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PARENTAL DIVORCE AND DISORDERED EATING: AN INVESTIGATION OF A GENE-ENVIRONMENT INTERACTION

By

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ABSTRACT

PARENTAL DIVORCE AND DISORDERED EATING: AN INVESTIGATION OF A GENE-ENVIRONMENT INTERACTION

By

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Objective: Previous research demonstrates an association between parental divorce and disordered eating that has traditionally been conceptualized as environmental in nature. However, it is possible that a gene-environment interaction may drive this relationship, whereby parental divorce serves as an environmental "trigger" for pre-existing genetic susceptibilities to disordered eating. This study examined this possibility by investigating whether the heritability of disordered eating is significantly higher in children living in divorced versus intact families. **Methods:** Participants included 1,810 adolescent and adult twins (mean age = 18.26) from the Michigan State University Twin Registry and the Minnesota Twin Family Study. Disordered eating was measured with the Minnesota Eating Behavior Subscale (MEBS). Univariate, twin constraint models were used to compare the heritability of disordered eating in divorced versus intact families. **Results:** A gene-environment interaction was not observed for most measures of disordered eating. However, a gene-environment interaction was suggested for body dissatisfaction, as the heritability of body dissatisfaction was higher in twins of divorced versus intact families. **Discussion**: Although gene-environment interactions do not appear to be important for relationships between divorce and most forms of disordered eating, the presence of these effects for body dissatisfaction requires further study. Future investigations should replicate our results and begin to identify the factors underlying the unique gene-environment relationships between divorce and body dissatisfaction.

TABLE OF CONTENTS

LIST OF TABLES

Parental Divorce and Disordered Eating: An Investigation of a Gene-Environment Interaction

Introduction

Research in the past several decades has examined the psychological effects of parental divorce on offspring (e.g., depression; Amato, 2001; Amato & Sobolewski, 2001; Hetherington, Bridges, & Insabella, 1998; Hetherington, Stanley-Hagan, & Anderson, 1989). One area of focus has been on the association between eating disorders and parental divorce (Billingham & Abrahams, 1998; Boumann & Yates, 1994; Herzog, 1982; Igoin-Apfelbaum, 1985; Martínez-González, et al., 2003; Yannakoulia, et al., 2008). For example, several studies have found higher rates of divorce in individuals with bulimia nervosa as compared to controls (Boumann & Yates, 1994; Herzog, 1982; Igoin-Apfelbaum, 1985), and that divorce prospectively predicts increased risk for onset of an eating disorder (e.g., eating disorder not otherwise specified, bulimia nervosa and anorexia nervosa; Martínez-González, et al., 2003). Associations between divorce and disordered eating extend to community samples as well, as body dissatisfaction and binge eating are significantly associated with parental divorce (Billingham & Abrahams, 1998; Yannakoulia, et al., 2008).

Taken together, the literature suggests that there are cross-sectional and prospective relationships between parental divorce and disordered eating that warrant additional investigation. Studies are needed to examine aspects of the relationship between divorce and disordered eating that would enhance understanding of divorce's effects. Researchers have not previously examined etiologic factors underlying associations between parental divorce and disordered eating. In general, divorce is

viewed as a stressful life event that results in the accumulation of many negative events, such as moving, decreases in socioeconomic status, changes in schools, changing relationships with parents, decreased social support, exposure to parental conflict, and loss of contact with extended family (Amato, 1993; Hetherington, 1993). Consistent with these ideas, it has been hypothesized that the effects of parental divorce on disordered eating are environmental in origin and lead to stress, increased negative affect, and dysphoria, which may increase risk for disordered eating (Martínez-González, et al., 2003; Welch, Doll, & Fairburn, 1997).

However, these associations could also be due to gene-environment interactions, where divorce serves as an environmental "trigger" for disordered eating in individuals who have existing genetic susceptibilities. Individuals who do not have these genetic predispositions, on the other hand, may be less likely to develop disordered eating in response to a parental divorce. The significant heritability of eating disorders (> 50% for Anorexia Nervosa and Bulimia Nervosa; Bulik, Sullivan, & Kendler, 1998; Bulik, et al., 2006; Bulik, Sullivan, Wade, & Kendler, 2000; Kendler, et al., 1991; Kendler, et al., 1995) and disordered eating (e.g., binge eating, body dissatisfaction, weight preoccupation; Bulik, et al., 1998; Culbert, Burt, McGue, Iacono, & Klump, 2009; Klump, Burt, McGue, & Iacono, 2007; Klump, Suisman, Burt, McGue, & Iacono, 2009; Sullivan, Bulik, & Kendler, 1998; Wade, Wilkinson, & Ben-Tovim, 2003) provides partial support for this hypothesis. Moderate-to-high heritability estimates support the possibility of a gene-environment interaction because they suggest some genetic influence on the trait in question, which is necessary for gene-environment interactions to occur. Despite calls from previous researchers to examine gene-environment interactions

for eating disorders (Bulik, 2005; see Bulik, et al., 2000; Klump, Wonderlich, Lehoux, Lilenfeld, & Bulik, 2002; Wade, Bulik, & Kendler, 2001), no studies to date have examined gene-environment interactions for disordered eating and divorce.

Parental divorce may interact with genetic risk as either a shared (i.e., co-twins within a twin pair have the same experiences and this increases sibling similarity) or nonshared (i.e., co-twins within a twin pair have different experiences that decrease sibling similarity) environmental risk factor. The *objective* experience of parental divorce is shared by co-twins within a twin pair, as both twins experience divorce within their family. However, parental divorce could also be described as an *effective* nonshared environmental factor, if the reaction to the divorce itself differs between siblings in a twin pair and results in sibling differences in behavior, mood, etc. (e.g., one twin experiences significant distress following the divorce while the other experiences very little distress; Turkheimer & Waldron, 2000). In the classical twin study in which gene-environment interactions are not specifically examined, gene-shared environment interaction effects load onto the heritability estimate (Purcell, 2002), which can lead to inflated heritability estimates and decreased estimates of shared environment. When the risk environment is instead nonshared in nature, the gene-nonshared environment interaction loads onto the estimate of the nonshared environment (Purcell, 2002).

Given that divorce may be operating as either an objective shared or an effective nonshared environmental factor, this particular interaction could be "hiding" in the heritability or nonshared environmental estimates in previous twin research. As noted above, heritability estimates have been moderate-to-high for disordered eating (see Bulik, et al., 2000), and interestingly, nonshared environmental factors have been found to

account for the remaining variance. These findings collectively suggest that gene-environment (either shared or nonshared) interactions may be present and could be contributing to one or both of these significant estimates. The present study will directly examine this possibility by investigating whether gene-environment interactions between divorce and disordered eating exist, and if so, whether divorce seems to be operating primarily as a shared or nonshared environmental factor.

