A Transdiagnostic Approach to Understanding Eating Disorders

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Abstract: Categorical models dominate the eating disorder field, but the tandem use of categorical and dimensional models has been proposed. A transdiagnostic dimensional model, number of lifetime eating disorder behaviors (LEDB), was examined with respect to (1) its relationship to a variety of indicators of the individual's functioning, (2) the degree to which it was influenced by genetic and environmental risk factors, and (3) exposure to specific environmental risk factors. Data from self-report and interview from 1002 female twins (mean age = 34.91 years, *SD* = 2.09) were examined. While 15.4% women met criteria for a lifetime eating disorder, 29% had at least one LEDB. The dimensional measure provided an indicator of associated functioning, and was influenced primarily by the nonshared environment. The number of LEDB was associated with the degree of impaired functioning. This impairment was associated with conflict between parents and criticism from parents when growing up.

Key Words: Eating disorder behaviors, transdiagnostic, genetic epidemiology, dimensional.

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There is debate about whether eating disorders have a dimensional or categorical nature. Taxometric analyses suggest inconsistency with a strictly dimensional model (Williamson et al., 2002), while other studies suggest that bulimia nervosa (BN) is a dimensional construct (Rowe et al., 2002). A recent review suggested that eating disorders that involve binge eating (and perhaps purging) are discontinuous with normalcy, whereas eating disorders not involving binge eating are dimensional in character (Williamson et al., 2005). However, even those disorders seen to conform to a discontinuous model are said to have latent dimensions that underlie the taxon (Waller and Meehl, 1998). These differing but not incompatible perspectives on eating disorders are consistent with a general call for the tandem use of categorical and dimensional assessments in mental illness to enhance clinical practice (Kessler, 2002).

To date, research of eating disorders has been dominated by categorical conceptualizations (i.e., using diagnosis to classify groups) with little attention paid to dimensional perspectives. No published study exists that examines the relationship between diagnosis and the number of lifetime eating disorder behaviors (LEDBs). In the current study, we sought to investigate the utility of a dimensional conceptualization of eating disorders, namely the number of LEDBs, defined for this purpose as objective binge eating, self-induced vomiting, laxative misuse, diuretic misuse, fasting, and low body weight (body mass index [BMI] ≤17.5). Our dimensional measure was consistent with a transdiagnostic perspective on eating disorders (Fairburn et al., 2003), with a focus on features that can occur across eating disorder groups, including eating disorder not otherwise specified (EDNOS), regardless of specific eating disorder diagnosis.

We therefore used data from a large twin population to examine this dimensional conceptualization in three ways. First, we compared patterns of diagnosis and functioning across the number of LEDBs. Second, we examined the contribution of genetic and environmental risk factors to our dimensional measure and compared this to a diagnostic measure, so we could examine any qualitative differences between the two. Third, we examined the relationship between LEDB and specific types of environment that have been previously implicated in the research literature as influencing the development of eating disorder behaviors.

METHODS

Participants

The data for the current study of female twin pairs came from three waves of data collection, summarized in Figure 1. The first two waves have previously been described (Heath et al., 2001), and the response rate in Figure 1 refers to all female twins, including females from dizygotic (DZ) female-

