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Genetic and environmental risk factors for the weight and shape concerns characteristic of bulimia nervosa

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ABSTRACT

Background. This study seeks to identify the genetic and environmental risk factors for the overvalued ideas that are characteristic of bulimia nervosa, using a biometrical model fitting approach with twin data.

Methods. The Eating Disorder Examination (EDE), which can be used to gain continuous measures of dietary restraint, eating concern, weight concern and shape concern, was administered to 325 female twins, both monozygotic (MZ) and dizygotic (DZ). For each subscale, questions were asked concerning the month prior to interview and lifetime prevalence ('ever').

Results. Model fitting indicated that there is a powerful role of the environment in shaping women's attitude towards weight, shape, eating and food, ranging from 38% to 100% of the variance. For all subscales, with the exception of weight concern, the best explanation for individual variation was one that incorporated additive genetic and non-shared environmental influences. In contrast, model fitting indicated that non-shared and shared environmental influences best explained the variance of weight concern.

Conclusions. With the exception of the Shape Concern subscale, environmental factors make a greater contribution than genetic factors to the development of the overvalued ideas that are seen to be one of the triggers for the development of bulimia nervosa. Given this substantial role of the environment influences, it seems likely that environmental manipulation can be effective in the prevention of bulimia nervosa.

INTRODUCTION

Bulimia nervosa is estimated to affect about 2% of women at some stage during their lifetime (Fairburn & Beglin, 1990). It is a disorder that is currently considered to consist of three major components, namely binge eating, innapropriate weight control behaviours (e.g. self-induced vomiting, laxative abuse, excessive exercise, starvation) and overvalued ideas regarding the importance of weight and shape in the definition of self-evaluation (American Psychiatric Association (APA), 1994). Within a cognitive model of bulimia nervosa, the overvalued ideas are argued to be the central cognitive substrate of bulimia nervosa (Cooper & Fairburn, 1993). These overvalued ideas of the importance of weight and shape can be distinguished from dissatisfaction with body shape, which is a labile feature that fluctuates in response to changes in shape, weight, control over eating and mood, and which can occur in other patient and non-patient groups (Cooper *et al*. 1987). In contrast, the overvalued beliefs are seen to be stable over time and are present only in eating disordered populations.

Disentanglement of the aetiology and influences on the development of bulimia nervosa, and indeed any psychiatric disorder, has proved to be a difficult and inconclusive

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task. Increasingly, researchers are turning to twin research to gain a greater understanding about the causes of variability in psychiatric morbidity (Kendler, 1993). There is no evidence, to date, to suggest that twins differ from singletons with respect to their risk for psychiatric disorders (Chitkara *et al.* 1988; Kendler *et al.* 1995*a*) and thus twin populations can provide a useful source of information about aetiological models. Although one study has found the concordance for bulimia nervosa to be the same across monozygotic (MZ) and dizygotic (DZ) twins (Treasure & Holland, 1991), the majority of twin studies of bulimia nervosa find evidence that MZ twins are more than twice as likely to be concordant for a diagnosis of bulimia nervosa than DZ twins (Fichter & Noegel, 1990; Hsu *et al.* 1990; Kendler *et al.* 1991). On the whole, this would suggest some genetic involvement in the development of bulimia nervosa. In terms of specific estimations, Kendler *et al.* (1991) found that about 50% of the variance in liability to bulimia nervosa was due to additive gene action and 50% due to individual-specific environment. However, the low prevalence rate of bulimia nervosa meant that this model's superiority over one replacing genetic factors with common-environmental factors was modest, and the authors concluded that it was not possible to rule out definitively the influence of shared environmental factors common to both twins.

In addition to the twin studies of women who have a diagnosis of bulimia nervosa, two twin studies exist that have used continuous measures of disordered eating and the attitudes and personality factors thought to predispose women to developing bulimia nervosa or anorexia nervosa. In the first of these, Holland *et al.* (1988) examined scores on the Eating Disorder Inventory (EDI, Garner *et al.* 1983), a commonly used indicator of disturbed attitudes and personality traits thought to be associated with eating disorders, in a twin population in which at least one twin was affected by anorexia nervosa. Findings suggested heritability estimates of almost 100% for the drive for thinness and body dissatisfaction subscales of the EDI. However, this study used a clinical population, which makes generalization of the results to a normal population difficult.

The second study, that of Rutherford *et al.* (1993) has addressed this weakness by examining the eating attitudes of a series of 246 normal twins pairs, using both the EDI and the Eating Attitudes Test (EAT, Garner & Garfinkel, 1979). This research found that a model suggesting additive genetic and unique environmental influences was likely to be the best-fitting, and that shared environment could be constrained to zero. From their preferred model, the derived heritability of the total EAT score was 41% and ranged from 25% to 52% for the EDI subscales, with the body dissatisfaction and drive for thinness subscales showing the highest heritabilities. Notably, the heritably estimates for the bulimia nervosa of the EDI and EAT were among the lowest, at 28% and 26% respectively, but the variance of shared-environment was able to be constrained to zero. This study suggests that environmental influences have a large role to play in the development of disordered eating in the general population. Neither of these studies, however, examined the overvalued ideas central to the cognitive formulation of bulimia nervosa (Cooper & Fairburn, 1993).

