is at the top of the range of the heritability estimates reported for other appetitive traits. In comparison, estimates between 0.18 and 0.40 have been reported for disinhibited eating (27, 28), and estimates between 0.24 and 0.33 have been reported for total caloric intake (29) among adults. Estimates between 0.22 and 0.45 for food preferences (14) and between 0.76 and 0.79 for food neophobia (25) have been reported among children. These findings suggest that genetically determined differences in eating rate could be steps on the pathway from genes to higher body weight, but multivariate genetic studies are needed to discover whether the same genes that influence weight also influence eating rate.

The modeling did not identify any significant effect of shared environment, but that lack of effect could be due to a lack of power in the relatively small sample size. In contrast, the more pronounced nonshared environment effect (and measurement

**TABLE 3**

Model fit and parameter estimates (and 95% CIs) for heritability of eating rate

| Model | Additive genetic effect (a²) | Shared environment effect (c²) | Nonshared environment effect (e²) | −2LL | df | ΔAIC | Δχ² | P
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>ACE</td>
<td>0.62 (0.18, 0.74)</td>
<td>0.00 (0.00, 0.35)</td>
<td>0.38 (0.26, 0.56)</td>
<td>648.943</td>
<td>237</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CE</td>
<td>—</td>
<td>0.43 (0.28, 0.57)</td>
<td>0.57 (0.43, 0.72)</td>
<td>655.750</td>
<td>238</td>
<td>4.807</td>
<td>6.807</td>
<td>0.009</td>
</tr>
<tr>
<td>AE</td>
<td>0.62 (0.45, 0.74)</td>
<td>—</td>
<td>0.38 (0.26, 0.55)</td>
<td>648.943</td>
<td>238</td>
<td>-2.000</td>
<td>0.001</td>
<td>—</td>
</tr>
<tr>
<td>E</td>
<td>—</td>
<td>1.00 (1.00, 1.00)</td>
<td>—</td>
<td>680.691</td>
<td>239</td>
<td>27.747</td>
<td>31.747</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

1 n = 242. LL, log likelihood; Δ, change; AIC, Akaike Information Criterion. Standard ACE model–fitting analyses were used to estimate additive genetic (a²), shared environment (c²), and nonshared environment (e²) effects.

2 CE, AE, and E submodels are nested within the ACE model. The ACE model dissects the phenotypic variance into a², c², and e²; the CE model drops the a² parameter and assesses the variance explained by the c² and e² parameters only; the AE model drops the c² parameter and assesses the variance explained by the a² and e² parameters only; the E model drops both the a² and c² parameters and assesses the variance explained by the e² parameter only. A better-fitting submodel shows a change in the model with less variance explained by the a² parameter only. A better-fitting submodel shows a change in χ² that does not represent a significant worsening of fit as designated by the P value (for a change in df of 1, the statistically significant change in χ² is 3.84).

3 Includes measurement error.

4 Two fit indexes are reported from the structural equation modeling analyses: P value based on the likelihood ratio χ² test and on AIC (22).

5 Probability was incalculable because of the lack of change in fit statistics.
error) of 0.38 may indicate that different treatment from the parents influences eating rate over the developmental trajectory.

Although the cross-sectional design of the present study precluded conclusions about causal direction, the results, when evaluated in combination with evidence from longitudinal studies in infants (11, 12), are consistent with the idea that rapid eating may be a common inherited phenotype that plays a role in the development of adiposity. It has been suggested that eating rate and eating deceleration represent distinct and opposing forces of facilitatory and inhibitory neural processes that mediate motivated behavior: i.e., eating rate represents facilitatory processes such as hunger, and deceleration represents inhibitory processes such as satiety (30, 31). In the present study, however, we found no evidence of differential deceleration.

A burgeoning literature is starting to document the genetic and molecular bases of human eating behavior (15). Of particular interest is the recent finding that the FTO gene, which is associated with an average weight difference of 1.5 kg per risk allele in many populations (32), is highly expressed in the human hypothalamus (33), which suggests that it plays a role in the control of hunger. Associations between FTO genotype and satiety sensitivity were already reported (34), and there may also be links with eating rate. Binge eating—characterized by excessive hunger, food-seeking behavior, and hyperphagia—is a major phenotypic expression of a melanocortin 4 receptor gene mutation (35), and eating rate also may reflect a common variant of this genotype.

There are practical implications of these findings. If eating rate can be modified, and if it results in the consumption of less food, then early promotion of slower eating for all children could lower the population mean weight and help to control current obesity trends (36). Whereas an estimated environmental effect of 0.38 suggests room for environmental modification, intervention studies that have specifically targeted the eating rate among children are rare. A simple intervention by Epstein et al (37) to encourage children to put their utensils down between bites succeeded in both slowing the eating rate and reducing the amount of food consumed over a 6-mo period. More research manipulating eating rates and assessing food consumption alongside objective indicators of hunger and satiety (such as appetite hormones) would be useful.

This study has a number of strengths. As far as we are aware, it is the first assessment of the heritability of eating rate among children. We assessed the association between eating rate and the full range of adiposity, rather than just comparing obese and normal-weight subjects, and this approach allowed the nature of the relation to be determined. In addition, the study used a naturalistic design that permitted the influence of ecologically valid factors, such as conversation between 2 siblings, on the eating rate.

There were limitations to the study design. Cross-sectional designs preclude conclusions about causation, and, without experimental study designs, we cannot exclude the possibility that higher adiposity drove faster eating. In terms of estimating heritability by using the twin design, it has been argued that shared environments are more similar for MZ twins (whether uterine or familial) than for DZ twins, which inflates estimates of heritability (38); however, research into other eating behaviors has supported the assumption of equal environments (39). Others have argued that the experience of growing up as a twin is too different from that of growing up as a singleton to allow generalizations from one condition to the other, although personality research has shown few differences between twins and singletons (40).

Filming the children was chosen as a method of observing eating that was superior to the use of a researcher to record behavior, but the presence of the camera may still have modified behavior, as indicated by some instances of “showing off.” Furthermore, eating rate may differ as a function of the food type (41), and therefore the findings should be replicated with foods other than sandwiches. Finally, the study sample was racially-ethnically homogenous (white), which limits the generalization of these findings to other races-ethnicities.

These findings indicate that differences in motivation to eat, characterized by a faster eating rate, are quantitatively associated with higher adiposity—the thinnest children eat the slowest, and the fattest eat the fastest. The high heritability of eating rate shown in this twin sample is consistent with the idea that faster eating forms part of a genetically determined cluster of behavioral risk factors for weight gain that may be amenable to environmental intervention.

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