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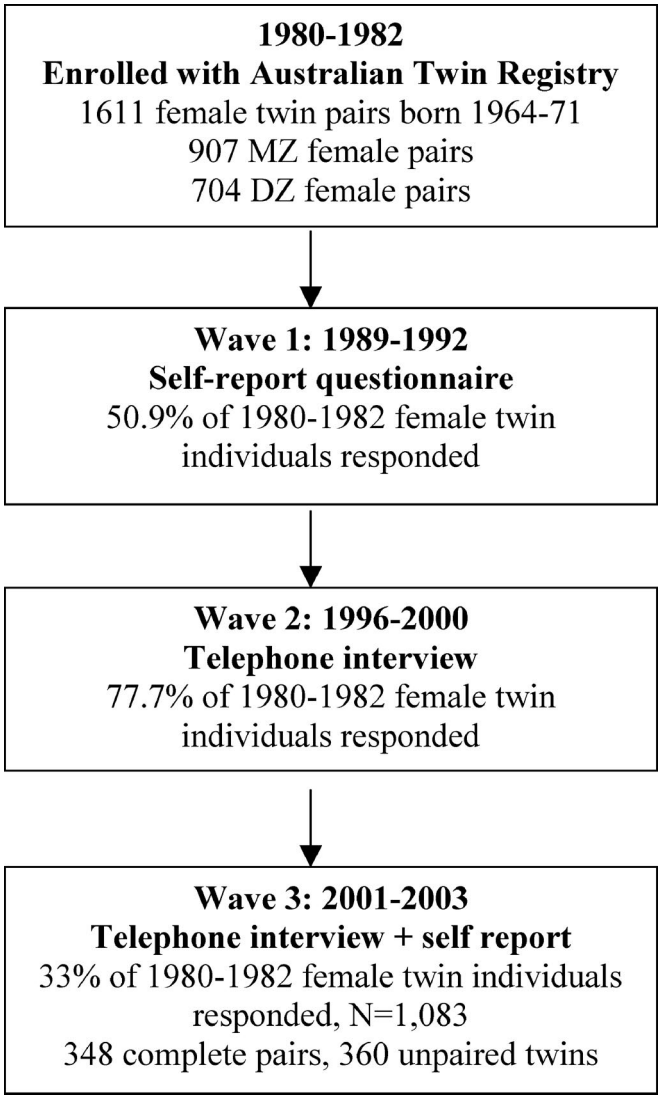

FIGURE 1. Sample size and waves of assessment of female-female MZ and DZ twins.

male pairs (Heath et al., 2001). The sample was originally derived from a cohort of 8536 twins (4268 pairs) born 1964 to 1971 who were registered as children with the Australian Twin Registry (ATR) over 1980 to 1982 in response to media appeals and systematic appeals through schools. In the third wave, there were 907 monozygotic (MZ) and 704 DZ female twin pairs.

Wave 1 data collection occurred in 1989 to 1992 when the twins were aged 18 to 25 years, when a self-report questionnaire was mailed that included questions about the presence of a variety of eating disorders. The response rate for women in the whole sample was 50.9%. Wave 2 data collection occurred over 1996 to 2000 using a diagnostic interview assessment over the telephone, which did not include assessment of eating disorder status. In this case, 77.7% of the original female sample completed interviews. While education below university level and being a DZ rather than an MZ twin predicted reduced likelihood of participating in the self-report questionnaire, associations between psychiatric history and health behavior variables were modest, and there was no association between BMI and questionnaire nonresponse (Heath et al., 2001).

The total protocol for wave 3 consisted of two parts: a self-report questionnaire and a telephone interview. Women were approached to participate if they had either participated in either the wave 1 or wave 2 data collection and came from female-female twin pairs. In all, 2320 twins or 1140 complete pairs were approached by the ATR, of which 1083 twins consented to participate (47%), 568 declined (24%), and 669 did not respond (29%). At least one further telephone call or letter was used to contact the nonresponders. Of those who consented, 1002 (43% of the total sample) completed the interview, and 1016 (44% of the total sample) completed the self-report questionnaire, with 962 women completing both the interview and the questionnaire, 54 completing the questionnaire only, and 40 completing the interview only. In all, 1056 females (46% of the total sample) participated in at least one of the data collection components, of whom 613 were from MZ pairs and 443 were from DZ pairs. The final sample included 348 complete pairs (221 MZ and 127 DZ) and 360 incomplete pairs (171 MZ and 189 DZ) in which only one of the twins participated. The Flinders University Social and Behavioral Ethics Committee approved wave 3 data collection.

Zygosity was determined on the basis of responses to standard questions about physical similarity and confusion of twins by parents, teachers, and strangers, methods that have been found to give better than 95% agreement with results of genotyping (Eaves et al., 1989).

Assessment

Wave 1

A mailed self-report questionnaire was used to assess lifetime eating disorders at Wave 1 of the same format reported previously for an older group of twins (Wade et al., 1996). This included 16 questions using a yes/no format designed to identify the presence of lifetime eating disorders, including difficulty controlling weight, presence of BN, anorexia nervosa (AN), obesity, or binge eating. A previous investigation has shown the measure to be associated with five latent factors, including syndromes consistent with overweight and overeating, AN, BN, and weight control including nonpurging and purging behaviors (Wade et al., 1996). Reliability over 10 years when using brief self-report measures of disordered eating show a κ of around 0.45 to 0.50 (Field et al., 1996), which can be compared with a κ of 0.61 for major depression over a 6-year period using a structured psychiatric assessment (Rice et al., 1992). For the purpose of the current study, the mean number of eating problems was calculated. Additionally, data relating to temperament and life events were investigated (Table 1).