Previous research examining disordered eating and divorce has also not examined the age of offspring at the time of the divorce, despite literature suggesting that there tends to be differences in response to divorce in children versus adolescents (Hetherington, et al., 1989). Given that the peak age of onset of disordered eating is adolescence and early adulthood (American Psychiatric Association, 2000), it seems likely that parental divorce during these periods would be more strongly related to disordered eating than parental divorce during early childhood, since there is closer proximity between the divorce itself and the typical age of onset of disordered eating. This hypothesis is supported by evidence that, though there are long term impacts of divorce, an especially risky period seems to occur within the first two years following divorce. This increased period of risk seems to be driven by the sheer number of stressful events occurring during this time (e.g., changing living arrangements, decreased contact with one parent, attempts to understand the divorce, etc. Kelly & Emery, 2003).

Given the above, the present study examined the possible presence of a gene-environment interaction for symptoms of disordered eating and divorce in 1,810 adolescent and young adult female twins. The presence of a gene-environment interaction was examined by comparing all divorced to all intact families. It was hypothesized that a

significant gene-environment interaction would exist, where the heritability of disordered eating symptoms would be higher in twins who experienced parental divorce than those who did not. Exploratory, secondary analyses were also conducted to examine the effects of twins' age at the time of divorce. For these secondary analyses, it was hypothesized that the strongest gene-environment interaction effects would be observed in twins who experienced divorce during adolescence/early adulthood (i.e., age 13 or greater) versus childhood.

Method

Participants

This study used archival data drawn from two population based twin studies, the Michigan State University Twin Registry (MSUTR; Klump & Burt, 2006), and the Minnesota Twin Family Study (MTFS; Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Iacono, McGue, & Krueger, 2006). Sample characteristics from each of these studies are described in Table 1.

Recruitment procedures for the MSUTR are detailed elsewhere (Klump & Burt, 2006), and therefore will only briefly be described here. The MSUTR recruited adolescent and young adult twins (ages 10-28) using flyers/paid advertisements (25%), recruitment mailings through the MSU Office of the Registrar (27%) and recruitment mailings using birth records (48%) through the Michigan Department of Community Health (MDCH). Although most participants completed all study procedures in the laboratory (95%), some subjects who were not able to travel to the lab participated by completing a mailed packet of questionnaires. Importantly, participants from the MSUTR have been shown to be representative of the population from which they were drawn in

terms of racial and ethnic background (i.e., 83% Caucasian; Culbert, Breedlove, Burt, & Klump, 2008; Klump & Burt, 2006).

Previous research demonstrates differences in the heritability of eating disorder symptoms in pre-pubertal versus adolescent twins and adults (Culbert, et al., 2009; Klump, Burt, et al., 2007; Klump, McGue, & Iacono, 2003; Klump, Perkins, Burt, McGue, & Iacono, 2007) Therefore, MSUTR twins under the age of 14 were excluded from the present study, and twins between the ages of 14-15 years were included only if they were in mid-puberty or beyond at the time of study participation. Mid-puberty was indicated by a score ≥ 2.5 (see Culbert, et al., 2009; Klump, et al., 2003) on the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). The PDS asks participants to report on the extent to which physical markers e.g., body hair growth, breast changes, onset of menarche) of puberty have occurred. The PDS exhibits good psychometric properties (Petersen, et al., 1988), and the PDS total score correlates highly ($r = .61$-$.67$) with physician ratings of pubertal development (Petersen, et al., 1988).

The second source of data comes from the MTFS, a population based, longitudinal twin study of same-sex twins and their parents (Iacono, et al., 1999; Iacono, et al., 2006). The MTFS data used in the present study includes 1,456 twins at approximately age 17. At the time of recruitment for the study, researchers identified twins who were either 11 or 17 years old using Minnesota birth certificates. Recruitment efforts resulted in the recruitment of 91% of twins who met age criteria for the study. Some twin families were later excluded because they 1) lived further than one day's drive from the MTFS lab or 2) the twins had been diagnosed with a mental or physical handicap that would prevent them from participating in the day long laboratory visits.

Like the MSUTR sample of twins, the MTFS sample is representative of the population from which they were drawn in terms of racial and ethnic background (i.e., 98% Caucasian; Iacono, et al., 1999; Iacono, et al., 2006), and is also comparable to Minnesota census data across multiple demographic domains (e.g., urban/rurual split, parent age, ethnicity, and marital status; Holdcraft & Iacono, 2004). Further details on recruitment methods for this study are available elsewhere (Iacono, et al., 1999; Iacono, et al., 2006).

For the current study, data from both the 11 year-old and 17 year-old MTFS cohorts were used. Age 17 assessments were used for both cohorts, which corresponds to the second follow-up assessment for 11 year-old twins and the intake assessment for the 17 year-old twin cohort. Including data from MTFS participants at age 17 is advantageous for several reasons. It maximizes the sample size of post-pubertal twins in the study, which is essential given the differential heritability of disordered eating in pre- versus post-pubertal twins (Culbert, et al., 2009; Klump, Burt, et al., 2007; Klump, et al., 2003). It also allows for the examination of twins during peak periods of risk for eating disorders (American Psychiatric Association, 2000) and closely matches the average age of the MSUTR sample of twins (see Table 1).

Measures

Zygosity determination. Zygosity determination methods differed slightly for the MSUTR and the MTFS. Both samples used the Physical Similarity Questionnaire (Lykken, Bouchard, McGue, & Tellegen, 1990; Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998), which correlates 95-99% with zygosity measured via genotyping. In addition to this zygosity questionnaire, the MTFS also used a staff opinion (based on physical similarity of face shape, ear shape, hair color, and eye color), and an algorithm

based on measurements of cephalic index (i.e., ratio of head width to length), fingerprint ridge counts, and the ponderal index (i.e., a measure of leanness calculated as height in inches/$\sqrt[3]{}$weight in pounds) to determine zygosity. When the three MTFS measures were not in agreement, a serological sample was taken to determine correct zygosity. Importantly, cases of mistaken zygosity in either the MSUTR or MTFS would decrease estimates of both heritability and gene-environment interactions, rather than increase estimates. This is because mistaken zygosity leads to the inclusion of some DZ twins within the MZ twin category, and some MZ twins in the DZ twin category. These errors would decrease overall differences between MZ and DZ twin correlations, thereby driving down estimates of genetic effects (given that genetic effects are implicated when MZ twins are more similar than DZ twins on a given trait). These decreases in genetic effects would then, by definition, decrease the gene-environment interaction as well (since a trait must be heritable for the interaction to occur).

