Published in final edited form as:
J Abnorm Psychol. 2009 November ; 118(4): 797–805. doi:10.1037/a0017204.

Genetic and Environmental Influences on Disordered Eating: An Adoption Study

Kelly L. Klump, Ph.D.[1], **Jessica L. Suisman, B.S.**[1], **S. Alexandra Burt, Ph.D.**[1], **Matt McGue, Ph.D.**[2], and **William G. Iacono, Ph.D.**[2]

[1]Department of Psychology, Michigan State University, East Lansing, MI

[2]Department of Psychology, University of Minnesota, Minneapolis, MN

Abstract

Twin studies indicate significant genetic, but little shared environmental, influences on eating disorders. However, critics argue that study limitations constrain the conclusions that can be drawn. Adoption studies avoid many of these limitations, but to date, no adoption studies of eating pathology have been conducted. The current study was the first adoption study to examine genetic/environmental effects for disordered eating. Participants included 123 adopted and 56 biological female sibling pairs. Disordered eating (i.e., overall eating pathology, body dissatisfaction, weight preoccupation, binge eating) was assessed using the Minnesota Eating Behaviors Survey. Biometric model-fitting indicated significant genetic influences (59–82%) on all forms of disordered eating, with nonshared environmental factors accounting for the remaining variance. Shared environmental factors did not contribute significantly to any disordered eating symptom. Our findings bolster those from twin studies and provide critical evidence of significant genetic effects on disordered eating symptoms.

Keywords

eating disorders; anorexia nervosa; bulimia nervosa; genetic; adoption study

Twin studies suggest moderate-to-high heritability (i.e., ~50–85%) of anorexia nervosa (AN; Bulik, et al., 2006; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik; 2001; Mazzeo, et al., 2009; Wade, Bulik, Neale, & Kendler, 2000) bulimia nervosa (BN; Baker, Mazzeo, & Kendler, 2007; Bulik, Sullivan, & Kendler, 1998; Bulik, Sullivan, Wade, & Kendler, 2000; Kendler, et al.,1991; Kendler, et al., 1995; and disordered eating symptoms (e.g., weight preoccupation, body dissatisfaction, binge eating, the use of compensatory behaviors; Keski-Rahkonen, et al., 2005; Klump, McGue, & Iacono, 2002; Klump, Burt, McGue, & Iacono, 2007; Reichborn-Kjennerud et al., 2003; Rutherford, McGuffin, Katz, & Murray, 1993; Silberg & Bulik, 2005; Spanos, Burt, & Klump, submitted; Sullivan, Bulik, & Kendler, 1998; Wade, Martin, et al., 2000; Wade et al., 1999) during adolescence and adulthood. Indeed, over 30 such twin studies have been conducted, and all but two (focusing on cognitive criteria for AN and BN; see Reichborn-Kjennerud et al., 2004 and Wade, Martin, & Tiggeman, 1998) found evidence for significant genetic effects.

Nonetheless, past critics of these studies have suggested that methodological limitations constrain the conclusions that can be drawn. For example, some (Fairburn, Cowen, & Harrison,

Corresponding Author: Kelly Klump, Ph.D., Department of Psychology, Michigan State University, 107B Psychology Building, East Lansing, MI 48824-1116, PH: 517-432-9861, FAX: 517-432-2476, klump@msu.edu.

1999) have noted that heritability estimates vary across twin studies and even within the same twin study (see Bulik, et al., 1998; Kendler, et al., 1991; Kendler, et al., 1995), making it difficult to determine the actual contribution of genetic factors. Others point out that the 95% confidence intervals for genetic effects have sometimes included 0, suggesting a lack of statistically significant effects (Levine & Smolak, 2006). The validity (or invalidity) of the Equal Environments Assumption is also frequently cited as a stumbling block to establishing the presence of genetic influences (Fairburn et al., 1999; Levine & Smolak, 2006). Despite evidence to the contrary (Bulik et al., 1998; Bulik et al., 2000; Kendler, Neale, Kessler, Heath & Eaves, 1993; Klump, Holly, Iacono, McGue, & Willson, 2000; Wade & Bulik; 2007), increased physical and/or environmental similarity of MZ relative to DZ twins has been theorized to artificially inflate MZ twin concordance and heritability estimates for eating disorders (Fairburn et al., 1999; Levine & Smolak, 2006). Although some critics have since acknowledged the likely presence of genetic effects (Fairburn & Harrison, 2003), earlier critiques continue to be cited and lingering doubts about the heritability of eating disorders remain (see Levine & Smolak, 2006).

Researchers also question the ability of twin studies to detect shared environmental effects on eating disorders. Shared environmental factors are those environmental influences that create behavioral similarities between siblings (Plomin, Asbury, & Dunn, 2001). Because both genetic and shared environmental influences make twins similar, the power of the classical twin study to detect shared environmental effects is relatively low, particularly in the presence of additive genetic influences (Martin, Eaves, Kearsey, & Davies, 1978). This diminished power may have contributed to the somewhat discrepant results regarding shared environmental influences on eating pathology. Indeed, estimates of shared environmental influences during adolescence and young adulthood have been variable. Some studies have found evidence for shared environmental influences (Bulik et al., 2000; Bulik et al., 2006; Kendler et al., 1991; Kendler et al., 1995; Klump, Burt, et al., 2007; Mazzeo et al., 2009; Reichborn-Kjennerud et al., 2004; Wade et al., 1998; Wade, Martin et al., 2000), but most have not (Bulik et al., 1998; Javaras, et al., 2008; Keski-Rahkonen et al., 2005; Klump et al., 2001; Klump et al., 2002; Kortegaard et al., 2001; Reichborn-Kjennerud et al., 2003; Reichborn-Kjennerud et al., 2004; Rutherford et al., 1993; Spanos et al., submitted; Sullivan et al., 1998; Wade et al., 1999; Wade, Bulik, et al., 2000).

A powerful method for addressing these concerns is an adoption study design. Adoption studies compare similarity for a trait or disorder in biological versus adoptive relatives to determine the degree to which genetic and shared environmental factors contribute to a phenotype (Plomin et al., 2001). Genetic factors are implicated if biological relatives are more highly correlated for a trait or disorder than are adoptive relatives. By contrast, shared environmental effects are implicated if adoptive and biological relatives are similarly correlated for a trait or disorder. Perhaps more importantly, because they do not share segregating genetic material, significant associations between adoptive siblings function as a “direct” estimate of shared environmental influences. Adoption studies are thus a particularly powerful method for the detection of shared environmental effects as compared to any other behavioral genetic design (Plomin et al., 2001).

Despite the power of the adoption study to confirm the presence of genetic and shared environmental effects, to date, no adoption studies have examined eating pathology. Thus, the aim of the current study was to use an adoption study design to examine genetic and environmental influences on disordered eating. We compared similarity for a range of disordered eating symptoms (e.g., overall levels of disordered eating, weight preoccupation, body dissatisfaction, binge eating) in biological versus adopted female sibling pairs.

