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# The structure of genetic and environmental risk factors for three measures of disordered eating

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## ABSTRACT

**Background.** The study explored the genetic and environmental risk factors for both the behaviours and attitudes characteristic of disordered eating.

**Methods.** In three waves of data collection, information was collected from female twins regarding their eating and attitudes towards eating, weight and shape. The first assessment consisted of a self-report questionnaire (1988–9) with 1682 women. The second assessment consisted of a semi-structured psychiatric interview schedule (1992–3), completed by 1852 women, many of whom had completed Wave 1 assessment. The third assessment, with 325 women chosen from Waves 1 and 2 (1995–6), consisted of a semi-structured interview (the Eating Disorder Examination).

**Results.** As only one twin pair was concordant for lifetime bulimia nervosa at Wave 3 assessment, ordinal measures of all assessments were used in a multivariate genetic analysis. Results indicated that additive genetic and non-shared environmental influences best explained variance in liability to disordered eating, with about 60% (95% CI 50–68) of the variance explained by genetic factors. Comparison with a model allowing for the effects of shared environment indicated genetic factors accounted for a similar degree of variance (59%, 95% CI 36–68).

**Conclusion.** Liability to the development of the behaviours and attitudes characteristic of eating disorders is best explained by both environmental and genetic factors, with covariation between the three measures best explained by a single latent phenotype of disordered eating which has a heritability of 60%.

## INTRODUCTION

Bulimia nervosa has been described as a heterogeneous disorder (Fairburn, 1991) that differs widely between patients with regard to background, affect, coping skills and personality variables (Vitousek & Manke, 1994). Given this diversity, it might be expected that a range of genetic and environmental risk factors fashion liability towards the development of bulimia nervosa. One of the most efficacious ways of investigating such risk factors is through the use of twins and a biometrical genetic model-fitting approach (Neale & Cardon, 1992; Kendler, 1993), which decomposes the variance of a behaviour or trait into four types of general influence: (1) additive genetic factors (A); (2) non-shared environmental factors (E); (3) shared environmental factors (C); and (4) dominant genetic effects (D).

To our knowledge, there are eight published studies that specifically examine the genetic epidemiology of bulimia nervosa in twin populations. The first three (Fichter & Noegel, 1990; Hsu *et al.* 1990; Treasure & Holland, 1991) examine small numbers of twins referred to

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treatment clinics or who responded to advertisements. Results from these studies indicate either higher concordance rates of bulimia nervosa in monozygotic (MZ) than dizygotic (DZ) twins or similar concordance rates, suggesting the possibility of some genetic involvement in the development of bulimia nervosa. However, none of these studies formally tests this hypothesis against the alternative of shared environment as the cause of familial aggregation and these clinically ascertained studies are subject to unknown ascertainment biases.

Four studies use Caucasian twins from the Virginia Twin Registry (Kendler *et al.* 1991, 1995; Bulik *et al.* 1998; Sullivan *et al.* 1998), and take advantage of more powerful analyses, utilizing a population-based twin sample ($N$ = 2163 female twins) and the more informative and sophisticated methodological approaches outlined by Neale & Cardon (1992). From the first study the best-fitting model indicated that about 50% of the variance in liability was due to A and 50% due to E. The second, more powerful study found the best-fitting model to also include C (41% of the variance), as well as A (30%) and E (29%). The third study, an examination of bingeing and vomiting, suggests these are complex traits resulting from an interplay of multiple genes and individual specific environment (E), with heritabilities ranging from 46–72%. The last study, using a bivariate measurement model (respondents were assessed on two occasions, 5 years apart) that eliminates measurement error, found that bulimia nervosa had a heritability of 83% (95% CI 0·64–1·00).

Finally, a recent study (Wade *et al.* 1998) using a small sample of women ($N$ = 325) from an Australian population-based twin registry, examined the subscale scores of the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993). The Weight and Shape Concern subscales measure the attitudinal components of bulimia nervosa, as defined by DSM-IV: 'self-evaluation is unduly influenced by weight and shape'. While the variance in the Shape Concern subscale was best explained by genetic influence (62%) and non-shared environment (38%), the Weight Concern subscale was best explained by environmental variance, both C (52%) and E (48%). However, the number of subjects in this study meant that there was insufficient power to definitively choose between models in the model-fitting process. In summary, it appears that the influence of both genetic and environmental factors on the development of bulimia nervosa may be important.

