Body Dissatisfaction and Drive for Thinness in Young Adult Twins

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ABSTRACT
Objective: We explored correlates of the Eating Disorder Inventory subscales Body Dissatisfaction (BD) and Drive for Thinness (DT) and genetic and environmental influences on these traits.
Method: In a population-based sample of 4,667 Finnish twins aged 22–27 years, we conducted twin modeling to explore genetic and environmental contributions to body dissatisfaction and drive for thinness. Logistic regression was used for the correlational analysis.
Results: Various eating and body size-related factors and psychosomatic symptoms were significantly associated with high body dissatisfaction and drive for thinness in both genders. In women, early puberty onset, early initiation of sexual activity, and multiple sex partners were statistically significant risk factors of body dissatisfaction. In gender-specific univariate twin models, additive genes accounted for 59.4% (95% confidence interval [CI] = 53.2–64.7%) of the variance in body dissatisfaction and for 51.0% (95% CI = 43.7–57.5%) of the variance in drive for thinness among females, but for none of the variance among males.
Discussion: There are very distinct gender differences in the heritability patterns of body dissatisfaction and drive for thinness in young adults. © 2005 by Wiley Periodicals, Inc.
Keywords: body dissatisfaction; drive for thinness; twin study; gender differences

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Introduction

The discrepancy between Western society’s obsession with body shape and thinness ideals and the reality of steadily increasing body weight in the current obesogenic environment causes widespread dissatisfaction with body shape and weight. This often emerges by preadolescence and is widespread in adolescent and young adult populations (Heatherton, Mahamed, Striepe, Field, & Keel, 1997; Jaeger et al., 2002; Moore, 1988; Sands, Tricker, Sherman, Armatas, & Maschette, 1997; Schur, Sanders, & Steiner, 2000).

Weight or body dissatisfaction is associated with depressive symptoms and dysphoria (Lautenbach et al., 1992; McCabe & Marwit, 1993; Rierdan & Koff, 1997), although the nature of the direction of causality is complex and unclear. Body dissatisfaction and related body image disturbances predict subsequent increases in depressive symptoms. These factors operate independent of risk factors of depression among girls (Stice & Bearman, 2001). However, low mood also influences body size perception and increases body dissatisfaction (Taylor & Cooper, 1992). The effect of low mood on appearance dissatisfaction also manifests before puberty (McCabe & Marwit, 1993).

The Body dissatisfaction (BD) and Drive for Thinness (DT) subscales of the Eating Disorder Inventory...
Inventory (EDI; Garner, 1991) are widely used and validated self-report measures of dissatisfaction with body shape. In a recent linkage study of anorexia, drive for thinness was shown to cluster in affected sibpairs and to be a valuable covariate to be included in linkage analyses (Devlin et al., 2002). Body dissatisfaction and drive for thinness may mark endophenotypes of eating disorders (for a review on endophenotypes in psychiatry, see Gottesman & Gould, 2003). For these reasons, it is important to understand the genetic and environmental contributions underlying body dissatisfaction and drive for thinness. The most direct way to do so is by studying a representative sample of twins.

We are aware of three previously published studies of the use of the EDI in twins: a volunteer sample of 492 adult female twins from the United Kingdom (Rutherford, McGuffin, Katz, & Murray, 1993), a population study of 1,282 female Minnesotan twins between the ages of 11 and 17 (Klump, McGue, & Iacono, 2000) using the BD and DT subscales modified for adolescents, and a Japanese study of 162 female twin pairs (Kamakura, Ando, Ono, & Maekawa, 2003), which did not include the BD and DT subscales. These studies suggest that EDI subscale scores are moderately heritable, but the small sample populations and the absence of confidence intervals (CIs) limit conclusions (Neale & Miller, 1997), setting a context for further investigation in a large population of both male and female twins.

The current investigation explores correlates of body dissatisfaction and drive for thinness in a large population-based sample of male and female twins. In addition, genetic and environmental contributions to a liability to drive for thinness and body dissatisfaction are assessed in both males and females separately.

**Methods**

**Sample**

The data reported are from FinnTwin16, a population-based study of five consecutive nationwide birth cohorts of Finnish twins born between 1975 and 1979 (Rose, Kaprio, Winter, Koskenvuo, & Viken, 1999). Data collection was conducted by postal questionnaire, at the age of 16 years, with follow-ups at 17 years, 18.5 years, and 22–27 years. The questionnaires assessed personality, social relationships, health, and health habits. Response rates were high (approximately 90%) across all occasions. The current study is based on responses to questionnaires administered to 16-year-old twins and on follow-up questionnaires administered to them when they were 22–27 years old (i.e., the first and fourth waves of data collection). EDI subscale responses and cross-sectionally correlated behaviors were assessed at age 22–27 years, as were possible predictors of future EDI scores at age 16–17 years. Our study sample comprises 4,667 twins (2,545 females and 2,122 males).

Twin zygosity was determined by standard items included in the baseline questionnaire (Sarna, Kaprio, Sistonen, & Koskenvuo, 1978). When necessary, questionnaire information was supplemented with photographs, fingerprints, and DNA-marker studies (Sarna & Kaprio, 1980). The zygosity of a few same-gender pairs remains uncertain and all twin pairs were classified as monozygotic (MZ), dizygotic (DZ), or unknown zygosity.