Methods

Participants

Participants were all female-female sibling pairs drawn from the first follow-up wave of the Sibling Interaction and Behavior Study (SIBS) from the Minnesota Center for Twin and Family Research (MCTFR). The SIBS study is a population-based, longitudinal study of Minnesota adoptive (i.e., non-genetically related) and biological adolescent siblings and their parents (McGue et al., 2007). Adoptive families were recruited in collaboration with three major Minnesota adoption agencies and were required to have: 1) an adopted sibling between the ages of 11 and 21 at the time of the intake assessment who had been placed in the adoptive home prior to age 2; and 2) a second sibling in the home who was not biologically related to the adopted individual (i.e., the second child was either biologically related to one or both parents or had been adopted prior to age 2). Adopted siblings were placed in the adoptive homes at a mean age of 4.7 months (SD = 3.4 months). Non-adoptive families were located using Minnesota state birth records and were selected to have sibling pairs that compared in age and gender to the adoptive sibling pairs. Both types of families were excluded if they did not live within driving distance of the University of Minnesota laboratory, siblings differed by more than 5 years in age, or either sibling had a physical or mental handicap that would prevent them from completing the intake assessment.

Once families were identified through birth records or adoption agencies, public records (e.g., phone directories) were used to locate current addresses for the families. This method resulted in the correct identification of addresses for 90% of the adoptive and 85% of the biological families. A parent was then interviewed to determine eligibility. Of the eligible families invited to participate, 63.2% (n = 409) of adopted families and 57.3% (n = 208) of non-adoptive families elected to participate in the study. Participating families were generally representative of the surrounding population (i.e., the broader Minneapolis and St. Paul, MN area) and of eligible families from which they were drawn. Among a broad range of variables examined (e.g., parental education, occupational status, reported child behavioral disorders), only one difference emerged between participating families and either non-participating families or families with two or more children in the surrounding area: participating mothers from biological families were significantly more likely to have a college degree (43.8%) than non-participating biological mothers (28.6%).

Participating biological and adoptive families also appeared to be similar to each other on several key characteristics. Rates of psychological disorders (e.g., major depression, alcohol use disorders) generally did not differ significantly between parents of adoptive and non-adoptive families. Adopted and biological children were not significantly different on a range of measured outcome variables (e.g., IQ, delinquency, and drug use). However, parents from adoptive families were older, more likely to have a college degree, and had higher occupational status than parents from non-adoptive families (McGue et al., 2007). In addition, due to high rates of international adoptions, adopted children were primarily of Asian ancestry (66%), while non-adoptive children were primarily Caucasian (95%). Disordered eating measures were administered to female siblings during the first follow-up assessment (i.e., they were not administered to males and they were not administered at the intake assessment). Thus, the sample for the present study was smaller than that for the full SIBS project and included 123 adopted female sibling pairs and 56 biological female sibling pairs (29% of the full sample). Twenty-two adopted pairs and 5 biological pairs were “incomplete” with one sibling missing data on the disordered eating measure (see metrics for determining missing data below). However, levels of disordered eating did not differ significantly between complete and incomplete sibling pairs (all p’s > .05; Cohen’s effect size d = .11–.23).

Participants ranged in age from 14–24 years, with a mean age of 18.70 (SD = 2.25). Table 1 includes the breakdown of ethnicity by sibling pair type. The vast majority of biological siblings were Caucasian (> 90%). By contrast, half (50%) of the adopted pairs were Asian, with a smaller number who were either Caucasian (15%) or "Other" (e.g., Hispanic, East Indian, Native American Indian, Pacific Islander) (5%). There was also a subsample (< 30% of total sample) of adopted pairs with siblings of different ethnicity, including pairs in which one sibling was Caucasian while the other was Asian or of "Other" ethnicity, and pairs in which one sibling was Asian whereas the sibling was of "Other" ethnicity (see Table 1). Finally, although the majority of adopted pairs (59%) included siblings who were both adopted, some pairs included one adopted sibling and one sibling who was biologically related to one or both of her rearing parents. Importantly, however, results did not differ when these later pairs were excluded (data not shown), suggesting that their inclusion did not unduly influence our findings.

Measures

Disordered Eating—Disordered eating was assessed with the 30-item Minnesota Eating Behaviors Survey (Klump, McGue, & Iacono, 2000; von Ranson, Klump, Iacono, & McGue, 2005). The MEBS[a] includes a total score assessing overall levels of disordered eating, as well as four subscales measuring body dissatisfaction (6 items assessing dissatisfaction with the size and/or shape of one's body), weight preoccupation (8 items assessing preoccupation with dieting, weight, and the pursuit of thinness), binge eating (7 items assessing the tendency to engage in episodes of overeating as well as thoughts about binge eating), and compensatory behavior (6 items assessing the tendency to use or to contemplate using inappropriate compensatory behaviors such as self-induced vomiting and laxatives to control weight). The MEBS is scored in the traditional pathological direction, with higher scores indicating more of the measured construct.

Although we focused on disordered eating rather than eating disorder diagnoses per se, disordered eating symptoms contribute to diagnostic criteria for AN and BN (e.g., weight preoccupation, binge eating) and they have been studied extensively in previous twin research (Culbert, Burt, McGue, Iacono, & Klump, submitted; Klump, McGue, et al., 2000; Klump et al., 2002; Klump, McGue, & Iacono, 2003; Klump, Burt, et al., 2007). The use of symptoms rather than diagnoses also allowed for relatively greater statistical power, given that the number of siblings who would be expected to have AN or BN would be low. Moreover, continuous (rather than categorical) measures provide greater statistical power for detecting genetic effects in behavioral genetic models (see Neale & Cardon, 1992).

With the exception of the compensatory behavior subscale (see below), the MEBS scales exhibit good psychometric properties. Internal consistency in adolescent girls in past studies (Klump, McGue, et al., 2000; von Ranson et al., 2005) and the current sample (see Table 2) is adequate, and convergent validity with related self-report measures of disordered eating (e.g., the Eating Disorders Examination Questionnaire; von Ranson et al., 2005) is strong (i.e., r's > .65; von Ranson et al., 2005). Three-year stability during middle and late adolescence has been shown to be moderate (~.40; Klump, McGue, et al., 2000), which is reasonable given the length of the test-retest interval and the likelihood of some change in disordered eating across this time period. The MEBS total score, body dissatisfaction, weight preoccupation, and binge eating scales also have been found to discriminate successfully between women with AN, BN, and control subjects (Klump, McGue, et al., 2000; von Ranson et al., 2005), suggesting that

[a]The Minnesota Eating Behavior Survey (previously known as the Minnesota Eating Disorder Inventory) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

they tap clinically relevant disordered eating symptoms. The lone exception is the compensatory behavior scale. In current (see Table 2) and past (e.g., see Klump, McGue, et al, 2000) studies of young adolescent girls, the internal consistency of the compensatory behavior subscale is poor (particularly in adoptive siblings) and item endorsement is low. Consequently, we excluded the compensatory behavior subscale from further analysis.

