Genetic and environmental influences on eating behavior: the Swedish Young Male Twins Study

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ABSTRACT

Background: Eating behavior may be implicated in the increasing prevalence of overweight and obesity, presumably in relation to easy access to energy-dense and highly palatable foods.

Objective: The aim of the present study was to disentangle genetic and environmental influences on eating behavior in a population-based cohort of male twins.

Design: The study included 326 dizygotic and 456 monozygotic male twin pairs aged 23–29 y from Sweden. The revised 21-item version of the Three-Factor Eating Questionnaire (TFEQ-R21) was used to assess eating behavior. This validated instrument consists of 3 dimensions: cognitive restraint, emotional eating, and uncontrolled eating. Structural equation modeling was used to estimate the heritability of eating behavior.

Results: Cognitive restraint was the only TFEQ-R21 scale that significantly correlated with BMI ($r = 0.39, P < 0.0001$). The best-fitted models gave a heritability of 59% (95% CI: 52%, 66%) for cognitive restraint, 60% (95% CI: 52%, 67%) for emotional eating, and 45% (95% CI: 36%, 53%) for uncontrolled eating.

Conclusions: These results show the great importance of genetic factors in the eating behavior of a large, unselected population of young adult male twins. Nonshared environmental factors were also important, whereas shared environmental factors did not contribute to eating behavior. Am J Clin Nutr 2005;81:564–9.

KEY WORDS Eating behavior, twins, males, genetics, heritability, structural equation models

INTRODUCTION

The global increase in the occurrence of overweight and obesity during the past few decades is probably explained by environmental factors and interactions between genes and environments. Previous twin and adoption studies reported heritabilities of body mass index (BMI) and obesity in the range of 30–80% (1, 2). Differences in genetic susceptibility to obesity may explain why some individuals develop obesity while others do not (3).

Hereditry seems to affect nearly all aspects of food intake regulation (4, 5). A study of middle-aged and older US male and female twins showed that ≥48% of the variability in healthy eating patterns could be attributed to genetic factors (6). Interestingly, genetic effects were more prominent in men than in women. Twin research from the United States has shown that genetics has important effects on meal size and meal frequency (7, 8). Two recent US studies assessed eating behavior by using the Three-Factor Eating Questionnaire (TFEQ) (9), which is a widely used self-assessment instrument that measures 3 domains: cognitive restraint, hunger, and disinhibition. A family study, which included 624 Amish men and women, estimated the heritability of restraint, disinhibition, and hunger to be 28%, 40%, and 23%, respectively (10). A second study, which included 210 pairs of female twins aged 25–64 y, found that additive genetic factors had a significant effect on disinhibition, whereas cognitive restraint and hunger were influenced mainly by nonshared environmental factors (11).

The Amish family study by Steinele et al (10) reported higher disinhibition and restraint scores in obese persons than in normal-weight subjects. Another study of 293 US women also found a positive association between disinhibition and BMI that was modified by an inverse association between restraint and BMI (12). In that study, symptoms of an eating disturbance were more strongly associated with disinhibition than with restraint; high disinhibition scores seem to predict overeating (13). The construct validity of the TFEQ was investigated in >4300 middle-aged obese men and women from Sweden (14). In line with findings by Mazzeo et al (15), the original TFEQ factor structure could not be replicated. A shorter, revised, 18-item instrument was developed that covered 3 domains: cognitive restraint, uncontrolled eating, and emotional eating. A further refined 21-item version (TFEQ-R21) that includes 3 additional items on emotional eating was used in the present study.

Investigation of the genetic and environmental influences on eating behavior is important for extending our understanding of food intake regulation, energy balance, and development of overweight and obesity. Our aim was to evaluate the role of genetic and environmental influences on eating behavior in a Swedish cohort of young adult male twins.
SUBJECTS AND METHODS

Subjects and determination of zygosity

The Swedish Young Male Twins Register was created in 1997 by identifying all male twins in the Swedish Medical Birth Register born from 1973 to 1979. In 1998, all twins in the register who were living in Sweden that year (n = 3566) were mailed a questionnaire about body size and physical activity. A total of 79% of the twins returned the questionnaire (16). Both twins in a pair were excluded from the cohort if one of the twins had died (n = 12) or refused to participate (n = 40). Six twins were excluded because of a severe handicap and 41 twins had no known address and could therefore not be contacted.