Disordered eating symptoms. Disordered eating in both samples was measured using the Minnesota Eating Behavior Survey (MEBS; Klump, McGue, & Iacono, 2000; von Ranson, Klump, Iacono, & McGue, 2005). The MEBS is a 30-item, self-report, true/false questionnaire. It was designed for use with children as young as 10 years, and has been shown to have excellent reliability and validity in adolescent and young adult females (see below). The MEBS includes a total score (i.e., overall measure of disordered eating) that is comprised of four subscales: body dissatisfaction (i.e., dissatisfaction with body weight/shape), weight preoccupation (i.e., preoccupation with weight and dieting), binge eating (i.e., thoughts of and/or engaging in binge eating), and compensatory behaviors (i.e., the use of inappropriate compensatory behaviors in order to change body

weight/shape). The present study did not include the compensatory behaviors subscale due to low item endorsement and low internal consistency reliability in younger subjects (Klump, et al., 2000).

Previous studies have demonstrated good internal consistency for the total score, body dissatisfaction, and weight preoccupation subscales in children and adolescents (alphas =.78-.89). Binge eating demonstrates a slightly lower alpha, (.65-.69) that is still within the acceptable range (von Ranson, et al., 2005). Studies also demonstrate satisfactory concurrent validity, as scores from the MEBS and Eating Disorder Examination-Questionnaire (EDE-Q) correlate moderately to highly on subscales that measure similar constructs (correlation = .83 in 14 year old girls; von Ranson, et al., 2005). Finally, girls with eating disorders generally report significantly higher scores than girls without eating disorders on all of the MEBS subscales (von Ranson, et al., 2005).

Parental marriage history. History of twin exposure to biological parental divorce was measured via twin self report in both studies. Twins were asked whether their parents were divorced and, if so, their age at the time of the divorce.

Data Analysis

Primary analyses: Independent sample t-tests were used to examine whether the current data replicate previous studies demonstrating increased rates of disordered eating in women who experienced parental divorce (Billingham & Abrahams, 1998; Boumann & Yates, 1994; Herzog, 1982; Igoin-Apfelbaum, 1985; Martínez-González, et al., 2003; Yannakoulia, et al., 2008). These tests examined whether there were mean differences in MEBS scores in twins from divorced versus intact families.

The possibility of a moderating effect of parental divorce on the heritability of disordered eating was then examined using twin intraclass correlations and biometric model-fitting. Twin intraclass correlations were used to examine initial indications of differences in genetic and environmental effects in twins from divorced versus intact families. Genetic influences are implied if the monozygotic (MZ) twin correlations are significantly greater than the dizygotic (DZ) twin correlations. Shared environmental influence is suggested when the MZ and DZ twin correlations are approximately equal and are also significant. Finally, nonshared environmental influence (which also includes measurement error) is inferred when the MZ correlation is less than 1.00, and/or both the MZ and DZ twin correlations are small and non-significant.

Univariate, twin constraint models were then used to examine the relative influence of additive genetic effects (A), shared environmental effects (C), and nonshared environmental effects (E) both *within* the divorced and intact groups independently of one another, as well as *differences* between the divorced and intact groups. Models were fit to raw data using Mx (Neale, 1995). Using raw data allows for the inclusion of all twin pairs, as it treats missing data as random (Little & Rubin, 1987). This is an advantage over the use of covariance matrices (where pairwise deletion occurs for missing data), as twin pairs can still be included in analyses even if one twin is missing data.

For the primary analyses, I initially estimated variances, means, and covariances of the raw data to obtain a baseline estimate of fit (-2lnL) for each subscale of the MEBS. I then fit fully unconstrained (i.e., A, C, and E are allowed to vary across the divorced and intact groups) and fully constrained (i.e., A, C, and E are constrained to be equal across the divorced and intact groups) biometric models to the data to examine possible

group differences in genetic and environmental effects. The fit of these biometric models was compared to that of the baseline model (i.e., the -2lnL of the baseline model was subtracted from the -2lnL of the biometric models), resulting in a likelihood-ratio chi-square test of goodness of fit for the model. This chi square was used to calculate Akaike's information criterion (AIC; χ^2-2*df*), a measure of model fit versus model parsimony for the constrained and unconstrained models separately. The fully unconstrained model provided estimates of the relative influence of A, C, and E *within* each group (i.e., without taking into account the effects in the other group). In order to examine *differences* between the two groups, the relative fit of the unconstrained and constrained models were compared using AIC (i.e., the smallest AIC indicated the best fitting model) and an additional likelihood-ratio chi-square test of goodness of fit. This second chi-square test compared the fully unconstrained model to the constrained model by subtracting the -2lnL of the fully unconstrained model from the -2lnlL of the constrained model(s).

Importantly, these model fit comparisons allowed for the determination of the presence versus absence of moderating effects of divorce. For example, if the fully unconstrained models provided a better fit to the data, it would suggest that there are differences in the influences of A, C, and/or E across divorced and intact groups. By contrast, if the fully constrained model provided the best fit to the data, then there would be no evidence for genetic moderation of divorce on disordered eating, as it would suggest that A,C, and E do not vary across groups.

Notably, before conducting the model-fitting analyses described above, I first examined potential differences in genetic and environmental influences on MEBS scores

between the MTFS and MSUTR samples. A fully unconstrained (i.e., A, C, and E were allowed to vary across the MTFS and MSUTR samples) and a fully constrained (i.e., A, C, and E were constrained to be equal across the MTFS and MSUTR samples) model were fit to the data. The fully constrained model provided a good fit to the data for all subscales, suggesting minimal sample differences in genetic or environmental effects. Given this high degree of similarity, the samples were combined in all subsequent analyses (data not shown).

Secondary analyses: Given previous research and theory suggesting that there are differences in responses to divorce in children versus adolescents (Hetherington, et al., 1989), secondary analyses were also conducted to examine the potential influence of twin age at the time of divorce. First, independent samples t-tests were run to examine mean level differences in disordered eating between twins who experienced divorce in childhood versus adolescence. Second, potential differences in the influences of A, C, and/or E were examined across three groups: childhood divorce (i.e., divorce at age 12 or under), adolescent divorce (i.e., divorce at age 13 and up), and intact families. Twin correlations and constraint models were used as described above to examine whether the heritability of disordered eating is moderated by age at divorce. Three additional "partially" constrained models were also computed for these analyses, where two groups were constrained while the other group was allowed to vary. Specifically, childhood and adolescence were constrained while allowing intact families to vary, childhood and intact families were constrained while allowing divorced families to vary, and adolescent and divorced families were constrained while allowing intact families to vary. These additional constraint models indicated whether subgroups of divorced families could be

constrained to be equal to each other while still varying from the intact families (e.g., Can childhood and adolescent divorce be constrained to be equal while still varying from intact families?).

Results

Prior to all analyses, the MEBS body dissatisfaction and binge eating scales were transformed ($\log_{10} x + 1$) due to the positive skew of the data. Age was then regressed out of all MEBS scores given the wide age range of our sample and research suggesting that mean levels of disordered eating varies by age, with lower rates at younger ages and an increase across adolescence (American Psychiatric Association, 2000; Jones, Bennett, Olmsted, Lawson, & Rodin, 2001; Klump, et al., 2000).