Statistical Analyses

The MEBS total scores were prorated for siblings who were missing ≤10% of the items. Scores were coded as missing for siblings missing more than 10% of items. Given the relatively small number of items on the MEBS subscales (range = 6 – 8 items), subscale scores from siblings with one or more missing items were not prorated, but were instead coded as missing. Thus, sample sizes vary somewhat across subscale analyses. Scores on the binge eating subscale were transformed ($\log_{10} x + 1$) prior to analysis due to the positive skew of the data.

Our sample was somewhat diverse in age and ethnicity. We therefore controlled for age by regressing out age from each sibling's MEBS score prior to analysis. This ensured that our results were not unduly influenced by differences in age across or within sibling pairs. We controlled for ethnicity by conducting analyses in the full sample, as well as in two subsamples: 1) pairs in which siblings "matched" on ethnicity (i.e., Caucasian-Caucasian or Asian-Asian pairs); and 2) pairs in which both siblings were Caucasian. Notably, we were unable to separately examine pairs in which both siblings were Asian because of the lack of biological pairs (N = 0 Asian-Asian biological siblings) to use for a comparison group. Nonetheless, sibling correlations in the adopted Asian pairs (range = .08 - .007) were similar to those of the other adoptive groups (see Table 3), and model fitting results in the other two subsamples (see above) allowed us to directly evaluate whether differences in ethnicity within or between pairs could account for our results.

We first calculated descriptive statistics to examine potential differences in means and/or variances for the MEBS scales between biological and adoptive sibling pairs. Sibling correlations were computed by sibling type to provide initial indications of genetic and environmental effects. Univariate structural equation models were then fit to the raw sibling data using the full maximum-likelihood method (FIML) and the Mx software program (Neale, 1995). The raw data option in Mx treats missing data as missing at random (MAR). Although it is sometimes difficult to determine if data are truly MAR (i.e., the probability of missing data on an item is unrelated to an individual's level of disordered eating), the use of FIML produces less biased and more efficient and consistent estimates than other techniques (e.g., pairwise or listwise deletion) in the face of missing data (Little & Rubin, 1987).

We used the univariate sibling models to estimate the relative contribution of additive genetic (A; genetic influences that add across segregating genes), shared environmental (C; environmental influences that are shared by siblings and are a source of behavioral similarity regardless of genes shared), and nonshared environmental (E; environmental influences that are not shared by siblings and are a source of behavioral dissimilarity) factors to the total phenotypic variance in each MEBS scale. Notably, because measurement error will make siblings different from each other, the E estimate includes this error of measurement. Under the full ACE model, the expected biological sibling covariance is ½A + C, the expected adopted sibling covariance is C, and the expected variance is A + C + E for biological and adoptive siblings.

The full ACE model was initially fit to the data and then compared to three submodels: one including additive genetic and nonshared environmental factors only (AE), another including only shared and nonshared environmental effects (CE), and a final model including only nonshared environmental factors (E). Model fit was determined by examining the difference

in the minimized value of minus twice the log-likelihood (−2lnL) between nested models. This difference yields a likelihood-ratio χ^2 test that is compared to the standard chi-square distribution to test the significance of the more restrictive model. A nonsignificant χ^2 indicates that the more restrictive model provides an appropriate fit to the data. In addition, models that minimize Akaike's information criterion (AIC; AIC=χ^2-2*Δdf; Akaike, 1987) are preferred, as the AIC balances model fit with parsimony.

Results

The MEBS subscale means and standard deviations by sibling pair type are presented in Table 2. There were no significant mean or variance differences in levels of disordered eating between biological and adoptive siblings. In both samples, there was ample variability in the range of scores as well as the percent of siblings (i.e., 8–29%) scoring above the MEBS scale clinical cut-offs (determined using means of individuals with eating disorders – see von Ranson et al., 2005). Indeed, a range of disordered eating (including clinical levels) was present in our sample. Comparisons of these data to those published previously suggest that disordered eating level and variability were similar to those of twins from the same recruitment region (see von Ranson et al, 2005). Collectively, these findings rule-out the possibility that our sample of adoptees had a restricted range of disordered eating symptoms, an issue that has been raised as a limitation of past adoption study research of other psychological phenotypes (see Stoolmiller, 1999).

In terms of the covariance between siblings, in the full sample, biological sibling correlations were uniformly greater than zero and moderate in magnitude (r's = .29–.46; see Table 3). By contrast, none of the adoptive sibling correlations were statistically distinguishable from zero. Differences between adoptive and biological sibling correlations were statistically significant or approached significance, suggesting the presence of significant genetic, but little shared environmental, influences on disordered eating. Importantly, the pattern of sibling correlations in the two subsamples was highly similar to that of the full sample (see Table 3), suggesting that genetic influences were prominent in these samples as well.

Findings from the univariate models are presented in Table 4. Parameter estimates from the ACE model in the full sample corroborated the sibling correlations by suggesting substantial genetic effects but little-to-no shared environmental influences on all forms of disordered eating. Indeed, while shared environmental estimates tended to be low and include 0 in the 95% confidence intervals, estimates of genetic effects were greater than 50% and rarely included 0. Model fit comparisons confirmed these impressions. The best fitting model for all types of disordered eating was the AE model, as evidenced by non-significant chi-square change tests and the lowest AIC values. The improved fit of this model offers further support for the notion that shared environmental factors did not meaningfully contribute to the variance in any type of disordered eating. By contrast, parameter estimates in the AE model indicated significant genetic influences (59–82%) on all types of disordered eating symptoms, with nonshared environmental factors accounting for the remaining variance.

Notably, findings in the subsamples were essentially identical to those of the full sample. Indeed, in all cases, the AE model provided the best fit to the data. Although confidence intervals were broader, and genetic estimates were higher in the subsamples as compared to the full sample (likely due to smaller samples, loss of power, and correspondingly imprecise point estimates), genetic and nonshared environmental effects were again prominent with little shared environmental influence.

Discussion

This study is the first adoption study of disordered eating. Findings provide critical evidence of substantial genetic effects on a range of disordered eating. Our heritability estimates were similar to those found in twin studies of eating disorders (Bulik et al., 1998; Klump et al., 2001; Kortegaard et al., 2001; Wade, Bulik, et al., 2000) and disordered eating symptoms (Keski-Rahkonen et al., 2005; Klump et al., 2002; Reichborn-Kjennerud et al., 2003; Rutherford et al., 1993; Spanos et al., submitted; Sullivan et al., 1998; Wade et al., 1998; Wade et al., 1999). This is somewhat surprising, given that heritability estimates from adoption studies tend to be lower than those from twin studies (Burt, in press). Nonetheless, the consistency of results suggests that twin study findings are unlikely to be unduly influenced by limitations cited by critics (Fairburn et al., 1999; Levine & Smolak, 2006).

