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

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Abstract

Objective—We investigated gene-environment interactions (G×E) for associations between parental divorce and disordered eating (DE).

Method—Participants were 1,810 female twins from the Michigan State University Twin Registry and the Minnesota Twin Family Study. The Minnesota Eating Behaviors Survey was used to assess DE. We tested for G×E by comparing the heritability of DE in twins from divorced versus intact families. It was hypothesized that divorce would moderate the heritability of DE, in that heritability would be higher in twins from divorced than twins from intact families.

Results—As expected, the heritability of body dissatisfaction was significantly higher in twins from divorced than intact families. However, genetic influences were equal in twins from divorced and intact families for all other forms of DE.

Discussion—Although divorce did not moderate heritability of most DE symptoms, future research should replicate G×Es for body dissatisfaction and identify factors underlying this unique relationship.

Keywords

Disordered Eating; Parental Divorce; Gene-Environment Interaction; Eating Disorders; Twins

Parental divorce has been implicated as an important risk factor for eating disorders (1-2). Higher rates of divorce have been observed in individuals with bulimia nervosa as compared to controls (1,3-4), and divorce prospectively predicts increased risk for onset of an eating disorder (e.g., eating disorder not otherwise specified, bulimia nervosa and anorexia nervosa; 5). Associations between divorce and disordered eating extend to community samples as well, as body dissatisfaction, weight control behaviors (e.g., dieting, exercise), and binge eating are significantly associated with parental divorce (2,6-7).

In general, researchers have not examined etiologic factors underlying associations between parental divorce and disordered eating. Divorce tends to be viewed as a stressful life event that results in the accumulation of many negative events for the offspring, such as moving, decreases in socioeconomic status, changes in schools, changing relationships with parents,

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decreased social support, exposure to parental conflict, and loss of contact with extended family (8-9). 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 (5,10).

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; 11,12-15) and disordered eating (e.g., binge eating, body dissatisfaction, weight preoccupation; 11,16-20) provides partial support for this hypothesis. If gene-environment interactions are present for divorce and disordered eating, divorce would be expected to moderate the heritability of disordered eating. Specifically, the heritability of disordered eating in twins experiencing parental divorce would be higher relative to twins who did not experience parental divorce, as the environmental trigger of divorce would be expected to enhance genetic predispositions for disordered eating.

Despite calls from previous researchers to examine gene-environment interactions for eating disorders (13,21-23), no studies to date have examined gene-environment interactions for disordered eating and divorce. The current study directly addressed this gap by investigating whether divorce moderates the heritability of disordered eating in 1,810 adolescent and young adult female twins from divorced versus intact families. It was hypothesized that the heritability of disordered eating symptoms would be higher in twins who experienced parental divorce than those who did not, suggesting the presence of gene-environment interaction effects.

Method

Participants

This study used archival data drawn from two population based twin studies, the Michigan State University Twin Registry (MSUTR; 24), and the Minnesota Twin Family Study (MTFS; 25,26). Sample characteristics from each of these studies are described in Table 1.

Recruitment procedures for the MSUTR are detailed elsewhere (24), 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; 24,27).

Previous research demonstrates differences in the heritability of eating disorder symptoms in pre-pubertal versus adolescent twins and adults (16-17,28-29). 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 (16,28) on the Pubertal Development Scale (PDS; 30). 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 (30), and the total score correlates highly (r = .61-.67) with physician ratings of pubertal development (30).

The second source of data comes from the MTFS, a population based, longitudinal twin study of same-sex twins and their parents (25-26). The MTFS data used in the present study included 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; 25,26) and is also comparable to Minnesota census data across multiple demographic domains (e.g., urban/rurual split, parent age, ethnicity, and marital status; 31). Further details on recruitment methods for this study are available elsewhere (25-26).

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 (16-17,28). It also allows for the examination of twins during peak periods of risk for eating disorders (32) 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 studies used the Physical Similarity Questionnaire (33-34), 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.