Independent samples t-tests were conducted to examine whether there were mean differences in MEBS scores between the MTFS and MSUTR. Results suggested that there were no significant differences in mean MEBS scores across studies for all subscales, providing further support for combining the two samples. Independent samples t-tests were also used to examine whether mean levels of disordered eating differed between intact and divorced families. Results indicated differences at the trend level for the total score and weight preoccupation, such that scores appeared to be higher in divorced compared to intact families. Mean level differences did not emerge for body dissatisfaction and binge eating (see Table 2), although the mean differences were in the expected direction (i.e., higher in divorced group). Importantly, the lack of strong phenotypic associations between divorce and disordered eating does not preclude the possibility of etiologic moderation, as gene-environment interactions may attenuate phenotypic associations.

Primary Analyses. As expected, twin correlations suggested genetic influences on disordered eating regardless of divorce status, as the MZ twin correlations were, in general, significantly greater than the DZ twin correlations (see Table 3). Significant nonshared environmental influences were also implied, as evidenced by MZ twin correlations that were less than 1.0. Finally, the shared environment appeared to be negligible for all MEBS subscales, as the MZ twin correlations were generally at least double the DZ twin correlations (i.e., the MZ-DZ correlations are not equal). Twin correlations for the total score, weight preoccupation, and binge eating subscales were quite similar between intact and divorced families (i.e., the difference between MZ and DZ twin correlations did not appear to differ greatly across family type). Therefore a gene-environment interaction is unlikely for these subscales. However, twin correlations for the body dissatisfaction subscale suggested that a gene-environment interaction may be present, as the difference between MZ and DZ twin correlations was greater in divorced compared to intact families, suggesting increased heritability in divorced families.

Results of model fitting analyses are presented in Table 4. Notably, C was estimated at 0 in all of the models (data not shown). These results are consistent with previous research suggesting that the influence of C on disordered eating is negligible in pubertal and post-pubertal twins (Bulik, et al., 1998; Culbert, et al., 2009; Javaras, et al., 2008; Kendler, et al., 1991; Klump, Perkins, et al., 2007; Reichborn-Kjennerud, et al., 2003). Therefore, models were re-run with C constrained to zero (i.e., estimating only A and E). These models uniformly provided the best fit to the data (lower AICs and non-

significant chi-square difference tests) as compared to the ACE models. Thus, only the AE models are presented in Table 4.

Overall, model fitting analyses indicated that there were no significant differences in heritability in divorced versus intact families. The fully constrained AE models provided the best fit to the data for the total score, weight preoccupation, and binge eating subscales, as indicated by the lower AIC value and the non-significant change in chi-square. Thus, while both genetic and nonshared environmental influences were important for these types of disordered eating symptoms, there were no gene-environment interactions that differentially influence genetic or environmental influences in the two groups.

The one exception to this general rule was body dissatisfaction. The fully unconstrained AE model provided a better fit to the data than the fully constrained model, as indicated by the lower AIC and significant change in chi-square. This finding suggests that A and/or E significantly differed between the divorced and intact groups. In order to further elucidate the nature of the effects, sub-models were fit to the data to examine whether group differences in A and E were statistically significant. These models estimated A while constraining E to be equal across groups and vice-versa. The fit of these models was then compared to the fully unconstrained model to determine the final, best fitting model. Neither A nor E could be constrained across group, as evidenced by the significant change in chi-square and increased AIC as compared to the fully unconstrained model. Therefore, a moderating influence of divorce on the genetic and nonshared environmental influences on disordered eating (i.e., a gene-environment interaction) appears to be present for body dissatisfaction. Specifically, parameter

estimates indicated that genetic effects are greater in the divorced group ($a^2 = .76$) than in the intact group ($a^2 = .56$). Conversely, effects of the nonshared environment were greater in the intact group ($e^2 = .44$) than in the divorced group ($e^2 = .24$).

Secondary Analyses: Age of Divorce. Findings for age of divorce were remarkably similar to those described above. T-tests revealed no significant differences in mean levels of symptoms of disordered eating by age at divorce (Table 5), and twin correlations suggested that timing of divorce is unlikely to have an impact on heritability of disordered eating symptoms (Table 3). The pattern and magnitude of MZ and DZ correlations for the childhood and adolescent groups were similar (see Table 3). Model fitting analyses are presented in Table 6. These analyses support indications from the twin correlations, as the fully constrained model (i.e., intact, childhood divorce, and intact divorce groups constrained to be equal) was the best fitting for all scales except for body dissatisfaction. For body dissatisfaction, the model constraining childhood and adolescent age at divorce to be equal provided the best fit, while the model constraining intact families to be equal as well did not provide a good fit. These results suggest that a gene-environment interaction was present for body dissatisfaction only, and age at divorce did not impact these findings.

Discussion

This was the first study to examine gene-environment interaction effects of parental divorce on the heritability of disordered eating. Evidence for a gene-environment interaction did not emerge for most symptoms of disordered eating (i.e., total score, binge eating, and weight preoccupation). Only body dissatisfaction exhibited these effects, in that that the heritability of body dissatisfaction was higher in offspring of divorced than

intact families. Age at the time of parental divorce did not impact any findings. Taken together, the present study suggests that the experience of divorce is associated with increased heritability of body dissatisfaction, but not other forms of disordered eating, regardless of age at divorce.

At the phenotypic level, results suggested only modest associations between disordered eating and divorce. No significant mean differences across divorce status were observed for binge eating and body dissatisfaction, and only trend-level differences were present for total score and weight preoccupation. These findings suggest that mean level effects, if present, are likely quite small. Indeed, effect sizes for all mean differences ranged from .05-.11 (see Table 2). Small effect sizes may explain inconsistencies in previous research, where some studies found associations between divorce and disordered eating (Billingham & Abrahams, 1998; Boumann & Yates, 1994; Herzog, 1982; Martínez-González, et al., 2003; Yannakoulia, et al., 2008) while others did not (Dolan, Lieberman, & Evans, 1990; Johnson & Flach, 1985; Mitchell, Hatsukami, Eckert, & Pyle, 1985). Large sample sizes would be needed to detect these small phenotypic effects, and thus, some studies with smaller samples (Dolan, et al., 1990; Johnson & Flach, 1985) may have failed to identify significant associations.