In aggregate then, twin and adoption studies have established genes as significant risk factors for the development of eating disorders. Heritability estimates obtained in this and other studies are on par with those for other forms of psychopathology (e.g., schizophrenia, Sullivan, Kendler, & Neale, 2003; bipolar disorder, Edvardsen et al., 2008) considered to be biologically based. The consistency of findings is striking and underscores the need for molecular genetic work identifying genes contributing to the genetic variance. Importantly, genetic research to date has produced somewhat inconsistent results (Bulik & Tozzi, 2004; Hinney, Friedel, Ramschmidt, & Hebebrand, 2004; Mazzeo, Slof-Op't Landt; van Furth, & Bulik, 2006), particularly with regard to BN (Klump & Culbert, 2007). Although this is not unlike other areas of psychiatry (e.g., schizophrenia; Crow, 2008) it has led some to argue against genetic effects on disordered eating (Levine & Smolak, 2006). Our data suggest a different conclusion, in that discrepancies likely reflect the need for more sophisticated approaches (e.g., genome-wide association studies; studies of epigenetic effects) rather than a lack of genetic effect per se. Our results should be interpreted as critical support for these investigations.

Our findings also provide an important lack of support for shared environmental influences on disordered eating. Correlations between adoptive siblings, which function as "direct" estimates of shared environmental effects, were uniformly small and non-significant. Moreover, shared environmental estimates in the full ACE model were non-significant and, not surprisingly, were able to be dropped in the final model (i.e., the AE model). These results provide **strong** support for findings from most twin studies that have suggested negligible shared environmental main effects on the development of disordered eating during adolescence and young adulthood.

However, there are three important caveats to these findings. First, although our findings suggest that shared environmental factors do not exert strong main effects during adolescence, they may impact the development of disordered eating through interactions with other etiologic factors. For example, family conflict may interact with genetic risk to increase susceptibility to disordered eating in both siblings (for an example in other areas of psychopathology, see Burt, McGue, Krueger, & Iacono, 2007). Likewise, family conflict could interact with a nonshared environmental factor (i.e., peer group) to increase susceptibility to disordered eating in one sibling relative to another. Within twin and adoption designs, gene × shared environment interactions are partitioned into the additive genetic variance (Burt, in press), while shared × nonshared environment interactions are partitioned into the nonshared environmental estimate. In both cases, there will be a corresponding decrease in the main effects of shared environmental effects in traditional decompositions of variance. Thus, while our findings rule out the main effects of shared environment, they do not rule out interactive effects that may be equally important and potent.

Second, and building upon the above points, the lack of shared environmental effects says very little about the role of culture in the development of eating disorders. A common misconception is to think that broad sociocultural factors (e.g., the thin beauty ideal for women) are implicated if shared environment is significant, and are excluded if shared environment is negligible. To the contrary, cultural factors may influence disordered eating in the face of non-significant shared environmental effects. For example, cultural factors (i.e., pressures for thinness) may increase risk for disordered eating in genetically susceptible siblings via gene × environment interactions. Indeed, in the absence of a culture that emphasizes thinness, phenotypic and genetic expression of disordered eating may be diminished (Keel & Klump, 2003). In addition, cultural factors may also influence disordered eating through nonshared environmental mechanisms. The prototypical example in this regard is participation in weight-focused sports. A sibling who participates in ballet will presumably experience more pressures to be thin than her sister who is a softball player. In this example, the cultural factor is a nonshared rather than shared environmental effect and is therefore included in the nonshared environmental variance that is significant (see Table 4) for the development of disordered eating.

Finally, while shared environment does not appear to be important during late adolescence/young adulthood, it has been found to be critical during other developmental stages. Data from two twin registries (i.e., see Culbert et al, submitted; Klump, Burt, et al., 2007; Klump, McGue, et al., 2000; Klump, Perkins, Burt, McGue, & Iacono, 2007; Klump, et al., submitted; Klump, McGue, & Iacono, 2003) confirm that the shared environment accounts for approximately 50% of the variance in disordered eating in girls before puberty. Our sample consisted of adolescent and young adult siblings; thus, our findings do not speak to the presence of shared environmental effects at other developmental stages (i.e., before adolescence or in middle adulthood). Additional adoption studies in these age ranges are needed to confirm twin study results. However, given the consistency of prior findings, it is likely that disordered eating shows important shared environmental effects during the pre-adolescent and pre-pubertal periods.

We should note several limitations of our work. First, sample sizes were relatively small, resulting in wide confidence intervals for some estimates. Thus, point estimates for genetic and nonshared environmental factors may be somewhat unstable, particularly in the subsamples where sample sizes were reduced. However, estimates for these effects are similar to those obtained in large, population-based twin studies (Bulik et al., 1998; Bulik et al., 2006; Klump, McGue, et al., 2000; Wade & Bulik, 2007). Moreover, it should be noted that our sample size likely did not affect our selection of the best-fitting models or estimates of shared environmental effects, as the adoptive sibling correlations (which are direct estimates of shared environmental effects) were estimated to be 0. Thus, regardless of our sample size, model-fit comparisons would have preferred the AE model and indicated no significant shared environmental effects on disordered eating.

Second, our sample was more diverse in age and ethnicity than those included in most twin studies of disordered eating. However, past twin data suggest no differences in genetic and environmental influences on disordered eating from post-puberty (Klump, Burt, et al., 2007) into young adulthood (Klump et al., submitted). Even so, we regressed out age from all sibling scores to address this concern. We also conducted subsample analyses to confirm that ethnicity did not affect findings. Thus, we feel our findings were not unduly influenced by our inclusion of a wider range of ages and ethnicities than examined in prior twin work. Nonetheless, it is important to note that we only examined siblings residing in Minnesota from primarily middle class backgrounds. Although this sample appeared to be representative of families in the surrounding areas (see Participants above), additional research is needed to confirm that our results are generalizable to other regions of the U.S. and world. Heritability estimates are population statistics that can vary by population and environmental circumstances. For

example, a recent study of female twins in Japan suggested significant shared environmental (but little genetic) influences on personality traits (e.g., interoceptive awareness, maturity fears, feelings of ineffectiveness) associated with eating disorders (Kamakura, Ando, Ono, & Maekawa, 2003). Likewise, research in other areas of psychology (e.g., IQ) found lower heritability in samples from disadvantaged backgrounds (e.g., low family income) (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). In the case of disordered eating, heritability estimates may vary when examining individuals from other cultures (e.g., non-Western nations; see Keel & Klump, 2003) and/or socioeconomic backgrounds (e.g., low family income). Future research should directly examine this possibility. This is particularly true given that twin studies of eating disorder and disordered eating focus almost exclusively on middle class samples from Western nations (e.g., the U.S., United Kingdom, Australia, Sweden; Bulik et al., 2000). These studies clearly show the importance of genetic influences for disordered eating within these cultures, but little is known about the heritability of disordered eating outside of a Western context.