Disordered eating symptoms—Disordered eating in both samples was measured using the Minnesota Eating Behavior Survey[a] (35-36). 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

[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.

present study did not include the compensatory behaviors subscale due to low item endorsement and low internal consistency reliability in younger subjects (36).

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 (35). 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; 35). Finally, girls with eating disorders generally report significantly higher scores than girls without eating disorders on all of the MEBS subscales (35).

Parental marriage history—History of twin exposure to biological parental divorce was measured via twin self report in both studies.

Data Analysis

Independent sample t-tests were used to examine whether the current data replicates previous studies demonstrating increased rates of disordered eating in females who experienced parental divorce (1,3-7). 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. Additive genetic effects (A) are implied if the monozygotic (MZ) twin correlations are significantly greater than the dizygotic (DZ) twin correlations. Shared environmental influence (C) is suggested when the MZ and DZ twin correlations are approximately equal and are also significant. Finally, nonshared environmental influence (E) (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 models were then used to examine the relative influence of genetic and environmental factors on disordered eating 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 (37). The use of raw data allows for the inclusion of all twin pairs, as it treats missing data as random (38). 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.

Variances, means, and covariances of the raw data were estimated to obtain a baseline estimate of fit (-2lnL) for each subscale of the MEBS. Fully unconstrained (i.e., genetic and environmental influences are allowed to vary across the divorced and intact groups) and fully constrained (i.e., all effects constrained to be equal across the divorced and intact groups) biometric models were then fit 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 genetic and environmental influences *within* each group, 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 genetic and/or environmental influences 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 genetic and environmental influences do not vary across groups.

Notably, before conducting the model-fitting analyses described above, we first examined potential differences in disordered eating/divorce associations between the MTFS and MSUTR samples. Independent samples t-tests (see Table 1) suggested that there were no significant differences in mean MEBS scores across studies for all subscales. We then fit twin constraint models to confirm that there were no etiological differences between the samples as well. 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 (data not shown). Given this high degree of similarity, the samples were combined in all subsequent analyses.

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. Given the wide age range of participants, and research suggesting increased rates of disordered eating across adolescence, (32,36,39), age was regressed out of all MEBS scores prior to analyses.

Independent samples t-tests were used to examine whether mean levels of disordered eating differed between intact and divorced families. Results indicated significant differences for the total score and weight preoccupation, such that these scores appeared to be higher in divorced compared to intact families. Mean level differences did not emerge for body dissatisfaction or 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 body dissatisfaction and binge eating does not preclude the possibility of etiologic moderation, as gene-environment interactions may attenuate phenotypic associations.

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 typically double the DZ twin correlations. These negligible influences of C are consistent with previous research suggesting that C is not important for disordered eating in female twins after puberty (11,14,16,29,40-41).

Twin correlations for the total score, body dissatisfaction, and weight preoccupation suggested potential moderation by divorce status, as the difference between MZ and DZ

twin correlations was greater in divorced compared to intact families, suggesting increased heritability in divorced relative to intact families. Twin correlations for the binge eating subscale showed the reverse pattern, as there was a greater difference between MZ and DZ twin correlations in the intact group.

Results of model fitting analyses are presented in Table 4. Due to the lack of indication of significant influence of the shared environment in any of the twin correlations, only genetic and nonshared environmental effects were estimated in these models (i.e., AE models). Overall, 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 ($e^2 = .24$).[b]

Discussion

This was the first study to examine gene-environment interaction effects of parental divorce on the heritability of disordered eating. Divorce status did not moderate the heritability of 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. 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.

At the phenotypic level, results suggested only modest associations between disordered eating and divorce. Significant mean-level differences across divorce status were present for the total score and weight preoccupation, but no significant mean differences were observed for binge eating and body dissatisfaction. Regardless of level of significance, mean level effects for all phenotypes are likely quite small, as effect sizes for all mean differences

[b]We also examined whether age at the time of the divorce (i.e., during childhood (≤12 years old) versus adolescence (≥13 years old)) significantly influenced results. Results remained unchanged from those reported herein, in that gene-environment interactions were only observed for body dissatisfaction. The heritability of body dissatisfaction was again higher in twins from divorced families, regardless of twin age at the time of divorce.

ranged from .05-.11 (see Table 2). Small effect sizes may explain some inconsistencies in previous research, where several studies found associations between divorce and disordered eating (1-2,4-7) while others did not (42-44). Large sample sizes would be needed to detect these small phenotypic effects, and thus, some studies with smaller samples (42-43) may have failed to identify significant associations.