Wave 2

A structured diagnostic interview designed for genetic studies on alcoholism, the Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994), was adapted

TABLE 1. Summary and Description of the Measures of Function

Variable	Description and Cronbach α
Wave 3	
Self esteem	Rosenberg Self-Esteem Scale (Rosenberg, 1960), 10 items, $\alpha = .87$
Impulsivity	Barratt Impulsiveness Scale (Barratt, 1959), 30 items, $\alpha = .81$
Perfectionism: concern about mistakes, personal standards, doubts about actions, parental expectations,[a] parental criticism[a]	Frost Multidimensional Perfectionism Scale (Frost et al., 1990), respectively 9 items, $\alpha = .90$; 7 items, $\alpha = .85$; 4 items, $\alpha = .82$; 5 items, $\alpha = .86$, and 4 items, $\alpha = .89$
Parental conflict[a]	Revised Moos Family Environment Scale, conflict subscale (Moos, 1974), 9 items, $\alpha = .73$
Comments about weight (from family, other adults, peers)[a]	Risk Factor Interview (Fairburn et al., 1997), 5 items, $\alpha = .88$
Body dissatisfaction	Adapted figural stimuli (Stunkard et al., 1983)
Weight concern, shape concern, eating concern and dietary restraint	Eating Disorder Examination (Fairburn and Cooper, 1993), respectively 5 items, $\alpha = .74$; 7 items, $\alpha = .86$; 5 items, $\alpha = .72$; 5 items, $\alpha = .60$
Body mass index, self-report	Weight (kg)/height $(m)^2$
Wave 2	
Major depression	Semi-Structured Assessment for the Genetics of Alcohol (SSAGA: Bucholz et al., 1994), DSM-IV criteria
Suicidality	SSAGA 0–3 scale: 0 = no suicidal ideation, 1 = any suicidal thoughts, 2 = persistent thoughts, 3 = suicide attempt
Wave 1	
Neuroticism	Eysenck Personality Questionnaire (Eysenck et al., 1985), 12 items, $\alpha = .80$
Harm avoidance, novelty seeking, reward dependence	Tridimensional Personality Questionnaire (Cloninger et al., 1991), 18 items each scale, respective $\alpha = .84, .74, .62$
Interpersonal dependency	Interpersonal Sensitivity Measure (Boyce and Parker, 1989), 12 items, $\alpha = .51$
Parental care and protectiveness[a]	Parental Bonding Inventory (Parker et al., 1979), 3 care items and 4 protectiveness items each for mother and father, respective $\alpha = .69, .65, .69, .58$

[a]Assessed for the period of the first 16 years of age.

for telephone use with an Australian sample and updated for DSM-IV diagnostic criteria (American Psychiatric Association, 1994). The interviewers were trained lay people. The diagnosis of major depression and a measure of suicidality were included as further dimensions of functioning (Table 1).

Wave 3

The content of the self-report questionnaire is summarized in Table 1. The telephone interview consisted of the Eating Disorder Examination (EDE; Fairburn and Cooper, 1993), revised according to the wording of the 14th edition. As the EDE assesses the previous 3 months of functioning, the interview was adapted so that it also assessed the lifetime presence of eating disorder features. Thus, our assessment allowed for the calculation of current eating disorder status and lifetime eating disorder status. Agreement between telephone and face-to-face diagnostic interviews has been found to be excellent and is likely to be highest for behaviorally anchored items (Rohde et al., 1997). All interviewers were postgraduate clinical psychology trainees who had been trained in use of the EDE by the first or second author. Each of the interviews was taped. Each new interviewer submitted these tapes as they were completed to the second author (J. B.), who provided corrective feedback until the interviewer had acquired the skills required to complete the interview independently. When interviewers were uncertain of ratings, they requested a second opinion. The first two authors made the final rating decisions. Monthly group meetings with T. W. and J. B. to discuss the interview process ensured interview fidelity.