The majority of studies of disordered eating using twins and biometrical modelling techniques rely on questions embedded within psychiatric interview schedules that use a binary definition of an eating disorder (i.e. has an eating disorder or not). Evidence would suggest that such measures of bulimia nervosa are approximate, identifying disordered eating but not bulimia nervosa in particular (Wade *et al.* 1997), and are less likely to identify binge eating correctly and the core psychopathology than interviews tailored specifically for the identification of bulimia nervosa. In addition, liability to disordered eating is assumed to be continuous and normally distributed in the general female population, as specified by the liability threshold model (Falconer, 1960; Kendler *et al.* 1991) and the use of a binary measure reduces the power of analyses to identify accurately the genetic and environmental risk factors to bulimia nervosa (Hewitt, 1997).

The current study provides an alternative approach to the majority of studies by using three different ordinal measures of disordered eating and the attitudes associated with bulimia nervosa in a multivariate analysis, with a large sample of female twins from a volunteer twin registry. These measures were assessed over three waves of data collection: the third measure represents a detailed assessment of disordered eating with a small sample, while the measures from the first two waves used more approximate measures of disordered eating but with large samples.

## METHOD

Three waves of data were collected that assessed the lifetime presence of disordered eating and attitudes. The relationship between these three waves of data is summarized in Fig. 1. Participants were twins who had volunteered to join the Australian National Health And Medical Research Council Twin Register (ATR) and who had signed a consent to be approached for scientific studies. Each twin received an introductory letter that fully explained the pro-

