

Genetic Relationships Between Personality and Eating Attitudes and Behaviors

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Genetic and environmental factors underlying relationships between personality traits and disordered eating were examined in 256 female adolescent twin pairs (166 monozygotic, 90 dizygotic). Eating behaviors were assessed with the Total Score, Body Dissatisfaction, Weight Preoccupation, Binge Eating, and Compensatory Behavior subscales from the Minnesota Eating Disorders Inventory (M-EDI; K. L. Klump, M. McGue, & W. G. Iacono, 2000). Personality characteristics were assessed with the Negative Emotionality, Positive Emotionality, and Constraint scales from the Multidimensional Personality Questionnaire (MPQ; A. Tellegen, 1982). Model-fitting analyses indicated that although genetic factors were more likely to contribute to MPQ and M-EDI phenotypic associations than environmental factors, shared genetic variance between the 2 phenotypes was limited. MPQ personality characteristics may represent only some of several genetic risk factors for eating pathology.

Phenotypic relationships between eating pathology and personality characteristics have been noted for decades (Wonderlich, 1995). Researchers examining these relationships have used a wide variety of personality measures, rendering comparisons of results problematic. Nonetheless, in aggregate, findings point to discernible patterns of personality characteristics in individuals with eating pathology. Specifically, elevated levels of negative emotionality (Casper, Hedeker, & McClough, 1992; Pryor & Wiederman, 1996), constraint (Casper et al., 1992; Pryor & Wiederman, 1996), and harm avoidance (Brewerton, Hand, & Bishop, 1993; Bulik, Sullivan, Fear, & Pickering, 2000; Casper, 1990; Casper et al., 1992; Kleifield, Sunday, Hurt, & Halmi, 1994a, 1994b; Klump, Bulik, et al., 2000; O'Dwyer, Lucey, & Russell, 1996) have been observed in individuals with anorexia nervosa (AN) or restricting forms of eating pathology, suggesting that these individuals tend to be anxious in nature and unusually fearful of novel stimuli and negative consequences. By contrast, individuals with bulimia nervosa (BN) or binge-purge subtypes of eating pathology have been found to have decreased levels of constraint (Bulik, Sullivan, Weltzin, & Kaye, 1995; Pryor & Wiederman, 1996) while maintaining the elevated levels of negative emotionality (Lilenfeld et al., 2000) and harm avoidance (Bulik, Sullivan, Joyce, & Carter, 1995) observed in women with AN. This pattern suggests that women with BN tend to be both anxious and impulsive, a person-

ality profile that may contribute to cycles of binge eating and purging frequently observed in these individuals.

Etiologic factors underlying these phenotypic relationships remain unclear. Initial hypotheses that associations were merely artifacts of malnutrition (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950) or consequences of chaotic eating behaviors (Vitousek & Manke, 1994) have generally not been supported by empirical research. Recent studies have indicated that personality disturbances (most notably, negative emotionality and harm avoidance) persist after weight and symptom recovery from both AN (Casper, 1990; Klump, Bulik, et al., 2000; O'Dwyer et al., 1996; Ward, Brown, Lightman, Campbell, & Treasure, 1998) and BN (Kaye et al., 2001; Lilenfeld et al., 2000), suggesting that they may be trait-related rather than state-related disturbances resulting from the illness itself. In addition, prospective data have shown personality to be the most significant predictor of the development of disordered eating over a 1- to 4-year period (Leon, Fulkerson, Perry, & Cudek, 1993; Leon, Fulkerson, Perry, Keel, & Klump, 1999), highlighting the possible role of personality as a risk factor for the later development of eating pathology. Thus, although definitive conclusions await additional longitudinal research, findings suggest the presence of enduring phenotypic relationships that likely reflect the role of personality as a risk factor for eating disturbances.

What types of etiologic factors contribute to these enduring relationships, biological or environmental? Environmental associations would suggest that changes in environmental contexts may prevent personality disturbances and thereby decrease risk for eating pathology. By contrast, information regarding biological relationships could aid neurobiological and genetic research aimed at identifying biological substrates of these disorders.

Indirect evidence from behavioral genetic research suggests that genetic factors may partially underlie phenotypic associations, as significant genetic influences have been found for both personality and eating pathology. For example, personality characteristics

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have been shown to be moderately heritable at 30–60% (Bouchard & McGue, 1990; Loehlin, Horn, & Willerman, 1981; Loehlin & Nichols, 1976; Scarr, Webber, Weinberg, & Wittig, 1981; Tellegen et al., 1988). In addition, twin studies suggest that greater than 50% of the risk for eating pathology is attributable to genetic factors (Bulik et al., 1998; Holland, Sicotte, & Treasure, 1988; Kendler et al., 1991; Klump, McGue, & Iacono, 2000; Klump, Miller, Keel, McGue, & Iacono, 2001; Wade, Bulik, et al., 2000). These estimates apply to both AN (Klump et al., 2001; Wade, Bulik, et al., 2000) and BN (Bulik et al., 1998; Kendler et al., 1991), as well as to disordered eating attitudes and behaviors such as body dissatisfaction (Klump, McGue, & Iacono, 2000; Rutherford, McGuffin, Katz, & Murray, 1993; Wade, Martin, & Tiggeman, 1998), weight preoccupation (Klump, McGue, & Iacono, 2000), binge eating (Bulik et al., 1998; Klump, McGue, & Iacono, 2000; Sullivan, Bulik, & Kendler, 1998), and the use of compensatory behavior (Klump, McGue, & Iacono, 2000; Sullivan et al., 1998).