Wave 3 Eating Disordered Behavior

Diagnoses

Lifetime DSM-IV diagnostic criteria (American Psychiatric Association, 1994) as reported from the EDE were used to form lifetime diagnoses. Consistent with previous authors, amenorrhea was not required for a diagnosis of AN (McIntosh et al., 2005). Given the lack of agreed diagnostic criteria for the DSM-IV diagnosis EDNOS (Fairburn and Bohn, 2005), we derived two subcategories designed to represent many of those belonging to this group. The first was binge eating disorder, in which there had to have been two objective binges a week for 3 months but concurrent weight control behavior was absent or did not meet the frequency criteria. Second, women who met the frequency criteria for purging weight control behavior but who had not experienced concurrent objective binges were given the diagnosis EDNOS–purging subtype (EDNOS-p). The prevalence of these eating disorders in the current sample can be found in some detail elsewhere (Wade et al., 2006) and are summarized in Table 2. The prevalence of eating disorders in this community sample was similar to that found in recent investigations of such samples using comprehensive assessments of eating behavior (Favaro et al., 2003; Striegel-Moore et al., 2003), where respective prevalence for AN was 1.5% and 2% (or 3.3% when amenorrhea was not required), and for BN, 2.3% and 4.6%.

LEDBs

The presence of six LEDBs was assessed using the EDE, namely objective binge eating, self-induced vomiting, laxative misuse, diuretic misuse, fasting, and self-reported low body weight (BMI $\leq$17.5). In each case, the behavior had to occur at the same frequency and duration threshold as required to make one or other of the specific DSM-IV eating disorder diagnoses (e.g., low body weight was sustained over a 3-month period, objective binge eating occurred at least twice a week over a 3-month period). Excessive exercise was not included as definition of this construct is difficult to

TABLE 2. Eating Disorder Diagnostic Groups Represented in the Sample and Number of Lifetime Behaviors Associated With Each Diagnosis

Lifetime DSM-IV Diagnosis	Number (%)	Number of Lifetime Behaviors		
		1	2	3–5
Anorexia nervosa and bulimia nervosa[a]	5 (0.5)	0	0	5
Anorexia nervosa and EDNOS-purging[b]	5 (0.5)	0	1	4
Anorexia nervosa purging type	15 (1.5)	0	2	13
Anorexia nervosa restricting type	18 (1.8)	13	4	1
Bulimia nervosa purging type[c]	22 (2.2)	0	11	11
Bulimia nervosa non-purging type	7 (0.7)	6[d]	1	0
Binge eating disorder	29 (2.9)	23	6	0
EDNOS, purging type[c]	53 (5.3)	28	22	3
No lifetime eating disorder diagnosis	848 (84.6)	137	3	0
Total	1002	207	50	37

[a]Met criteria for lifetime anorexia nervosa and bulimia nervosa at separate times.
[b]Met criteria for lifetime anorexia nervosa and EDNOS-purging type at separate times.
[c]Excludes those who also had lifetime anorexia nervosa.
[d]Women who used excessive exercising as the only compensatory behavior for the binge eating.

identify reliably. Hence, the maximum number of lifetime behaviors per person was six.

Statistical Analyses

To achieve our first aim, we used logistic regressions to examine impairment of function associated with the different levels of eating disorder behaviors. Women with no LEDB were compared with women who had one, two, and three to five behaviors, respectively. Continuous measures were standardized so that the resulting odds ratios (ORs) and 95% confidence intervals (95% CIs) indicated the change in risk for the dependent measures for every *SD* change in the independent variable. As our observations were correlated, the assumption of independent sampling was violated. Given that our statistical package (SPSS) had no way of adjusting standard errors for nonindependent observations that involve dichotomous variables, we used the conservative adjustment procedure outlined previously by Kendler and Gardner (1998), and thus corrected *p* values are provided. We did not adjust our 95% CI as the purpose of these analyses was comparative rather than for the provision of absolute values.