In 2002 all twins in the register who were living in Sweden that year (n = 3484) were mailed a follow-up questionnaire on body size, physical activity, eating behavior, and frequency of contact between twin brothers. At the time of the survey, the twins were aged 23–29 y (±: 25.8 y). Responses were obtained from 2173 (62%) of the twins. The Ethics Committee at the Karolinska Institute, Stockholm, approved this study.

Data on zygosity were collected in 1998 and 2002 by using 2 classic questions concerning how similar the twins were in childhood and how often teachers had difficulty distinguishing the brothers. These validated questions have been widely used in previous Swedish twin studies (17, 18). Twin pairs who answered both “as like as two peas in a pod” to the first question and “always or nearly always” to the second question were categorized as monozygotic (MZ). Twin pairs who answered both “not more than siblings in general” to the first question and “seldom” or “never or almost never” to the second question were classified as dizygotic (DZ). In 73% of the complete twin pairs, zygosity could be determined as either DZ (302 pairs) or MZ (388 pairs) on the basis of these questions. The remaining 260 pairs (including those with differing answers in 1998 and 2002) were classified as being of undetermined zygosity.

A DNA test was performed to determine zygosity in consenting twin pairs with undetermined zygosity. The twins rinsed their mouths with Scope mouthwash solution (Procter & Gamble, Cincinnati, OH). DNA was purified from buccal cells as described elsewhere (19). Zygosity was determined by using 16 highly polymorphic microsatellite markers from Webserst 6 (Uppsala Genome Center, Uppsala, Sweden). A twin pair was considered MZ if concordant for all 16 markers and was considered DZ if they differed in more than one marker. No twin pair differed in one marker. The analysis software used was GENESCAN version 3.5.1 and GENOTyper version 3.7 NT (Applied Biosystems, Foster City, CA). All 114 pairs who completed the DNA test were classified as either MZ or DZ.

In the present study we excluded individuals who 1) did not have complete data on the variables used, 2) had undetermined zygosity, or 3) belonged to an incomplete pair of twins. Altogether, 782 twin pairs, 326 DZ pairs and 456 MZ pairs, with complete information on zygosity, BMI, and eating behavior composed the study population.

Measures

Body size

BMI was calculated by dividing weight (in kg) by height squared (in meters). The cutoff for overweight was 25 and that for obesity was 30 according to World Health Organization criteria. The twins were instructed to measure their waist circumference midway between the lower border of the chest and the anterior iliac crest by using a tape provided with the questionnaire. An illustration helped them to perform this task properly.

Eating behavior

The TFEQ is a 51-item self-assessment instrument of eating behavior that measures cognitive restraint, disinhibition, and hunger (9). The questionnaire has been widely used in both overweight and normal-weight subjects (20). However, several psychometric studies have failed to replicate the factor structure of the TFEQ, and the construct validity of the measure has been questioned (14, 15).

The original TFEQ was translated, adapted, and validated for Swedish conditions (21) and, later, a shortened, revised 18-item version was constructed (14, 22). Further development of the construct validity of the short form resulted in the 21-item version (TFEQ-R21) used in the present study. The TFEQ-R21 covers 3 eating behavior domains: the cognitive restraint scale (6 items) assesses control over food intake to influence body weight and body shape; the emotional eating scale (6 items) measures the propensity to overeat in relation to negative mood states, eg, when feeling lonely, anxious, or depressed; and, the uncontrolled eating scale (9 items) assesses the tendency to lose control over eating when feeling hungry or when exposed to external stimuli. The dichotomized response format used in the original TFEQ was changed to a 4-point response scale in the TFEQ-R21. Higher scores indicate greater cognitive restraint, uncontrolled eating, or emotional eating.

Correlations between the original TFEQ scales and the revised short-form scales have been presented (14). In that Swedish study, a strong association was observed between the original cognitive restraint scale and the revised 6-item restraint scale (r = 0.89). The revised 9-item uncontrolled eating scale, which combines items from the hunger (6 items) and disinhibition (3 items) scales of the original TFEQ, was strongly associated with hunger (r = 0.89) and somewhat less strongly associated with disinhibition (r = 0.77). The new emotional eating scale, which was constructed from items included in the disinhibition scale, was most strongly associated with disinhibition (r = 0.69) and was moderately associated with the hunger scale (r = 0.41).