Nevertheless, heritability alone does not provide sufficient evidence of a genetic association between personality and disordered eating. Phenotypic associations are as likely to be due to environmental as genetic factors. For example, it could be argued that the overprotective, enmeshed family environment of women with AN (Foulkes, 1996; Horesh et al., 1996) contributes to the development of anxious, harm-avoidant traits as well as disordered eating. Likewise, the often chaotic, disruptive, and impulsive family environments of women with BN (Schmidt, Humfress, & Treasure, 1997) could contribute to these individuals' negative affectivity, impulsive natures, and use of food to ameliorate these states. Differentiation between genetic and environmental contributions to associations can only be achieved through large-scale behavioral genetic studies in which both personality and eating pathology are assessed.

To our knowledge, only one such study has investigated genetic and environmental influences on relationships between personality and disordered eating. In a twin study of 537 monozygotic (MZ) and 344 dizygotic (DZ) twins, Wade, Martin, et al. (2000) examined relationships between neuroticism and a general measure of disordered eating assessing symptoms of AN, BN, binge eating, and obesity. Findings suggested that modest, positive relationships between these two phenotypes were primarily the result of common nonshared environmental rather than common genetic factors. The authors concluded that the relatively low magnitude of the phenotypic correlations, coupled with their use of a heterogeneous measure of disordered eating, may have prohibited their detection of significant shared genetic variance.

We sought to extend these findings by performing multivariate genetic analyses of personality and relatively homogeneous disordered eating scales in a population-based sample of twins. The specific aims of this study were to (a) examine phenotypic associations between personality characteristics and disordered eating attitudes and behaviors such as body dissatisfaction, weight preoccupation, binge eating, and the use of compensatory behaviors; (b) determine the extent to which genetic or environmental factors underlie relationships; and (c) examine whether common genes and environmental factors account for the total variance in disordered eating or whether there are genetic and environmental influences on eating pathology that are independent of those for personality.

We have chosen to focus on continuous rather than categorical definitions of disordered eating for several reasons. First, continuous phenotypes provide greater power to detect genetic associations over categorical definitions (Neale & Cardon, 1992). Second, the eating variables examined appear to be as heritable as clinical diagnoses (Klump, McGue, & Iacono, 2000) and have been shown to be correlates of, and risk factors for, clinical eating pathology (Leon et al., 1993; Striegel-Moore, Silberstein, & Rodin, 1986). Indeed, in the current twin sample, levels of body dissatisfaction, weight preoccupation, binge eating, and compensatory behaviors were found to discriminate between women with and without AN and BN (Miller von Ranson, 1998). Finally, relatively few studies have examined phenotypic associations between personality and individual disordered eating attitudes and behaviors, despite their importance for understanding risk for a range of eating pathology.

Method

Participants

The sample consisted of female twin participants in the Minnesota Twin Family Study (MTFS), a population-based, longitudinal study of the development of substance use and related disorders in reared-together male and female twins and their parents. Twins were ascertained from State of Minnesota birth records, and they were located using public databases, including telephone directories and driver license registrations (Lykken, Bouchard, McGue, & Tellegen, 1990). Although recruitment procedures and exclusion criteria are detailed elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999), it is notable that the MTFS has been successful in locating more than 90% of twin births in the state of Minnesota in any given birth year.

The initial sample of 676 included 16-, 17-, and 18-year-old twins (mean age = 17.46 years, $SD = 0.51$). One hundred eleven twins (16%) did not complete either the personality or eating assessments and thus were excluded from analysis. Reasons for this noncompletion included computer malfunctions (leading to an inability to retrieve data from the computer-based eating pathology assessment) or the failure of twins to return paper copies of either assessment by mail when they were unable to complete them during their in-person assessment. Another 27 (4%) twins were excluded because of missing data on more than 10% of the items on the measure of eating attitudes and behaviors. Only pairs whose co-twins completed both the eating pathology and personality measures were included in analyses, leading to the exclusion of an additional 25 twins (4%) and a final sample of 512 twins (76% of initial sample), including 166 MZ and 90 DZ twin pairs. The excess of MZ relative to DZ twin pairs is partially due to an increased rate of participation in MZ twins. In addition, this excess likely reflects the increase in MZ twin births in the state of Minnesota across time (Hur, McGue, & Iacono, 1995).

Zygosity Determination

Zygosity was established using a parental zygosity questionnaire regarding physical similarity of the twins, a project staff zygosity estimate, and an algorithm diagnosis calculated from ponderal index, cephalic index, and fingerprint ridge count. Disagreements among these estimates were resolved through serological examination of genetic polymorphisms. In a validation study using 50 pairs of twins, all of whom had the serological test, in every case where the three zygosity estimates agreed, the serological analysis confirmed the agreement.

Measures

Eating attitudes and behaviors. Self-reported eating attitudes and behaviors were assessed with a computer-administered, 30-item Minnesota

Eating Disorders Inventory (M-EDI; Klump, McGue, & Iacono, 2000). Several modifications were made to the original EDI (Garner, Olmstead, & Polivy, 1983) to tailor it for use with preadolescent twins, who are included in the MTFS but who are not the focus of this article. These modifications included (a) selecting a subset of EDI items to create a shorter measure, with an emphasis placed on items assessing eating attitudes and behaviors rather than personality traits; (b) simplifying the language of items to increase comprehensibility to girls as young as 10 years old; and (c) altering the scoring convention to true–false to simplify administration and interpretation. The M-EDI consists of 24 true–false items drawn primarily from three subscales of the original EDI: Drive for Thinness, Body Dissatisfaction, and Bulimia. Two items from the original EDI Interoceptive Awareness scale that were behavioral in nature and were similar to constructs tapped by other scales were also included. In addition, six items created by MTFS researchers to assess the use of compensatory behaviors, such as self-induced vomiting, laxatives, diuretics, diet pills, and exercise, were also added to the measure.

