# Genetic and environmental risk factors shared between disordered eating, psychological and family variables

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## Abstract

The aim of this paper is to examine the sources and structure of covariation between disordered eating, neuroticism, parental care and protection, self-esteem and emotional reliance on other people. The eating, personality and family functioning measures, obtained from 537 MZ and 344 DZ female twin pairs aged 30–45 years, were examined using multivariate biometrical genetic modeling techniques. The best-fitting independent pathway model suggested that the measure of disordered eating shared unique environmental risk factors with neuroticism and perceptions of parental care. Neuroticism, self-esteem and emotional reliance on others shared genetic risk factors. The specific sources of individual variation for the six variables included a mixture of: (a) genetic, common and unique environment (disordered eating and the parental perception variables), (b) genetic and unique environment (neuroticism) and (c) unique environment only (self-esteem and emotional reliance on others). Disordered eating did not share genetic risk factors with any of the measured variables. The implications of these findings for our understanding of disordered eating are discussed. © 2000 Elsevier Science Ltd. All rights reserved.

*Keywords:* Eating disorders; Behaviour genetics; Risk factors; Twins

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## 1. Introduction

It is widely agreed that the development of eating disorders is influenced by many different variables (Vitousek, 1996). However, identification of these risk factors has proved more problematic. Commonly, it has been postulated that in western cultures there exist general and pervasive influences on women's eating and attitudes, such as sociocultural influences and dietary restraint (Brownell & Rodin, 1994; Stice, 1994; Polivy & Herman, 1985). While these factors are thought to be common in the development of any eating disorder, the heterogeneity of disordered eating (and bulimia nervosa in particular) suggest that a variety of other influences may be instrumental in the development of specific eating disorders, influences which are not necessarily common to all eating disorders. Speculation and research about these latter influences most commonly focuses on the nature and quality of family relationships and the personality that the individual brings to their particular situation. It is these variables which are of interest in this current study.

With respect to disordered eating and family functioning, it has been well documented that women with bulimia nervosa perceive their parents as being less caring than control women (Kendler, MacLean, Neale, Kessler, Heath & Eaves, 1991; McNamara & Loveman, 1990; Woodside, Shekter-Wolson, Garfinkel, Olmsted, Kaplan & Maddocks, 1995). Similarly, with regard to protectiveness, women with eating disorders have been found to judge their families as less encouraging of independence than other women with psychiatric conditions and control women (Williams, Chamove & Millar, 1990; Johnson & Flach, 1985). However, the precise role of family functioning in the development of bulimia nervosa has been hotly debated in the literature. Perhaps the most commonly held viewpoint is that family functioning has no direct causal relationship with disordered eating, but rather that it moderates attitudes toward the self and the extent to which people regulate their emotionality (Strober & Humphrey, 1987). In other words, it exerts a more direct effect on personality, which in turn influences the development of disordered eating.

With respect to personality, women with bulimia nervosa have been found to be more neurotic than control women (Feldman & Eysenck, 1986; Kendler et al., 1991; Wade, Tiggemann, Heath, Abraham & Martin, 1995). Further research suggests that neuroticism is an enduring characteristic of temperament, which shares 70% of its genetic risk factors with major depression (Kendler, Neale, Kessler, Heath & Eaves, 1993). In turn, we know that depression shares around 46% of its genetic liability with bulimia nervosa (Walters, Neale, Eaves, Heath, Kessler & Kendler, 1992).

Low self-esteem is also seen as a crucial personality factor influencing the subsequent development of an eating disorder. It has been found to be one of the most consistent indicators of poor treatment outcome for bulimia nervosa (Fairburn, Peveler, Jones, Hope & Doll, 1993) and has also been indicated as a risk factor for the development of bulimia nervosa (Kendler et al., 1991). Self-esteem is moderately heritable (52% of the variance), with the remaining influences consisting of the nonshared environment (Roy, Neale & Kendler, 1995). A somewhat related personality characteristic seen to influence the development of an eating disorder is locus of control, or the degree to which women feel that they are personally effective in their lives and their relationships. The results consistently suggest that an external

locus of control and feelings of ineffectiveness act as risk factors for bulimia nervosa (Kendler et al., 1991; Shisslak, Pazda & Crago, 1990; Williams et al., 1990).