Gene-environment interaction effects were only observed for body dissatisfaction in this study. Results suggested significantly greater heritability of this measure of disordered eating in the divorced group. It will be important for future studies to both replicate our results and examine mechanisms underlying these effects. For example, one body of literature suggests that body dissatisfaction is linked to depression, and generally suggests that individuals who are depressed have increased levels of body dissatisfaction

(Allgood-Merten, Lewinsohn, & Hops, 1990; M. Cooper & Hunt, 1998; P. J. Cooper & Fairburn, 1993; Joiner, Schmidt, & Singh, 1994; Joiner, Schmidt, & Wonderlich, 1997; Joiner, Wonderlich, Metalsky, & Schmidt, 1995; Keel, Fulkerson, & Leon, 1997; Keel, Mitchell, Davis, & Crow, 2001; Leon, Fulkerson, Perry, & Cudeck, 1993; McCabe & Marwit, 1993; Rierdan & Koff, 1997; Roth & Armstrong, 1993; Taylor & Cooper, 1986). Associations between body dissatisfaction and depression appear to be unique, as there are links between depression and body dissatisfaction even in the absence of other symptoms of disordered eating (e.g., Keel, et al., 2001). This may help explain why body dissatisfaction, but not other forms of disordered eating, showed unique gene-environment interaction effects. Importantly, depression is also associated with parental divorce (Hetherington, et al., 1998; Hetherington, et al., 1989; Huurre, Junkkari, & Aro, 2006; Størksen, Røysamb, Moum, & Tambs, 2005; Strohschein, 2005), and there is some evidence that separation events in childhood (including parental divorce) exhibit gene-environment interaction effects for depression (i.e., childhood separation events increase risk for depression only if high latent genetic risk is present; Zimmermann, et al., 2008).

Given the above, it may be that a gene-environment interaction emerges for body dissatisfaction due to a particularly robust association between body dissatisfaction and depression, and interactions between depression and divorce. Unfortunately, this hypothesis could not be directly tested in the present study, as different measures of depression were used across twin registries (e.g., depression symptom counts in MTFS; Beck Depression Inventory (BDI) in MSUTR) and age groups (BDI in adult MSUTR twins; Children's Depression Inventory (CDI) in adolescent MSUTR twins). Future studies should directly investigate this hypothesis.

The gene-environment interaction effects observed for body dissatisfaction did not emerge for any other measures of disordered eating (i.e., MEBS total score, binge eating, and weight preoccupation). It appears that the modest phenotypic effects of divorce on disordered eating do not translate into etiological differences between groups for these measures, despite theories proposing gene-environment interaction effects (Bulik, 2005; Klump, et al., 2002). Similar results have emerged for other psychological variables, whereby environmental rather than genetic causal mechanisms have been linked to divorce, even given significant heritability of the phenotype (e.g., delinquency; Burt, Barnes, McGue, & Iacono, 2008; D'Onofrio, et al., 2007; D'Onofrio, et al., 2005, 2006; O'Connor, Caspi, DeFries, & Plomin, 2000). Future studies should measure divorce in combination with other life stressors (e.g., decreased family income, changes in family composition) to determine whether gene-environment interaction effects emerge for these more comprehensive measures of divorce "stress". Composite measures of life stress have demonstrated gene-environment interactions for other disorders (e.g., Caspi, et al., 2003; Silberg, Rutter, Neale, & Eaves, 2001), and should be explored in future research of disordered eating.

Given the lack of gene-environment effects for most disordered eating symptoms, it will be important for future research to consider explanations for these null effects. One possibility is that the link between divorce and disordered eating is driven entirely by environmental mechanisms. For instance, given that divorce is associated with numerous stressful life events (e.g., moving, changing relationships with peers, decrease in socioeconomic status, etc.), and that stressful life events are linked to disordered eating (Ball & Lee, 2000; Welch, et al., 1997), it is possible that the stress of divorce directly

increases levels of disordered eating. (Jacobi, Hayward, De Zwaan, Kraemer, & Agras, 2004). If the main effects of the environment are driving the association, environmental risk factors are likely to be nonshared in nature, as the shared environment is generally non-significant for disordered eating in adolescence and adulthood (Klump, et al., 2009) and the lack of significant gene-environment interaction suggests that shared environmental effects are not "hiding" in the additive genetic estimates. An alternative possibility is that the association is driven entirely through the main effects of genetic risk factors. Indeed, classical twin studies have suggested that the experience of divorce is heritable (McGue & Lykken, 1992). The heritability of divorce is unlikely to operate through a set of divorce "genes" per se, but may instead be transmitted through heritable psychological traits (e.g., personality characteristics) that increase risk for divorce (Jockin, McGue, & Lykken, 1996). With regard to disordered eating, it is possible that the same heritable personality characteristics that increase risk for divorce may also increase risk for disordered eating (e.g., high levels of negative affect). If this were the case, main effects of genetic risk factors would be operating, as the same genes would increase risk for divorce in the parents and disordered eating in offspring.

One final possibility is that an association between divorce and disordered eating is driven by gene-environment correlations rather than gene-environment interactions. This hypothesis could not be tested in the present study, as statistical models do not exist to test for gene-environment correlations when the moderator (i.e., divorce) does not vary between twin pairs (Purcell, 2002). However, one particular type of gene-environment correlation, passive gene-environment correlations, may be at play in this case. Passive gene-environment correlations occur when offspring passively receive genes from their

parents that are associated with the environment in which they are raised. In the case of disordered eating, it is possible that children receive genes from their parents that increase their vulnerability to disordered eating while, at the same time, increases the likelihood of exposure to divorce. Specifically, it is possible that a parent high in levels of disordered eating may be more likely to experience marital dissatisfaction and divorce (Friedman, Dixon, Brownell, Whisman, & Wilfley, 1999; Van den Broucke & Vandereycken, 1989) than a parent low on disordered eating. In this case, the offspring would be exposed to both the environmental (i.e., parental divorce) and genetic (i.e., genes for disordered eating) risk for disordered eating, and the two correlated risk factors together may increase the likelihood of disordered eating in the offspring.

Future studies are needed to directly examine whether any of the effects discussed above are operating, i.e., whether the main effects of the nonshared environment, the main effects of genetic factors, or passive gene-environment correlations contribute to disordered eating and divorce associations.. Studies using other designs, such as adoption studies, will be especially useful in examining which of these effects may be important. For instance, an adoption study of divorced parents and adopted children could give some indication of the importance of all three mechanisms. If the divorce of adoptive parents still influences disordered eating of adoptive children, genes and passive gene-environment correlations must not be contributing to the association, as adoptive children and their parents by definition do not share genetic material. In this case, the main effects of the environment would be paramount, as adoptive parental divorce could only have environmental effects on their adoptive children's disordered eating risk.

In addition to examining the overall effects of divorce on disordered eating risk, the present study also was the first to investigate whether timing of divorce is important. Age at divorce failed to have an impact on mean levels of disordered eating symptoms or gene-environment interactions between divorce and disordered eating. These findings were somewhat surprising, given robust associations between adolescence and the development of disordered eating (American Psychological Association, 2000). However, these results may have been influenced by the timing of the assessment of disordered eating. Disordered eating was assessed some time after divorce in this study ($M = 10.25$ years post-divorce, $SD = 5.23$). It is possible that timing of divorce may actually be important, but the effects are only detectable when measured directly after the divorce, rather than many years later. Future research should investigate whether the lack of effect found in the present study persists when disordered eating is examined during the time period directly following the divorce.