A previous factor analysis (Klump, McGue, & Iacono, 2000) of the M-EDI yielded the following four subscales: Body Dissatisfaction (dissatisfaction with the size or shape of one's body), Weight Preoccupation (preoccupation with dieting, weight, and the pursuit of thinness), Binge Eating (the tendency to engage in episodes of overeating as well as having attitudes conducive to binge eating), and Compensatory Behavior (the tendency to use or to contemplate using inappropriate compensatory behaviors such as self-induced vomiting and laxatives to control weight). The M-EDI and its subscales are scored in the traditional "pathological" direction with high scores indicating greater degrees of the measured construct. Previous studies have supported the reliability and validity of the revised measure and its subscales (Klump, McGue, et al., 2000; Miller von Ranson, 1998).

M-EDI total scores were prorated for 4 (< 1%) twins who were missing one to three (>10%) of the M-EDI items. Given the relatively small number of items on each subscale (range = 6–8 items), data from participants with one or two missing items on any given subscale were not included in analyses of that scale. M-EDI scores were transformed ($\log_{10} x + 1$) prior to analyses because of the positive skew of the data.

Personality. Self-reported personality characteristics were assessed with the 198-item version of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). The MPQ is a true–false measure of normal range personality functioning that was constructed through an iterative sequence of rational and factor analytic procedures. The MPQ measures 11 primary scales and three higher order factor scales that are linear weighted composites of the primary scales. The three higher order factor scales tap three broad dimensions of personality: Positive Emotionality, or the tendency for pleasurable, active engagement in one's work and social environment; Negative Emotionality, or the tendency to experience negative mood states, including emotional tension, anxiety, anger, and resentment; and Constraint, or the tendency to exercise general restraint and caution, to inhibit behavioral impulses, and to endorse traditional moral values. Four of the MPQ primary scales (Well-Being, Social Potency, Achievement, and Social Closeness) load principally on Positive Emotionality, three (Stress Reaction, Alienation, and Aggression) load principally on Negative Emotionality, and three (Control, Harm Avoidance, and Traditionalism) load principally on Constraint; the 11th primary scale, Absorption, does not load principally on any of the higher order factors. In analyses presented here, we focus exclusively on the higher order MPQ scales that have been examined more extensively in eating disorders research (Casper, 1990; Pryor & Wiederman, 1996).

Statistical Analyses

We used Pearson product–moment correlations between M-EDI and MPQ scores to assess phenotypic associations. Cross-twin, cross-trait correlations were then calculated and compared to within-individual cor-

relations to provide initial indications of genetic and environmental influences underlying phenotypic associations. It should be noted that intraclass correlations and model-fitting analyses (see below) were only conducted for MPQ and M-EDI phenotypic relationships that were statistically significant, because it is meaningless to examine etiologic commonalities between two traits that have minimal associations.

Cholesky decomposition models were fit to twin variance–covariance matrices using the maximum-likelihood method and the Mx software program (Neale, 1995). Cholesky decomposition is a multivariate technique based on the principles of factor analysis that provides estimates of additive genetic (A: genetic influences that add across genes), shared environmental (C: environmental influences that are shared by reared-together twins and are thus a source of their behavioral similarity), and nonshared environmental (E: environmental influences that are not shared by reared-together twins and are thus a source of their behavioral dissimilarity) contributions to variance in and to covariance between MPQ and M-EDI scales.

The ACE Cholesky model is depicted in Figure 1. As described by Loehlin (1996), although the ordering of the variables in the three-factor Cholesky model is arbitrary with respect to how well the model fits the observed data (i.e., both orderings fit the data well), it is not arbitrary with respect to the parameter estimates that are produced. In the present study, we were interested in determining the extent to which genetic and environmental effects on the personality measures could account for genetic and environmental effects on the M-EDI scales. Consequently, priority was accorded the MPQ scores; it is represented on the left-hand side of Figure 1. In this case, the genetic and environmental variance in the M-EDI scales is decomposed into components attributable to the genetic and environmental effects on the MPQ (represented as a_{21} , c_{21} , and e_{21}) and

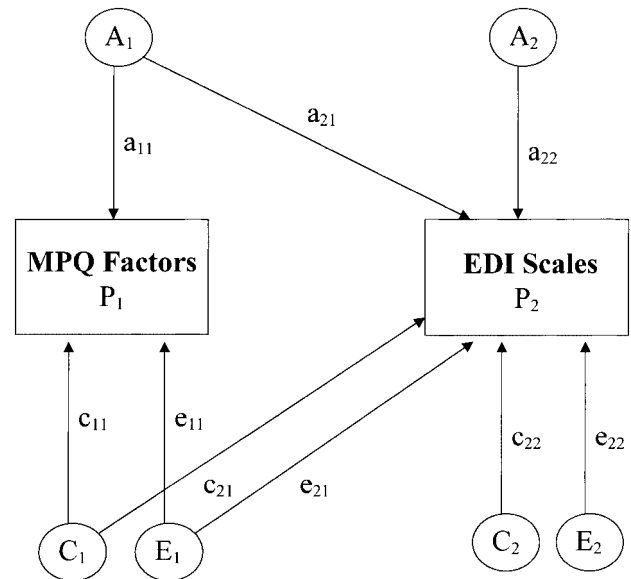


Figure 1. Path diagram of Cholesky ACE model for Multidimensional Personality Questionnaire (MPQ) and Minnesota Eating Disorders Inventory (M-EDI) scales. Variance in each attribute is assumed to be determined by the additive combination of three latent factors: additive genetic (A), shared environmental (C), and nonshared environmental (E) effects. The additive genetic, shared environmental, and nonshared environmental variances in M-EDI scores are partitioned into those components attributable to the genetic and environmental effects on MPQ scores (a_{21} , c_{21} , e_{21}) and residual components that are independent of the genetic and environmental effects on MPQ scores (a_{22} , c_{22} , e_{22}). P₁ = Phenotype 1 (MPQ scores); P₂ = Phenotype 2 (M-EDI scores).