One twin study exists that dissents from these general findings. Kendler *et al.* (1995*b*) investigated the genetic and environmental risk factors for bulimia nervosa in conjunction with five other psychiatric disorders, namely specific phobias, generalized anxiety disorder, panic disorder, major depression and alcoholism, thereby generating more power for detecting effects. Their analyses revealed a substantial role for shared-environmental factors in bulimia nervosa, which contributed 41% of the variance. Genetic factors and individual-specific factors accounted for around 30% and 29% respectively. These results were in striking contrast to the other psychiatric disorders, where virtually no common-environmental factors were found to play a role.

In summary, there is agreement that the influence of environmental factors on bulimia nervosa is substantial. However, there is some confusion about the extent and type of these environmental risk factors. Those analyses utilizing greater methodological sophistication and larger subject numbers would indicate that 50–70% of the variance is made up of en-

vironmental influence and there is some suggestion that both shared and non-shared environment are substantial contributors to the development of bulimia nervosa. In addition, there is a general weakness in the assessments of bulimia nervosa in previous twin studies of disordered eating. In some of these studies, measures of disordered eating have been reliant on questions embedded within psychiatric interview schedules, which use a binary definition of an eating disorder (i.e. has an eating disorder or not). Such interview schedules have been shown to provide an indication of disordered eating in general rather than an indication of bulimia nervosa in particular (Wade *et al.* 1997*a*). The remainder of the studies have used continuous measures that arguably do not measure the core, stable psychopathology of bulimia nervosa, but focus instead on body dissatisfaction.

To date, there has been no specific examination of the genetic influences on the overvalued ideas associated with bulimia nervosa, which are hypothesized to be the core cognitive substrate of bulimia nervosa and central to the development of bulimia nervosa (Fairburn & Cooper, 1989; Cooper & Fairburn, 1993). The major aim of the present study is to examine the genetic and environmental risk factors for the attitudes associated with bulimia nervosa, as measured on a continuous scale. The method of analysis in this paper will use the more informative and powerful statistical approaches of Neale & Cardon (1992).

METHOD

Measure

The Eating Disorder Examination (EDE, Fairburn & Cooper, 1993) was in part formulated precisely to measure weight and shape concerns and the extent to which people equate body shape and weight with self-worth. Hence, this represents an assessment tool that measures the core psychopathology of bulimia nervosa (Wilson, 1993). The EDE is a semi-structured, investigator-based interview. Each item of the EDE is measured on a seven-point scale of severity. The final total score is the average of all items, and ranges from 0 to 6, with 0 indicating no problems and 6 indicating frequent and severe problems. Items are rated for the preceding month. In addition, the EDE assesses for the presence of a DSM-IV (APA, 1994) diagnoses of bulimia nervosa and anorexia nervosa in the preceding 3 months.

The continuous EDE measure is comprised of four subscales. The first of these is the Restraint Subscale, which examines restraint over eating, avoidance of eating, particular food avoidance, dietary rules and desire to have an empty stomach. The second subscale is called Eating Concern, which looks at preoccupation with food, eating or calories, fear of losing control over eating, ability to eat in public, eating in secret and guilt about eating. The third subscale is the Shape Concern subscale, examining issues related to dissatisfaction and preoccupation with body shape, fear of weight gain, the importance of body shape to the person, feelings of fatness and discomfort with the body. The final subscale is called Weight Concern, which examines issues relating to dissatisfaction and preoccupation with weight, reaction to prescribed weighing, and desire to lose weight.

The subscales of the EDE have shown sensitivity in distinguishing between different subgroups of women, including: women with eating disorders and controls (Cooper *et al.* 1989); restraining non-patient controls and women with bulimia nervosa (Wilson & Smith, 1989; Rosen *et al.* 1990); women experiencing high levels of body dissatisfaction with no accompanying eating disorder symptomatology and women with bulimia nervosa (Rosen *et al.* 1995); obese binge eaters and normal weight women with bulimia nervosa (Marcus, *et al.* 1992).

The EDE was administered as a telephone interview. In addition to assessing the presence of eating psychopathology in the previous month, we also asked if the person had ever experienced the behaviour or attitude. We did this for both the items making up the continuous scale and the diagnostic questions. With regard to each item, one of three ratings was possible: (1) never a problem; (2) a moderate problem; or (3) a severe problem. A moderate problem signified that the person had experienced the attitude or behaviour but that it had not caused much intrusion in their life. A severe problem indicated the presence of the attitude and

Table 1. *Patterns of intra-pair correlations and the sources of variance implicated*

Pattern of correlations	Sources of variance implicated
$r_{MZ} = r_{DZ} = 0$	Non-shared environment
$r_{MZ} = r_{DZ} > 0$	Non-shared environment + shared environment
$r_{MZ} = 2r_{DZ}$	Additive genetic + non-shared environment
$r_{MZ} < 2r_{DZ}$	Additive genetic + non-shared environment + shared environment
$r_{MZ} > 2r_{DZ}$	Additive genetic + non-shared environment + dominance

behaviour as an intrusive problem. These scores were calculated for each of the four subscales as well as a total score. In all, this gave eight continuous measures to analyse: four subscale scores in the past month and four 'ever' subscale scores.

