BRIEF REPORT

Genetic and Environmental Associations between Body Dissatisfaction, Weight Preoccupation, and Binge Eating: Evidence for a Common Factor with Differential Loadings Across Symptom Type

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ABSTRACT

Objective: Prior twin studies provide support for a single "common factor" that contributes genetic and environmental risk to a range of disordered eating symptoms. However, the common factor may be indexed less well by binge eating (BE) than other symptoms of eating disorders [i.e., body dissatisfaction (BD) and weight preoccupation (WP)]. We sought to explore the presence of a common factor and test whether loadings differed across three key symptoms (i.e., BE, BD, WP).

Method: Disordered eating was assessed via self-report in 631 female twin pairs from the Michigan State University Twin Registry.

Results: We detected a common disordered eating factor that was influenced primarily by additive genetic and non-shared environmental influences. However, we observed different loadings on this common factor by symptom type, as factor loadings for BD and WP were stronger than that for BE. Moreover, the residual environmental and/or genetic variances (i.e., those that are independent of the common factor) were larger in BE than those of BD or WP.

Discussion: Although all three symptoms share a common set of genetic and environmental influences, risk for BE may involve additional genetic, biological, and environmental factors that are not shared with other symptoms of eating pathology. © 2016 Wiley Periodicals, Inc.

Keywords: body dissatisfaction; binge eating; weight preoccupation; common factor; eating disorders; twins

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Introduction

Previous studies have explored whether disordered eating symptoms are etiologically distinct or share an underlying latent common factor.[1,2] Twin studies are useful for exploring etiologic structures as they examine whether symptoms/disorders share common additive genetic (A; genetic influences that add across genes), shared environmental (C; environmental influences that are shared by reared together twins that contribute to similarity), and/or non-shared environmental (E; environmental influences that are not shared by reared together twins that contribute to dissimilarity, including measurement error) influences. To date, three twin studies investigated shared etiologic effects and found evidence for a common disordered eating factor[1–3] that was influenced primarily by genetic and non-shared environmental influences.

Interestingly, however, careful review of these studies' findings reveals that binge eating behavior and cognitions (e.g., disinhibition) may load less

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strongly on the common factor than other disordered eating symptoms (e.g., drive for thinness, dietary restraint).[1] Differential loadings for binge eating could suggest that there are unique genetic and/or environmental factors (in addition to the common factors) that lead specifically to binge eating but not other types of disordered eating symptoms. This information could be very valuable for future classification systems and etiologic theories that attempt to understand similarities/differences between eating disorders that share binge eating as a common symptom (e.g., bulimia nervosa) versus those that do not (e.g., purging disorder).

Unfortunately, no twin study to date has examined differential loadings on the common factor by symptom type. The aim of the current study was to explore differences between cognitions/behaviors associated with binge eating (BE) and other disordered eating symptoms [i.e., body dissatisfaction (BD), weight preoccupation (WP)].

Methods

Participants

Participants were 631 female twin pairs (MZ = 354 (56.1%), DZ = 277 (43.9%); see Supporting Information Table 1 for sample characteristics) ages 10–28 ($M = 17.51$, $SD = 3.12$) drawn from archival datasets in the Michigan State University Twin Registry.[4,5] We only included twins who were in mid-puberty or beyond (i.e., scored above 2.5 on the pubertal development scale[6]) in order to control for possible differences in genetic/environmental effects between pre-pubertal and pubertal twins.[7,8] Details about recruitment, response rates, and demographics of the MSUTR can be found elsewhere.[4,5,9] Zygosity was determined using a physical similarity questionnaire[6] and established methods[9] that are 95–99% accurate.[10]

Measures

The body dissatisfaction (BD; discontent with body size and/or shape), weight preoccupation (WP; preoccupation with weight/dieting), and binge eating (BE; engaging in and/or thinking about binge eating) subscales of the *Minnesota Eating Behavior Survey (MEBS;*[11])* were examined in analyses. These subscales demonstrate good convergent validity with the Eating Disorder Examination Questionnaire[12] and discriminate between individuals with versus without eating disorders.[11] The subscales exhibited strong internal consistency in our sample (αs = .87–93).