Importantly, the presence of only modest phenotypic associations does not negate the possibility of moderation of *heritability* by divorce status. The presence of such gene-environment interactions may attenuate phenotypic effects, as the phenotypic association is less likely to be present in individuals without genetic predispositions. Indeed, despite modest phenotypic associations, the heritability of body dissatisfaction was higher in divorced relative to intact families, suggesting the presence of a gene-environment interaction. It will be important for future studies to both replicate this result and examine mechanisms underlying the effect. 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 (45-57). 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 (47). 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 (58-62), 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; 63).

Given the above, it may be that a gene-environment interaction emerged 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 extend to other measures of disordered eating (i.e., MEBS total score, binge eating, and weight preoccupation). Reasons for these non-significant results are unclear, particularly given theories of gene-environment interactions for disordered eating, divorce, and other family-related variables (21-23). However, measurement issues have may have limited our ability to detect significant gene-environment associations with the other disordered eating variables. It is possible that our dichotomous measure of parental divorce did not adequately capture the stress of divorce, leading to non-significant results in the majority of our AE models. Indeed, twin correlations suggested potential moderation by divorce status for all DE subscales, (i.e., the difference between the MZ and DZ correlations appeared to differ across groups). However, effects in the AE twin models were only significant for body dissatisfaction. More comprehensive measures of divorce stress (e.g., decreased family income, changes in family composition, parental remarriage) might reveal gene-environment interactions for divorce and disordered eating that did not emerge as significant with our dichotomous measure. In addition, stressors before the divorce (i.e., marital discord, parental separation) may impact the heritability of disordered eating more than the divorce itself. Although parental divorce and these variables are strongly related (see 64), they are often conceptualized and studied separately. Future studies should include measures of parental

divorce, general divorce “stress”, and marital discord in order to examine their potential interactive effects with genetic and environmental risk factors for disordered eating.

An additional limitation of this study was the examination of disordered eating in 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 (13), suggesting that 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.

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Int J Eat Disord. Author manuscript; available in PMC 2012 March 1.

Table 1

MTFS, MSUTR, and Combined Twin Sample Characteristics

				MTFS vs. MSUTR Mean Comparisons	
	Combined Sample	**MTFS**	**MSUTR**	***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%)	313 (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 (358.63)	**<.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)	0.28 (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. Although 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)	.04	.10
Weight Preoccupation	2.86 (2.44)	3.14 (2.59)	1.85 (559.27)	.03	.11
Body Dissatisfaction	2.35 (2.17)	2.57 (2.29)	1.33 (1721)	.09	.10
Binge Eating	1.32 (1.55)	1.39 (1.53)	1.05 (1720)	.15	.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. All T-tests were one-tailed.

Int J Eat Disord. Author manuscript; available in PMC 2012 March 1.

Table 3

Twin Correlations for Disordered Eating in Intact and Divorced Families

Family Type	Intact Families			Divorced Families		
	MZ	DZ	z	MZ	DZ	z
Total Score	.61**	.20**	6.3++	.71**	.20*	4.24++
Weight Preoccupation	.56**	.15**	5.99++	.52**	.06	3.2++
Body Dissatisfaction	.58**	.19**	5.85++	.75**	.21*	4.7++
Binge Eating	.38**	.08	3.98++	.38**	.16	1.48

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, and MZ = 105-106; DZ = 64.

* $p = .05$,

** $p=.01$; the twin correlation is significantly different from zero

++ $p = .01$; the MZ and DZ correlations are significantly different from each other

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)**	--	--	--	--
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)	--	--	--	--

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	.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**

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 compare the fit of the model relative to the fully unconstrained model.

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