To achieve our second aim, we used bivariate twin modeling procedures to investigate the overlap between genetic and environmental risk factors for the wave 3 dimensional measure and the wave 1 questionnaire report of lifetime eating disorders. We used a bivariate model including the self-reported eating disorders at wave 1 and the wave 3 LEDB. Such bivariate models are more powerful than univariate models as they allow for covariance relationships to aid in optimization of the parameters (Neale et al., 2003). Additionally, our use of both wave 1 and wave 3 data meant that the wave 3 measure was corrected for any ascertainment bias resulting from differential attrition with respect to eating problems since wave 1 (Little and Rubin, 1987). We used raw data rather than variance-covariance matrices so that data from incomplete twin pairs could be included and used in the analyses. As the data were positively skewed, the normal weights of the raw scores were used (Wade et al., 1999). To examine the correlations between each twin (cross-twin) and each variable (cross-trait), the data were analyzed using maximum likelihood estimation with Mx (Neale, 1997). Second, to examine the sources of individual difference of these scales, the data were examined using bivariate Cholesky decomposition procedures (Neale and Cardon, 1992) with Mx. Three latent influences on each measure are modeled: additive genes (A), common or shared environment (C), and nonshared or unique environment (E). The full ACE model was first fitted to the data, followed by an AE, CE, and E model, including correlation terms between the latent sources of variance. The goal of model fitting was to explain the observed data as an optimal combination of goodness-of-fit and parsimony. These models and this process as it applies to eating disorders have been fully explained previously (Bulik et al., 2000).

To achieve our third aim, specific sources of retrospectively reported environment were examined across the three levels of eating disordered behavior and compared with women with no eating disorder behavior in logistic regressions as described for our first aim.

RESULTS

Participation at Wave 3

Of those women participating in wave 3, 78% had completed the eating questions at wave 1, and all but two women (99.8%) had completed an interview at wave 2. This sample represents 33% of the original group of female children enrolled in the ATR, 45% of those women who participated at wave 1, and 40.2% of those women who participated at wave 2. Participation at wave 3 was not predicted by the number of eating problems at wave 1 ($t\,[960] = 1.00$, $p = 0.32$), nor by any personality variables, including neuroticism ($t\,[1001] = -0.96$, $p = 0.34$), harm avoidance ($t\,[988] = -0.93$, $p = 0.36$), or novelty seeking ($t\,[978] = -0.46$, $p = 0.65$). Neither was participation at wave 3 predicted by lifetime depression reported at wave 2 ($\chi^2[1] = 0.45$, $p = 0.50$). However, those women who reported higher levels of lifetime suicidality at wave 2 were significantly more likely to participate at wave 3 ($t\,[700.70] = 1.98$, $p = 0.049$). The mean age at the time of wave 3 data collection was 34.97 years ($SD = 2.11$), ranging from 28.10 years to 39.98 years.

Eating Disorder Behaviors and Their Relationship to Diagnosis

The majority of women ($N = 708$, 70.7%) had not experienced any LEDB. A total of 207 (20.7%) women had experienced one such behavior, 50 (5%) had experienced

two, and 37 (3.7%) reported three behaviors. The relationship between diagnosis and eating disorder behaviors is summarized in Table 2, where it can be seen that around 15% of the sample met criteria for a lifetime eating disorder.

Relationship With Lifetime Behaviors and Function

The comparisons between those women with no LEDB with those with one, two, or three to five LEDBs are shown in Table 3. In this case, there was a clear general trend in which more LEDB was associated with incrementally increased impairment for 12 of the 18 variables. Given that the greatest proportion of women with three to five lifetime LEDBs had AN (comprising 57% of the total group membership), we investigated whether the group with the most lifetime behaviors was being influenced solely by AN. We therefore selected the women with AN and compared those women with one to two behaviors to those women with three to five behaviors. Despite less power to find significant results, the same trend applied, with incremental increases in psychopathology across the lifetime behavior groups. In particular, there was a significant difference between these two groups (using one-tailed tests) with respect to self-esteem (OR = 0.41, 95% CI: 0.19–0.88); concern about mistakes (OR = 1.85, 95% CI: 0.92–3.69); impulsivity (OR = 1.85, 95% CI: 1.00–3.57); comments from others about food, weight, or eating (OR = 1.85, 95% CI: 0.92–3.69); parental expectations (OR = 1.83, 95% CI: 0.93–3.63); parental criticism (OR = 1.67, 95% CI: 0.93–3.00); major depression (OR = 4.55, 95% CI: 1.33–15.57); suicidality (OR = 2.70, 95% CI: 1.37–5.30); and novelty seeking (OR = 2.18, 95% CI: 1.01–4.72).