**Hypotheses**

Based on existing literature, we expected that body dissatisfaction and drive for thinness would be associated with depressive symptoms (Lautenbacher et al., 1992; Rierdan & Koff, 1997), poor general health (Neumark-Sztainer, Story, Resnick, Garwick, & Blum, 1995), larger current body size (Casey et al., 1991; Pingitore, Spring, & Garfield, 1997; Allaz, Bernstein, Rouget, Archinard, & Morabia, 1998), dietary restraint (Davis, Durnin, Gurevich, Le Maire, & Dionne, 1993), smoking (Wiseman, Turco, Sunday, & Halmi, 1998), and higher levels of education (Allaz et al., 1998). These key variables were measured at age 16 years and at ages 22–27 years with the exception of dietary restraint and depressive mood, which were, unfortunately, not included in the questionnaires administered to 16-year-olds. Furthermore, we used our data to test an earlier report that suggested pubertal timing is not significantly associated with body dissatisfaction and drive for thinness (Ackard & Peterson, 2001).

**Measures**

EDI. The questionnaire administered to the 22–27-year-olds included three subscales of the EDI-1 (Garner, 1991): DT, BD, and Bulimia. A Finnish version of this instrument has been translated and validated (Charpentier, Pia, personal communication, 2003). The EDI responses were scored 1 to 6 to ensure a normal distribution. The DT subscale has seven items with a coefficient alpha of .87 for females and .75 for males. The BD subscale has eight items with alpha values of .92 in females and .86 in males. In contrast to DT and BD, Bulimia subscale scores are not included in the current article because they were elevated in very few individuals. For the correlational analysis and male twin models, DT and BD scores were dichotomized into high versus low groups, using 75% percentiles as cutoff points.
serving as the reference category.

dichotomized, with responses of "very or rather good"

year and 22–27-year assessments using the question,

of women). Self-reported health was measured at the 16-

reported high GHQ-12 scores (12.4% of men and 24.6% 

(Holi, Marttunen, & Aalberg, 2003), 19.1% of twins

versus "weekly or more." For the multivariable models, a 

continuous sum score of the aforementioned symptoms 

was calculated.

In addition, as a mental health screen at the 22–27-

year assessment, we included the 12-item General Health 

Questionnaire (GHQ-12; Goldberg et al., 1997), scored 

with the usual 0–0–1–1 method. Using the 3/4 cutoff 

point recently recommended for the Finnish population 

(Holi, Marttunen, & Aalberg, 2003), 19.1% of twins 

reported high GHQ-12 scores (12.4% of men and 24.6% 

of women). Self-reported health was measured at the 16-

year and 22–27-year assessments using the question, 

“What do you think about your health right now?” with 

response categories “very good,” “rather good,” “average,” 

“rather bad,” and “very bad.” This response was 

dichotomized, with responses of “very or rather good” 

serving as the reference category.

Body Size. At the 16 and 22–27-year assessments, the 

twins self-reported their current weight and height, 

from which their body mass index (BMI) was calculated. 

At the 22–27-year assessment, they also measured their 

waist circumference using a tape measure that was 

mailed with the questionnaire. Detailed instructions, 

which were also mailed with the questionnaire, included 

a body drawing that indicated the site of measurement.

For a subsample of 212 young adult twins, their height, 

weight, and waist circumference were measured by 

trained personnel using a stadiometer, calibrated beam 

balance, and a tape measure. The agreement between 

these standardized measurements and self-report was 

excellent (correlations of .96 for height, .94 for weight, 

and .88 for waist; Silventoinen et al., 2003). The median 

time interval between self-report and the standardized 

measurement was 356 days. In addition to their current 

weight, the twins also reported their ideal and maximum 

weights at adult height.

Dietary Restraint/Disinhibition. Dietary restraint was 

assessed at the 22–27-year assessment with the question, 

“Which of the following best describes you?” “It’s easy 

for me to eat about the amount I need to” (normal eating, 

reference category); “I quite often eat more than I 

actually need” (overeating); “I often try to restrict my 

eating” (restrictive eating); and “At times, I’m on a strict 
diet, at others I overeat” (alternating restrictive eating / 

overeating). The 22–27-year questionnaire included 

items specifically designed to assess nutrition, eating 

patterns, dieting, body shape, exercise, sexual behavior, 

and life satisfaction. Thus, rich information across these 

domains was available. For illustrative purposes, we 

chose to include several of these variables in the correla-

tional analyses.

Smoking. Smoking was assessed at ages 16 and 22–27,

dichotomizing the response categories to having never 

smoked versus having ever smoked or still smoking 

regularly.

Education Level. We measured education level at ages 

17 and 22–27. The dichotomy used was mandatory 

school only versus higher education (vocational school, 

high school, polytechnic school, or university). The edu-

cation level in adolescence was taken from the 17-year 

questionnaire (when 93.0% of the twins had finished 

mandatory education) instead of the 16-year question-

naire (when only 57.5% of the twins had finished man-

datory education). The education level at 17 is more 

informative of academic success in mid-adolescence 

than the education level at 16, because further education 

is voluntary after 16, and the choice of educational paths 

reflects academic performance.

Puberty. Information on the age of puberty onset 

(menarche in females, voice break in males), reported 

by the twins at age 16, was available from 1,939 males 

and 2,465 females. If the individual had not undergone 

puberty onset by age 16 (118 [6.1%] males and 27 [1.1%] 

females), the age of puberty onset was coded as 16.5 

years. As the age of puberty onset differs by gender, 

different age thresholds were used for females and 

males. For example, early menarche (before age 12 

years) was reported by 297 (12.1%) women and late 

menarche (at 14 years or later) was reported by 558 

(22.6%) women. Early voice break (before age 13 years) 

was reported by 196 (10.1%) men and late voice break (at 

15 years or later) was reported by 516 (26.6 %) men.