residual components that are independent of the genetic and environmental variance in the MPQ scales (represented as a_{22} , c_{22} , and e_{22}), whereas there is no decomposition of genetic and environmental effects on the MPQ scales (which are represented as a_{11} , c_{11} , and e_{11} in the figure). The following statistics were derived from the estimated parameters: the heritability of each of the three MPQ scores (h_1^2); the heritability of each of the five M-EDI scales, both overall (h_2^2) as well as decomposed into a portion attributable to genetic effects on MPQ scores (h_3^2) and a residual component (h_4^2); the correlation between the genetic components for each M-EDI scale and each MPQ scale (r_a); the correlation between the environmental components for each M-EDI scale and each MPQ scale (r_e); and the bivariate heritability (i.e., the proportion of phenotypic correlations between M-EDI and MPQ scores accounted for by genetic factors) and environmentality (i.e., the proportion of the phenotypic correlation accounted for by environmental factors).

To decrease the number of model comparisons, we initially fit three models to the data: the full ACE, CE, and AE models. The overall fit of these models was assessed with the chi-square goodness-of-fit statistic, with large (statistically significant) chi-square values leading to a rejection of the model. If more than one of these models provided an appropriate fit to the data, they were compared using the chi-square difference test and Akaike's information criteria (AIC; Akaike, 1987). The chi-square difference test compares nested models by examining differences in chi-square values (χ^2 diff), using as its degrees of freedom the difference in the degrees of freedom for the two models. The more parsimonious model is preferred when χ^2 diff is nonsignificant. AIC ($\chi^2 - 2$ degrees of freedom), a statistic that weighs model fit against model parsimony, was also used to select the best-fitting initial model as indicated by the lowest AIC value.

After selecting the best-fitting initial model, we tested the significance of genetic correlations, environmental correlations, and residual genetic components by constraining each of these parameters to zero and again comparing model fit using the chi-square difference test. The overall, best-fitting model was then determined on the basis of this difference test.

The assumptions of these models include the following: (a) All genetic effects are additive, (b) there is no assortative mating for the measures in question, and (c) the degree of shared environmental influence is equal for MZ and DZ twins so that a greater correlation between MZ as compared with DZ twins is due to genetic factors. It is customary to assume additivity of genetic effects in the analysis of twin data (Eaves, Eysenck, & Martin, 1989), because it is not possible to estimate nonadditive genetic, additive genetic, and shared environmental effects simultaneously with reared-together twin data. Although there is little evidence for spousal similarity on most psychological characteristics (Price & Vandenberg, 1980), recent data suggest a small but significant assortative mating effect for body weight (Allison et al., 1996), a significant predictor of eating pathology (Fairburn, Welch, Doll, Davies, & O'Connor, 1997). Nonetheless, assortative mating leads to an underestimation of heritability. Thus, if assortative mating were significant for eating pathology, it would lead to lower heritability estimates than the substantial ones obtained for these traits and disorders thus far (Bulik et al., 2000).

Finally, the assumption of equal environmental similarity for MZ and DZ twins has generally been supported by behavioral genetic research (see Plomin, DeFries, & McClearn, 1990). Violations of this assumption would lead to overestimations of heritability that reflect genetic as well as environmental variance. Despite evidence to the contrary (Hettema, Neale, & Kendler, 1995), a recent study of the influence of physical similarity on twin resemblance for M-EDI scores in the current sample showed negligible effects (Klump, Holly, Iacono, McGue, & Willson, 2000). In addition, several other studies investigating the influence of childhood environmental similarity (Bulik et al., 1998; Kendler et al., 1991; Sullivan et al., 1998), perceived versus real zygosity (Kendler, Neale, Kessler, Heath, & Eaves, 1993), and the frequency of contact as adults (Kendler et al., 1991) have also provided support for the Equal Environments Assumption in twin studies of eating pathology. Nonetheless, a recent investigation of

the influence of childhood and adolescent co-socialization on twin concordance for BN showed that twins with higher co-socialization have increased concordance for the disorder (Bulik et al., 1998). Direction of effects is difficult to determine from this study; twin similarity may lead to increased co-socialization, rather than the reverse. Thus, additional research is needed to replicate findings and examine their relevance for twin studies of eating attitudes and behaviors.

Results

Associations Between M-EDI and MPQ Scores

Table 1 presents the Pearson product-moment correlations between the M-EDI and MPQ scales. Negative Emotionality showed significant positive relationships with all five M-EDI scales, whereas Positive Emotionality was also significantly but modestly correlated with M-EDI total score, Body Dissatisfaction, and Binge Eating. Compensatory Behavior was the only subscale showing significant associations with Constraint.

Bivariate Analyses

Intraclass correlations for M-EDI and MPQ scales are presented in Table 2. Significantly increased MZ relative to DZ twin correlations for all five M-EDI scales and most MPQ scales suggest genetic effects. These findings indicate that both the M-EDI and MPQ scales are heritable constructs that may share genetic influence.

Supportive of this hypothesis are the MZ cross-twin correlations. Most were statistically significant and approached the within-person correlations (see Table 3). These correlations suggest that M-EDI scores can be predicted nearly as well by the MPQ profile of one's MZ co-twin as by one's own MPQ profile. The small and nonsignificant DZ cross-twin, cross-trait correlations suggested weak predictive relationships between personality composites and DZ co-twin M-EDI scores.

Model-fitting analyses were used to confirm initial impressions of genetic mediation of personality and M-EDI scale relationships. Comparisons of the six Cholesky models are presented in Table 4. Of the initial three models (ACE, CE, AE) examined, the AE model provided the best fit to the data for all of the MPQ and M-EDI score relationships. These findings suggest that shared environmental factors contribute little to phenotypic associations between MPQ and M-EDI scales.