There are some limitations of the present study that should be noted. Parental divorce, but not marital discord, was examined. Parental divorce and marital discord are strongly related (see Amato & Sobolewski, 2001), but they are often conceptualized and studied separately, as divorce results in changes in family composition and other stressors that are not present for marital discord alone (Amato & Sobolewski, 2001). Studies including measures of both marital discord and divorce suggest that they have similar effects on various outcome variables (e.g., psychological well being, relationships with mother and father; Amato & Sobolewski, 2001), although this has never been examined for eating disorders. Thus, it is unknown whether results of the present study would differ if marital discord, rather than divorce, were examined for its moderating effects.

Future studies should include measures of parental divorce and marital discord in order to elucidate the relationship between these two variables and commonalities and differences in their association with disordered eating symptoms.

This study examined disordered eating using a non-clinical sample of subjects. Given that subjects were not clinically diagnosed with an eating disorder, it is unknown if present findings will generalize to clinical populations. However, estimates of genetic and environmental effects from non-clinical samples are nearly identical to those from clinical samples (Bulik, et al., 2000), suggesting the present results would likely be similar in clinical populations. Further, it would be difficult to directly examine moderating effects of divorce on disordered eating symptoms in a clinical sample, as all subjects would have high levels of disordered eating (e.g., body dissatisfaction, binge eating, etc.), reducing variability in the outcome variables. However, future studies could investigate the clinical significance of these findings by investigating whether the interaction of divorce and body dissatisfaction is predictive of the later development of clinical eating disorders.

In summary, this was the first study to directly examine gene-environment interaction effects of parental divorce on disordered eating. Future research is needed to clarify the magnitude and clinical significance of phenotypic effects of divorce on risk for disordered eating, given the small effect sizes detected in this sample. Studies using large samples should also replicate gene-environment interaction effects for body dissatisfaction and investigate potential mechanisms that drive the interaction (e.g., depression). Further, these findings should be extended to measures of marital discord and other symptoms of disordered eating.

Table 1.
MTFS, MSUTR, and Combined Sample Characteristics

	Combined Sample	MTFS	MSUTR	MTFS vs. MSUTR Mean Comparisons	
				t(df)	*p*
N	1,810	1,456	354	--	--
MZ Pairs	1,125	925	200	--	--
DZ Pairs	685	531	154	--	--
Divorced Families: N (%)	397 (22%)	303 (21%)	94 (27%)	--	--
Intact Families: N (%)	1,413(78%)	1,153(79%)	260 (73%)	--	--
Current Age					
Range	14-28	16-20	14-28	--	--
Mean (SD)	18.26 (1.76)	17.85 (0.70)	19.94 (3.20)	12.15 (358.33)	**<.001**
Age at Divorce					
Range	1-22	1-18	1-22	--	--
Mean (SD)	7.92(5.13)	7.68 (4.92)	8.74 (5.75)	1.70 (382)	.09
MEBS Total Score					
Range	0-29	0-27	0-29	--	--
Mean (SD)	7.72 (6.06)	7.62 (6.04)	8.11 (6.12)	0.69 (1735)	.49
MEBS Body Dissatisfaction					
Range	0-6	0-6	0-6	--	--
Mean (SD)	2.40 (2.20)	2.35 (2.20)	2.58 (2.22)	.29 (1735)	.78
MEBS Weight Preoccupation					
Range	0-8	0-8	0-8	--	--
Mean (SD)	2.93 (2.48)	2.93 (2.50)	2.93 (2.39)	.28 (1734)	.86
MEBS Binge Eating					
Range	0-7	0-7	0-7	--	--
Mean (SD)	1.34 (1.55)	1.30 (1.51)	1.52 (1.72)	1.53 (1734)	.13

Note. MTFS = Minnesota Twin Family Study; MSUTR = Michigan State University Twin Registry; MEBS = Minnesota Eating Behavior Survey; MZ = Monozygotic Twins; DZ = Dizygotic Twins. N = Number of individuals included in sample. Though raw data are presented for descriptive purposes, log transformed scores for weight preoccupation and binge eating were used for T-tests in order to account for positive skew. Age was regressed out of all MEBS variables prior to T-tests.

Table 2.
Mean Differences in Levels of Disordered Eating in Intact versus Divorced Families

MEBS Scale	Intact MEBS Mean (SD)	Divorced MEBS Mean (SD)	*t* (*df*)	*p*	Cohen's *d*
Total Score	7.57 (6.01)	8.20 (6.16)	1.78 (1721)	.08	.10
Weight Preoccupation	2.86 (2.44)	3.14 (2.59)	1.85 (559.27)	.07	.11
Body Dissatisfaction	2.35 (2.17)	2.57 (2.29)	1.33 (1721)	.19	.10
Binge Eating	1.32 (1.55)	1.39 (1.53)	1.05 (1720)	.29	.05

Note. MEBS = Minnesota Eating Behavior Survey. Age was regressed out of all scores prior to analyses, and log transformed scores were used for the binge eating and body dissatisfaction subscales. Raw means and standard deviations are presented here for interpretive purposes.

Table 3.
Twin Correlations for Disordered Eating in Intact, Divorced, and Subgroups of Divorced Families

Family Type	Intact Families			Divorced Families			Childhood Divorce			Adolescent Divorce		
	MZ	DZ	*z*	MZ	DZ	*z*	MZ	DZ	*z*	MZ	DZ	*z*
Total Score	.61**	.20**	6.3^{++}	.71**	.20*	4.24^{++}	.71**	.18	3.58^{++}	.70**	.05	2.29^{+}
Weight Preoccupation	.56**	.15**	5.99^{++}	.52**	.06	3.2^{++}	.49**	.09	2.26^{+}	.53**	-.07	1.85^{+}
Body Dissatisfaction	.58**	.19**	5.85^{++}	.75**	.21*	4.7^{++}	.75**	.23*	3.75^{++}	.79**	-.10	3.29^{++}
Binge Eating	.38**	.08	3.98^{++}	.38**	.16	1.48	.43**	.18	1.41	.26	.03	0.66

Note. MZ = Monozygotic. DZ = Dizygotic. z = Fisher one-tailed r-to-z transformation test of equality. Sample sizes (in twin pairs) for correlations were, for intact families, MZ = 404-405, DZ = 249; for divorced families, MZ = 105-106; DZ = 64; for childhood divorce MZ = 75-76; DZ = 43; and for adolescent divorce MZ = 26; DZ = 15. Sample sizes vary slightly within family type due to missing data.