Equal environment assumption and contact frequency

Because MZ twins share all their genes, whereas DZ twins, on average, share one-half their genes, the difference between MZ and DZ within-pair correlations on the 3 TFEQ-R21 scales is a measure of the genetic contribution to these traits. However, this may not necessarily be true if environmental factors that affect eating behavior are more strongly correlated in MZ than in DZ twins (23). If so, the equal environment assumption (EEA) might be invalid and the heritability of eating behavior might then be overestimated. To explore this assumption and the robustness of our results, we stratified our statistical analyses (models) by contact frequency.

The twins’ contact frequency was assessed with the question, “How often do you usually get together with or speak to your twin brother?”; 9 response choices were given that ranged from “every day” to “once a year.” Twin pairs who answered daily or several times a week were classified as having frequent contact, and pairs who answered once a week or less were classified as...
having less frequent contact. The remaining pairs were not classified with respect to contact frequency. Information on contact frequency was available for 84% of the twin pairs. In 56% of the DZ pairs and 81% of the MZ pairs, both brothers reported that they had frequent contacts with each other.

When testing the EEA, Klump et al. (24) did not find support for the hypothesis that within-pair resemblance in eating behavior is affected differently in MZ and DZ pairs by degree of physical resemblance or other similarities. Evans et al. (25) found evidence strongly suggesting that twin studies do not violate the EEA.

Statistical analysis

Because scores on the 3 eating behavior scales were measured on ordinal scales, we used Spearman rank correlations for descriptive purposes. Structural equation modeling (SEM) (23) was used in univariate heritability analyses of the 3 eating behavioral traits. The total phenotypic variation can be decomposed into 4 latent (unmeasured) variance components: A, additive genetic effects; D, nonadditive (dominance) genetic effects; C, environmental effects shared by twin brothers; and E, environmentally effects not shared by twin brothers. As mentioned above, MZ twins share all their genes whereas DZ twins, on average, share one-half of their additive genes and one-fourth of their dominant (nonadditive) genes. Heritability in the broad sense is defined as the proportion of the total phenotypic variation that is due to the genetic components A, D, or both (26). Under this genetic model, SEM can estimate the variance components from which heritability estimates can be derived.

The 3 TFEQ-21 scales are highly skewed and truncated in the tails of the distributions and could not be considered to be normally distributed. We therefore categorized the variables and handled them as ordinal and used liability-threshold models in our analyses. A liability is a continuously distributed variable underlying the observed ordinal variable, and individuals lying between certain thresholds on this liability distribution are assumed to have the same status of trait. To get an unbiased heritability estimate of the liability, it is assumed that the observed categorized outcomes reflect such an underlying bivariate normally distributed liability of the trait (27). We found that dividing the scores into quartiles gave acceptable agreement with this assumption for all 3 scales. The assumption of an underlying bivariate normal distribution was tested by using the root mean square error of approximation (RMSEA) measure available in PRELIS/LISREL 8.5 (28). These analyses gave RMSEA = 0.017 (P = 0.86) for emotional eating, and RMSEA = 0.085 (P = 0.000) for uncontrolled eating. Therefore, the assumption of underlying normal distributions could not be rejected at the 0.05 level. To estimate the variance components and CIs, SEM was fitted directly to the contingency (frequency) tables, with categories according to the quartiles, by maximum likelihood with the software Mx (27, 29). Data preparation for contingency tables and other descriptive statistics was done in SAS (30). The SEM models ADE, ACE, AE, DE, CE, and E were fitted and then compared by Akaike’s Information Criteria (AIC) (31) to find the model that represented the best compromise between goodness-of-fit and parsimony. We also conducted analyses with different numbers of thresholds and even analyzed the variables on eating behavior as continuous. These analyses gave similar estimates and we therefore present only the results from the analyses based on quartiles.

The 1564 subjects (782 twin pairs) included in the analyses were compared with the 1920 subjects not included in the analysis (because of nonparticipation in the survey in 2002, missing data, or incomplete pair) by using birth date, BMI from conscription examination at age 18 y, and educational level from the 1998 survey. BMI was available for 91% of the twins included in the analyses and for 90% of the twins not included. Level of education was available for 91% of the twins included in the analyses and for 66% of the twins not included. Comparisons of mean differences for date of birth, BMI, and proportion of individuals with high versus low education, as well as comparisons between MZ and DZ, were done with t tests derived by using generalized mixed models adjusted for within-twin-pair correlation as well as heterogeneity between zygosity groups.