Third, we did not assess the biological relatives (i.e., parents, siblings) of the adoptees. Although identification and assessment of these types of relatives present significant logistical difficulties, their inclusion would have allowed us to rule out the possibility that our heritability estimates were inflated due to increased environmental similarity in biological versus adoptive siblings. Essentially, we would have been able to rule out the equivalent of the Equal Environments Assumption for adoption studies. It is important to note, however, that our heritability estimates are on par with those from previous large-scale twin studies where the Equal Environments Assumption has been upheld (Bulik et al., 2000; Bulik et al., 1998; Kendler et al., 1993; Klump, Holly, et al., 2000; Wade & Bulik, 2007). In addition, violations of the Equal Environments Assumption would not affect our estimates of shared environment, as the near zero adoptive sibling correlations confirm a lack of shared environmental effects in our sample. Thus, although future work should aim to include biological relatives of adoptees, our findings likely represent reasonable estimates of the degree of genetic and environmental influences on disordered eating

Finally, we examined disordered eating symptoms rather than AN or BN per se, and we only examined them in women. Findings therefore may not generalize to men or populations of individuals with clinical eating disorders. However, our genetic and environmental estimates are highly consistent with those obtained in most twin studies of men (see Slane, Burt, & Klump, submitted; Reichborn-Kjennerud et al., 2003; , Reichborn-Kjennerud et al., 2004; Tholin, Rasmussen, Tynelius, & Karlsson, 2005) as well as twins studies of AN and BN (i.e., heritability ~ 50–83%). Thus, while the limitation still deserves note, accumulating data suggest that the genetic and environmental architecture underlying risk for eating pathology is similar across sex and the continuum of severity of eating disorders.

Before ending, it is important to highlight implications of our findings. Perhaps most importantly, we hope that our work will help change perceptions of eating disorders as purely psychosocial phenomena. Currently, some US legislation and insurance statutes limit mental health parity for eating disorders because they are not considered to be "biologically based" (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). While no psychiatric disorder is entirely "biologically based", we hope that our findings will contribute to increased recognition of the genetic basis for eating disorders and the ways in which genes may interact with environmental factors to create differential risk.

Acknowledgments

This research was supported by US Public Health Service grants (AA 11886; MH 066140) from the National Institute of Health awarded to Drs. McGue and Iacono.

References

Akaike H. Factor analysis and aic. Psychometrika 1987;52:317–332.

Baker JH, Mazzeo SE, Kendler KS. Association between broadly defined bulimia nervosa and drug use disorders: Common genetic and environmental influences. International Journal of Eating Disorders 2007;40:673–678. [PubMed: 17868121]

Bulik CM, Sullivan PF, Kendler KS. Heritability of binge-eating and broadly defined bulimia nervosa. Biological Psychiatry 1998;44:1210–1218. [PubMed: 9861464]

Bulik CM, Sullivan PF, Wade TD, Kendler KS. Twin studies of eating disorders: A review. International Journal of Eating Disorders 2000;27:1–20. [PubMed: 10590444]

Bulik CM, Tozzi F. Genetics in eating disorders: State of the science. CNS Spectrums 2004;9:511–515. [PubMed: 15208510]

Bulik CM, Sullivan PF, Tozzi F, Furberg H, Lichtenstein P, Pedersen NL. Prevalance, heritability, and prospective risk factors for anorexia nervosa. Archives of general psychiatry 2006;63:305–312. [PubMed: 16520436]

Burt SA, McGue M, Krueger RF, Iacono WG. Environmental contributions to adolescent delinquency: A fresh look at the shared environment. Journal of Abnormal Child Psychology 2007;35:787–800. [PubMed: 17505878]

Burt SA. Rethinking environmental contributions to child and adolescent psychopathology: A meta-analysis of shared environmental influences. Psychological Bulletin. (in press).

Crow TJ. The emperors of the schizophrenia polygene have no clothes. Psychological Medicine 2008;38:1681–1685. [PubMed: 18423075]

Culbert KM, Burt SA, McGue M, Iacono WG. Puberty and the genetic diathesis of disordered eating. (submitted).

Edvardsen J, Torgersen S, Roysamb E, Lygren S, Skre I, Onstad S, et al. Heritability of bipolar spectrum disorders. Unity or heterogeneity. Journal of Affective Disorders 2008;106:229–240. [PubMed: 17692389]

Fairburn CG, Cowen PJ, Harrison PJ. Twin studies and the etiology of eating disorders. International Journal of Eating Disorders 1999;26:349–358. [PubMed: 10550776]

Fairburn CG, Harrison PJ. Eating disorders. The Lancet 2003;361:407–416.

Hinney A, Friedel S, Remschmidt H, Hebebrand J. Genetic risk factors in eating disorders. American Journal of Pharmacogenomics 2004;4:209–223. [PubMed: 15287815]

Javaras KN, Laird NM, Reichborn-Kjennerud T, Bulik CM, Pope HG, Hudson JI. Familiality and heritability of binge eating disorder: Results of a case-control family study and a twin study. International Journal of Eating Disorders 2008;41:174–179. [PubMed: 18095307]

Kamakura T, Ando J, Ono Y, Maekawa H. A twin study of genetic and environmental influences on psychological traits of eating disorders in a Japanese female sample. Twin Research 2003;6:292–296. [PubMed: 14511435]

Keel PK, Klump KL. Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. Psychological Bulletin 2003;129:747–769. [PubMed: 12956542]

Kendler KS, MacLean C, Neale MC, Kessler RC, Heath AC, Eaves LJ. The genetic epidemiology of bulimia nervosa. American Journal of Psychiatry 1991;148:1627–1635. [PubMed: 1842216]

Kendler KS, Neale MC, Kessler RC, Heath AC, Eaves LJ. A test of the equal-environment assumption in twin studies of psychiatric illness. Behavior Genetics 1993;23:21–27. [PubMed: 8476388]

Kendler KS, Walters EE, Neale MC, Kessler R, Heath A, Eaves L. The structure of genetic and environmental risk factors for six major psychiatric disorders in women. Archives of general psychiatry 1995;52:374–383. [PubMed: 7726718]

Keski-Rahkonen A, Bulik CM, Neale BM, Rose RJ, Rissanen A, Kaprio J. Body dissatisfaction and drive for thinness in young adult twins. International Journal of Eating Disorders 2005;37:188–199. [PubMed: 15822080]