Modeling Twin Data

Structural equation modeling was performed using the 

computer program Mx (Neale, Boker, Xie, & Maes, 2002).

To ensure that missing data did not unduly distort para-

meter estimates, we used the raw data maximum likelihood 
estimation option in Mx (Neale et al., 2002) that enabled 
inclusion of unmatched twin pairs (approximately 10% of 

our sample). Among females, DT and BD scores were nor-

dally distributed. However, among males, DT and BD dis-

tributions were extremely nonnormal, possibly reflecting 

underlying bimodality (most of the observations were in 

the low end of the distribution, with few high scores in the
upper end), and Box-Cox transformations were not successful. Thus, the male EDI subscales were dichotomized using 75% percentile points as cutoffs. In females, we used DT and BD scores as a continuous variable, from which variance-covariance matrices were calculated and fitted using Mx. The distributions and variances for EDI subscale scores in each gender were so different that gender-limitation modeling was not applied. We were therefore unable to include data from opposite-gender twin pairs in the structural equation modeling portion of the current article. Instead, we modeled each gender separately. Bivariate Cholesky decompositions were also performed to obtain genetic and environmental correlations.

Using data on twins reared together, four separate parameters can be modeled: additive genetic effects (A), dominance genetic effects (D), common environmental effects (C), and unique environmental effects (E). C effects are those that are shared by both twins and E effects are those that are individual specific and uncorrelated within the twin pair. E effects also include measurement error. Based on the different combinations of these parameters, AE, ACE, ADE, and CE models can be fitted. Effects caused by D and C effects cannot be modeled simultaneously (Falconer & Mackay, 1996). Based on twin correlations (tetrachoric correlations for males, Pearson correlations for females, calculated using SAS, Version 8.0), ACE and its submodels were deemed the most probable explanatory models for body dissatisfaction in women and men and drive for thinness in men. Female pairwise DT correlations showed possible dominance effects. For example, both ACE and ADE and submodels were fitted. Model fitting started with the most parsimonious models, that is, AE and CE, and then advanced to ACE/ADE models. However, in this setting, the power to detect D effects is relatively low (Falconer & Mackay, 1996).

To assess how well a model fits the data, we used the −2 log-likelihood statistic. The likelihood ratio of alternative models was calculated by the difference in their chi-square values. The difference between two such chi-square goodness-of-fit statistics is itself distributed as a chi-square statistic with degrees of freedom equal to the difference in degrees of freedom of the two models being compared (Neale & Cardon, 1992). If the difference in the chi-square values of two models was not statistically significant, the principle of parsimony was applied. The model with fewer parameters was preferred.

In addition, gender-specific bivariate Cholesky decompositions were performed with Mx (Neale et al., 2002) using the raw data maximum likelihood estimation option that enabled inclusion of unmatched twin pairs. In the bivariate twin model, the correlation between BD and DT scores can be divided into the correlations due to A, C, D, and E effects.

Results

Correlates

The sociodemographic characteristics of our sample are presented in Table 1. As a probable result of multiple waves of participation, women (54.5%) and individuals with a higher education (more than the mandatory 9 years) were somewhat overrepresented in the fourth wave of follow-up data (94.6% in the sampled twins vs. 83.6% in the 20–29-year age group of the general Finnish population, as reported by Statistics Finland, 2001). Overall, women exhibited substantially higher levels of BD and DT than did men.

BD and DT scores correlated (r = .72 in females and r = .65 in males). Drive for thinness and intentional weight loss of ≥5 kg on at least one occasion were correlated in both females (r = .47) and males (r = .59). The relationship between BMI and EDI subscales was nonlinear in both genders (Figure 1).

Associations of eating disorders and intentional weight loss with BD and DT are presented in Table 2. Age was not associated with variation in BD or DT scores.

Table 1. Sociodemographic characteristics of Finnish young adult twins (N = 4,667)