Submodels of the AE model (i.e., AE $-r_a$, AE $-r_e$, AE $-residual_a$) were compared to determine the relative influence of common genetic, common nonshared environmental, and residual

Table 1
Pearson Correlations Between M-EDI and MPQ Scores

MPQ higher order scales	M-EDI total score	Body Diss.	Weight Preocc.	Binge Eating	Comp. Behavior
Positive Emotionality	-.09*	-.09*	-.06	-.14**	-.03
Negative Emotionality	.33***	.20***	.23***	.32***	.16***
Constraint	-.07	-.07	-.04	-.07	-.15***

Note. $N = 512$ twins. M-EDI = Minnesota Eating Disorder Inventory; MPQ = Multidimensional Personality Questionnaire; Diss. = Dissatisfaction; Preocc. = Preoccupation; Comp. = Compensatory.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2
Intraclass Correlations for M-EDI and MPQ Higher Order Scales

Scale	MZ (n = 166 pairs)	DZ (n = 90 pairs)
M-EDI scale		
Total score	.58*†	.17*
Body Dissatisfaction	.60*†	.13
Weight Preoccupation	.55*†	.08
Binge Eating	.36*†	.11
Compensatory Behavior	.52*†	.24*
MPQ higher order factor		
Negative Emotionality	.34*	.21*
Positive Emotionality	.56*†	.34*
Constraint	.54*†	.17

Note. M-EDI = Minnesota Eating Disorders Inventory; MPQ = Multidimensional Personality Questionnaire; MZ = monozygotic; DZ = dizygotic.

* $p < .05$, one-tailed. The correlation is significantly different from 0. † $p < .05$, one-tailed. The MZ correlation is significantly larger than the DZ correlation.

genetic factors on M-EDI and MPQ phenotypic relationships. For the majority of MPQ and M-EDI relationships, the AE model without common nonshared environmental influences (i.e., AE $-r_c$) provided the best fit to the data, as indicated by nonsignificant chi-squares and chi-square difference tests, as well as low AIC values. These results suggest that relationships between MPQ and M-EDI scores are primarily the result of common genetic rather than common nonshared environmental factors. Two exceptions to this finding are the relationships between Negative Emotionality and total score and between Negative Emotionality and Binge Eating. For these associations, the full AE model was the best fitting, suggesting that both common genetic and common nonshared environmental influences contribute to phenotypic associations.

Parameter estimates from the best-fitting AE models are summarized in Table 5. Similar to previous findings (Tellegen et al., 1988), the heritability of all three MPQ factors ranged from .34 to .55. Estimates of the heritability of the M-EDI scales were also very similar to those reported previously from univariate analyses (Klump, McGue, et al., 2000). The contribution of multivariate models is not in providing more precise estimates of heritability, but rather in helping to characterize the nature of the relationship between two measures.

Bivariate estimates, genetic correlations, and nonshared environmental correlations suggest that relationships between M-EDI scales and MPQ factors are primarily mediated through shared genes (bivariate heritabilities = .49–1.00), and that 2–22% of the proportion of the total heritability of M-EDI scales is attributable to the genetic component shared with MPQ factors (i.e., genetic correlations [r_a] = $-.15$ to $.47$). Common nonshared environmental influences were negligible for most MPQ and M-EDI score relationships, with the exception of Negative Emotionality and Total Score relationship and the Negative Emotionality and Binge Eating relationship, where 40–51% of the variance was attributable to the common nonshared environmental factors.

Despite these common effects, findings also suggested that the majority of genetic and nonshared environmental influences on

M-EDI scales appear to be independent of those affecting MPQ scores. Residual heritable and nonshared environmental components ($h_r^2 = .27-.59$; $e_r^2 = .39-.67$) were consistently larger than the attributable effects ($h_a^2 = .00-.11$; $e_a^2 = .00-.04$). Thus, although relationships between MPQ and M-EDI scores are mediated primarily through shared genes, other genetic and nonshared environmental effects that are independent of personality contribute primarily to the development of these attitudes and behaviors.

Discussion

Personality and Disordered Eating Attitudes and Behaviors

This study is one of the first to examine etiologic relationships between personality characteristics and disordered eating attitudes and behaviors. Phenotypic associations generally corroborated findings from previous work in clinical populations. For example, clinical studies have found elevated negative affectivity scores in individuals with both AN and BN (Brewerton et al., 1993; Bulik, Sullivan, Weltzin, & Kaye, 1995; Casper, 1990; Casper et al., 1992; Kleinfeld et al., 1994a, 1994b; Klump, Bulik, et al., 2000; Lilienfeld et al., 2000; O'Dwyer et al., 1996; Pryor & Wiederman, 1996). Similarly, we found the most significant and consistent correlations between the MPQ Negative Emotionality scale and all five M-EDI scales.

Significant negative associations between Positive Emotionality and M-EDI total score, Body Dissatisfaction, and Binge Eating also mimic those of other studies that have suggested that women with eating disorders have decreased levels of positive affect and optimism (Pryor & Wiederman, 1996). In addition, previous work suggests that women who binge and purge tend to score higher on measures of impulsivity, novelty seeking, and behavioral disinhibition (Bulik, Sullivan, Joyce, & Carter, 1995; Lilienfeld et al., 2000). Although we did not find significant associations with Binge Eating, significant negative relationships between MPQ Constraint and M-EDI Compensatory Behavior corroborate these prior results.