RESULTS

Twins not included in the analysis were on average 1.6 mo younger than the study population (P = 0.043); however, no significant differences were found with respect to BMI (difference = -0.03; P = 0.80) at the conscription examination at age 18 y. The study population included a larger proportion of highly educated subjects (educational level higher than upper secondary school) than among the twins not included in the analysis (40% and 34.9%, respectively; P = 0.020).

Means of body size and shape for the MZ and DZ twins are shown in Table 1. Means of body weight and waist circumference were slightly lower among the MZ than among the DZ twins. According to World Health Organization criteria, 27.5% of the DZ twins and 23% of the MZ twins were overweight (25 ≤ BMI < 30), whereas 3.5% of the DZ twins and 2.8% of the MZ twins were obese (BMI ≥ 30). There were no significant differences between MZ and DZ twins in proportions of overweight (P = 0.088) or obesity (P = 0.516). Cognitive restraint was the only TFEQ-R21 scale that was significantly correlated with BMI (r = 0.39, P < 0.0001).

About one-half of the twins (51%) had the lowest possible score on the emotional eating scale, which indicated a low rate of emotional-related eating behavior disturbances in the young men in the population. The distribution of cognitive restraint was also skewed: almost 23% of the twins had the lowest possible score. The distribution of uncontrolled eating was less skewed than the distributions of the other 2 scales.
The within-pair correlations of the 3 scales of the TFEQ-R21 were more than twice as high in the MZ twins as in the DZ twins (Table 2). These results strongly indicate the presence of genetic effects on eating behavior. The within-pair correlations were also more than twice as high for the MZ pairs than for the DZ pairs for the single items included in the 3 scales.

The results of the heritability analyses are shown in Table 3. Heritability estimates from the ADE, ACE, AE, and DE models were similar. The ADE, AE, and DE models gave similar fits, with AIC differences of only 2.0 in favor of the more simple models. This difference, however, was not significant (P = 0.16), and judging from the CIs, it seems that both additive and nonadditive components are of importance. Consequently, we choose the ADE models for all 3 traits. Both the ACE and the CE models provided worse goodness-of-fits than did the ADE models. AIC differences in favor of the ADE model over the ACE model were 6.6 for cognitive restraint, 0.6 for emotional eating, and 2.8 for uncontrolled eating; differences in favor of the ADE model over the CE model were 15.7 (P < 0.0001) for cognitive restraint, 8.8 (P = 0.003) for emotional eating, and 16.4 (P < 0.0001) for uncontrolled eating (P values from likelihood ratio test with 1 df). From the ADE model, broad sense heritability was estimated to 59% (95% CI: 52%, 66%) for cognitive restraint, 60% (95% CI: 52%, 67%) for emotional eating, and 45% (95% CI: 36%, 53%) for uncontrolled eating.

To assess whether the EEA might be violated, we performed analyses stratified by amount of contact within twin pairs. Heritability estimates of twins with frequent versus less frequent contact differed by only ~0–8% from the overall heritability under the ADE model (data not shown). We thus found no evidence or indication that the EEA was violated in our analyses of these eating behavioral traits.

### Table 2

Within-twin-pair Spearman correlations of the eating behavior items on the 3 scales of the revised 21-item version of the Three-Factor Eating Questionnaire

<table>
<thead>
<tr>
<th>Item</th>
<th>Cognitive restraint scale score</th>
<th>Emotional eating scale score</th>
<th>Uncontrolled eating scale score</th>
</tr>
</thead>
<tbody>
<tr>
<td>I deliberately take small helpings to control my weight.</td>
<td>0.14 (0.03, 0.25)</td>
<td>0.10 (–0.01, 0.21)</td>
<td>0.09 (0.00, 0.18)</td>
</tr>
<tr>
<td>I consciously hold back at meals to keep from gaining weight.</td>
<td>0.11</td>
<td>0.02</td>
<td>0.00 (0.00, 0.16)</td>
</tr>
<tr>
<td>I don’t eat some foods because they make me fat.</td>
<td>0.18</td>
<td>0.04</td>
<td>0.05 (0.00, 0.15)</td>
</tr>
<tr>
<td>How often do you avoid “stocking up” on tempting foods?</td>
<td>0.11</td>
<td>0.01</td>
<td>0.01 (0.00, 0.06)</td>
</tr>
<tr>
<td>How likely are you to make an effort to eat less than you want?</td>
<td>0.07</td>
<td>0.02</td>
<td>0.02 (0.00, 0.08)</td>
</tr>
<tr>
<td>On a scale of 1 to 8, where 1 means no restraint in eating and 8 means total restraint, what number would you give yourself?</td>
<td>0.15</td>
<td>0.07</td>
<td>0.02 (0.00, 0.08)</td>
</tr>
<tr>
<td>Factors contributing to dietary restraint:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dizygotic twins</td>
<td>Monozygotic twins</td>
<td></td>
</tr>
<tr>
<td>Cognitive restraint scale score</td>
<td>0.55 (0.48, 0.62)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional eating scale score</td>
<td>0.36 (0.28, 0.45)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uncontrolled eating scale score</td>
<td>0.43 (0.35, 0.51)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 3