<table>
<thead>
<tr>
<th>Correlates</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (at 4th wave of follow-up)</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>24.4 years (SD 0.8)</td>
</tr>
<tr>
<td>Range</td>
<td>22.8–27.2 years</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>2,122 (45.6)</td>
</tr>
<tr>
<td>Female</td>
<td>2,545 (54.5)</td>
</tr>
<tr>
<td>Zygosity</td>
<td></td>
</tr>
<tr>
<td>Monozygotic</td>
<td>1,408 (30.2)</td>
</tr>
<tr>
<td>Same-gender dizygotic</td>
<td>1,470 (31.5)</td>
</tr>
<tr>
<td>Opposite-gender dizygotic</td>
<td>1,510 (32.4)</td>
</tr>
<tr>
<td>Unknown</td>
<td>279 (6.0)</td>
</tr>
<tr>
<td>Living arrangements</td>
<td></td>
</tr>
<tr>
<td>Married or cohabiting</td>
<td>2,163 (46.3)</td>
</tr>
<tr>
<td>Single</td>
<td>1,347 (28.9)</td>
</tr>
<tr>
<td>Lives with parent(s)</td>
<td>502 (10.8)</td>
</tr>
<tr>
<td>Lives with child(ren)</td>
<td>76 (1.6)</td>
</tr>
<tr>
<td>Lives with someone else</td>
<td>574 (12.3)</td>
</tr>
<tr>
<td>Lives with cotwin</td>
<td>376 (8.1)</td>
</tr>
<tr>
<td>Highest education completed</td>
<td></td>
</tr>
<tr>
<td>Mandatory education only</td>
<td>251 (5.4)</td>
</tr>
<tr>
<td>Vocational school or training</td>
<td>1,300 (27.9)</td>
</tr>
<tr>
<td>Vocational college</td>
<td>387 (8.3)</td>
</tr>
<tr>
<td>Senior high school</td>
<td>1,870 (40.1)</td>
</tr>
<tr>
<td>Polytechnic</td>
<td>554 (11.9)</td>
</tr>
<tr>
<td>University</td>
<td>300 (6.4)</td>
</tr>
<tr>
<td>Current abode</td>
<td></td>
</tr>
<tr>
<td>Helsinki metropolitan area</td>
<td>1,151 (24.7)</td>
</tr>
<tr>
<td>Large city</td>
<td>2,288 (49.0)</td>
</tr>
<tr>
<td>Smaller city</td>
<td>449 (9.6)</td>
</tr>
<tr>
<td>Rural town or village</td>
<td>485 (10.4)</td>
</tr>
<tr>
<td>Countryside</td>
<td>276 (5.9)</td>
</tr>
</tbody>
</table>
The correlates of body dissatisfaction for each gender are presented in Table 3. In young adulthood, restrictive eating, overeating, alternating restricting-binging, and various other unhealthy eating patterns were statistically and significantly associated with high BD and DT scores in both genders. Individuals with high BD and DT scores exhibited, on average, a higher BMI and greater waist circumference than those with low BD or DT scores. Women who belonged to the high BD group in young adulthood were significantly more likely to smoke, drink, skip breakfast, belong to the highest 10th percentile for BMI, and report frequent sleeping difficulties, headaches, anxiety symptoms and neck and back pain in late adolescence at age 16, and have a lower education level at age 17. Also, men with high BD scores were also more likely to be overweight at 16, and they reported more sleeping difficulties, fatigue, and nervousness in adolescence. The within-individual correlation of psychosomatic symptoms at age 16 with psychosomatic symptoms at 22–27 was $r = .15$ to $r = .26$ in both genders; for smoking, these correlations were $r = .48$ in women and $r = .44$ in men; and for alcohol use, $r = .13$ in women and $r = .23$ in men. Correlates of drive for thinness (Table 4) were similar, with a very strong relationship between overweight at 16 and drive for thinness in young adulthood, and with more associations with psychosomatic features for girls than boys. However, neither tobacco nor alcohol use in adolescence was significantly associated with drive for thinness in either males or females.

Psychosomatic symptoms, frequent feelings of loneliness, and dissatisfaction with parental relationships were statistically and significantly more common in women and men with high BD and DT scores. In women, early puberty onset was a statistically very significant risk factor of body dissatisfaction, whereas late puberty onset protected them from body dissatisfaction in young adulthood. In men, body dissatisfaction was not significantly associated with puberty. Drive for thinness was higher in men with early puberty and lower in women with late puberty, but these associations were statistically weak. High body dissatisfaction was associated with an earlier age at first sexual intercourse and more sexual partners in women, but not in men. These associations remained very significant ($p < .001$) even when controlling for age of pubertal onset.

When the young adulthood correlates of body dissatisfaction and drive for thinness were adjusted...
## Table 3. Correlates of body dissatisfaction and drive for thinness: ORs from univariate female and male logistic regression models