To investigate whether the number of LEDBs was being confounded with duration of disordered eating, and thus accounting for the relationship to function, we investigated whether duration predicted the number of lifetime behaviors using a series of ANOVAs. There was no relationship between the number of lifetime behaviors and duration of low weight for women with AN ($F\,[2, 40] = 1.82$, $p = 0.18$), duration of binge eating for women with binge eating disorder ($F\,[1, 25] = 0.60$, $p = 0.44$), or duration of vomiting ($F\,[2, 17] = 0.33$, $p = 0.72$) or diuretic use ($F\,[2, 8] = 1.38$, $p = 0.31$) for women with EDNOS-p. However, duration of binge eating did predict the number of behaviors for women

TABLE 3. Logistic Regression Comparing Women With No Lifetime Eating Disorder Behaviors (LEDBs) to Women Who Had 1, 2 or 3 to 5 LEDBs

Variable (standardized)	0 LEDB/1 LEDB OR (95% CI)	0 LEDB/2 LEDB OR (95% CI)	0 LEDB/3–5 LEDB OR (95% CI)
Wave 3 measures of temperament and eating			
Self-esteem	0.86 (0.73–1.01)	0.84 (0.62–1.15)	0.36 (0.26–0.51)*
Impulsivity	1.10 (0.93–1.29)	0.99 (0.73–1.34)	1.43 (1.02–2.01)***
Concern about mistakes	1.16 (0.98–1.36)	1.35 (1.01–1.81)	3.03 (2.13–4.30)*
Personal standards	1.00 (0.85–1.18)	1.23 (0.89–1.68)	2.39 (1.74–3.28)*
Doubts about actions	1.11 (0.94–1.30)	1.04 (0.75–1.44)	2.08 (1.54–2.81)*
Body dissatisfaction	0.81 (0.68–0.96)*	1.04 (0.77–1.41)	1.33 (1.00–1.78)
Weight concern	1.18 (1.01–1.38)***	1.73 (1.35–2.22)*	1.96 (1.48–2.60)*
Shape concern	1.23 (1.05–1.43)***	1.78 (1.38–2.31)*	1.89 (1.41–2.54)*
Eating concern	1.25 (1.07–1.45)***	1.33 (1.07–1.66)***	1.46 (1.19–1.79)**
Dietary restraint	1.14 (0.97–1.33)	1.78 (1.41–2.26)*	1.20 (0.87–1.67)
BMI	0.59 (0.47–0.73)*	0.97 (0.71–1.32)	0.86 (0.59–1.26)
Wave 2Wave 2 measures of lifetime depressive psychopathology			
Major depression	1.96 (1.42–2.69)*	2.17 (1.22–3.87)***	7.65 (3.54–16.53)*
Suicidality	1.23 (1.03–1.46)***	1.63 (1.23–2.16)**	2.39 (1.79–3.20)*
Wave 3 measures of temperament			
Neuroticism	1.14 (0.95–1.37)	1.24 (0.88–1.74)	1.50 (1.03–2.20)***
Interpersonal sensitivity	1.14 (0.95–1.37)	1.22 (0.86–1.73)	1.42 (0.95–2.12)
Harm avoidance	1.01 (0.84–1.22)	0.86 (0.60–1.24)	1.27 (0.87–1.87)
Novelty seeking	1.02 (0.85–1.23)	1.35 (0.96–1.89)	1.49 (1.02–2.17)***
Reward dependence	1.33 (1.10–1.61)***	1.13 (0.79–1.62)	1.05 (0.71–1.54)
Wave 3 and Wave 1 measures of retrospectively reported life events in the first 16 years of life			
Parental conflict	1.14 (0.97–1.33)	1.50 (1.14–1.99)***	1.94 (1.44–2.61)*
Comments about weight	1.38 (1.17–1.63)**	1.51 (1.11–2.04)***	2.95 (2.09–4.17)*
Parental expectations	0.92 (0.78–1.09)	1.01 (0.75–1.37)	1.76 (1.31–2.37)**
Parental criticisms	1.12 (0.96–1.32)	1.20 (0.90–1.60)	1.94 (1.44–2.61)*
Care from mother	0.91 (0.76–1.07)	0.71 (0.55–0.90)***	0.69 (0.53–0.90)***
Overprotection from mother	1.07 (0.90–1.29)	1.27 (0.92–1.76)	1.23 (0.85–1.77)
Care from father	1.08 (0.88–1.32)	0.69 (0.52–0.92)***	0.64 (0.47–0.88)***
Overprotection from father	0.97 (0.80–1.17)	1.07 (0.77–1.49)	1.16 (0.79–1.70)