Selection of participants

A sample of 325 women was chosen for the EDE interview, which was carried out by telephone over 1994 and 1995. To be eligible for selection for interview, the women had to meet three broad criteria. First, at least one of the twin pair had to have participated in an earlier Wave 1 assessment (self-report questionnaire from 1988–9) or Wave 2 assessment (semi-structured psychiatric interview from 1992–3). Further details of these assessments and their outcomes is available (Wade *et al.* 1996). Secondly, only women from female–female pairs (both MZ and DZ) were approached. Thirdly, in order to maximize the chance that any eating disorder was both likely to have already occurred and that it was also more likely to be accurately recalled, only women who were aged between 30 and 45 years at Wave 1 data collection were interviewed. The selection of this age range was guided by the consideration that the mean age of onset of bulimia nervosa is around 20 years (Keck *et al.* 1990; Kendler *et al.* 1991), with a linear increase in the number of women experiencing their first symptom between the ages of 14 and 25 (Bushnell *et al.* 1990) and few new cases of bulimia nervosa occurring after age 25 (Woodside & Garfinkel, 1992).

Two samples of twins were chosen for the EDE telephone interview. First, a random sample of twin pairs was chosen ($N = 225$). The second sample contained all twin pairs where one or both met criteria for a possible lifetime diagnosis of bulimia nervosa at Wave 1 or Wave 2 i.e. an ascertained sample ($N = 100$). Those who had already been selected for the random sample ($N = 7$ pairs) were deleted from this second sample. Given that bulimia nervosa is a low prevalence disorder, the ascertained group was selected in order to increase the number of women likely to have had a lifetime diagnosis of bulimia nervosa. The random sample was chosen in order to increase the power of the analyses and to ensure that the sample represented the full continuum of eating concerns that exist in the female population.

Of the 206 twin pairs approached for interview, 151 complete twin pairs were interviewed (106 MZ and 45 DZ pairs) and 23 incomplete pairs were interviewed where only one twin was interviewed as the other twin refused interview or could not be traced (13 MZ and 10 DZ twins). Therefore, of the twin pairs that were approached for interview, at least one twin was interviewed in 84·5% of the twin pairs approached. There was no difference between women who agreed or refused to participate in the interview, in terms of their eating problems or weight ranges (Wade *et al.* 1997*b*).

Biometrical model fitting

Structural equation models are applied to twin data in order to decompose the overall phenotypic (or observed) variance of a behaviour or trait into three types of general influence (Roy *et al.* 1995): (1) additive genetic factors that reflect the additive effect of alleles at the genetic loci influencing the trait (A); (2) non-shared environmental factors, which represent aspects of the environment that only one of each twin pair experiences (E); and (3) shared environmental factors common to both twins of a pair (C). In addition, dominant genetic effects can also contribute to the variance (D) (Martin *et al.* 1978). The non-shared environment can not be distinguished from error of measurement in univariate analyses; this requires the use of a multivariate analysis.

Since MZ, or identical, twin pairs share all their genes and DZ, or non-identical, twins on average share only half their genes, different patterns of intra-pair correlations between zygosities can be used to indicate the presence of different influences (Martin *et al.* 1988), as

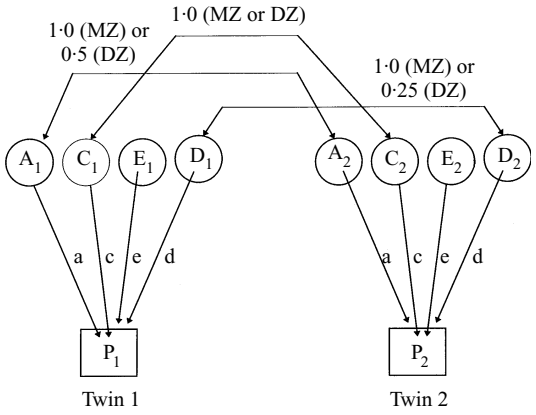

FIG. 1. Univariate model for data from monozygotic (MZ) or dizygotic (DZ) twins reared together – Twin 1 (T1) and Twin 2 (T2). A_1, C_1, E_1, D_1 refers to the sources of postulated variance for T1 and A_2, C_2, E_2, D_2 refers to the sources of postulated variance for T2, where A refers to the additive genetic influences, C refers to the shared environmental influences, E refers to the unique environmental influences and D refers to dominant genetic influences. These latent variables influence the phenotypes P_1 and P_2 by amounts a, c, e and d (path coefficients). Single headed arrows from the latent variables to the phenotype (P_1 and P_2) represent the causal impact of genes and environment on behaviour. Partial regression coefficients (a, c, e, d) reflect the degree of relationship between the two variables – fixed in the model to be equivalent in T1 and T2 and MZ and DZ twins. Since the path coefficients (a, c, e, d) are standardized, they can be interpreted as correlation coefficients and the variance of the phenotype can be estimated by squaring these coefficients, which are then called standardized variance components.

summarized in Table 1. Biometrical model fitting using structural equation modelling (SEM) uses these correlations to estimate parameters of the genetic and environmental variance underlying a disorder. It also provides formal statistical tests which assess the significance and adequacy of models that test different combinations of postulated liabilities (Silberg *et al.* 1994).