<table>
<thead>
<tr>
<th>Correlate</th>
<th>Body Dissatisfaction</th>
<th>Drive for Thinness</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body size and shape</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current BMI (OR per unit increase)</td>
<td>1.36 (1.31–1.42) ***</td>
<td>1.17 (1.13–1.20) ***</td>
</tr>
<tr>
<td>BMI at 16 (OR per unit increase)</td>
<td>1.34 (1.28–1.41) ***</td>
<td>1.21 (1.16–1.26) ***</td>
</tr>
<tr>
<td>Current waist (OR per unit increase)</td>
<td>1.08 (1.07–1.10)</td>
<td>1.04 (1.03–1.05) ***</td>
</tr>
<tr>
<td>Overweight at 16</td>
<td>0.92 (0.97–1.04)</td>
<td>0.88 (0.82–0.96)</td>
</tr>
<tr>
<td><strong>Eating-related variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overeating</td>
<td>7.88 (6.12–10.14) ***</td>
<td>5.69 (4.39–7.38) ***</td>
</tr>
<tr>
<td>Restrictive eating</td>
<td>8.06 (5.96–10.89) ***</td>
<td>15.37 (11.35–20.82)***</td>
</tr>
<tr>
<td>Alternating overeating and restrictive eating</td>
<td>15.18 (10.34–22.29) ***</td>
<td>23.30 (15.48–35.06)***</td>
</tr>
<tr>
<td>International weight loss of ≥5 kg at least once</td>
<td>3.99 (3.24–4.90) ***</td>
<td>4.03 (3.30–4.92) ***</td>
</tr>
<tr>
<td>Self-reported ideal BMI (OR per unit increase)</td>
<td>3.13 (2.52–3.76) ***</td>
<td>1.11 (0.94–1.36) ***</td>
</tr>
<tr>
<td><strong>Puberty and sexuality</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early puberty (&lt;12 for girls, &lt;13 for boys)</td>
<td>1.64 (1.23–1.91) ***</td>
<td>1.31 (0.98–1.74) NS</td>
</tr>
<tr>
<td>Late puberty (≥14 for girls, ≥15 for boys)</td>
<td>0.47 (0.36–0.62) ***</td>
<td>0.75 (0.59–0.95) *</td>
</tr>
<tr>
<td>Number of sex partners (OR per each partner)</td>
<td>1.11 (1.04–1.20) **</td>
<td>1.09 (1.01–1.17) *</td>
</tr>
<tr>
<td>Age at first sexual intercourse</td>
<td>0.90 (0.86–0.94) ***</td>
<td>0.96 (0.92–1.01) NS</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td>1.43 (1.16–1.77) ***</td>
<td>1.04 (0.83–1.30) NS</td>
</tr>
<tr>
<td>Education level in adulthood: mandatory education only (vs. more than mandatory education)</td>
<td>1.50 (1.21–1.86) ***</td>
<td>1.02 (0.81–1.28) NS</td>
</tr>
<tr>
<td>Education level at 17: mandatory education only (vs. more than mandatory education)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Substance use</strong></td>
<td>1.23 (0.96–1.58) NS</td>
<td>1.31 (1.03–1.68) *</td>
</tr>
<tr>
<td>Illegal drug use</td>
<td>1.52 (1.24–1.86) ***</td>
<td>1.30 (1.07–1.57) **</td>
</tr>
<tr>
<td>Smoking at 16</td>
<td>1.14 (0.87–1.49) NS</td>
<td>1.17 (0.95–1.45) NS</td>
</tr>
<tr>
<td>Frequent alcohol use</td>
<td>1.38 (1.11–1.73) **</td>
<td>1.18 (0.95–1.46) NS</td>
</tr>
<tr>
<td>Frequent (weekly or more) alcohol use at 16</td>
<td>1.23 (0.98–1.55) NS</td>
<td>1.02 (0.81–1.28) NS</td>
</tr>
<tr>
<td><strong>Psychological and psychosomatic symptoms</strong></td>
<td>1.99 (1.50–2.63) ***</td>
<td>2.62 (2.01–3.43) ***</td>
</tr>
<tr>
<td>Current unhappiness (vs. current happiness)</td>
<td>2.13 (1.69–2.68) ***</td>
<td>1.84 (1.46–2.31) ***</td>
</tr>
<tr>
<td>Self-perceived poor health</td>
<td>2.27 (1.77–2.92) ***</td>
<td>1.88 (1.48–2.41) ***</td>
</tr>
<tr>
<td>Current frequent stomach pain (vs. monthly or less)</td>
<td>1.75 (1.36–2.26) ***</td>
<td>1.90 (1.47–2.44) ***</td>
</tr>
<tr>
<td>Frequent stomach pain at 16 (vs. monthly or less)</td>
<td>1.40 (0.99–1.96) NS</td>
<td>1.80 (1.30–2.49) ***</td>
</tr>
<tr>
<td>Current headaches (vs. monthly or less)</td>
<td>1.81 (1.47–2.24) ***</td>
<td>1.44 (1.17–1.89) ***</td>
</tr>
<tr>
<td>Frequent headaches at 16 (vs. monthly or less)</td>
<td>1.46 (1.17–1.81) ***</td>
<td>1.44 (1.15–1.81) ***</td>
</tr>
<tr>
<td>Current nervousness</td>
<td>1.68 (1.36–2.08) ***</td>
<td>1.79 (1.44–2.23) ***</td>
</tr>
<tr>
<td>Nervousness at 16</td>
<td>1.25 (1.01–1.56)</td>
<td>1.78 (1.44–2.19) ***</td>
</tr>
<tr>
<td>Current depressive mood</td>
<td>1.60 (1.30–1.96)</td>
<td>1.78 (1.37–2.31) ***</td>
</tr>
<tr>
<td>Current sleeping difficulties</td>
<td>1.95 (1.59–2.39) ***</td>
<td>1.95 (1.46–2.56) ***</td>
</tr>
<tr>
<td>Sleeping difficulties at 16</td>
<td>1.52 (1.21–1.91) ***</td>
<td>1.27 (1.02–1.59) *</td>
</tr>
<tr>
<td>Current satisfaction with partner</td>
<td>1.20 (0.97–1.49) NS</td>
<td>1.38 (1.12–1.70) **</td>
</tr>
<tr>
<td>Current satisfaction with mother</td>
<td>1.38 (1.04–1.83)</td>
<td>1.42 (1.09–1.85) **</td>
</tr>
<tr>
<td>Current satisfaction with father</td>
<td>1.57 (1.25–1.96) ***</td>
<td>1.44 (1.16–1.79) ***</td>
</tr>
<tr>
<td>Current frequent feelings of loneliness</td>
<td>1.42 (1.17–1.73) ***</td>
<td>1.63 (1.34–1.98) ***</td>
</tr>
</tbody>
</table>

Note: OR = odds ratio; BMI = body mass index; NS = not significant.

aConfidence intervals adjusted for clustered sampling. Rounding error in sums of some percentages.

*p < .05. **p < .01. ***p ≤ .001.
for concurrent BMI in logistic regression models (detailed results not shown), the general pattern of correlates did not change, save for an overall decrease of odds ratios (OR) of eating and body size-related correlates, and an overall increase of ORs in the psychological health domain. After controlling for BMI, waist circumference and early age of puberty onset were no longer statistically and significantly associated with body dissatisfaction and drive for thinness in women, and education level in adulthood was no longer statistically and significantly associated with body dissatisfaction in either gender. The association of body dissatisfaction and education level in adulthood was no longer statistically and significantly associated with body dissatisfaction in either gender. The association of body dissatisfaction and education level in adulthood reflects a greater prevalence of overweight (12.7% vs. 7.1%, p < .00001 in women; 16.9% vs. 11.8%, p = .0018 in men) among individuals with a low level of education. Additional adjustments for self-reported history of anorexia and bulimia in addition to BMI did not affect the ORs beyond what was achieved by BMI adjustment alone, except for a very slight overall decrease in magnitude (approximately 0.1) for most variables. There were no changes in the patterns of statistical significance.