$^{*}p = .05$, $^{**}p = .01$, the twin correlation is significantly different from zero

$^{+}p = .05$, $^{++}p = .01$; the MZ and DZ correlations are significantly different from each other

Table 4

Note. a^2 = additive genetic effects, e^2 = nonshared environmental effects. Binge eating and body dissatisfaction scores were log transformed prior to analyses to account for positive skew. Columns for a^2 and e^2 indicate standardized parameter estimates with confidence intervals in parenthesis. Baseline models were first fit to the raw data by estimating variances, covariances, and means. These models provided a baseline index of fit ($-2\ln L^a$) that was then compared to the biometric models, providing a likelihood-ratio chi-square test of goodness of fit for the model. Akaike information criteria (AIC) were then computed from these likelihood-ratio chi-squares. Dashed lines for estimates of a^2 and e^2 in the constrained models indicate that parameter estimates were constrained to be equal to those for the preceding group. The best fitting model is noted in bolded text.

[a]Difference in -2lnL from the baseline model; used to calculate AIC

[b]Difference in -2lnL from the fully unconstrained model; *p* values comparing fit relative to the fully unconstrained model are calculated from this statistic

Table 4. Model fitting results in Intact versus Divorced families.

MEBS Subscale	Intact Families N = 697 Pairs		Divorced Families N = 195 Pairs		Model Fit Statistics				
	a^2	e^2	a^2	e^2	-2lnL (df)	$-2\ln L_{\Delta}$ (df)[a]	$-2\ln L_{\Delta}$ (df)[b]	*p*	AIC
Total Score									
Baseline Model	--	--	--	--	4573.86 (1695)	--	--		--
Fully Unconstrained AE	.60 (.53-.65)	.40 (.35-.47)	.69 (.59-.77)	30 (.23-.41)	4595.53 (1709)	21.67 (14)	--		-6.33
Fully Constrained AE	**.62 (.57 -.67)**	**.38 (.33-.43)**	--	--	**4598.23 (1711)**	**24.37 (16)**	**2.70 (2)**	**.26**	**-7.63**
Weight Preoccupation									
Baseline Model	--	--	--	--	4644.15 (1694)	--	--	--	--
Fully Unconstrained AE	.53 (.47-.60)	.46 (.40-.53)	.49 (.35-.61)	.51 (.39-.65)	4663.77 (1708)	19.62 (14)	--		-8.38
Fully Constrained AE	**.53 (.47-.58)**	**.47 (.42-.53)**	--	--	**4666.97 (1710)**	**22.82 (16)**	**3.20 (2)**	**.20**	**-9.18**
Body Dissatisfaction									
Baseline Model	--	--	--	--	4597.41 (1695)	--	--	--	--

Table 4 (cont'd).

MEBS Subscale	Intact Families N = 697 Pairs		Divorced Families N = 195 Pairs		Model Fit Statistics				
	a^2	e^2	a^2	e^2	-2lnL (df)	$-2\ln L_{\Delta}$ (df)[a]	$-2\ln L_{\Delta}$ (df)[b]	*p*	AIC
Fully Unconstrained AE	**.56 (.50-.62)**	**.44 (.37-.50)**	**.76 (.67-.83)**	**.24 (.17-.33)**	**4612.32 (1709)**	**14.91 (14)**	--		**-13.09**
Constrain A	.60 (.55-.65)	.40 (.35-.45)	.68 (.60-.75)	.32 (.25-.40)	4619.48 (1710)	22.07 (15)	7.16 (1)	.008	-7.93
Constrain E	.60 (.54-.65)	.40 (.35-.46)	.64 (.57-.71)	.36 (.29-.43)	4622.13 (1710)	24.72 (15)	9.81 (1)	.002	-5.28
Fully Constrained AE	.62 (.57-.66)	.38 (.34-.43)	--	--	4623.98 (1711)	26.57 (16)	11.66 (2)	.003	-5.43
Binge Eating									
Baseline Model	--	--	--	--	4758.45 (1694)	--	--	--	--
Fully Unconstrained AE	.36 (.28-.44)	.64 (.56-.72)	.37 (.21-.51)	.63 (.48-.79)	4777.04 (1708)	18.59 (14)	--		-9.41
Fully Constrained AE	**.37 (.29-.44)**	**.63 (.57-.71)**	--	--	**4777.14 (1710)**	**18.69 (16)**	**0.10 (2)**	.95	**-13.31**

Table 5.
Mean Differences in Levels of Disordered Eating Symptoms in Childhood Divorce versus Adolescent Divorce Families

MEBS Scale	Childhood Divorce Mean (SD) (N = 259-260)	Adolescent Divorce Mean (SD) (N = 89)	t (df)	*p*	Cohen's *d*
Total Score	8.10 (6.12)	8.22 (6.30)	.09 (347)	.93	.02
Weight Preoccupation	3.06 (2.54)	3.19 (2.70)	.44 (347)	.66	.05
Body Dissatisfaction	2.58 (2.32)	2.45 (2.22)	.37 (347)	.71	.06
Binge Eating	1.38 (1.51)	1.39 (1.60)	.15 (346)	.88	.006

Note. MEBS = Minnesota Eating Behavior Survey. Age was regressed out of all scores prior to analyses, and log transformed scores were used for the binge eating and body dissatisfaction subscales. Raw means and standard deviations are presented here for interpretive purposes.

Table 6.
Model fitting analyses for intact families, childhood divorce, and adolescent divorce.

	Intact Families N = 714 Pairs		Childhood Divorce N = 140 Pairs		Adolescent Divorce N = 46 Pairs		Model Fit Statistics				
MEBS Subscale	a^2	e^2	a^2	e^2	a^2	e^2	-2lnL (df)	$-2\ln L_{\Delta}$ (df)[a]	$-2\ln L_{\Delta}$ (df)[b]	*p*	AIC
Total Score											
1. Baseline Model	--	--	--	--	--	--	4506.00 (1663)	--	--	--	--
2. Fully Uncon	.60 (.53-.65)	.40 (.35-.47)	.69 (.56-.78)	.31 (.22-.44)	.68 (.39-.83)	.32 (.17-.61)	4533.49 (1684)	27.49 (21)	--	--	-14.51
3. Child/Adol Con	.60 (.53-.65)	.40 (.35-.47)	.69 (.58-.77)	.31 (.23-.42)	--	--	4533.93 (1686)	27.93 (23)	0.44 (2)	.80	-18.07
4. Intact/Child Con	.61 (.56-.66)	.39 (.34-.44)	--	--	.68 (.39-.83)	.32 (.17-.61)	4535.46 (1686)	29.46 (23)	1.97 (2)	.37	-16.54
5. Intact/Adol Con	.60 (.54-.66)	.40 (.34-.46)	.69 (.56-.78)	.31 (.22-.44)	--	--	4534.19 (1686)	28.19 (23)	0.70 (2)	.70	-17.81
6. Fully Con AE	**.62 (.56-.66)**	**.38 (.34-.44)**	**--**	--	**--**	**--**	**4536.05 (1688)**	**30.05 (25)**	**2.56 (4)**	**.46**	**-19.95**
Weight Pre.											
1. Baseline Model	--	--	--	--	--	--	4573.69 (1662)	--	--	--	--

Table 6 (cont'd).