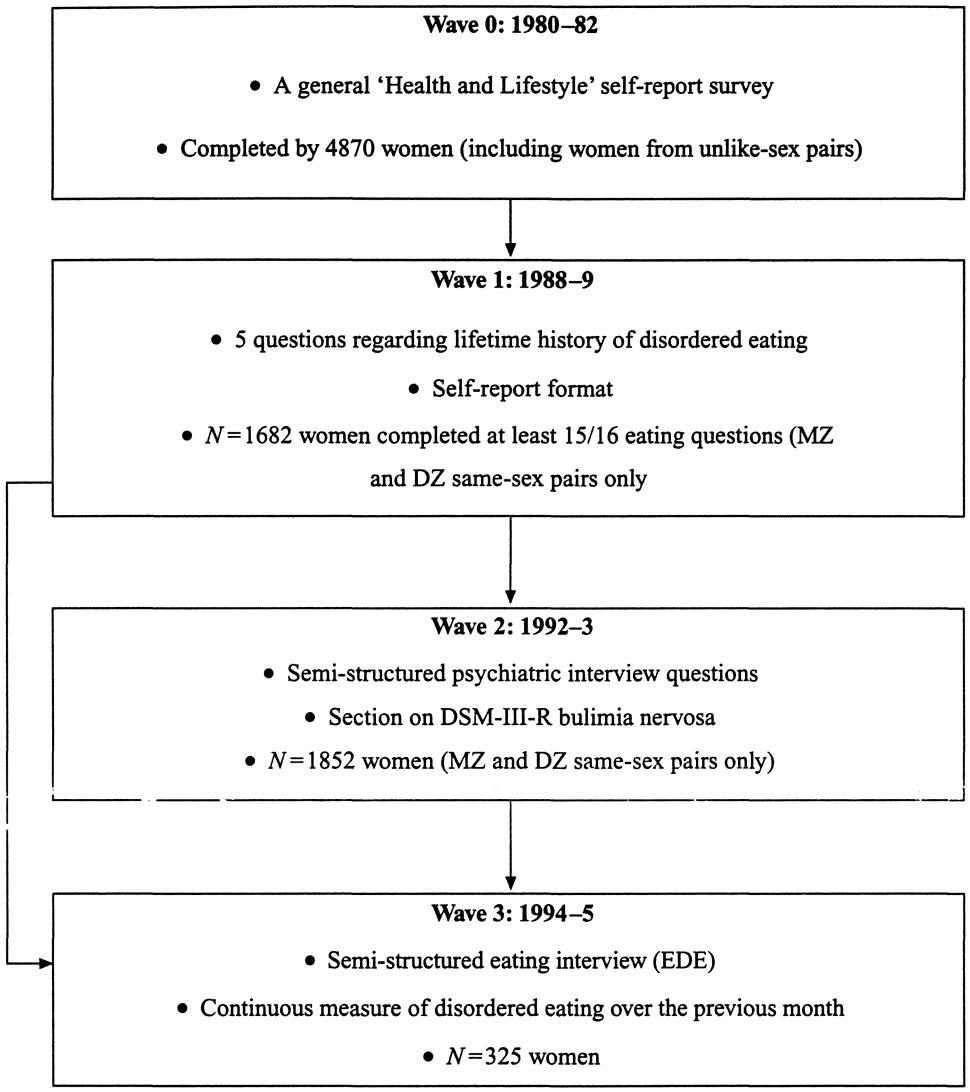


FIG. 1. Flowchart representing the relationship between the three waves of data collection on eating disorders with women aged 30–45 years at Wave 1.

cedures of the study and at a follow-up phone call, gave verbal consent for this specific study.

### Wave 1 sample and assessment

In 1988–9 a self-report questionnaire concerning general health was sent out to twin pairs who had completed a general 'Health and Lifestyle' self-report questionnaire 8 years earlier (Health *et al.* 1994). In order to maximize the chance that any eating disorder was likely to have already occurred and that it was also more likely to be accurately recalled, only data from women who were aged between 30 and 45 years was subsequently analysed. Of the 4116 women who returned questionnaires, 1976 were aged between 30 and 45, the mean age (on 1 January 1989) being 36·61 years (S.D. = 4·72). The self-report questionnaire contained five questions relating to eating problems (Wade *et al.* 1996), summarized in Table 1. Sixteen responses were derived from the questions, each item relating to whether the woman had *ever* (i.e. now or previously) experienced the problem. As answers to the questions did not permit the formulation of an eating disorder diagnosis, an index of disordered eating was developed for later use in the multivariate analysis: data for those pairs who could be scored for at least 15 out of the 16 items were summarized. The 'yes' responses for the items were added and then divided by the number of items, giving a score between 0 and 1, were 0 equals no eating problems. This was

Table 1. *Questions used in the Wave 1 and 2 assessment of disordered eating*

Wave 1: 5 questions, 16 items with a yes/no response
1 Do you feel you have difficulty controlling your weight?
2 Do you feel you have problems with disordered eating?
3 Have you used any of the following methods to control your body weight: (1) starvation; (2) self-induced vomiting; (3) excessive exercise; (4) laxatives; (5) fluid tablets; (6) slimming tablets?
4 Do you feel you have been preoccupied with thoughts of food or body weight?
5 Have you ever suffered from or been treated for: eating disorder; low body weight; binge eating; obesity; weight loss; anorexia nervosa; bulimia?

Wave 2: 5 questions
1 Were you ever greatly concerned about eating too much, looking too fat, or gaining too much weight?
2 Has there ever been a time in your life when you went on eating binges – eating a large amount of food in a short period of time (usually less than 2 hours)?
3 Did you go on eating binges as often as twice a week for at least 3 months?
4 During these binges were you afraid you could not stop eating, or that your eating was out of control?
5 Did you do anything to prevent weight gain from binge eating such as: making yourself vomit; taking laxatives or diuretics; dieting strictly; fasting; exercising vigorously; anything else?

Wave 2: Scoring key – questions [illegible] answered affirmatively
1 Bulimia nervosa: questions 1 to 5
2 Probable bulimia nervosa: questions 2, 3 and 5 and either question 1 or question 4
3 Binge-eating: questions 1 and 2 and perhaps 3 and 4
4 Weight preoccupation: question 1
5 No problems: none

computed for 1682 women, with a mean age (on 1 January 1989) of 36·54 years (S.D. = 4·65). The internal reliability of this measure (Cronbach's alpha) was 0·75.

## Wave 2 sample and assessment

The second wave of data collection consisted of telephone interviews using a general psychiatric interview schedule. Of the 4870 women who completed the original 1980–81 mailed survey, 4116 returned the self-report questionnaire in 1988–9 and 3845 completed the follow-up telephone interview in 1992–3 (79·0% of the original 4870). The completion rate for the telephone interview among the respondents to the 1988/9 survey was 93·4%. The psychiatric interview schedule was the Semi-Structured Assessment for the Genetics of Alcoholism, as modified for use in Australia, SSAGA-OZ (Bucholz *et al.* 1994; Health *et al.* 1997), which primarily comprises items previously validated by other research interviews, such as the Composite International Diagnostic Interview (CIDI) (WHO, 1993). The Wave 2 interview was administered, on average, 3·8 years (S.D. = 0·6) after the Wave 1 questionnaire was completed. The section relating to eating behaviour (Table 1) asked questions relating to DSM-III-R criteria (APA, 1994) for bulimia nervosa. For purposes of data analysis, responses were divided into five categories: bulimia nervosa, probable bulimia nervosa, binge eating, concern with eating or weight, no problems (Wade *et al.* 1996). The interviewer rater was blind to the diagnosis of the co-twin.