Klump KL, Holly A, Iacono WG, McGue M, Willson LE. Physical similarity and twin resemblance for eating attitudes and behaviors: A test of the Equal Environments Assumption. Behavior Genetics 2000;30:51–58. [PubMed: 10934799]

Klump KL, McGue M, Iacono WG. Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. Journal of Abnormal Psychology 2000;109:239–251. [PubMed: 10895562]

Klump KL, Miller KB, Keel PK, McGue M, Iacono WG. Genetic and environmental influences on anorexia nervosa syndromes in a population-based twin sample. Psychological Medicine 2001;31:737–740. [PubMed: 11352375]

Klump KL, McGue M, Iacono WG. Genetic relationships between personality and disordered eating. Journal of Abnormal Psychology 2002;111:380–389. [PubMed: 12003459]

Klump KL, McGue M, Iacono WG. Differential heritability of eating pathology in pre-pubertal versus pubertal twins. International Journal of Eating Disorders 2003;33:287–292. [PubMed: 12655625]

Klump KL, Burt SA, McGue M, Iacono WG. Changes in genetic and environmental influences on disordered eating across adolescence. Archives of general psychiatry 2007;64:1409–1415. [PubMed: 18056549]

Klump KL, Culbert KM. Molecular genetic studies of eating disorders: Current status and future directions. Current Directions in Psychological Science 2007;16:37–41.

Klump KL, Perkins PS, Burt SA, McGue M, Iacono WG. Puberty moderates genetic influences on disordered eating. Psychological Medicine 2007;37:627–634. [PubMed: 17335640]

Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for Eating Disorders position paper: Eating disorders are serious mental illnesses. International Journal of Eating Disorders 2009;42:97–103. [PubMed: 18951455]

Klump KL, Burt SA, Spanos A, McGue M, Iacono WG, Wade TM. Genetic and environmental influences on eating disorder symptoms: Developmental differences from childhood to middle adulthood for weight and shape concerns. (submitted).

Kortegaard LS, Hoerder K, Joergensen J, Gillberg C, Kyvik KO. A preliminary population-based twin study of self-reported eating disorder. Psychological Medicine 2001;31:361–364. [PubMed: 11232922]

Levine, MP.; Smolak, L. The prevention of eating problems and eating disorders: Theory, research, and practice. Marwah, NJ: Lawrence Erlbaum Associates; 2006.

Little, RJA.; Rubin, DB. Statistical Analysis with Missing Data. New York: John Wiley and Sons; 1987.

Martin NG, Eaves LJ, Kearsey MJ, Davies P. The power of the classical twin study. Heredity 1978;40:97–116. [PubMed: 272366]

Mazzeo, SE.; Slof-Op't Landt, MCT.; van Furth, EF.; Bulik, CM. Genetics of eating disorders. Annual Review of Eating Disorders: Part 2. In: Wonderlich, S.; Mitchell, J.; de Zwaan, M.; Steiger, H., editors. Annual Review of Eating Disorders. Oxford: Radcliffe Publishing Ltd.; 2006.

Mazzeo SE, Mitchell KS, Bulik CM, Reichborn-Kjennerud T, Kendler KS, Neale MC. Assessing the heritability of anorexia nervosa symptoms using a marginal maximal likelihood approach. Psychological Medicine 2009;39:463–473. [PubMed: 18485259]

McGue M, Keyes M, Sharma A, Elkins I, Legrand L, Johnson W, et al. The environments of adopted and non-adopted youth: Evidence on range restriction from the sibling interaction and behavior study (SIBS). Behavior Genetics 2007;37:449–462. [PubMed: 17279339]

Neale, MC. Mx: Statistical Modeling. Vol. 3rd Edition. Richmond, VA: Department of Psychiatry; 1995. 23298

Neale, MC.; Cardon, LR. Methodology for Genetic Studies of Twins and Families. Dordrecht, The Netherlands: Kluwer Academic Publishers BV; 1992.

Plomin R, Asbury K, Dunn J. Why are children in the same family so different? Nonshared environment a decade later. Canadian Journal of Psychiatry 2001;46:225–233.

Reichborn-Kjennerud T, Bulik CM, Kendler KS, Roysamb E, Maes H, Tambs K, et al. Gender differences in binge-eating: A population based twin study. Acta Psychiatrica Scandinavica 2003;108:196–202. [PubMed: 12890274]

Reichborn-Kjennerud T, Bulik CM, Kendler KS, Roysamb E, Tambs K, Torgersen S, et al. Undue influence of weight on self-evaluation: A population-based twin study of gender differences. International Journal of Eating Disorders 2004;35:123–132. [PubMed: 14994348]

Rutherford J, McGuffin P, Katz RJ, Murray RM. Genetic influences on eating attitudes in a normal female twin pair population. Psychological Medicine 1993;23:425–436. [PubMed: 8332659]

Silberg JL, Bulik CM. The developmental association between eating disorders symptoms and symptoms of depression and anxiety in juvenile twin girls. Journal of Child Psychology and Psychiatry 2005;46:1317–1326. [PubMed: 16313432]

Slane JD, Burt SA, Klump KL. Genetic and environmental influences on disordered eating: Sex similarities and differences. (submitted).

Spanos A, Burt SA, Klump KL. Do weight and shape concerns exhibit genetic effects? Investigating discrepant findings. (submitted).

Stoolmiller M. Implications of the restricted range of family environments for estimates of heritability and nonshared environment in behavior-genetic adoption studies. Psychological Bulletin 1999;125:392–409. [PubMed: 10414224]

Sullivan PF, Bulik CM, Kendler KS. Genetic epidemiology of binging and vomiting. The British Journal of Psychiatry 1998;173:75–79. [PubMed: 9850207]

Sullivan PF, Kendler KS, Neale MC. Schizophrenia as a complex trait: Evidence from a meta-analysis of twin studies. Archives of general psychiatry 2003;60:1187–1192. [PubMed: 14662550]

Tholin S, Rasmussen F, Tynelius P, Karlsson J. Genetic and environmental influences on eating behavior: The Swedish Young Male Twins Study. American Journal of Clinical Nutrition 2005;81:564–569. [PubMed: 15755823]

Turkheimer E, Haley A, Waldron M, D'Onofrio B, Gottesman II. Socioeconomic status modifies heritability of IQ in young children. Psychological Science 2003;14:623–628. [PubMed: 14629696]

von Ranson KM, Klump KL, Iacono WG, McGue M. The Minnesota Eating Behavior Survey: A brief measure of disordered eating attitudes and behaviors. Eating Behaviors 2005;6:373–392. [PubMed: 16257811]

Wade TD, Martin NG, Tiggemann M. Genetic and environmental risk factors for the weight and shape concerns characteristic of bulimia nervosa. Psychological Medicine 1998;28:761–771. [PubMed: 9723134]

Wade TD, Martin NG, Neale MC, Tiggemann M, Treloar SA, Bucholz KK, et al. The structure of genetic and environmental risk factors for three measures of disordered eating. Psychological Medicine 1999;29:925–934. [PubMed: 10473319]

Wade TD, Bulik CM, Neale M, Kendler KS. Anorexia nervosa and major depression: Shared genetic and environmental risk factors. American Journal of Psychiatry 2000;157:469–471. [PubMed: 10698830]

Wade TD, Martin NG, Tiggemann M, Abraham S, Treloar S, Heath AC. Genetic and environmental risk factors shared between disordered eating, psychological and family variables. Personality and Individual Differences 2000;28:729–740.