	Intact Families N = 714 Pairs		Childhood Divorce N = 140 Pairs		Adolescent Divorce N = 46 Pairs		Model Fit Statistics				
MEBS Subscale	a^2	e^2	a^2	e^2	a^2	e^2	-2lnL (df)	$-2\ln L_\Delta$ $(df)^a$	$-2\ln L_\Delta$ $(df)^b$	*p*	AIC
2. Fully Uncon	.54 (.47-.60)	.46 (.40-.53)	.47 (.30-.61)	.53 (.39-.70)	.46 (.11-.70)	.54 (.30-.89)	4596.02 (1683)	22.33 (21)	--	--	-19.67
3. Child/Adol Con	.54 (.47-.60)	.46 (.40-.53)	.47 (.31-.60)	.53 (.40-.69)	--	--	4596.48 (1685)	22.79 (23)	0.46 (2)	.79	-23.21
4. Intact/Child Con	.53 (.46-.58)	.47 (.42-.54)	--	--	.46 (.11-.70)	.54 (.30-.89)	4597.68 (1685)	23.99 (23)	1.66 (2)	.44	-22.01
5. Intact/Adol Con	.53 (.47-.59)	.47 (.41-.54)	.47 (.30-.61)	.53 (.39-.70)	--	--	4598.27 (1685)	24.58 (23)	2.25 (2)	.32	-21.42
6. **Fully Con**	**.52 (.46-.58)**	**.48 (.42-.54)**	**--**	**--**	**--**	**--**	**4599.56 (1687)**	**25.87 (25)**	**3.54 (4)**	**.32**	**-24.13**
Body Diss.											
1. Baseline Model	--	--	--	--	--	--	4524.06 (1663)	--	--	--	--
2. Fully Uncon	.56 (.50-.62)	.44 (.38-.50)	.76 (.66-.84)	.24 (.16-.34)	.79 (.57-.90)	.21 (.10-.43)	4542.98 (1684)	18.92 (21)	--	--	-23.08
3. Child/Adol Con	**.56 (.50-.62)**	**.44 (.38-.50)**	**.77 (.68-.83)**	**.22 (.17-.32)**	**--**	**--**	**4543.08 (1686)**	**19.02 (23)**	**0.10 (2)**	**.95**	**-26.98**

Table 6 (cont'd).

	Intact Families N = 714 Pairs		Childhood Divorce N = 140 Pairs		Adolescent Divorce N = 46 Pairs		Model Fit Statistics				
MEBS Subscale	a^2	e^2	a^2	e^2	a^2	e^2	-2lnL (df)	$-2\ln L_\Delta$ (df)[a]	$-2\ln L_\Delta$ (df)[b]	*p*	AIC
4. Intact/Child Con	.60 (.54-.65)	.40 (.35-.46)	--	--	.79 (.57-.90)	.21 (.10-.43)	4552.58 (1686)	28.52 (23)	9.60 (2)	.008	-17.48
5. Intact/Adol Con	.58 (.51-.63)	.42 (.37-.49)	.76 (.66-.84)	.24 (.16-.34)	--	--	4546.93 (1686)	22.87 (23)	3.95 (2)	.14	-23.13
6. Fully Con	.61 (.55-.66)	.39 (.34-.45)	--	--	--	--	4555.77 (1688)	31.71 (25)	12.79 (3)	.005	-18.29
Binge Eating											
1. Baseline Model	--	--	--	--	--	--	4691.34 (1662)	--	--	--	--
2. Fully Uncon	.36 (.28-.44)	.64 (.56-.72)	.44 (.25-.59)	.56 (.41-.75)	.17 (0-.50)	.83 (.50-1.0)	4712.40 (1683)	21.06 (21)	--	--	-20.94
3. Child/Adol con	.36 (.28-.44)	.64 (.56-.72)	.37 (.20-.52)	.63 (.48-.80)	--	--	4715.03 (1685)	23.69 (23)	2.63 (2)	.27	-22.31
4. Intact/Child Con	.38 (.30-.45)	.62 (.55-.70)	--	--	.17 (0-.50)	.83 (.50-1.0)	4713.45 (1685)	22.11 (23)	1.05 (2)	.59	-23.89
5. Intact/Adol Con	.35 (.27-.43)	.65 (.57-.73)	.44 (.25-.59)	.56 (.41-.75)	--	--	4713.80 (1685)	22.46 (23)	1.4 (2)	.50	-23.54

Table 6 (cont'd).

	Intact Families N = 714 Pairs		Childhood Divorce N = 140 Pairs		Adolescent Divorce N = 46 Pairs		Model Fit Statistics				
MEBS Subscale	a^2	e^2	a^2	e^2	a^2	e^2	-2lnL (df)	$-2\ln L_\Delta$ (df)[a]	$-2\ln L_\Delta$ (df)[b]	*p*	AIC
6. Fully Con	**.36 (.29-.43)**	**.64 (.57-.71)**	--	--	--	--	**4715.12 (1687)**	**23.78 (25)**	**2.72 (3)**	**.44**	**-26.22**

Note. Fully Uncon = Fully unconstrained; Con = constrained; a^2 = additive genetic effects; e^2 = nonshared environmental effects. Binge eating and body dissatisfaction scores were log transformed prior to analyses to account for positive skew. Columns for a^2 and e^2 indicate standardized parameter estimates with confidence intervals in parenthesis. Baseline models (model 1) were first fit to the raw data by estimating variances, covariances, and means. These models provided a baseline index of fit (-2lnL) that was then compared to the biometric models, providing a likelihood-ratio chi-square test of goodness of fit for the model. Akaike information criteria (AIC) were then computed from these likelihood-ratio chi-squares. Model 2 (fully unconstrained AE) gives unique estimates for each group as they are all allowed to vary. Model 3 constrains the child and adolescent divorce groups to be equal. Model 4 constrains the intact and child groups to be equal. Model 5 constrains the intact and adolescent groups to be equal. Model 6 constrains estimates for all three groups to be equal. Dashed lines for estimates of a^2 and e^2 in the constrained models indicate that parameter estimates were constrained to be equal to those for a preceding group. The best fitting model is noted in bolded text.

[a]Difference in -2lnL from the baseline model; used to calculate AIC

[b]Difference in -2lnL from the fully unconstrained model; *p* values comparing fit relative to the fully unconstrained model are calculated from this statistic

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