The total number of women from same-sex pairs women who completed satisfactory interviews with regard to eating problems was 3845, and of these, 1852 were aged between 30 and 45 years at Wave 1, with mean age (on 1 January 1993) being 40·61 years (S.D. = 4·72). The interviews were administered by trained lay interviewers, all of whom were female. The scoring of the interview was subsequently carried out by the senior author, a clinical psychologist with a number of years postgraduate experience running an eating disorders clinic.

## Wave 3 sample and assessment

A sample of 325 women was chosen to be interviewed with the Eating Disorder Examination (EDE) (Fairburn & Cooper, 1993) over the telephone, carried out during 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 either Wave 1 or Wave 2. Secondly, only women from female–female pairs (MZ and DZ) were approached. Thirdly, only women aged between 30 and 45 years at Wave 1 data collection were interviewed (i.e. aged 36 to 51 years at Wave 3).

Two samples of twins were chosen for the EDE telephone interview. First, a random sample of twin pairs was chosen. Secondly, all twin pairs within the age range where one or both met criteria for a possible lifetime diagnosis of bulimia nervosa on Wave 1 or Wave 2 data were selected. This included women who: (1) at Wave 1 had admitted to suffering from bulimia or binge eating and who had also admitted to having problems with disordered eating and had been preoccupied with body weight or food; or (2) at Wave 2 had been assessed as having

bulimia nervosa or probable bulimia nervosa. Those who had already been selected for the random sample were deleted from this sample ($N = 7$ pairs). We refer to the latter sample as the ascertained sample because these pairs have been selected for interview because at least one of the pair is thought to be affected by the disorder of interest. In all, 225 women from the random sample and 100 women from the ascertained sample agreed to be interviewed, a total of 200 MZ twins and 125 DZ twins, including 94 complete MZ pairs and 57 complete DZ pairs.

The EDE (12th edition) is a semi-structured, investigator-based interview which provides a continuous measure of the core psychopathology associated with eating disorders (Fairburn & Cooper, 1993). Each item of the EDE is measured on a seven-point scale of severity and the final total score on the EDE, which is the average of all items, ranges from 0 to 6, with 0 indicating no problems and a 6 indicating frequent and severe problems. Items are rated for the preceding month and it is the total EDE score that is used in the multivariate analyses. In addition, the EDE assesses the presence of a DSM-IV diagnosis of bulimia nervosa and anorexia nervosa in the preceding 3 months. For the purposes of this study, the questions pertaining to diagnostic criteria were also asked as lifetime questions and women were eliminated from the study if they had developed an eating disorder between Waves 1 and 3.

Both samples (i.e. the random and ascertained) were combined; this method is described in detail elsewhere (Wade *et al.* 1999). One of each twin pair was randomly chosen to be interviewed first. The order by which these twins were to be interviewed was determined by using a computer-generated random listing of names. Once all the twins had been interviewed, the co-twins were also randomly chosen for interviewing. Therefore, the interviewer was blind to the results of the assessments from Wave 1 and Wave 2 data and any halo effects from talking to the co-twin were minimized.

### Preparation of the data

As the Wave 3 data only drew from female–female pairs (MZ and DZ) who were aged between 30 and 45 at Wave 1, only data for such pairs were selected from Waves 1 and 2. Data was analysed using PRELIS 2·03 (Joreskog & Sorbom, 1993) and Mx (Neale, 1997). We analysed the raw data directly by evaluating the likelihood of each pedigree under the model and summing the logarithms of the individual terms according to Lange *et al.* (1976). The model was written in terms of the expected means and the expected covariance matrix for all individuals in the pedigree. Each pedigree can be a different structure, so the algorithm as interpreted in Mx can cope with missing values. Provided values at later waves are missing either completely at random, or as a function of values at earlier waves, then an unbiased Maximum Likelihood Estimate of the full expected covariance matrix of values at Waves 1, 2 and 3 for both twins (i.e. $6 \times 6$ matrix) may be obtained (Little & Rubin, 1987).