To determine whether there were differences in the correlational profiles of drive for thinness and body dissatisfaction in each gender, we entered the statistically most significant and theoretically most interesting correlates (BMI at 16 and 22–27; dietary restraint, GHQ-12 score, global scores of psychosomatic symptoms at 16 and 22–27; and ages of puberty onset and first sexual intercourse) in multivariable logistic regression models. Using the principle of parsimony, we removed from each model all independent variables that could be removed without a statistically significant reduction in model fit. The resulting best-fitting models of body dissatisfaction and drive for thinness in women and men are detailed in Table 4. Early puberty and early initiation of sexual activity remain unique correlates of female body dissatisfaction in these analyses.

**Twin Modeling**

The intrapair correlations of body dissatisfaction were .59 in MZ and .29 in DZ female pairs, .45 in MZ and .23 in DZ male pairs, and .07 for opposite-gender DZ twin pairs. For drive for thinness, these correlations were .51 and .21 in female pairs, .51 and .46 in male pairs, and .19 in opposite-gender DZ pairs. Neither the means nor variances of body dissatisfaction or drive for thinness between MZ and DZ twins in either gender were significantly different.

For body dissatisfaction in women (Table 5), the purely environmental CE model fit our data extremely poorly compared with the full ACE model. In the ACE model, the C estimate was negligibly small, and when it was omitted in an AE model, there were no changes in the overall model fit, so the more parsimonious AE model was preferred. For body dissatisfaction in men (Table 5), both ACE and CE models had acceptable fits, but CE provided the most parsimonious fit. Our gender-specific univariate twin modeling results indicate that there are significant additive genetic contributions to body dissatisfaction in females (59.4%; 95% CI = 53.2–64.7%), but not in males.
For drive for thinness in women (Table 6), twin correlations suggested that both ADE and ACE models might fit the data. ADE fit the data better than ACE, but AE was the most parsimonious model ($\Delta \chi^2 = 1.5$, $\Delta df = 1$, $p = .63$). The CE model fit the data extremely poorly. In the ACE model, the C estimate was negligibly small. When it was omitted altogether (AE model), there was no change in the overall model fit, and the more parsimonious AE model was preferred. For drive for thinness in men (Table 6), the A estimate in the ACE model was very small. When it was omitted in the CE model, the model fit did not deteriorate significantly ($\Delta \chi^2 = .3$, $\Delta df = 1$, $p = .58$). Accordingly, the best-fitting DT models were AE for women and CE for men. There were significant additive genetic contributions to drive for thinness in females (51.0%; 95% CI = 43.7–57.5%), but not in males.

In women, the genetic effects of drive for thinness correlated with the genetic effects of body dissatisfaction at the level of approximately .80 (95% CI = .74–.85) and the E effects of BD and DT had a correlation of .59 (95% CI = .53–.65) in the bivariate Cholesky decompositions. Body dissatisfaction and drive for thinness in males were influenced solely by environmental factors in our models, and genetic and environmental correlations of body dissatisfaction and drive for thinness in males were not estimated.

**Discussion**

The heritability patterns of body dissatisfaction and drive for thinness were highly gender specific. In women, these traits exhibited moderate to high heritability, whereas in men, they were purely environmental. Gender differences were much less conspicuous in the patterns of correlates of body dissatisfaction and drive for thinness. Disordered eating patterns, depressive symptoms, poor self-perceived

**TABLE 5. Comparison of alternative univariate gender-specific twin models of body dissatisfaction**

<table>
<thead>
<tr>
<th>goodness-of-Fit Tests</th>
<th>Heritability</th>
<th>Common Environment</th>
<th>Unique Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females (continuous)b</td>
<td>$-2LL$</td>
<td>$df$</td>
<td>$\Delta \chi^2$</td>
</tr>
<tr>
<td>ACE (saturated)</td>
<td>11,749.811</td>
<td>1,606</td>
<td>—</td>
</tr>
<tr>
<td>CE</td>
<td>11,786.945</td>
<td>1,607</td>
<td>37.1</td>
</tr>
<tr>
<td>AEc</td>
<td>11,749.811</td>
<td>1,607</td>
<td>0</td>
</tr>
<tr>
<td>Males (dichotomous)c</td>
<td>$2LL$</td>
<td>$df$</td>
<td>$\Delta \chi^2$</td>
</tr>
<tr>
<td>ACE (saturated)</td>
<td>2,133.926</td>
<td>1,222</td>
<td>—</td>
</tr>
<tr>
<td>CEc</td>
<td>2,137.552</td>
<td>1,223</td>
<td>3.6</td>
</tr>
<tr>
<td>AE</td>
<td>2,243.468</td>
<td>1,223</td>
<td>109.5</td>
</tr>
</tbody>
</table>

Note: $A$ additive genetic effects; $C$ common environmental effects; $E$ environmental effects. Rounding error in sums of percentages.

$^a$The $p$ value associated with $\Delta \chi^2$ (change in model fit compared to the saturated model).

$^b$For females, the C estimate in ACE models is very small, and good model fit is obtained by omitting the C estimate altogether: AE models give female data the best fit.

$^c$Best fitting model.