Table 3
Cross-Twin, Cross-Trait Correlations Between M-EDI and MPQ Scales Showing Significant Phenotypic Correlations

M-EDI scale	Negative Emotionality		Positive Emotionality		Constraint	
	MZ	DZ	MZ	DZ	MZ	DZ
Total score	.30*	.03	$-.15^*$.08	NA	NA
Body Dissatisfaction	.25*	.04	$-.12$.05	NA	NA
Weight Preoccupation	.26*	$-.05$	NA	NA	NA	NA
Binge Eating	.18*	.07	$-.14^*$.03	NA	NA
Compensatory Behavior	.23*	.04	NA	NA	$-.14^*$.01

Note. $N = 256$ pairs: 166 monozygotic (MZ) and 90 dizygotic (DZ). M-EDI = Minnesota Eating Disorders Inventory; MPQ = Multidimensional Personality Questionnaire; NA = not applicable (because the phenotypic correlation between variables was not statistically significant). None of the MZ and DZ cross-twin, cross-trait correlations were significantly different from each other.

* $p < .05$, one-tailed. The correlation is significantly different from 0.

Table 4
Comparison of Bivariate Cholesky Decomposition Models

Model	$\chi^2(df)$	p	AIC	χ^2 diff (df) ^a	p
Negative Emotionality					
M-EDI total score					
ACE	10.08 (11)	.52	-11.92	—	—
CE	30.11 (14)	.005	2.11	20.03 (3)	<.001
AE	10.49 (14)	.73	-17.51	0.41 (3)	ns
$-r_a$	—	—	—	12.17 (1)	<.001
$-r_e$	—	—	—	10.74 (1)	<.001
$-residual_a$	—	—	—	19.08 (1)	<.001
Body Dissatisfaction					
ACE	9.02 (11)	.62	-12.99	—	—
CE	32.46 (14)	.003	4.46	23.44 (3)	<.001
AE	9.42 (14)	.80	-18.58	0.40 (3)	ns
$-r_a$	—	—	—	7.73 (1)	.05
$-r_e$	—	—	—	1.64 (1)	ns
$-residual_a$	—	—	—	19.86 (1)	<.001
Binge Eating					
ACE	8.14 (11)	.70	-13.86	—	—
CE	13.68 (14)	.40	-14.32	5.54 (3)	ns
AE	8.36 (14)	.82	-19.64	0.22 (3)	ns
$-r_a$	—	—	—	8.99 (1)	.05
$-r_e$	—	—	—	13.23 (1)	<.01
$-residual_a$	—	—	—	14.04 (1)	<.001
Compensatory Behavior					
ACE	3.25 (11)	.99	-18.76	—	—
CE	9.39 (14)	.74	-18.61	6.14 (3)	ns
AE	4.24 (14)	.99	-23.76	0.99 (3)	ns
$-r_a$	—	—	—	9.94 (1)	<.05
$-r_e$	—	—	—	0.24 (1)	ns
$-residual_a$	—	—	—	17.39 (1)	<.001
Weight Preoccupation					
ACE	9.49 (11)	.58	-12.51	—	—
CE	27.32 (14)	.01	.68	17.83 (3)	<.001
AE	10.06 (14)	.69	-17.94	0.57 (3)	ns
$-r_a$	—	—	—	8.06 (1)	ns
$-r_e$	—	—	—	3.21 (1)	ns
$-residual_a$	—	—	—	19.55 (1)	<.001
Positive Emotionality					
M-EDI total score					
ACE	9.78 (11)	.55	-12.22	—	—
CE	32.56 (14)	.002	4.56	22.78 (3)	<.001
AE	10.67 (14)	.64	-17.33	0.89 (3)	ns
$-r_a$	—	—	—	3.82 (1)	<.05
$-r_e$	—	—	—	0.05 (1)	ns
$-residual_a$	—	—	—	52.39 (1)	<.001
Body Dissatisfaction					
ACE	8.15 (11)	.70	-13.85	—	—
CE	34.24 (14)	<.001	6.24	26.09 (3)	<.001
AE	9.19 (14)	.76	-18.81	1.04 (3)	ns
$-r_a$	—	—	—	1.79 (1)	ns
$-r_e$	—	—	—	0.36 (1)	ns
$-residual_a$	—	—	—	68.13 (1)	<.001
Binge Eating					
ACE	13.81 (11)	.24	-8.19	—	—
CE	21.47 (14)	.06	-6.53	7.66 (3)	ns
AE	15.16 (14)	.30	-12.84	1.35 (3)	ns
$-r_a$	—	—	—	4.28 (1)	<.05
$-r_e$	—	—	—	1.03 (1)	ns
$-residual_a$	—	—	—	18.99 (1)	<.001
Constraint					
Compensatory Behavior					
ACE	13.00 (11)	.29	-9.00	—	—
CE	29.49 (14)	.006	1.49	16.49 (3)	<.001
AE	13.60 (14)	.40	-14.40	0.60 (3)	ns
$-r_a$	—	—	—	8.02 (1)	<.001
$-r_e$	—	—	—	1.00 (1)	ns
$-residual_a$	—	—	—	40.00 (1)	<.001

Note. AIC = Akaike's information criteria; χ^2 diff = chi-square difference test; M-EDI = Minnesota Eating Disorders Inventory; ACE, CE, and AE models include all genetic (r_a) and environmental (r_e) correlations; $-r_a$ is the full AE model minus the genetic correlation; $-r_e$ is the full AE model minus the nonshared environmental correlation; $-residual_a$ is the full AE model minus residual genetic effects. The best-fitting model is indicated in boldface.

^a AE and CE models are compared with the full ACE model for the χ^2 diff test; $-r_a$, $-r_e$, and $-residual_a$ are compared with the full AE model for this test.