*$p < 0.05$; **$p < 0.001$; ***$p < 0.0001$.

with BN (F [2, 31] = 5.05, p = 0.01), with women with one behavior having significantly longer duration of disorder than women with two or more behaviors. Duration of laxative abuse predicted the number of behaviors for women with EDNOS-p (F [2, 30] = 6.50, p = 0.005), where women with three or more behaviors had longer duration of disorder than the other women. Overall the data were not consistent with the suggestion that chronicity and the number of behaviors are confounded.

Twin Analyses

Cross-Trait and Cross-Twin Correlations

These correlations are displayed in Table 4. Within each twin in the pair, the wave 1 eating measure and the wave 3 LEDB were correlated at 0.33 for twin 1 and 0.16 for twin 2 for MZ twins, and 0.31 for twin 1 and 0.38 for twin 2 for DZ twins. Cross-twin correlations for eating behavior at wave 1 were 0.54 for MZ twins and 0.11 for DZ twins. The maximum likelihood estimates of the cross-twin correlations for wave three lifetime behaviors were 0.11 for MZ twins and −0.01 for DZ twins.

Bivariate Genetic Analysis

To examine the genetic epidemiology of our lifetime behavior measure, a bivariate genetic analysis with the wave 1 eating measure was performed. In the ACE model, the parameter estimate for the shared environment for both measures was zero, and the AE model was not significantly worse fitting than the ACE model (χ^2 [3] = 0, $p > 0.99$). However, both the CE (χ^2 [3] = 40.76, $p < 0.01$) and the E (χ^2 [6] = 136.16, $p < 0.01$) models were significantly worse fitting than the full model, and therefore, these models were not examined further. As shown in Table 5, variations of the AE models were further examined by systematically removing parameters related to the correlations between the genetic and nonshared environment latent factors. The best fitting and most parsimonious model was one that postulated additive genes and nonshared environment contributing to both the phenotypes but that only included a correlation between the latent additive genetic variable. The additive genetic action (52%, 95% CI: 44%–58%) and nonshared environment (48%, 95% CI: 41%–56%) accounted for roughly equal variance in the wave 1 phenotype, whereas the majority of the variance of the lifetime behaviors was accounted for by the nonshared environment (90%, 95% CI: 79%–100%) rather than the additive genetic action (10%, 95% CI: 0.002%–21%). Around 18% of the variance between the additive genetic component (r = 0.42) for the wave 1 and wave 3 measures was shared.

TABLE 4. Maximum Likelihood Estimates for Cross-Twin, Cross-Trait Correlations Between the Wave 1 Measure of Disordered Eating and the Wave 3 Number of Lifetime Eating Disorder Behaviors[a]

	Twin 1		Twin 2	
	Wave 1	Wave 3	Wave 1	Wave 3
Twin 1				
Wave 1	1.00	0.33	**0.54**	0.09
Wave 3	0.31	1.00	0.08	**0.11**
Twin 2				
Wave 1	**0.11**	−0.02	1.00	0.16
Wave 3	0.03	**−0.01**	0.38	1.00

[a]Twin pair correlations are in bold, MZ twin correlations are in the upper diagonal, and DZ twin correlations are in the lower diagonal.

TABLE 5. Comparative Fit of the Various Bivariate Models Examining Wave 1 and Wave 3 Measures

Model	−2 Log Likelihood	*df*	Versus Model #	Δ χ^2 (*df*) *p*
1. ACE-$r_a r_c r_e$	6820.71	2719	—	—
2. AE-$r_a r_e$	6820.71	2722	1	0 (3) $p > 0.99$
3. AE-r_a	6820.71	2723	2	0 (1) $p > 0.99$
4. AE-r_e	6825.46	2723	2	4.75 (1) $p < 0.05$

Specific Environmental Risk Factors

Sources of possible specific environmental risk factors, reported retrospectively for the first 16 years, are reported in Table 3. While neither of the overprotection variables was significantly related to the number of lifetime behaviors, there were positive associations between the number of eating disorder behaviors and parental conflict, comments about weight, parental expectations, parental criticism, and negative associations with both maternal and paternal care.