Wade TD, Bulik CM. Shared genetic and environmental risk factors between undue influence of body shape and weight on self-evaluation and dimensions of perfectionism. Psychological Medicine 2007;37:635–644. [PubMed: 17176504]

Table 1

Breakdown of Ethnicity by Sibling Pair Type.

Sibling Pair Type	Number of Sibling Pairs N (%)
Biological Siblings:	
Caucasian	51 (91%)
Asian	0 (0%)
"Other"	5 (9%)
Total	**56 (100%)**
Adoptive Siblings:	
Same Ethnicity	
Caucasian	19 (15%)
Asian	62 (50%)
"Other"	6 (5%)
Subtotal	*87 (71%)*
Different Ethnicity	
Caucasian-Asian	30 (24%)
Caucasian-"Other"	3 (2%)
Asian-"Other"	3 (2%)
Subtotal	*36 (29%)*
Total	**123 (100%)**

Table 2

Means, Standard Deviations, and Score Ranges for the Minnesota Eating Behavior Survey (MEBS) Scales.

	Biological Siblings (n = 107)				Adoptive Siblings (n = 224)				Mean Comparisons			Levene's Test of Variances	
MEBS Scales	**M (SD)**	**Range**	**% ≥ cut-off**	**α**	**M (SD)**	**Range**	**% ≥ cut-off**	**α**	**t (329)**	***p***	**d**	**F (1, 329)**	***p***
Total Score **(Possible score = 0–30)**	7.56 (6.17)	0–28	**11%**	.90	7.07 (5.62)	0–24	**10%**	.88	−.73	.47	.08	1.22	.27
Weight Preoccupation **(Possible score = 0–8)**	2.95 (2.58)	0–8	**12%**	.84	2.65 (2.37)	0–8	**10%**	.81	.15	.29	.12	2.04	.15
Body Dissatisfaction **(Possible score = 0–6)**	2.22 (2.00)	0–6	**29%**	.79	1.83 (1.96)	0–6	**24%**	.82	−.68	.09	.20	.39	.54
Binge Eating **(Possible score = 0–7)**	1.40 (1.60)	0–7	**18%**	.71	1.56 (1.55)	0–7	**20%**	.67	.85	.40	.10	.03	.87
Compensatory Behavior **(Possible score = 0–5)**	0.31 (0.79)	0–5	**8%**	.67	0.35 (0.75)	0–4	**8%**	.54	.49	.62	.06	.28	.59

Note. MEBS = Minnesota Eating Behavior Survey; % ≥ **cut-off = percent of siblings who scored above the clinical cut-offs on the MEBS scales (Cut-offs: Total Score = 15.55; Weight Preoccupation = 6.11; Body Dissatisfaction = 3.95; Binge Eating = 2.53; Compensatory Behaviors = 1.53);** d = Cohen's **effect size for mean comparisons**. Although log-transformed Binge Eating scores were used in analyses, raw values are listed here for descriptive purposes.

Table 3

Sibling Correlations for Disordered Eating.

MEBS Scales	Biological Pairs	Adoptive Pairs	Test of Equality	
			z	One-tailed *p*
Full Sample:	N = 51 pairs	N = 101 pairs		
Total Score	.42**	.02	2.43	**.008**
Weight Preoccupation	.29**	.07	1.30	.10
Body Dissatisfaction	.30**	−.06	2.10	**.02**
Binge Eating	.31**	−.06	2.16	**.02**
Same Ethnicity:	N = 51 pairs	N = 66 pairs		
Total Score	.42**	−.03	2.49	**.006**
Weight Preoccupation	.29**	−.04	1.77	**.04**
Body Dissatisfaction	.30**	−.05	1.88	**.03**
Binge Eating	.31**	−.08	2.09	**.02**
Caucasian Pairs Only:	N = 47 pairs	N = 17 pairs		
Total Score	.46**	−.15	2.11	**.02**
Weight Preoccupation	.33**	−.17	1.68	.05
Body Dissatisfaction	.34**	−.18	1.75	**.04**
Binge Eating	.32**	−.12	1.47	.07

Note. MEBS = Minnesota Eating Behavior Survey; Same Ethnicity = pairs who “match” on ethnicity (i.e., either Caucasian-Caucasian or Asian-Asian pairs).

** $p < .001$. The correlation is significantly greater than zero.

Table 4

Univariate Sibling Models.

MEBS Scale	Parameter Estimates A	C	E	Model Fit Statistics −2lnL (df)	$-2\ln L_{\Delta}$ (df)	*p*	AIC
	Full Sample (*n* = 51 Biological Pairs; *n* = 101 Adoptive Pairs)						
Total Score							
ACE	.82 (.21–1.00)	.02 (.00–.20)	.17 (.00–.69)	925.09 (327)	--	--	271.09
AE	**.85 (.35–1.00)**	--	**.15 (.00–.65)**	**925.12 (328)**	**.03 (1)**	**.86**	**269.12**
CE	--	.14 (.00–.29)	.86 (.71–1.00)	931.81 (328)	6.72 (1)	.009	275.81
E	--	--	1.00 (−1.00–1.00)	935.21 (329)	10.12 (2)	.006	277.21
Weight Preoccupation							
ACE	.47 (.00–1.00)	.07 (.00–.25)	.46 (.00–.97)	930.23 (327)	--	--	276.23
AE	**.61 (.05–1.00)**	--	**.39 (.00–.95)**	**930.73 (328)**	**.50 (1)**	**.48**	**274.73**
CE	--	.14 (.00–.29)	.86 (.71–1.00)	932.19 (328)	1.96 (1)	.16	276.19
E	--	--	1.00 (−1.00–1.00)	935.24 (329)	5.01 (2)	.08	277.24
Body Dissatisfaction							
ACE	.59 (.03–1.00)	.00 (.00–.14)	.41 (.00–.94)	930.58 (327)	--	--	276.58
AE	**.59 (.06–1.00)**	--	**.41 (.00–.94)**	**930.58 (328)**	**.00 (1)**	**1.00**	**274.58**