One methodology, which offers a powerful examination of the casual relationship between variables, is the twin study (Silberg, Erickson, Meyer, Eaves, Tutter & Hewitt, 1994), particularly with recent advances in biometrical modeling techniques (Neale & Cardon, 1992). The only twin study to date to examine the relationship between disordered eating and psychological variables is that of Kendler et al. (1991). Using logistic regression analysis, their study found that psychological risk factors for bulimia nervosa included lower levels of perceived care from the father in the first 15 years of life, low self-esteem, an external locus of control and high levels of neuroticism. The purpose of the present study was to investigate in an Australian sample the genotypic and environmental causes of resemblance among various personality variables, including self-esteem, neuroticism and emotional reliance on others, and measures of perceived family functioning, together with a measure of disordered eating, using more powerful biometrical genetic modeling techniques.

## 2. Method

### 2.1. Participants

Women registered with the Australian National Health and Medical Research Council (NHMRC) Twin Registry who participated in a self-report questionnaire mailed in 1988–1989 were included in these analyses. Women were selected if they were from MZ and DZ same-sex pairs and aged 30–45 years ($M = 36.5$, S.D. $= 4.7$) at the time of data collection. Data were available for 537 MZ and 344 DZ twin pairs, where each woman had provided data on at least one of the six variables of interest (i.e. the measure of eating problems, three personality variables and two measures of parental functioning).

### 2.2. Measures

Initially, a 'Health and Lifestyle Survey for Twins' was sent out to approximately 1500 pairs of twins. This survey consisted of a large battery of different questionnaires and hence many of the measures included were shortened versions of the original. Sixteen items examined disturbed eating and attitudes. In addition to these questions, of particular relevance to the present study were questions relating to perceived parental care and protection in the first fifteen years of life and the personality measures of neuroticism, self-esteem and interpersonal dependency.

The eating questions examined the women's experience with both previous and current problems involving eating, eating disorders and weight (Wade, Heath, Abraham, Treloar, Martin & Tiggermann, 1996a). Although answers to the questions did not permit the formation of diagnostic categories, a confirmatory factor analysis of the items showed them to yield five stable and generalisable factors, which accounted for 60% of the variance of disordered eating (Wade, Tiggerman, Heath, Abraham, Treloar & Martin, 1996b). The items were formed into an index of disordered eating, in which the 'yes' responses to the 16 items

were added together and then divided by the number of items included in the algorithm (at least 15 out of 16 items had to be answered in order for the data to be included in these analyses). This gave a final score between 0 and 1, the closer the score to 0 indicating fewer eating problems. The internal reliability (Cronbach's alpha) of the index was 0.75.

Perceptions of parental style were measured by the short version (Todd, Boyce, Heath & Martin, 1994) of the parental bonding instrument (PBI: Parker, Tupling & Brown, 1979). A retrospective measure should reduce contamination of perceived family functioning by any current eating problems. Three items assessed parental care (e.g. "seemed emotionally cold", "appeared to understand my problems and worries") and four items measured parental protectiveness (e.g. "let me do those things I liked doing", "liked me to make my own decisions"). Each item was rated on a four-point Likert scale. For each measure, items were completed separately for mother and father. Here the scales for mother and father were combined, providing one parental care measure and one parental protection measure. The correlation between the full length PBI care scale and the three-item version used in this study has been reported as 0.94 (father) and 0.93 (mother), with the correlation between the full length PBI protection scale and the four-item version as 0.89 (father) and 0.91 (mother), and the shortened version has been judged to be acceptable for use in epidemiological studies (Todd et al., 1994). The internal reliabilities of the short-version PBI measures used in this study were 0.78 (parental care) and 0.77 (parental protectiveness).

With regard to the three personality variables, neuroticism was assessed with the neuroticism subscale of the short form of the Eysenck Personality Questionnaire-Revised (EPQ-R: Eysenck, Eysenck & Barrett, 1985). The internal reliability in this study was 0.82. The measure of self-esteem was the ten-item Rosenberg self-esteem scale (RSE: Rosenberg, 1965) which had an internal reliability of 0.68. The third personality variable selected for examination was the emotional reliance on another person dimension of the interpersonal dependency inventory (IDI: Hirschfeld, Klerman, Gough, Barnett, Korchin & Chodoff, 1977). This scale represents a measure of locus of control, in that it measures the extent to which the person relies on others for their emotional regulation. The scale used consisted of ten of the eighteen items from the IDI, including items such as "I tend to expect too much from others" and "I would feel helpless if deserted by someone I love". Items were rated on a four-point Likert scale and the internal reliability of the measure was 0.77.