As all three waves of data were positively skewed, the normal weights of the raw scores were used (i.e. using the liability threshold model). Although the type of assessment of disordered eating differs between Waves 1, 2 and 3, the underlying assumption of the following analyses is that all three waves are assessments of the same latent liability. Given this position, one would expect moderate correlations between the three different measures of disordered eating. The pairwise correlations between the three waves of data are all moderate at around 0·5, with the highest correlations between Waves 1 and 2 ($r = 0{\cdot}54$).

### Multivariate analyses

A fundamental problem is the huge range of potential models which can be fitted. Therefore, some systematic approach to reduce the hazards of multiple hypothesis testing is required. The first step is to decide on the sources of variation and covariation (i.e. A, C or E) and having done this, to then explain the structure of covariation within each source. We therefore begin by testing a range of Cholesky decomposition models (Neale & Cardon, 1992), which have the same combination of sources of variance as for univariate model fitting. Once these sources of variation and covariation are identified, the structure of covariation within each source is investigated. This is achieved by examining the fit of an independent pathways (IP) model, in which each of the latent factors has its own path to each observed variable, and comparing it to

the fit of a common pathways (CP) model, a more stringent model than the IP model as it hypothesizes that covariation between the three measures is determined by a single phenotypic latent variable, which is itself determined by additive genetic and non-shared environmental sources of variance. Given that the IP and the Cholesky AE model both have 12 parameters identified, the fit statistics are the same. The CP model, however, has 11 parameters identified and the standardisation of the latent factor (i.e. making the variance equal to 1) adds another parameter, hence increasing the degrees of freedom by 2.

## RESULTS

### Estimation of lifetime bulimia nervosa

Of those selected for interview, a total of 23 out of 325 women (7·1 %) were diagnosed as having had a past diagnosis of bulimia nervosa using the EDE. Only one pair of twins was concordant for bulimia nervosa, and this was a MZ pair. This low concordance rate meant that the diagnostic category was too rare to be used for any genetic analyses, as had been used previously (Kendler *et al.* 1991).

### Twin correlations

Using maximum likelihood estimation, the correlations were calculated between the three waves of data and between Twin 1 and Twin 2, and are summarized in Table 2. The pattern of twin correlations, with the MZ correlations being more than double those of the DZ correlations (with the exception of Wave 1), suggests the presence of genetic factors influencing the development of disordered eating.

### Model fitting

Comparison of the Cholesky models is shown in Table 3. The ADE and ACE models are almost indistinguishable in their fit. Dropping C from the ACE model causes a change in fit of $\chi^2 = 1{\cdot}57$, indicating that an AE model is equally adequate, whereas dropping A changes fit by $\chi^2$

Table 2. *Maximum likelihood estimates of correlations between disordered eating measures in twins on three occasions (W1, W2, W3). MZ pairs are above the diagonal and DZ pairs are below the diagonal. Correlations between twin pairs are in bold*

| | Twin 1 | | | Twin 2 | | |
|---|---|---|---|---|---|---|
| | Wave 1 | Wave 2 | Wave 3 | Wave 1 | Wave 2 | Wave 3 |
| Twin 1 | | | | | | |
| Wave 1 | — | 0·61 | 0·41 | **0·43** | 0·31 | 0·34 |
| Wave 2 | 0·57 | — | 0·40 | 0·30 | **0·29** | 0·18 |
| Wave 3 | 0·49 | 0·40 | — | 0·32 | 0·31 | **0·55** |
| Twin 2 | | | | | | |
| Wave 1 | **0·27** | 0·18 | 0·16 | — | 0·44 | 0·38 |
| Wave 2 | 0·08 | **0·10** | 0·01 | 0·56 | — | 0·31 |
| Wave 3 | 0·14 | 0·06 | **0·22** | 0·40 | 0·30 | — |

Table 3. *Goodness-of-fit of multivariate models to the normal-transformed raw observations of the age restricted data*

| | Goodness-of-fit | | |
|---|---|---|---|
| Model | −2 × log likelihood (df) | $\chi^2$ (df)* | *P* |
| 1 Cholesky ACE | 9362·288 (3838) | — | — |
| 2 Cholesky AE | 9363·876 (3844) | 1·566 (6) | > 0·9 |
| 3 Cholesky CE | 9388·566 (3844) | 26·278 (6) | < 0·01 |
| 4 Cholesky E | 9539·879 (3850) | 177·591 (12) | < 0·01 |
| 5 Independent pathway AE | 9363·876 (3844) | — | — |
| 6 Common pathway AE | 9367·282 (3846) | 3·406 (2)† | > 0·1 |

* The likelihood ratio, $\chi^2$, is obtained by subtracting the fit function (df) of the ACE model.
† The measure of fit is calculated from subtracting the CP model from the IP model.