The genetic and environmental components of the 3 eating behavior scales as estimated by structural equation modeling

<table>
<thead>
<tr>
<th>Eating behavior scales</th>
<th>Best fitting model</th>
<th>Additive genetic factors (A)</th>
<th>Nonadditive genetic factors (D)</th>
<th>Nonshared environmental factors (E)</th>
<th>Heritability (A + D)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive restraint</td>
<td>ADE</td>
<td>0.00 (0.00, 0.43)</td>
<td>0.59 (0.15, 0.66)</td>
<td>0.41 (0.34, 0.48)</td>
<td>0.59 (0.52, 0.66)</td>
</tr>
<tr>
<td>Emotional eating</td>
<td>ADE</td>
<td>0.60 (0.24, 0.67)</td>
<td>0.00 (0.00, 0.37)</td>
<td>0.40 (0.53, 0.48)</td>
<td>0.60 (0.52, 0.67)</td>
</tr>
<tr>
<td>Uncontrolled eating</td>
<td>ADE</td>
<td>0.00 (0.00, 0.47)</td>
<td>0.45 (0.00, 0.53)</td>
<td>0.55 (0.47, 0.64)</td>
<td>0.45 (0.36, 0.53)</td>
</tr>
</tbody>
</table>

1 95% CI in parentheses.

2 Model with additive genetic factors (A), nonadditive genetic factors (D), and nonshared environmental factors (E).
DISCUSSION

To our knowledge, this is one of the first population-based, male twin study of the relative influences of genes and environment on eating behavior. Heritability was estimated to be 59% for cognitive restraint, 60% for emotional eating, and 45% for uncontrolled eating by the ADE model, which included additive genetic (A), nonadditive genetic (D), and nonshared environmental factors (E). The decision to include a D component in the final model is debatable because our structural equation models had limited statistical power to discriminate between the effects of A and D (32) as indicated by the broad CIs for the variance components. Without more precise biological knowledge of the links between genetic effects and eating behavioral traits, however, it seems imprudent to discount any of these genetic components.

The complexity of the true underlying genetic model might be underestimated in our study, which may have contributed to our difficulties in discriminating between the effects of A and D on eating behavior. As recently shown by Eaves et al (33), within-pair resemblance in a trait, which varies according to contact frequency, may mistakenly be interpreted as a nonadditive genetic effect. Such an effect may be due to genetic factors on niche selection, ie, simply that genetic factors influence how, where, and with whom twins choose to spend their time. Such genetic niche selection effects may mistakenly be interpreted as an effect of D on the studied trait. Even though the contributions of A and D are difficult to disentangle, the heritability estimates in the present study remained almost unchanged regardless of whether D was included in the model. In the present study, the ADE model represents the best compromise between goodness-of-fit and parsimony. The key message is that genetic factors have a strong effect on all 3 eating behavior domains measured by the TFEQ-R21, regardless of the patterns of inheritance.

Most young men in this study were of Swedish or other European origin and thus our results may not be generalizable to males of non-Caucasian ancestry. However, we have good reason to believe that the results are generalizable to singletons. Twins have higher morbidity and mortality during the first year of life, but thereafter the differences between twins and singletons are small or nonexistent. Evans and Martin (25) concluded that twins are representative of the general population with respect to most health and behavioral outcomes, and we believe this to also be true with respect to eating behavior.