*The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) 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 (1983) Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

Statistical Analyses

Pearson correlations were used to examine phenotypic associations among the symptoms, and twin intraclass correlations were computed to provide initial indications of additive genetic, shared environmental, and nonshared environmental influences. Similar to prior work,[1–3] we fit the common pathway model and independent pathway model to examine the presence of a common factor and differential loadings across symptom type. These models were fit to the raw twin data using full-information maximum likelihood in Mplus 7.3.[13] Modeling procedures are detailed elsewhere.[1] Briefly, in the common pathway model (Supporting Information Fig. 1), a single, higher-order latent variable was specified to account for covariation between the symptoms. The variance in the common factor, as well as the residual variance not accounted by the common factor, was subsequently decomposed into additive genetic, shared environmental, and nonshared environmental influences. Conversely, in the independent pathway model (Supporting Information Fig. 2), separate genetic and environmental factors were estimated for each symptom. Although these unique genetic and environmental factors may correlate with each other, no common factor accounts for their covariation.

To identify the best etiologic structure, we compared the fit of the full (i.e., models including A, C, and E) common pathway and independent pathway models using the root mean square error of approximation (RMSEA), Akaike's information criterion (AIC), Bayesian information criterion (BIC), and the Comparative Fit Index (CFI). Submodels of the best-fitting model were then compared using the Satorra–Bentler χ^2 difference Test[14] (i.e., the simpler model is preferred when the χ^2 is nonsignificant). Notably, age and body mass index (kg/m^2; calculated using measured height and weight) were included as covariates in all models to control for their possible effects.

Results

Stronger correlations were observed between BD and WP ($r = .63$, $p < .001$) than between BE and either BD ($r = .45$, $p < .001$) or WP ($r = .48$, $p < .001$). Nonetheless, twin correlations suggested significant genetic (i.e., the MZ twin correlation is significantly greater than the DZ correlation; BD: MZ $r = .64$, DZ $r = .44$, z test of independence =

TABLE 1. Model fit indices for the independent pathway model (IPM) and the common pathway models (CPM) (*N* = 631 twin pairs)

Model	χ^2	*df*	S-B $\Delta\chi^2$	Δdf	*P* values	RMSEA	CFI	AIC	BIC
IPM	82.80	74	–	–	–	0.020	.99	18638.62	18789.83
CPM	**82.82**	**77**	**0.84**	**3**	**0.840**	**0.015**	**1.00**	**18633.69**	**18771.56**
CPM–Constrain BD, WP, and BE factor loadings	183.49	79	311.36	2	<0.001	0.065	.92	18762.44	18891.42
CPM–Constrain BD and BE factor loadings	114.73	78	32.63	1	<0.001	0.039	.97	18675.40	18808.82
CPM–Constrain BD and WP factor loadings	100.36	78	41.12	1	<0.001	0.030	.98	18654.91	18788.33
CPM–Constrain BE and WP factor loadings	182.26	78	89.84	1	<0.001	0.065	.92	18766.45	18899.87

Note. IPM, independent pathway model; CPM, common factor model; CPM-Constrain BD, WP, and BE factor loadings = common factor model with factor loadings constrained to be the same across all three disordered eating constructs (i.e., BD, WP, and BE); CPM- Constrain BD and BE factor loadings = common factor model with BD and BE factor loadings constrained to be equal and WP allowed to vary; CPM-Constrain BD and WP = common factor model with BD and WP factor loadings constrained to be equal and BE allowed to vary; CPM-constrain BE and WP = common factor model with BE and WP factor loadings constrained to be equal and BD allowed to vary; χ^2, chi-squared; *df*, degrees of freedom; S-B $\Delta\chi^2$, Satorra–Bentler model difference test; Δdf, change in degrees of freedom from the full common factor model and reduced common factor model; RMSEA, Root Mean Square Error of Approximation; CFI, Comparative Fit Index; AIC, Akaike's information criteria; BIC, Bayesian information criterion. Lower AIC and BIC values indicate good model fit, whereas CFI values above .95 and RMSEA values below .05 indicate good model fit. The CPM submodels with constrained factor loadings were compared to the full CPM model. The best fitting model is bolded and outlined.