Table 4. *Genetic and unique environmental correlations for the three waves of data. The genetic correlations are in the lower half of the matrices and the environmental correlations are in the top half of the matrices*

| | Wave 1 | Wave 2 | Wave 3 |
|---|---|---|---|
| | Environmental correlations | | |
| Wave 1 | **1·00** | 0·37 | 0·18 |
| Wave 2 | 0·86 | **1·00** | 0·22 |
| Wave 3 | 0·66 | 0·59 | **1·00** |
| | Genetic correlations | | |

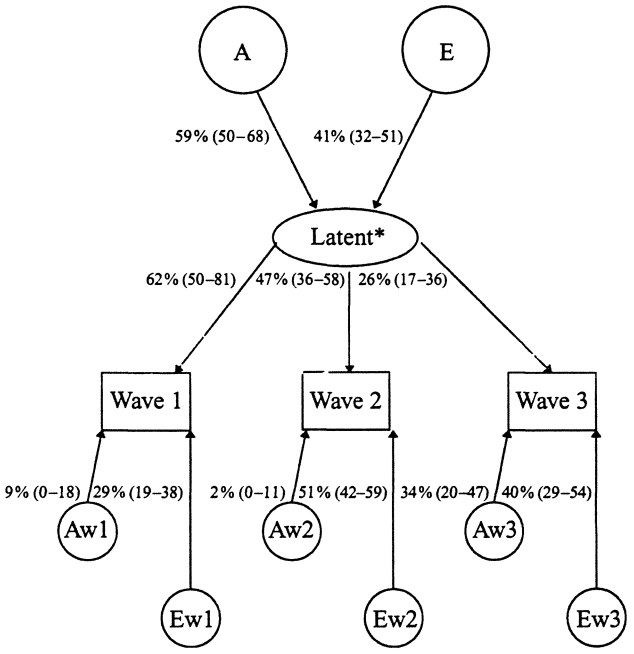


FIG. 2. The best-fitting model, the AE common pathway model for one twin. The percentages of variance contributed by each source to the observed variables (Wave 1 questionnaire, Wave 2 interview and Wave 3 EDE interview) are shown (95% confidence intervals are given (in parentheses) adjacent to each estimate). *Liability to disordered eating.

= 26·28, indicating that the CE model gives a poor description of the data. Thus, the best fitting model was the AE model, i.e. the best explanation of the covariation within and between twin pairs across the three waves of data are due to additive genetic and non-shared environmental influences.

## Genetic and environmental correlations

The genetic and non-shared environmental correlations between the three waves of data are shown in Table 4. There are high correlations between the genetic factors influencing the three waves of data, ranging from 0·59 to 0·86, the highest correlation being between the Wave 1 and 2 data. There are moderate correlations between the unique environmental factors influencing the development of the three measures of disordered eating over time, ranging from 0·18 to 0·37. Over the 7 year span between Waves 1 and 3, there is still a considerable amount (66%) of shared genetic risk factors between these two waves. The environmental correlation between the two measures is lower, at 18%.

## Common pathway model

With the best-fitting sources of covariation identified as A and E, the best-fitting structure of the model was then examined, first with an IP model, and then a CP model. The fit statistics of these models are shown in the lower part of Table 3 above. Comparison of these two models indicates that the CP structure is actually the most parsimonious description of the model structure, as it is not significantly different from the IP model.

The pathways from this model are summarized in Fig. 2. The latent variable has a broad heritability of 59% and the non-shared environmental influences account for 41% of the variance. This latent factor has an important phenotypic influence on Wave 1 data (the self-report questions about disordered eating) in particular (62% of its variance), and also on the Wave 2 data (the SSAGA interview). This factor has a reduced influence on the Wave 3 data (the EDE score), only contributing 26% of its variance.