In the case of the univariate model (presented in Fig. 1), the variable of interest must be measurable and can be either a continuous or an ordinal measure. Along the top of the Fig. 1 are the latent variables, the sources of postulated variance outlined above i.e. A, C, D and E. These latent variables are present for both Twin 1 (T1) and Twin 2 (T2) and are labelled A_1, C_1, D_1, E_1 and A_2, C_2, D_2, E_2 respectively. These latent variables are assumed to have a variance of 1 (i.e. they are standardized in the model) and the double headed arrows represent the correlation between them. These correlations are presented as fixed parameters. The correlations for the additive genetic effects and the dominant genetic effects are 1·0 for MZ twins (all genes in common) and 0·5 and 0·25 respectively for DZ twins (on average, sharing half their genes). An assumption of the twin model is that MZ or DZ twins experience common environment to the same degree and therefore $r_c = 1$ for both MZ and DZ twins. The correlation between the common environmental factors is fixed to unity for both twin groups given that both twins experience this environment equally. Non-shared environmental effects are, by definition, uncorrelated between T1 and T2, so there is no correlation between E_1 and E_2 specified in the model. In cross-sectional univariate models, E also reflects measurement error and short-term fluctuations. The tracing rules of path analysis are used to yield the expected variances and covariances among the variables. Univariate modeling is used in the current study.

Various computer programs exist which can be used for biometrical model fitting. The most commonly used combination of programmes in the analysis of twin data is PRELIS (Joreskog & Sorbom, 1993) and Mx (Neale, 1997), the latter a program specifically designed for the analysis of twin data. PRELIS is used as a tool of investigation and preparation and Mx is used for analysis of the data and model testing. The process of model testing initially fits the 'full' (ACE) model to the data as a way of evaluating the statistical properties of the data. The effect of dropping one of the parameters (i.e. testing an AE, CE or E model) is then statistically tested by computing the difference in χ^2 between the models (known as the chi-square test). It is now recognized that on its own this is an inadequate test of fit, partly because it is so dependent on N (Marsh & Smith, 1987) and many other goodness-of-fit statistics have been developed. In the case of Mx, the Akaike's Information Criterion (AIC) is used as a goodness-of-fit statistic in addition to the χ^2 statistic. The AIC, which is the χ^2 value minus twice the degrees of freedom, selects the model that best combines the features of parsimony and goodness of fit. Generally, the better the individual model, the smaller the χ^2 (and the less significant the corresponding P value) and the smaller (more negative) the AIC. The ultimate goal of model fitting is to account for the data with the smallest number of parameters possible (Silberg *et al.* 1994). The methodology for analysing the univariate data in this study requires a correction

for ascertainment term, as the twin correlations calculated from the ascertained group will be biased away from the population values (Martin & Wilson, 1982; Neale *et al.* 1989).

RESULTS

Heterogeneity of the data

We expect that twins sampled randomly from the population will have equal (or at least not significantly different) means between the MZ and DZ twins and the first and second twins of each pair. Any differences would indicate that sampling problems exist with respect to the variable under study. To ensure that no potential biases did exist, the heterogeneity of the means was investigated for each subscale, for both the random and ascertained groups. No significant differences were found between the means of: the MZ and DZ twins; or the first and second twins for any of the subscales. This indicates that there are no systematic differences between the means of the MZ and DZ twins or the first and second twin of each pair and therefore no sampling bias exists with respect to these variables.

Additionally, if sampling has been satisfactory, we would also expect that the means and variances of those twins where both completed the EDE (participant–concordant pairs), and those where only one twin participated (participant–discordant pairs), would be the same. The means and variances were only tested for those women from the random group as there were insufficient numbers of participant–discordant pairs from the ascertained sample.

Table 2. *Summary statistics from the ascertainment correction of the correlations from the univariate analysis of the* 4 *EDE subscales, including correlations with* 95% *confidence intervals of the corrected ascertained groups* (*AGC*), *random groups* (*RG*) *and pooled AGC* + *RG* (*MZ = monozygotic twins and DZ = dizygotic twins*). *Tests of heterogeneity of the twin correlations of the random group and the ascertained group are included*

Subscale	*r* (95% CI)	Overall fit function (df)
Dietary Restraint		
MZ AGC	0·08 (−0·36 to 0·47)	
DZ AGC	0·16 (−0·38 to 0·55)	
MZ RG	0·40 (0·17 to 0·56)	
DZ RG	0·05 (−0·36 to 0·42)	516·96 (327)
MZ (AGC + RG)	0·33 (0·12 to 0·49)	
DZ (AGC + RG)	0·09 (−0·26 to 0·39)	518·74 (329)
Heterogeneity test		$\chi^2 = 1{\cdot}77$ (2), $P > 0{\cdot}3$
Eating Concern		
MZ AGC	0·54 (0·31 to 0·69)	
DZ AGC	0·06 (−0·32 to 0·42)	
MZ RG	0·41 (0·13 to 0·58)	
DZ RG	0·29 (−0·14 to 0·56)	528·59 (327)
MZ (AGC + RG)	0·47 (0·31 to 0·59)	
DZ (AGC + RG)	0·17 (−0·13 to 0·42)	530·01 (329)
Heterogeneity test		$\chi^2 = 1{\cdot}42$ (2), $P > 0{\cdot}3$
Weight Concern		
MZ AGC	0·56 (0·29 to 0·72)	
DZ AGC	0·39 (0·03 to 0·61)	
MZ RG	0·56 (0·32 to 0·68)	
DZ RG	0·52 (0·22 to 0·69)	523·16 (327)
MZ (AGC + RG)	0·56 (0·42 to 0·66)	
DZ (AGC + RG)	0·46 (0·24 to 0·61)	523·60 (329)
Heterogeneity test		$\chi^2 = 0{\cdot}44$ (2), $P > 0{\cdot}8$
Shape Concern		
MZ AGC	0·62 (0·35 to 0·76)	
DZ AGC	0·40 (−0·01 to 0·63)	
MZ RG	0·62 (0·47 to 0·72)	
DZ RG	0·29 (−0·03 to 0·52)	515·02 (327)
MZ (AGC + RG)	0·62 (0·49 to 0·71)	
DZ (AGC + RG)	0·33 (0·09 to 0·51)	515·280 (329)
Heterogeneity test		$\chi^2 = 0{\cdot}26$ (2), $P > 0{\cdot}8$