## 2.3. Analysis strategy

In univariate analyses, structural equation models are applied to twin data in order to decompose the overall phenotypic (or observed) variance of a trait into four types of influence: (1) additive genetic factors (A) that reflect the additive effect of genetic loci influencing the trait, (2) genetic dominance (D) which refers to the interaction between alleles at the same locus, (3) nonshared environmental factors (E) which represent the aspects of the environment that only one twin experiences and (4) shared environment factors (C) common to both twins in the pair. The ultimate goal of model fitting is to account for the data with the smallest number of parameters possible (Neale & Cardon, 1992).

In a multivariate analysis, given that the structure of variance may be quite different for each trait, the goal of analysis is to delineate how the genetic and environmental sources of

variance influence covariation between traits. By comparing cross-twin, cross-variable correlations in MZ and DZ twins, and contrasting these to the cross-twin, within-variable and within-twin, cross-variable correlations, the covariation of two or more variables can be partitioned into genetic and environmental components. Two alternative models are tested here to describe how genetic and environmental factors may influence covariation or the *structure* of covariation. The primary focus of both these models is the contribution of the common genetic and environmental influences to the variance specific to each observed variable. The first of these models is the independent pathways model, where each of the latent genetic or environmental factors has its own path to each observed variable. The second model, the common pathways model, is a more stringent model, which hypothesises that the covariation between symptoms is determined by a single phenotypic latent variable (Neale & Cardon, 1992).

In the multivariate analyses outlined in this paper, a model fitting approach was used to distinguish the effects of genetic and environmental etiologic factors that were common to all the variables from the effects of genetic and environmental factors that were of etiologic importance only for the specific variables. The final aim of model testing is to find the most parsimonious model, which will explain the observed data. In this case, the overall fit function and the accompanying degrees of freedom of each model were calculated. The difference between the fit functions ($\chi^2$) and degrees of freedom of the full and best fitting model and the comparison model were then examined for significance.

The computer programme Mx (version 1.42) was used to carry out the analyses (Neale, 1997). Through the process of listwise deletion, use of maximum likelihood analysis with covariance matrices would have resulted in a substantial loss of subjects. Therefore maximum likelihood analysis was used on the raw data, thus ensuring that cases were not lost through listwise deletion and making maximum use of the available data (Neale, 1997). This method depends on the assumption that missingness of any of the six scale scores of interest here is completely random (Little & Rubin, 1987).

## 2.4. Preparation of the data

The six variables (eating index, two parental functioning and three personality variables) were first examined for univariate normality. While univariate normality is a necessary condition for multivariate normality, departures from which comprises the methodology of structural equation modeling, it does not guarantee this condition. The measures of self-esteem and emotional reliance on another person did not depart significantly from normality ($ps > 0.05$) but the other four variables (the eating index, neuroticism, parental care and parental protection) consistently departed from univariate normality. All variables were therefore transformed to normal weights.

## 3. Results

The maximum likelihood estimates (MLE) of the means and variances were first tested for equality between twin 1 and twin 2 and then between MZ and DZ twins. No significant

differences were found, consistent with the hypothesis that MZ and DZ twins have been drawn from the same population.

The MLE of the cross-twin, cross-trait correlations are presented in Table 1 separately for MZ and DZ pairs. Within this table are four different matrices of intra-individual (phenotypic) correlations between the 6 measures — for twin 1 and twin 2 within both MZ and DZ twins. Overall, the psychological variables were only very modestly correlated with the index of disordered eating. The most highly correlated was neuroticism, with correlations ranging from 0.10 to 0.34. In other words, the greater the problems with disordered eating, the higher the levels of neuroticism. The personality variables were more substantially associated with each other, the highest correlation being between neuroticism and the interpersonal dependency variable (emotional reliance on another) at around −0.60, i.e. the greater the neuroticism trait, the more the person was likely to look to other people for emotional regulation. Also of note were the correlations between neuroticism and self-esteem, self-esteem and interpersonal dependency, and parental protection and parental care, indicating that higher levels of neuroticism and greater external locus of control were associated with lower self-esteem, while less perceived parental care is associated with overprotectiveness.

The MLE of the MZ and DZ twin correlations for the six traits (also displayed in Table 1) are in bold in the top right quarter (MZ) and bottom left quarter (DZ). Since the $r_{MZ}$ is approximated equal to $2r_{DZ}$, this suggested that neuroticism, self-esteem and emotional reliance showed little or no effect of shared environmental influences (C), but rather determination by genetic and unique environmental influences (A and E). The twin correlations for the eating measure, parental care and parental protection measures, were suggestive of genetic, shared and unique environmental influences because the $r_{MZ} < 2r_{DZ}$. The effect of the shared environment was particularly suggested for the parental protection variable, where the MZ and DZ correlations are similar.