For the Wave 1 measure, 38% of the variance is occasion or instrument specific. Of this, 9% is additive genetic and 29% is non-shared environment (which will include measurement error). For the Wave 2 data, 53% of the variance is accounted for by specific influences with most accounted for by the non-shared environment at 51%. The Wave 3 data has 74% of its variance accounted for by specific influences; 34% of these specific influences are additive genetic and 40% are non-shared environment. These results indicate once again that while the presence of a mediating latent factor best fits the three waves of data, the EDE data are the least similar to the other two waves in that individual variance in the EDE data are

due mainly to genetic and environmental influences that are not shared by the other two waves of data. This indicates that the EDE is measuring a construct that is somewhat different from the first two measures of disordered eating.

## DISCUSSION

This study uses a multivariate analysis, incorporating three measures of disordered eating that were collected over a 7-year period with three waves of data collection, to investigate the epidemiological risk factors for eating disorders. The multivariate analysis, incorporating near complete data for the earlier two waves, estimates a correlation for the much more limited Wave 3 data, as if it were measured on the entire population rather than just a small random group and even smaller ascertained group, ascertained on the basis of Wave 1 and Wave 2 scores. It is of interest to note that the twin correlations for the EDE calculated using the multivariate analysis are similar to those calculated using a univariate analysis which corrects for ascertainment (a joint correlation estimate, using the Wave 3 data where an ascertainment correction is employed, gives a MZ correlation of 0·62 (95% CI = 0·41–0·70) and a DZ correlation of 0·30 (95% CI = 0·01–0·43), indicating that the multivariate analysis does indeed calculate unbiased estimates for the ascertained group.

Overall, the results of the multivariate analysis suggest that the most likely influences determining individual variation in disordered eating are additive genetic (A) and non-shared environment (E), at 60% and 40% of the variance respectively. It is worthy of note that non-shared environmental influences can include some aspects of the family environment (Silberg *et al.* 1994), as recent research makes it clear that children raised in the same family may experience surprisingly different environments (Dunn & Plomin, 1990; Scarr, 1992). Parents tend to stress the similarity with which they treat their children whereas children tend to stress the differences in parental treatment they receive (Kendler, 1996), perhaps partly in response to the different demands that their different genes make upon the apparently 'common' environment (Jinks & Fulker, 1970).

The phenotypic and maximum likelihood estimates of the correlations across the three waves of data are moderate, with Wave 1 and Wave 2 being most highly correlated. Each wave measures a different aspect of disordered eating: Wave 1 is a brief and general measure, Wave 2 is a brief measure specific to bulimia nervosa, Wave 3 is a more detailed assessment of the range of behaviours and attitudes that place a person at risk of developing an eating disorder. Follow-up analyses to investigate the best model to describe the covariation across the three waves of data indicated that the common pathway model was the most parsimonious. This model suggests that covariation between the three measures of disordered eating is determined by a single latent variable, which has a broad heritability of 59%. This latent variable can be interpreted to represent a latent liability to disordered eating.

Over the 7-year span between the collection of Waves 1 and 3, there was still a considerable amount of genetic risk factors shared between these two waves, with Wave 1 sharing 56% of its genetic risk factors with Wave 3, and 18% of its non-shared environmental influences. This may indicate that, despite the dissimilarity between the nature of data collection at Waves 1 and 3, there is considerable stability of vulnerability to disordered eating over time. This finding is consistent with clinical outcome studies, a review of which concludes that 'although many of these treatments result in dramatic reductions in the frequency of target eating behaviours, the majority of subjects still are symptomatic at the end of treatment' (Mitchell *et al.* 1993).

These results should be interpreted in the context of two limitations. The first is the high degree of error measurement associated with single assessment of eating disorders. However, though the three measures were different, it is likely that the estimates of the latent liability are accurate, as it relies on a multi-measure, longitudinal design. The second limitation pertains to the interpretation of the latent factor. We have chosen to interpret it as a latent liability to disordered eating, which can include bulimia nervosa, anorexia nervosa or eating disorder not otherwise specified. However, there is no definitive explanation offered by the methodology for this latent liability.

In summary, the multivariate analysis suggests

that that the underlying influences on individual variation in disordered eating are additive genetic and individual-specific environmental. Results indicate that there is a common phenotypic latent variable mediating the three measures of disordered eating, but that this affects the continuous measure of eating less than the two brief and approximate measures of eating. This suggests that different measures of disordered eating, and specific components of disordered eating, are associated with distinct and varying sources of liability. In other words, identification of genetic influence is not the problem – identification of the phenotype and the specific features of eating disorders which are being affected is the more challenging problem for future research.

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