Table 3. *Results of fitting genetic (additive: A and dominant: D) and environmental (shared: C and non-shared: E) models for variation to the corrected ascertained and random groups jointly*

Model	Standardized parameters A	C	E	D	Fit function (df)	Comparison of nested submodels to the full model*
Dietary Restraint						
ADE	0·0233	—	0·3060	0·6707	518·74 (329)	
AE	0·3190	—	0·6810	—	518·91 (330)	$\chi^2 = 0{\cdot}168$, df = 1, $P > 0{\cdot}5$
CE	—	0·2615	0·7385	—	520·24 (330)	$\chi^2 = 1{\cdot}330$, df = 1, $P > 0{\cdot}2$
E	—	—	1	—	527·78 (331)	$\chi^2 = 9{\cdot}039$, df = 2, $P < 0{\cdot}01$
Eating Concern						
ADE	0·2203	—	0·2485	0·5312	530·01 (329)	
AE	0·4610	—	0·5390	—	530·19 (330)	$\chi^2 = 0{\cdot}176$, df = 1, $P > 0{\cdot}5$
CE	—	0·3723	0·6277	—	533·84 (330)	$\chi^2 = 3{\cdot}650$, df = 1, $P > 0{\cdot}05$
E	—	—	1	—	553·73 (331)	$\chi^2 = 23{\cdot}72$, df = 2, $P < 0{\cdot}01$
Weight Concern						
ACE	0·2036	0·3549	0·4415	—	523·60 (329)	
AE	0·5794	—	0·4206	—	526·13 (330)	$\chi^2 = 2{\cdot}530$, df = 1, $P > 0{\cdot}1$
CE	—	0·5196	0·4804	—	524·51 (330)	$\chi^2 = 0{\cdot}911$, df = 1, $P > 0{\cdot}3$
E	—	—	1	—	572·53 (331)	$\chi^2 = 48{\cdot}93$, df = 2, $P < 0{\cdot}01$
Shape Concern						
ACE	0·5807	0·0394	0·3799	—	515·28 (329)	
AE	0·6222	—	0·3778	—	515·31 (330)	$\chi^2 = 0{\cdot}030$, df = 1, $P > 0{\cdot}8$
CE	—	0·5056	0·4944	—	522·13 (330)	$\chi^2 = 6{\cdot}824$, df = 1, $P < 0{\cdot}01$
E	—	—	1	—	564·73 (331)	$\chi^2 = 49{\cdot}45$, df = 2, $P < 0{\cdot}01$

* χ^2 (df) of the fit of the submodels are obtained by subtracting the fit function (df) of the full, best fitting model within which the submodel is nested from the fit function (df) of the submodel.

No sampling bias with respect to the means and variances of the participant–concordant and participant–discordant pairs was found for any of the four subscales (Dietary Restraint, $\chi^2 = 5{\cdot}44$ (df = 10), $P > 0{\cdot}8$; Eating Concern, $\chi^2 = 9{\cdot}68$ (df = 10), $P > 0{\cdot}3$; Weight Concern, $\chi^2 = 6{\cdot}94$ (df = 10), $P > 0{\cdot}7$; Shape Concern, $\chi^2 = 12{\cdot}48$ (df = 10), $P > 0{\cdot}2$).

Twin correlations

The twin correlations of the sample ascertained for their eating problems were corrected and a summary of these correlations is presented in Table 2 for all four subscales. In each case, the MZ ascertained (corrected), DZ ascertained (corrected), MZ random and DZ random are examined separately, in addition to the joint estimations of the correlations (i.e. when the ascertained and random groups are constrained to be the same). Tests of heterogeneity are also provided, indicating that the correlations between the twins in the ascertained corrected group and the random group are not significantly different.

It can be noted at this stage that the pattern of twin correlations for the Dietary Restraint, Eating Concern and Shape Concern subscales is $r_{MZ} > 2r_{DZ}$, therefore suggesting either the ADE or AE model, whereas the pattern of correlations for the Weight Concern subscale is $r_{MZ} < 2r_{DZ}$, indicating the presence of either an ACE or CE model. Resolution of these models will be achieved by parameterizing the correlations testing the models against each other.