$^d$For males, the A estimate in ACE models is very small, and omission of A improves model fit: CE models give male data the best fit.

**TABLE 6. Comparison of alternative univariate gender-specific twin models of drive for thinness**

<table>
<thead>
<tr>
<th>goodness-of-Fit Tests</th>
<th>Heritability</th>
<th>Common Environment or Dominance Effects</th>
<th>Unique Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females (continuous)b</td>
<td>$-2LL$</td>
<td>$df$</td>
<td>$\Delta \chi^2$</td>
</tr>
<tr>
<td>ACE (saturated)</td>
<td>10,840.145</td>
<td>1,589</td>
<td>—</td>
</tr>
<tr>
<td>CE</td>
<td>10,868.156</td>
<td>1,590</td>
<td>28.0</td>
</tr>
<tr>
<td>AEc</td>
<td>10,840.145</td>
<td>1,590</td>
<td>0</td>
</tr>
<tr>
<td>ADE (saturated)</td>
<td>10,838.675</td>
<td>1,589</td>
<td>—</td>
</tr>
<tr>
<td>DE</td>
<td>10,840.145</td>
<td>1,590</td>
<td>1.7</td>
</tr>
<tr>
<td>AE</td>
<td>10,840.145</td>
<td>1,590</td>
<td>1.5</td>
</tr>
<tr>
<td>Males (dichotomous)c</td>
<td>$2LL$</td>
<td>$df$</td>
<td>$\Delta \chi^2$</td>
</tr>
<tr>
<td>ACE (saturated)</td>
<td>2,084.051</td>
<td>1,207</td>
<td>—</td>
</tr>
<tr>
<td>CEc</td>
<td>2,084.351</td>
<td>1,208</td>
<td>0.3</td>
</tr>
<tr>
<td>AE</td>
<td>2,218.443</td>
<td>1,208</td>
<td>134.4</td>
</tr>
</tbody>
</table>

Note: $A$ additive genetic effects; $C$ common-environmental effects; $E$ environmental effects. Rounding error in sums of percentages.

$^a$The $p$ value associated with $\Delta \chi^2$ (change in model fit compared to the saturated model).

$^b$For females, ADE model fits the data better than ACE according to the $-2$ log likelihood statistic, but AE is the most parsimonious model.

$^c$Best fitting model.

$^d$For males, the A estimate in the ACE model is very small, and it can be removed altogether without a significant decrease in model fit. CE is the most parsimonious male model.
health, and larger body size were, as hypothesized, strongly and consistently associated with body dissatisfaction and drive for thinness in both men and women. Contrary to previous reports (Ackard & Peterson, 2001; Stice & Whitenton, 2002), early puberty onset, early initiation of sexual activity, and multiple sex partners were statistically significant risk factors of body dissatisfaction in women, but not in men.

**Twin Modeling**

In contrast to previous twin studies of body dissatisfaction and drive for thinness, we applied twin modeling to men as well as women. Our estimates of heritability of body dissatisfaction and drive for thinness in women were very close to those obtained from the British adult volunteer twin sample (best-fitting AE model: $a^2$ of body dissatisfaction = 52%, $d^2$ of drive for thinness = 44%; Rutherford et al., 1993) and were almost identical to the heritability estimates of the modified BD and DT subscales reported for Minnesota adolescents ($a^2$ of modified body dissatisfaction = 60% at age 17, $d^2$ of modified drive for thinness = 47% at both 11 years and 17 years; Klump et al., 2000). However, given our larger samples, we could provide 95% CIs that enrich estimates obtained in previous research. Heritability estimates of weight and body dissatisfaction in women measured using instruments other than the EDI have been fairly similar in magnitude (Wade, Martin, & Tiggemann, 1998; Wade, Wilkinson, & Ben Tovim, 2003).

The gender differences in heritability patterns were striking. We found no evidence of genetic influences on body dissatisfaction and drive for thinness in men. This finding is in line with our observation that intentional weight loss is more strongly and environmentally mediated in males than in females (Keski-Rahkonen et al., 2005). These differences may be real. Perhaps, female body shape ideals and weight control behavior much more closely reflect the strongly and genetically influenced biologic body composition, whereas male body shape ideals are more disconnected from the underlying biologic reality. More likely, though, the process of comparing body dissatisfaction and drive for thinness between women and men is an uneasy one.

Sources of body dissatisfaction and drive for thinness are likely different in males and females. The EDI focuses on core areas of female body and weight dissatisfaction. Thus, body dissatisfaction and drive for thinness, as measured in the EDI, are clearly not ideal measures of male body shape-related attitudes, because domains of core importance such as muscularity and stature are completely ignored (Cohane & Pope, 2001; McCabe, Ricciardelli, & Finemore, 2002). Further research is clearly required to accurately characterize these traits in males (Anderson & Bulik, 2004).

In interpreting our results, one must be mindful of differences in the body dissatisfaction and drive for thinness distributions in males and females, resulting in the use of continuous measures for women, dichotomous variables for men, and different modeling strategies for each gender. Because of these scaling issues, male and female results may not be directly comparable, and loss of power due to dichotomization is reflected in the wider CIs for male parameter estimates.

Our study was not able to assess potential genetic overlap between BMI and body dissatisfaction and drive for thinness because of apparent nonlinearity in the relationship of the BD and DT subscales of the EDI and BMI, which current structural equation modeling approaches are ill-equipped to handle. Overlooking the apparent nonlinearity would oversimplify and distort reality. In the future, it might also be interesting to assess the genetic overlap between eating disorders and body dissatisfaction and drive for thinness in this sample.