The TFEQ was initially constructed to evaluate the influences of cognitive, emotional, and external factors on eating behavior in obese subjects. A few items of the TFEQ-R21 emotional eating scale were part of the disinhibition scale of the original TFEQ. The emotional eating scale of the TFEQ-R21 comprises a solid, separate measure of overeating induced by negative mood and emotions. This scale is based on psychosomatic theory, which suggests that some individuals respond by overeating as opposed to appetite loss when experiencing negative emotions (eg, fear, anxiety, or anger). The so-called emotional eaters have difficulties in distinguishing between the psychological states of negative emotions on the one hand and hunger-satiety on the other. Interestingly, our results indicate that emotional eating as measured by the TFEQ-R21 is strongly influenced by genetic factors (heritability 60%). The theory suggests that obese persons exhibit more emotionally induced eating than do nonobese persons (34).

Steinle et al (10) reported considerably lower estimates of heritability for restraint eating (28%), disinhibition (40%), and hunger (23%) in their US Amish family study than those we found in our Swedish study. The comparison should, however, be interpreted cautiously because Steinle et al used the original version of the TFEQ (9), whereas we used the TFEQ-R21. Our estimates of the effects of A, D, and E on eating behavior were largely the same among twin pairs with frequent and less frequent contact. These analyses and the literature on the EEA and eating behavior (35) suggest that the higher estimates of heritability in our Swedish study are not due to a violation of the EEA. However, we cannot exclude the possibility that other environmental factors not captured by the frequency of contact, eg, maternal feeding practices in childhood, made the MZ twins more alike than the DZ twins. We can thus not exclude the possibility that a violation of the EEA may have contributed to the high heritability estimates.

Measurement error might partly explain differences in heritability estimates between our study and those by Steinle et al (10) and Mazzeo et al (15). Psychometric analysis of the original TFEQ constructs has shown weak item-scale internal consistency, especially for the 21-item cognitive restrained scale (14), and “noise” from items with poor item-scale correlations may have attenuated their heritability estimates.

Results from the female twin study by Neale et al (11) and Mazzeo et al (15) emphasize the urgent need for revision of items included in the original TFEQ. These authors included 36 of the original 51 items with a new 4-category Likert-type response format. Their factor analyses indicated poor fit of data to the original three-factor structure (15). The revised TFEQ-R21 used in the present study has been validated in population-based and obese clinical samples in Sweden and appears to be robust regarding factor structure and construct validity.

The factor structure of the TFEQ-R21 was satisfactorily replicated in the present study sample (unpublished data). The present male twin study showed strong genetic influences on all 3 eating behavioral traits. In contrast, the female twin study found genetic influences on only the disinhibition scale (heritability 45%) (11, 15), which may be compared with our results for uncontrolled eating (heritability 45%). The authors of that study concluded that the disinhibition and hunger scales of the original TFEQ covaried significantly in their model, indicating that both scales may be influenced by the same genetic factors (15). In fact, the uncontrolled eating scale of the TFEQ-R21 was constructed to solve the problem of overlap between these scales and therefore consists of the most efficient items from both constructs (14).

To date, the search for specific genes that increase the risk of common forms of obesity has been unsuccessful (36, 37). On the basis of the results presented above, it is clear that eating behavior is strongly influenced by genes. However, it is currently unknown how similar the genes affecting eating behavior are to those multiple genes involved in the development of overweight and obesity. In the present study, scores on the restraint eating scale were strongly associated with the BMI of the twins. Several other studies also reported close associations between dimensions of eating behavior and BMI (10, 12). Eating behaviors measured by the TFEQ-R21 may be promising phenotypes for future genetic research on the roots of the obesity epidemic. This is further supported by the findings of Steinle et al (10), who reported linkage to disinhibition and restraint on chromosomal links between genetic effects and eating behavioral traits.
regions in proximity to genes encoding for peroxisome proliferator activated receptor γ, glucagon-like peptide 1, and leptin. Studies of the simultaneous effects of multiple genes and gene-environment interactions are most likely to be successful. However, the difficulties of this enterprise are underscored by recent research on a comprehensive model of intake regulation that indicated that different genes seem to influence food intake regulation and body weight (5).

We are grateful to Marianne Sullivan for her constructive criticism and comments.

FR and JK developed the core idea. All authors participated in the design of the study. ST constructed the questionnaire with help from JK. ST had primary responsibility for data collection. ST and PT analyzed the data. ST drafted the paper and all authors contributed substantially to the final version. The authors had no conflicts of interest to declare.

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