Genetic and environmental risk factors for the EDE measures (last month)

The parameterization of the (corrected) correlations for MZ and DZ twins involves investigation of three potential latent sources of variation in liability to the phenotype: additive genetic affects (A); common environment (C); and individual-specific environment (E). Using the correlation ratios, submodels were tested against each other using an Mx script that included the MZ and DZ ascertained and the MZ and DZ randomly selected samples. The summary statistics for the parameterization analyses for the Dietary Restraint, Eating Concern, Weight Concern and Shape Concern subscales of the EDE are presented in Table 3.

For three of the four subscales, namely the Dietary Restraint subscale, Eating Concern subscale and Weight Concern subscale, the AE

Table 4. *Results from univariate testing of the best fitting models for the four subscales of the EDE, using the combined random and ascertained groups* (95% *confidence intervals are given for each parameter; A = additive genetic; C = shared environment; and E = non-shared environment*)

	Percentage of Variance					
	Last Month			Ever		
Subscale	A	C	E	A	C	E
Dietary Restraint (AE)	32 (12–48)		68 (52–89)	58 (45–67)		42 (33–55)
Eating Concern (AE)	46 (30–58)		54 (42–70)	50 (36–61)		50 (39–64)
Weight Concern (CE)		52 (43–64)	48 (39–60)		45 (34–57)	55 (45–67)
Shape Concern (AE)	62 (50–71)		38 (29–50)	51 (36–62)		49 (38–64)

and CE models do not differ significantly from the best-fitting full model. At this stage, we have insufficient power to choose between these two models. It can be noted that, in line with the twin correlations discussed earlier, the Weight Concern subscale appears to be different from all the other subscales, in that causes of individual variance are most likely to be due to environmental influences alone (a CE model), with 52% of the variance being due to shared environment and 48% of the variance being due to the non-shared environment. For Shape Concern, however, univariate analysis indicates that only one model does not differ significantly from the best fitting model and that is the AE model ($\chi^2 = 0{\cdot}03$, df = 1, $P > 0{\cdot}8$), with additive genetic influences accounting for 62% of the variance and unique environmental influences accounting for the majority of the variance at 38%.

Comparison of 'in the past month' and 'ever' subscale measures

The same procedures were carried out with each of the 'ever' measures. Essentially, the same results were found with these measures, with the exception that only model testing of the Weight Concern subscale offers insufficient power to choose between the two best fitting models (CE or AE). The best fitting models, that is those models with the lowest chi-square value, for the four subscales measuring the functioning over the last month and the four subscales measuring 'ever' are summarized in Table 4.

In the case of the Dietary Restraint subscale, the proportions of variance are actually quite different between the functioning over the last month and 'ever', as can be seen by the lack of overlap in the 95% confidence intervals, which may indicate a decrease of genetic influence on dieting behaviour over time. The interpretation, however, is uncertain, due to the lack of power to select a best-fitting model for functioning over the previous month. The three remaining subscales have roughly equivalent proportions of variance between the last month's functioning and 'ever', indicating the temporal stability of the liability to disordered eating and the associated attitudes.

DISCUSSION

This study has examined the four subscale measures of the EDE in order to investigate the genetic and environmental causes of individual variation in attitudes that predispose women toward disordered eating. These four subscales represent measures of Dietary Restraint, Eating Concern, Weight Concern and Shape Concern, and each was examined in terms of functioning both over the month prior to interview and 'ever'. In the case of all the subscales, a large degree of variance was contributed by environmental influences, thus indicating some potential role for environmental manipulations to change the prevalence of disordered eating.

The Dietary Restraint subscale is a behavioural index of restrained eating, that is eating aimed at reducing weight. The overall best fitting model for both measures (last month and 'ever') is the AE model, with the majority of variance accounted for by non-shared environmental influences at 52–89% and 33–55% respectively. The comparison of these two estimates suggest that the vulnerability to dietary restriction is moderately stable over time but that environmental influences become more important as women get older. This may simply

suggest that factors such as chronic health problems (e.g. type II diabetes) and natural weight gain with age may become greater issues for women as they grow older.

The Eating Concern subscale measures the concern surrounding eating and food. The overall best-fitting model for both measures is the AE model, with the majority of variance accounted for by non-shared environmental influences. In the case of the last month measure, this is estimated between 42–70%, and for the 'ever' measure 39–64%. The vulnerability to discomfort experienced by women about eating publicly appears to remain remarkably stable over time.

Turning now to the Weight Concern and Shape Concern scales, which represent the core psychopathology of bulimia nervosa. The Weight Concern subscale measures the degree of concern associated with weight, and a central concept of this subscale is the degree to which women equate their weight with their self-worth. Along with shape concern, it is the presence of a high degree of weight concern that acts as a trigger to the development of disordered eating. Taking into account the results from both the previous month and 'ever' analyses, the model most likely to account for individual variation is the CE model, with about 50% of the variance accounted for by each type of environmental influence. In contrast, the best fitting model for the Shape Concern subscale is the AE model, with the majority of variance accounted for by additive genetic influences (50–71% for the measure from the previous month and 36–62% for the 'ever' measure).

In summary, genetic analyses of three of the subscales of the EDE indicate that the model best explaining the individual variance in Dietary Restraint, Eating Concern and Shape Concern is one that incorporates additive genetic influences and non-shared environmental influences. The finding with regard to individual variation in the Weight Concern subscale measure is different. Univariate analyses of the previous month's functioning and 'ever' suggest that the individual variation is best explained (while not ruling out the AE model) by environmental influences only, both shared and non-shared. This finding may go some way to explaining the conflicting results from previous twin studies (Kendler *et al.* 1991; 1995*b*). The influence of shared environment does indeed seem to be affecting some, but not all, aspects of disordered eating. This finding clearly indicates that future studies of the genetic and environmental risk factors for bulimia nervosa should use a multifaceted measure of this disorder, and one that permits delineation of the overvalued ideas associated with bulimia nervosa.