	Parameter Estimates			Model Fit Statistics			
MEBS Scale	**A**	**C**	**E**	**−2lnL (df)**	**$-2\ln L_{\Delta}$ (df)**	***p***	**AIC**
CE	--	.06 (.00–.21)	.94 (.79–1.00)	934.78 (328)	4.20 (1)	.04	278.78
E	--	--	1.00 (−1.00–1.00)	935.33 (329)	4.75 (2)	.09	277.33
Binge Eating							
ACE	.61 (.05–1.00)	.00 (.00–.15)	.39 (.00–.91)	930.07 (327)	--	--	276.07
AE	**.61 (.09–1.00)**	**--**	**.39 (.00–.91)**	**930.07 (328)**	**.00 (1)**	**1.00**	**274.07**
CE	--	.07 (.00–.22)	.93 (.78–1.00)	934.51 (328)	4.44 (1)	.04	278.51
E	--	--	1.00 (−1.00–1.00)	935.25 (329)	5.18 (2)	.08	277.25
	Same Ethnicity Only (*n* = 51 Biological Pairs; *n* = 66 Adoptive Pairs)						
Total Score							
ACE	.95 (35–1.00)	.00 (.00–.19)	.05 (.00–.58)	699.01 (241)	--	--	217.01
AE	**.95 (.44–1.00)**	**--**	**.05 (.00–.56)**	**699.01 (242)**	**.00 (1)**	**1.00**	**215.01**
CE	--	.15 (.00–.32)	.85 (.68–1.00)	707.45 (242)	8.44 (1)	.004	223.45
E	--	--	1.00 (−1.00–1.00)	710.26 (243)	11.25 (2)	.004	224.26
Weight Preoccupation							
ACE	.68 (.02–1.00)	.00 (.00–.20)	.32 (.00–.91)	694.11 (241)	--	--	212.11
AE	**.68 (.11–1.00)**	**--**	**.32 (.00–.89)**	**694.11 (242)**	**.00 (1)**	**1.00**	**210.11**

	Parameter Estimates			Model Fit Statistics			
MEBS Scale	**A**	**C**	**E**	**−2lnL (df)**	**$-2\ln L_{\Delta}$ (df)**	***p***	**AIC**
CE	--	.11 (.00–.28)	.89 (.72–1.00)	698.12 (242)	4.01 (1)	.045	214.12
E	--	--	1.00 (−1.00–1.00)	699.46 (243)	5.35 (2)	.07	213.46
Body Dissatisfaction							
ACE	.68 (.05–1.00)	.00 (.00–.19)	.32 (.00–.88)	694.03 (241)	--	--	212.03
AE	**.68 (.14–1.00)**	**--**	**.32 (.00–.86)**	**694.03 (242)**	**.00 (1)**	**1.00**	**210.03**
CE	--	.11 (.00–.28)	.89 (.72–1.00)	698.43 (242)	4.40 (1)	.04	214.43
E	--	--	1.00 (−1.00–1.00)	699.85 (243)	5.82 (2)	.054	213.85
Binge Eating							
ACE	.64 (.04–1.00)	.00 (.00–.17)	.36 (.00–.91)	687.55 (241)	--	--	205.55
AE	**.64 (.09–1.00)**	**--**	**.36 (.00–.91)**	**687.55 (242)**	**.00 (1)**	**1.00**	**203.55**
CE	--	.09 (.00–.26)	.91 (.74–1.00)	691.79 (242)	4.24 (1)	.04	207.79
E	--	--	1.00 (−1.00–1.00)	692.66 (243)	5.11 (2)	.08	206.66
Caucasian Pairs Only (*n* = 47 Biological Pairs; *n* = 17 Adoptive Pairs)							
Total Score							
ACE	.93 (.20–1.00)	.00 (.00–.31)	.07 (.00–.62)	373.47 (130)	--	--	113.47
AE	**.93 (.42–1.00)**	**--**	**.07 (.00–.58)**	**373.47 (131)**	**.00 (1)**	**1.00**	**111.47**

	Parameter Estimates			Model Fit Statistics			
MEBS Scale	**A**	**C**	**E**	**−2lnL (df)**	**$-2\ln L_{\Delta}$ (df)**	***p***	**AIC**
CE	--	.28 (.05–.49)	.72 (.51–.95)	379.22 (131)	5.75 (1)	.02	117.22
E	--	--	1.00 (−1.00–1.00)	384.72 (132)	11.25 (2)	.004	120.72
Weight Preoccupation							
ACE	.65 (.00–1.00)	.00 (.00–.35)	.35 (.00–.93)	370.48 (130)	--	--	110.48
AE	**.65 (.10–1.00)**	--	**.35 (.00–.90)**	**370.48 (131)**	**.00 (1)**	**1.00**	**108.48**
CE	--	.21 (.00–.44)	.79 (.56–1.00)	372.96 (131)	2.48 (1)	.12	110.96
E	--	--	1.00 (−1.00–1.00)	375.81 (132)	5.33 (2)	.07	111.81
Body Dissatisfaction							
ACE	.68 (.00–1.00)	.00 (.00–.30)	.32 (.00–.90)	376.54 (130)	--	--	116.54
AE	**.68 (.14–1.00)**	--	**.32 (.00–.86)**	**376.54 (131)**	**.00 (1)**	**1.00**	**114.54**
CE	--	.20 (.00–.42)	.80 (.58–1.00)	379.69 (131)	3.15 (1)	.08	117.69
E	--	--	1.00 (−1.00–1.00)	382.38 (132)	5.84 (2)	.054	118.38
Binge Eating							
ACE	.62 (.00–1.00)	.00 (.00–.36)	.38 (.00–.95)	366.70 (130)	--	--	106.70
AE	**.62 (.09–1.00)**	--	**.38 (.00–.91)**	**366.70 (131)**	**.00 (1)**	**1.00**	**104.70**

	Parameter Estimates			Model Fit Statistics			
MEBS Scale	**A**	**C**	**E**	**−2lnL (df)**	**$-2\ln L_{\Delta}$ (df)**	***p***	**AIC**
CE	--	.21 (.00–.42)	.79 (.58–1.00)	368.78 (131)	2.08 (1)	.15	106.78
E	--	--	1.00 (−1.00–1.00)	371.80 (132)	5.10 (2)	.08	107.80

Note. MEBS = Minnesota Eating Behavior Survey; A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effect; −2lnL = −2 times the log likelihood; $-2\ln L_{\Delta}$ = the difference in −2lnL values between the full (i.e., ACE) and reduced model; AIC = Akaike's Information Criteria; Same Ethnicity = pairs who "match" on ethnicity (i.e., are either Caucasian-Caucasian or Asian-Asian pairs). The 95% confidence intervals for parameter estimates are included in parentheses. The best-fitting models are noted in bolded text.