Table 5
Parameter Estimates for the Best-Fitting Models

Estimate	M-EDI total score	Body Dissatisfaction	Binge Eating	Compensatory Behavior	Weight Preoccupation
Negative Emotionality					
Heritability estimate					
Negative Emotionality scale ^a					
Total (h^2)	.34 (.22, .46)	.34 (.22, .46)	.34 (.22, .46)	.34 (.22, .46)	.34 (.21, .46)
M-EDI scale					
Total (h^2)	.58 (.47, .67)	.61 (.51, .70)	.34 (.20, .46)	.57 (.47, .66)	.55 (.44, .65)
Attributable (h_a^2)	.10 (.02, .24)	.09 (.02, .21)	.07 (.01, .18)	.09 (.02, .20)	.11 (.04, .23)
Residual (h_r^2)	.48 (.35, .59)	.52 (.39, .63)	.27 (.23, .38)	.48 (.34, .59)	.44 (.30, .56)
Bivariate heritability	.60 (.31, .83)	1.00	.49 (.19, .75)	1.00	1.00
Environmentality estimate					
Negative Emotionality scale ^a					
Total (e^2)	.66 (.54, .79)	.66 (.54, .79)	.66 (.54, .79)	.66 (.54, .79)	.66 (.54, .79)
M-EDI scale					
Total (e^2)	.41 (.33, .53)	.39 (.30, .49)	.66 (.54, .80)	.43 (.34, .53)	.45 (.35, .56)
Attributable (e_a^2)	.02 (.004, .06)	0	.04 (.01, .10)	0	0
Residual (e_r^2)	.39 (.31, .50)	.39 (.30, .49)	.62 (.51, .75)	.43 (.34, .53)	.45 (.35, .56)
Bivariate environmentality	.40 (.16, .69)	0	.51 (.25, .81)	0	0
Correlation					
Genetic (r_a)	.42 (.20, .62)	.39 (.20, .58)	.47 (.19, .72)	.39 (.20, .59)	.45 (.25, .65)
Nonshared environmental (r_c)	.24 (.10, .37)	0	.25 (.12, .38)	0	0
Positive Emotionality					
Heritability estimate					
Positive Emotionality scale ^a					
Total (h^2)	.55 (.44, .64)	.55 (.44, .64)	.55 (.44, .64)	—	—
M-EDI scale					
Total (h^2)	.58 (.47, .67)	.61 (.51, .70)	.33 (.19, .45)	—	—
Attributable (h_a^2)	.02 (.00, .07)	.02 (.00, .06)	.03 (.003, .09)	—	—
Residual (h_r^2)	.56 (.45, .66)	.59 (.49, .69)	.30 (.16, .42)	—	—
Bivariate heritability	1.00	1.00	1.00	—	—
Environmentality estimates					
Positive Emotionality scale ^a					
Total (e_r^2)	.45 (.36, .56)	.45 (.36, .56)	.45 (.36, .56)	—	—
M-EDI scale					
Total (e_r^2)	.42 (.33, .53)	.39 (.30, .49)	.67 (.55, .81)	—	—
Attributable (e_a^2)	0	0	0	—	—
Residual (e_r^2)	.42 (.33, .53)	.39 (.30, .49)	.67 (.55, .81)	—	—
Bivariate environmentality	0	0	0	—	—
Correlation					
Genetic (r_a)	-.17 (-.34, -.01)	-.15 (-.30, .02)	-.31 (-.52, -.11)	—	—
Nonshared environmental (r_c)	0	0	0	—	—
Constraint					
Heritability estimate					
Constraint scale					
Total (h_r^2)	—	—	—	.53 (.42, .63)	—
M-EDI scale					
Total (h_r^2)	—	—	—	.56 (.45, .65)	—
Attributable (h_a^2)	—	—	—	.03 (.002, .09)	—
Residual (h_r^2)	—	—	—	.53 (.42, .63)	—
Bivariate heritability	—	—	—	1.00	—
Environmentality estimate					
Constraint scale					
Total (e_r^2)	—	—	—	.47 (.37, .58)	—
M-EDI scale					
Total (e_r^2)	—	—	—	.43 (.35, .54)	—
Attributable (e_a^2)	—	—	—	0	—
Residual (e_r^2)	—	—	—	.43 (.35, .54)	—
Bivariate environmentality	—	—	—	0	—
Correlation					
Genetic (r_a)	—	—	—	-.23 (-.39, -.06)	—
Nonshared environmental (r_c)	—	—	—	0	—

Note. M-EDI = Minnesota Eating Disorders Inventory; h^2 = total heritability; h_r^2 = total heritability of indicated Multidimensional Personality Questionnaire (MPQ) factor; h_a^2 = total heritability of indicated M-EDI scale; h_a^2 = heritability of indicated M-EDI scale that is attributable to genetic effects on the indicated MPQ score; h_r^2 = heritability of indicated M-EDI scale that is independent of genetic effects on the indicated MPQ scale; bivariate heritability = the proportion of phenotypic correlations between indicated M-EDI and MPQ scales accounted for by genetic factors; e^2 = environmental heritability; e_r^2 = total environmentality of the indicated MPQ scale; e_a^2 = total environmentality of the indicated M-EDI scale; e_a^2 = environmentality of indicated M-EDI scale that is attributable to environmental effects on the indicated MPQ score; e_r^2 = environmentality of indicated M-EDI scale that is independent of environmental effects on the indicated MPQ scale; bivariate environmentality = the proportion of phenotypic correlations between indicated M-EDI and MPQ scales accounted for by nonshared environmental factors; r_a = genetic correlation, or the correlation between the genetic components for the indicated M-EDI scale with the indicated MPQ scale; r_c = nonshared environmental correlation, or the correlation between the nonshared environmental components for the indicated M-EDI scale with the indicated MPQ scale.

^a Higher order factor scale of the MPQ.

In summary, the present analyses are significant in extending findings from clinical studies to individual disordered eating attitudes and behaviors. Understanding temperamental characteristics associated with these symptoms is likely to result in more comprehensive personality profiles of individuals suffering from a range of eating pathology.