These results do, however, beg the question of why the two measures of the overvalued ideas associated with disordered eating have different aetiological influences. While the Weight Concern and Shape Concern subscales do contain one similar item (i.e. how important is your weight/shape in influencing the way you feel about yourself as a person?), they do also give different information, with the Shape Concern subscale containing more items and focusing particularly on matters relating to appearance (e.g. in bathers, without any clothes). In contrast, the Weight Concern subscale deals only with matters relating to weight, a number on the scales and personal satisfaction with that number. In the end, it may be that weight is a much more concrete indicator of personal acceptability in that it can be assessed by a single, specific number (i.e. in kilograms), and may appeal to those women who are dichotomous and perfectionistic thinkers, traits which have been highly associated with eating disorders (Bastiano *et al.* 1995).

In summary, all four measures together, whether measuring the functioning over the last month or 'ever', suggest a role of the environment for shaping attitudes that predispose women to developing eating problems, as they account for 38% to 100% of the variance. It could be that one environmental influence these attitudes have in common is that provided by the general sociocultural *mileu* in Western society, which associates the weight and shape of women with desirable personality characteristics and indicators of self-worth (Stice, 1994). Postulated transmitters of these pressures are family, peers and the media. These sociocultural ideals are thought to interact with stable individual characteristics, such as perfectionism, asceticism and difficulties in affect regulation, to produce the overvalued ideas (Vitousek, 1996). It was beyond the scope of this study to identify particular environmental variables which influence disordered eating, but further research

should particularly seek to identify those variables that contribute to the development of the overvalued ideas, which in turn are so central to the development of bulimia nervosa.

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REFERENCES

American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* (*4th edn*). APA: Washington, DC.

Bastiani, A. M., Rao, R., Weltzin, T. & Kaye, W. H. (1995). Perfectionism in anorexia nervosa. *International Journal of Eating Disorders* **17**, 147–152.

Bushnell, J. A., Wells, E., Hornblow, A. R., Oakley-Browne, M. A. & Joyce, P. (1990). Prevalence of three bulimia syndromes in the general population. *Psychological Medicine* **20**, 671–680.

Chitkara, B., MacDonald, A. & Reveley, A. M. (1988). Twin birth and adult psychiatric disorder: an examination of the case records of the Maudsley Hospital. *British Journal of Psychiatry* **152**, 391–398.

Cooper, P. J. & Fairburn, C. G. (1993). Confusion over the core psychopathology of bulimia nervosa. *International Journal of Eating Disorders* **13**, 385–389.

Cooper, P. J., Taylor, M., Cooper, Z. & Fairburn, C. G. (1987). The development and validation of the Body Shape Questionnaire. *International Journal of Eating Disorders* **6**, 485–494.

Cooper, Z., Cooper, P. J. & Fairburn, C. G. (1989). The validity of the Eating Disorder Examination and its subscales. *British Journal of Psychiatry* **154**, 807–812.

Fairburn, C. G. & Beglin, S. J. (1990). Studies of the epidemiology of bulimia nervosa. *American Journal of Psychiatry* **147**, 402–408.

Fairburn, C. G. & Cooper, P. J. (1989). Eating disorders. In *Cognitive Behaviour Therapy for Psychiatric Problems. A Practical Guide* (ed. K. Hawton, P. M. Salkovskis, J. Kirk and D. M. Clark), pp. 277–314. Oxford University Press: Oxford.

Fairburn, C. G. & Cooper, Z. (1993). The Eating Disorder Examination. In *Binge Eating: Nature, Assessment and Treatment* (ed. C. G. Fairburn and G. T. Wilson), pp. 317–359.

Fichter, M. M. & Noegel, R. (1990). Concordance for bulimia nervosa in twins. *International Journal of Eating Disorders* **9**, 255–263.

Garner, D. M. & Garfinkel, P. E. (1979). The eating attitudes test: an index of the symptoms of anorexia nervosa. *Psychological Medicine* **9**, 273–279.

Garner, D. M., Olmsted, M. P. & Polivey, J. (1983). Details and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders* **2**, 15–34.

Holland, A. J., Sicotte, J. & Treasure, J. (1988). Anorexia nervosa: evidence for a genetic basis. *Journal of Psychosomatic Research* **32**, 561–571.

Hsu, L. K. G., Chesler, B. E. & Santhouse, M. S. W. (1990). Bulimia nervosa in eleven sets of twins: a clinical report. *International Journal of Eating Disorders* **9**, 275–282.

Joreskog, K. G. & Sorbom, D. (1992). *PRELIS 2.03*: Scientific Software International, Inc.: Chicago.

Keck, P. E., Pope, H. G., Hudson, J. I., McElroy, S. L., Yurgelen-Todd, D. & Hundert, E. M. (1990). A controlled study of phenomenology and family history in outpatients with bulimia nervosa. *Comprehensive Psychiatry* **31**, 275–283.