3.55, $p < .001$; WP: MZ $r = .54$, DZ $r = .35$, z test of independence = 2.96, $p < .01$; BE: MZ $r = .43$, DZ $r = .20$, z test of independence = 3.19, $p < .001$) and nonshared environmental influences (i.e., MZ twin correlations < 1.00) on all three symptoms.

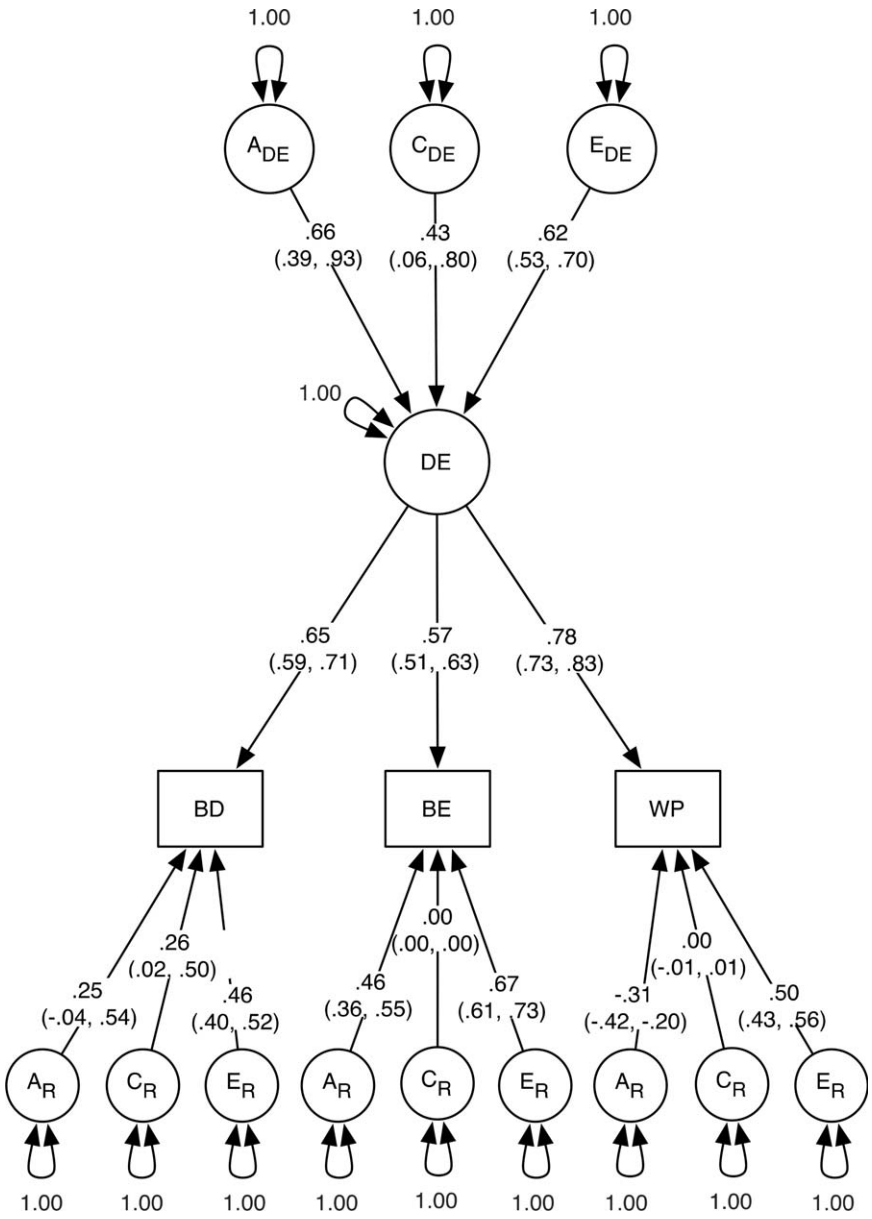
The full common pathway model provided a more parsimonious fit to the data than the full independent pathway model, as evidenced by a higher CFI value and lower RMSEA, AIC, and BIC values (**Table 1**). Similar to previous research, there were significant genetic (44%), shared environmental (19%), and nonshared environmental (38%) contributions to this common factor. Moreover, parameter estimates suggested that BE loaded less strongly on the common factor than the other disordered eating symptoms (see **Fig. 1**—common factor accounted for 42% of the variance in BD, 61% in WP, and only 32% in BE) and had larger residual estimates (i.e., 0–45% for BE, 6–21% for BD, and 0–25% for WP). To test differential loadings directly, we fit a submodel that constrained the factor loadings to be equal across the symptoms (i.e., CPM-Constrain factor loadings in **Table 1**). Results showed that BD, WP, and BE factor loadings could not be set to equal, as the Satorra–Bentler χ^2 difference test was highly significant. We then fit additional models to determine if some symptom factor loadings could be constrained (e.g., BE and BD), while others were allowed to vary (e.g., WP). None of these models fit the data better than the full common pathway model (i.e., all had significant χ^2s).