DISCUSSION

The focus of the current report is an exploration of a dimensional measure of eating disorders, namely the number of LEDBs reported by each woman. A strong pattern of increase of impaired functioning as the number of behaviors increased was indicated. More areas of life were impacted when three or more LEDBs were reported. Compared with women reporting fewer than three behaviors, women with three or more behaviors reported lower levels of self-esteem and higher levels of impulsivity, personal standards, concern over mistakes, doubts about actions, neuroticism, and novelty seeking. This pattern is similar to that found in depression, where psychosocial impairment is linearly related to symptom count, and reaching diagnostic symptom threshold has no special implications for the expected level of impairment (Pickles et al., 2001). Regardless of eating disorder status, and including the two EDNOS subdiagnoses, the number of lifetime behavioral features present provided a useful indicator of the severity of associated functional impairment.

The genetic epidemiology of our two eating measures suggests that they are reflecting different but related constructs that share some genetic risk factors. Our wave 1 measure reflects eating disorder groupings and is most similar in nature to a recent study of features associated with BN (Rowe et al., 2002), where a higher score indicated an increased likelihood of meeting diagnosis for an eating disorder. As with the current study, an AE model was found to be the best fitting model with very similar estimates of genetic influence to our measure (54%, 95% CI: 44–62 compared with our estimate, 52%, 95% CI: 48–58). This finding is consistent with other studies that have shown genetic influence to be an important factor with respect to the

development of an eating disorder (Bulik et al., 1998; Wade et al., 1999). In contrast, our wave 3 measure, the number of LEDBs, is not associated with any one eating disorder, and includes many people without a clinical eating disorder (almost one half of this group did not have an eating disorder diagnosis). Twin concordance on this measure can be seen to indicate a similar degree of impairment of function rather than an increased likelihood of meeting criteria for an eating disorder. Therefore, our findings suggest that it is the nonshared environment that has the major role in determining the severity of disordered eating and its associated impact on the person's function. Our finding that the environment is an important and major contributor to some dimensions of the eating disorder phenotype is not unique. Studies of two different twin populations have found that weight concern (the undue influence of body weight on self-evaluation, part of the diagnostic criteria for both AN and BN) is influenced solely by environmental factors (Reichborn-Kjennerud et al., 2004; Wade et al., 1998).

Most of the specific environmental variables included recall of the parental relationship in the first 16 years of life, and all except parental overprotection were associated with increasing lifetime behaviors in the predicted directions. These variables can potentially be seen to be part of the nonshared environment given that this type of environment can be either *objective,* an actual experience or event that is not shared by siblings, or *effective*, where the same event can be experienced uniquely by each family member, depending on a number of factors such as age and temperament, thus producing differential outcomes (Turkheimer and Waldron, 2000).

The results of this study should be interpreted in the context of four important limitations. First, we had a less than optimal response rate for our wave 1 and wave 3 data at 46%. This rate is commensurate with other large population studies in Australia (Brown et al., 1998) but lower than others (Hay, 2003). There was no indication that eating problems or personality influenced participation at wave 3. However, those with poor outcome with respect to the eating disorders may have been under-represented in the current study. Second, we had only a small number of twin pairs for our wave 3 data. This limited our power to accurately determine parameters in our genetic analyses. However, the bivariate estimations of these parameters provide more accurate parameter estimations. Third, our bivariate analysis used a self report measure and an interview based measure, which may contribute to the qualitative differences of the measures. Fourth, we examined only a limited aspect of impaired function. Further research is required to identify whether the number of LEDB is associated with impairment of other domains of life including social, vocational and quality of life. Future research should further investigate a variety of domains of function in addition to a wider range of variables that may explain the contribution of the nonshared environment to increased LEDB.

CONCLUSION

In summary, in this dimensional transdiagnostic study the number of LEDBs reported was associated with the degree of impairment of function experienced by the person, even though LEDBs were not necessarily indicative of the presence of an eating disorder and the number of LEDBs varied across different eating disorder diagnoses. This impairment seemed to be mainly influenced by the nonshared environment. Conflict between parents and criticism from parents when growing up appeared to be aspects of this effective nonshared environment, as were comments about eating and weight from others including peers.

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