Kendler, K. S. (1993). Twin studies of psychiatric illness. *Archives of General Psychiatry* **50**, 905–915.

Kendler, K. S., MacLean, C., Neale, M. C., Kessler, R., Heath, A. C. & Eaves, L. (1991). The genetic epidemiology of bulimia nervosa. *American Journal of Psychiatry* **148**, 1627–1637.

Kendler, K. S., Martin, N. G., Heath, A. C. & Eaves, L. J. (1995*a*). Self-report psychiatric symptoms in twins and their non-twin relatives: are twins different? *American Journal of Medical Genetics* **60**, 588–591.

Kendler, K. S., Walters, E. E., Neale, M. C., Kessler, R. C., Heath, A. C. & Eaves, L. J. (1995*b*). The structure of the genetic and environmental risk factors for six major psychiatric disorders in women. *Archives of General Psychiatry* **52**, 374–383.

Marcus, M. D., Smith, D. E., Santelli, R. & Kaye, W. (1992). Characterisation of eating disordered behaviour in obese binge eaters. *International Journal of Eating Disorders* **12**, 249–255.

Marsh, H. W. & Smith, D. (1987). Cross-national study of the structure and level of multidimensional self-concepts: an application of confirmatory factor analysis. *Australian Journal of Psychology* **39**, 61–77.

Martin, N. G. & Wilson, S. R. (1982). Bias in the estimation of heritability from truncated samples of twins. *Behavior Genetics* **12**, 467–472.

Martin, N. G., Eaves, L. J., Kearsey, M. J. & Davies, P. (1978). The power of the classical twin study. *Heredity* **40**, 97–116.

Martin, N. G., Jardine, R., Andrews, G. & Heath, A. C. (1988). Anxiety disorders and neuroticism: are there genetic factors specific to panic? *Acta Psychiatrica Scandinavica* **77**, 698–706.

Neale, M. C. (1997). *Mx: Statistical Modeling*, 4th edn. Department of Psychiatry, Box 710 MCV, Richmond VA 23298.

Neale, M. C. & Cardon, L. R. (1992). *Methodology for Genetic Studies of Twins and Families*. Kluwer Academic Publishers: Dordrecht.

Neale, M. C., Eaves, L. J., Kendler, K. S. & Hewitt, J. K. (1989). Bias in correlations from truncated samples of relatives. *Behavior Genetics* **19**, 163–169.

Rosen, J. C., Vara, L., Wendt, S. & Leitenberg, H. (1990). Validity studies of the Eating Disorder Examination. *International Journal of Eating Disorders* **9**, 519–528.

Rosen, J. C., Reiter, J. & Orosan, P. (1995). Assessment of body image in eating disorders with the body dysmorphic disorder examination. *Behaviour Research and Therapy* **33**, 77–84.

Roy, M., Neale, M. C. & Kendler, K. S. (1995). The genetic epidemiology of self-esteem. *British Journal of Psychiatry* **166**, 813–820.

Rutherford, J., McGuffin, P., Katz, R. J. & Murray, R. M. (1993). Genetic influences on eating attitudes in a normal female twin population. *Psychological Medicine* **23**, 425–436.

Silberg, J. L., Erickson, M. T., Meyer, J. M., Eaves, L. J., Rutter, M. L. & Hewitt, J. K. (1994). The application of structural equation modeling to maternal ratings of twins' behavioural and emotional problems. *Journal of Consulting and Clinical Psychology* **62**, 510–521.

Stice, E. (1994). Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanism of action. *Clinical Psychology Review* **14**, 633–661.

Treasure, J. & Holland, A. (1991). Genes and aetiology of eating disorders. In *The New Genetics of Mental Illness* (ed. P. McGuffin and R. Murray), pp. 198–211. Butterworth-Heinemann: Oxford.

Vitousek, K. (1996). The current status of cognitive-behavioural models of anorexia nervosa and bulimia nervosa. In *Frontiers of*

Cognitive Therapy (ed. P. M. Salkovskis), pp. 383–418. Guilford Press: New York.

Wade, T., Heath, A. C., Abraham, S., Treloar, S. A., Martin, N. G. & Tiggemann, M. (1996). Assessing the prevalence of eating disorders in an Australian twin population. *Australian and New Zealand Journal of Psychiatry* **30**, 845–851.

Wade, T., Tiggemann, M., Martin, N. G. & Heath, A. C. (1997*a*). A comparison of the Eating Disorder Examination and a general psychiatric schedule. *Australian and New Zealand Journal of Psychiatry* **31**, 852–857.

Wade, T., Tiggemann, M., Martin, N. G. & Heath, A. C. (1997*b*). Characteristics of interview refusers: women who decline to participate in interviews relating to eating. *International Journal of Eating Disorders* **22**, 95–99.

Wilson, G. T. (1993). Assessment of binge eating. In *Binge Eating: Nature, Assessment and Treatment* (ed. C. G. Fairburn and G. T. Wilson), pp. 227–249. Guilford Press: New York.

Wilson, G. T. & Smith, D. E. (1989). Assessment of bulimia nervosa: an evaluation of the Eating Disorder Examination. *International Journal of Eating Disorders* **8**, 173–179.

Woodside, D. B. & Garfinkel, P. E. (1992). Age of onset in eating disorders. *International Journal of Eating Disorders* **12**, 31–36.