Discussion

We found lower factor loadings for BE and a higher proportion of BE-specific genetic and nonshared environmental influences compared to other disordered eating symptoms. While factor loadings for WP and BD were not equal, both loaded more strongly on the common factor than BE. Thus, similar to studies that demonstrate cross-disorder gene sharing,[15] our results suggest that disordered eating symptoms share a set of common genetic and environmental risk factors. However, our findings substantially advance this literature by showing that BE also exhibits unique etiologic effects.

Moving forward, it will be important to identify the common and unique mechanisms contributing to different types of disordered eating. One potential framework is the National Institute of Mental Health's Research Domain Criteria (RDoC).[16] The common factor identified herein may be linked to the RDoC negative valence domain, which is characterized by systems primarily responsible for negative affect and anxiety.[17] These systems are important for all types of eating disorders, including those characterized by BE.[18,19] By contrast, positive valence domain systems have been more strongly linked to BE and overeating (e.g., Bello and Hajnal (2010)[20]). Indeed, these systems are responsible for appetitive responses to reward and may contribute to BE beyond the effects of the negative valence domain systems. Clearly, the mechanisms contributing to common/unique effects go beyond these neurological and biological processes, as residual nonshared environmental influences were much larger for BE than the other symptoms. Nonetheless, the RDoC negative and positive valence domains provide a unifying framework for examining the genetic, neurobiological, and environmental factors that likely contribute to common and unique risk factors across eating disorders and their symptoms.

FIGURE 1. Path diagram of the full common pathway model (CPM). DE = Common Disordered Eating Factor; BD = Body Dissatisfaction; BE = Binge Eating; WP = Weight Preoccupation. A_{DE} = additive genetic effects on the common disordered eating factor; C_{DE} = shared environmental effects on the common disordered eating factor; E_{DE} = nonshared environmental effects on the common disordered eating factor; A_R = residual additive genetic effects; C_R = residual shared environmental effects; E_R = residual nonshared environmental effects. Values are standardized parameter estimates with 95% confidence intervals in parentheses. Individual path estimates can be squared to estimate the proportion of variance accounted by the common factor for each disordered eating symptom (e.g., for body dissatisfaction $.65^2 = .42$, suggesting that 42% of the total variance in body dissatisfaction can be attributed to the common factor). Similarly, path estimates can be squared to estimate the proportion of variance in the common factor accounted for by genetic (i.e., $.66^2 = .44$, or 44%), shared environmental (i.e., $.43^2 = .18$, or 18%) and nonshared environmental (i.e., $.62^2 = .38$ or 38%) influences. The proportion of residual variance accounted for by genetic and nonshared environmental factors can be determined in the same way (i.e., by squaring the path estimates).



Our study was not without limitations. We used a population-based sample and it is unknown if results extend to clinical populations. However, recent data suggest that binge eating exists on a continuum with clinical eating disorders,[21] and thus, results likely extend to clinical populations as well. Disordered eating was assessed with a self-report questionnaire. Future studies should test whether these results extend to interview-based measures. Ideally, these investigations should investigate a wider array of symptoms to inform etiological models and classification systems across the spectrum of pathology.

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