Genetic Relationships

In general, results suggested that common genetic factors contribute more to relationships between personality and disordered eating attitudes and behaviors than common nonshared environmental factors. With the exception of the Binge Eating subscale, bivariate heritabilities indicated that more than half of all relationships between personality and eating phenotypes are due to shared genetic factors. These results highlight the potential role of temperament as a genetic risk factor for eating pathology. As mentioned above, accumulating evidence suggests that a tense, anxious disposition may predate the onset of (Leon et al., 1993; Leon et al., 1999), and persist after recovery from (Casper, 1990; Lilenfeld et al., 2000; O'Dwyer et al., 1996; Ward et al., 1998), both AN and BN. The present findings add to this literature by suggesting that the enduring nature of these personality disturbances may be the result of shared genetic variance and that personality characteristics may contribute to the genetic diathesis for these disorders. For instance, it may be that girls who are genetically predisposed to be nervous, anxious, and pessimistic are more likely to focus on their bodies during the pubertal changes that affect these characteristics than girls who are not tense and prone to worry. Similarly, these girls may be less able to deal with other forms of stressors, and this decreased ability may then interact with societal pressures for thinness and result in a body- and weight-focused adolescent who is at increased risk for clinical eating pathology. Both of these examples are speculative, but they highlight the potential role of personality as a genetic risk factor for eating pathology that interacts with environmental stress to create eating symptoms and disorders.

Nonetheless, findings also indicate that a large proportion of genetic influence on eating attitudes and behaviors is independent of MPQ scores. This finding highlights the multifactorial nature of the genetic diathesis for these symptoms and disorders. Personality is likely to be only one of many contributing factors. Previous studies suggest that body weight (Klump, McGue, et al., 2000), anxiety disorders (Kendler et al., 1995), major depression (Wade, Bulik, et al., 2000), and possibly ovarian hormones (Klump, McGue, & Iacono, in press) may also contribute to this genetic diathesis, although further research examining these and other traits is clearly needed.

Our findings conflict with those of Wade, Martin, et al. (2000), who found common nonshared environmental rather than common genetic effects influenced associations between neuroticism and overall disordered eating. Discrepant findings may be due to differences in personality constructs examined, differences in measures of eating pathology, or differences in the time period assessed. Although neuroticism has been found to be associated with MPQ Negative Emotionality (Church, 1994), subtle differences in the personality constructs could have led to differences in the results obtained here. Likewise, Wade, Martin, et al. used a more heterogeneous measure of eating pathology than the subscales

used here, a measure that included AN symptoms, BN symptoms, binge eating, and obesity characteristics all in the same scale. The authors noted (see p. 737) that their use of a more heterogeneous scale may have decreased their ability to detect specific phenotypic and genetic relationships between neuroticism and different types of disordered eating. Finally, the Wade, Martin, et al. twins were adults who reported on current personality characteristics but past and current disordered eating, whereas the MTFs twins were adolescents who focused entirely on current levels of both characteristics. Thus, there may be differences in etiologic effects across adolescence and adulthood. Alternatively, personality and eating pathology genetic relationships may have been attenuated in the Wade, Martin, et al. study because they assessed some participants at different times. Clearly, additional research in adolescent and adult twins is needed to clarify discrepant results and identify those personality characteristics showing the strongest genetic and environmental relationships with disordered eating.

In addition, research examining environmental contributions to eating pathology is also needed. Genetic diatheses are merely predispositions if environmental stressors do not trigger symptoms. Our findings and those of Wade, Martin, et al. (2000) suggest that nonshared environmental factors contribute significantly to certain personality and disordered eating relationships, particularly between neuroticism and negative emotionality and overall disordered eating and binge eating. To date, very little research has examined specific nonshared environmental factors, including differential familial, peer, sibling, and prenatal experiences, in the development of eating pathology, despite the importance of these influences for the development of eating disorders (Klump, Wonderlich, Lehoux, Lilenfeld, & Bulik, 2002). Future research should seek to identify nonshared environmental factors contributing most to these disorders and elucidate the mechanisms by which they interact with genetic factors to create dysfunctional eating.

Study Limitations

This study is unique in its examination of relationships between personality and individual disordered eating symptoms, as well as its use of multivariate twin techniques to disentangle genetic from environmental factors. Despite these strengths, several limitations should be noted. First, the generalizability of findings to clinical eating disorders is unknown. Although the importance of examining personality and eating disorder symptom relationships is clear, it is equally important to determine whether similar etiologic relationships exist in individuals with AN and BN. Second, the data are cross-sectional in nature, and thus we are unable to address causality or direction of effects (i.e., personality is a genetic risk factor for eating pathology or vice versa). Prospective data and larger sample sizes are needed to examine such causal models. Third, genetic correlations between two phenotypes are insufficient for making inferences regarding specific sets of genetic loci (Carey, 1988). Indeed, phenotypes with all of their genes in common can have low genetic correlations, and those with few genes in common can have large genetic correlations (Carey, 1988). Thus, we are unable to make inferences about specific shared genetic loci but can only instead comment on the proportion of shared genetic variance between personality and disordered eating attitudes and behaviors.

Finally, we were limited to self-report assessments of personality and eating attitudes and behaviors that are included on the MPQ and revised M-EDI, respectively. Additional research is needed to determine whether other measures (e.g., interview-based) and types of personality characteristics (e.g., neuroticism, extraversion) and disordered eating (e.g., dietary restraint, eating- and body-related obsessions) show similar etiologic relationships. Moreover, future research should examine other potential genetic risk factors for eating pathology using multivariate twin techniques. These investigations will have the greatest power to determine the relative impact of quantitative traits on the heritability of eating pathology and thereby provide an integrative understanding of the diathesis to these disorders.

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