**Correlational Analyses**

Despite the distinct gender differences in genetic architecture and the much greater overall level of body dissatisfaction and drive for thinness in women, their patterns of correlates were surprisingly similar, perhaps because body dissatisfaction and drive for thinness were highly correlated with each other. Gender differences in correlate patterns were unremarkable. In both women and men, body dissatisfaction and drive for thinness coexist with a larger body size, multiple psychosomatic symptoms, and signs of psychological distress. Although body dissatisfaction and drive for thinness shared most of their genetic determinants in women, body dissatisfaction was influenced by factors pertaining to sexual maturity, whereas drive for thinness was not. Early puberty onset and high BD scores were clearly associated in females in our sample, whereas late puberty onset was protective of body dissatisfaction in women. This effect has also been observed in other populations (Slof, Mazzeo, & Bulik, 2003). The association between body dissatisfaction and pubertal development was independent of current BMI, suggesting a complex relationship among sexual maturation, BMI, and body dissatisfaction. Early sexual maturation and a larger number of sexual partners were also associated with body dissatisfaction, but only in women, not in men.
A potential source of bias for comparisons of men and women is that menarche is a superior retrospective measure of pubertal timing than voice change. However, pubertal development was assessed at age 16, so recall bias should be modest. Menarche may also be more directly connected to body shape issues and feelings of physical awkwardness. Girls’ early sexual maturation may be a source of mixed feelings and shame, whereas boys’ early sexual maturation may be associated with power and status (Brown & Gilligan, 1992).

The direction of causality remains unclear, because we do not know at which age body dissatisfaction manifests in our sample. It could conceivably be bidirectional (early puberty increases body awareness, body dissatisfaction, and drive for thinness; extreme body dissatisfaction and drive for thinness may be related to weight loss or eating disorder behaviors that suppress or delay puberty). This merits further study in prospective cohorts, particularly because some of the previous attempts to study body dissatisfaction prospectively have not found an association between early menarche and body dissatisfaction (Stice & Whitenton, 2002).

Strengths and Limitations

The current study explored correlates of the BD and DT subscales in the EDI in the largest population sample yet reported. A remarkable strength of this sample is its excellent population coverage and high response rate, which help to minimize self-report bias.

The study also has limitations. It cannot reveal whether a causal link exists between psychosomatic symptoms and body dissatisfaction and drive for thinness, nor can it elucidate the direction of causality. Based on existing literature (Kostanski & Gullone, 1999; Sands, Tricker, Sherman, Armatas, & Maschette, 1997; Schur, Sanders, & Steiner, 2000), it is fair to assume that both of these processes start before puberty. Body image concerns may preclude depressive symptoms (Stice & Bearman, 2001). Further studies of prospective samples starting at an early age are needed to resolve this issue. It is also unclear whether individuals who report body dissatisfaction and drive for thinness are more prone to report problems with all aspects of their lives, exhibiting excessive sensibility or vulnerability to negative feelings, or whether their lives are actually fraught with unpleasant life experiences.

Because the current study population arises from the fourth wave of a longitudinal questionnaire-based follow-up, men and individuals with a low level of education were somewhat underrepresented in the study sample. Still, our population coverage was better than in many other twin studies (Lykken, McGue, & Tellegen, 1987). Furthermore, the EDI measures may not optimally target areas of male appearance concerns (Anderson & Bulik, 2004). Males and females may also interpret differently DT and BD questions in the EDI. Also, the BD and DT scores were more normally distributed and lent themselves more readily to twin modeling in females, meaning that parameter estimates and CIs for females are more precise than for males. In males, the dichotomization of the highly skewed BD and DT measures at the 75th percentile was essentially arbitrary, and this threshold of “affection status” could of course have been defined differently.

Some caveats apply to interpreting BMI-related measures derived from a twin population. MZ twins are, typically, smaller at birth than DZ twins. In an earlier analysis of this longitudinal sample, the size difference seemed to persist until the end of puberty in males (Pietiläinen et al., 1999). However, at 22–27, the differences in BMI means between male MZ and DZ twins were no longer statistically significant. Other studies of adult Finnish twins have also shown that the BMIs of twins and nontwins are comparable (Korkeila, Kaprio, Rissanen, & Koskenvuo, 1991; Rissanen, Heliovaaara, & Aromaa, 1988), and our EDI subscale means are in line with age and gender-specific norms from the Finnish nontwin EDI validation sample (Charpentier, Pia, personal communication, 2003). All this suggests that conclusions derived from the twin population can be extended to the entire population.

There are several other limitations to the current study. Our findings regarding body dissatisfaction and drive for thinness in a nonclinical population may be culture dependent. Levels of body dissatisfaction and drive for thinness may differ from those in other countries, and in particular, to those in non-Western cultures. Extending conclusions from this sample to other populations requires caution. However, our heritability estimates are very similar to those obtained in British and U.S. female samples, which supports the notion that heritabilities of these traits may be fairly robust within Western societies. Similarly, applicability of these results may be age and cohort specific. Finally, because our main outcome measures, body dissatisfaction and drive for thinness, were measured only once, the temporal course and stability of these traits are unclear in this sample, and the direction of causality remains unclear.

There are distinct gender differences in the heritability patterns of body dissatisfaction and drive
for thinness. In women, these traits are moderately to highly genetic, whereas in men, they are purely environmentally influenced. In both genders, high BD and DT scores are associated with marked psychological distress. To alleviate psychological suffering, environmental determinants of male body and weight dissatisfaction merit further study.

References