Table 1
Maximum likelihood estimates of the twin pair correlations between the twins for eating, parental care (pc) and parental protection (pp), neuroticism (neur), self-esteem (se) and emotional reliance on others (idi). MZ correlations (for 302–322 twin pairs) are placed in the top right of the diagonal and DZ correlations (for 280–310 twin pairs) are placed in the bottom left of the diagonal. Correlations between twin pairs for the same trait are in bold

| | eating/T1 | pc/T1 | pp/T1 | neur/T1 | se/T1 | idi/T1 | eating/T2 | pc/T2 | pp/T2 | neur/T2 | se/T2 | idi/T2 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Eating/T1 | *1.00* | −0.13 | 0.09 | 0.22 | −0.13 | −0.23 | **0.43** | −0.08 | 0.05 | 0.15 | −0.15 | −0.13 |
| pc/T1 | −0.15 | *1.00* | −0.43 | −0.24 | 0.21 | 0.21 | −0.18 | **0.54** | −0.26 | −0.13 | 0.17 | 0.18 |
| pp/T1 | 0.16 | 0.44 | *1.00* | 0.30 | −0.21 | −0.33 | 0.11 | −0.23 | **0.38** | 0.13 | −0.14 | −0.13 |
| Neur/T1 | 0.20 | −0.15 | 0.25 | *1.00* | −0.50 | −0.61 | 0.15 | −0.15 | 0.14 | **0.44** | −0.22 | −0.31 |
| se/T1 | −0.16 | 0.02 | −0.15 | −0.43 | *1.00* | 0.47 | −0.07 | 0.16 | −0.11 | −0.32 | **0.36** | 0.29 |
| idi/T1 | −0.21 | 0.12 | −0.18 | −0.58 | 0.40 | *1.00* | −0.12 | 0.12 | −0.17 | −0.33 | 0.21 | **0.38** |
| Eating/T2 | **0.26** | −0.13 | −0.01 | 0.02 | 0.07 | −0.04 | *1.00* | −0.17 | 0.09 | 0.34 | −0.23 | −0.27 |
| pc/T2 | −0.07 | **0.39** | −0.20 | 0.01 | −0.05 | −0.04 | −0.14 | *1.00* | −0.46 | −0.21 | 0.26 | 0.24 |
| pp/T2 | 0.06 | −0.18 | **0.34** | 0.08 | 0.02 | 0.01 | 0.10 | −0.46 | *1.00* | 0.19 | −0.16 | −0.19 |
| Neur/T2 | 0.12 | −0.03 | 0.09 | **0.20** | −0.04 | −0.06 | 0.25 | −0.15 | 0.16 | *1.00* | −0.48 | −0.60 |
| se/T2 | 0.02 | 0.04 | −0.09 | −0.19 | **0.07** | 0.11 | −0.14 | 0.10 | −0.19 | −0.47 | *1.00* | 0.50 |
| idi/T2 | −0.06 | 0.10 | −0.09 | −0.23 | 0.00 | **0.11** | −0.23 | 0.23 | −0.14 | −0.56 | 0.53 | *1.00* |

## 3.1. Multivariate genetic analysis

The model fitting process is summarised in Table 2. We begin with a full independent pathway model that included additive genetic, shared and unique environmental factors both unique to each trait and common to each trait (model 1). We then fit a common pathway model of the same type to the data (model 2) which produced a significantly worse fit ($p < 0.01$). In model 3, the shared environmental common factor was eliminated and this model was not found to be significantly worse than the full model, and was therefore more parsimonious. Model 4 eliminated the additive genetic common factor from the full model; the fit was significantly worse than the full model ($p < 0.01$). The final model tested (model 5) eliminated the common environmental influences unique to each trait and common to each trait. While this model was not significantly worse-fitting than the full model, it did give a significantly worse fit than model 3, which has only three free parameters ($\chi^2 = 13.03$, d.f. $= 3$, $p < 0.01$).

Figure 1 summarises the structure and the magnitude of each path of the best fitting model, allowing for both additive genetic and unique environmental influences in common to the variables and also additive genetic, unique environmental and shared environmental influences specific to each variable. Those pathways which were effectively zero (i.e. to 3 decimal points) are not shown. It can be noted that, consistent with the twin correlations, it was only disordered eating, parental care and parental protection that have shared environmental specific influence as well as the genetic and nonshared environmental specific influences.

The sources of variance in liability to the six traits under this best fitting multivariate model are displayed in Table 3. The path, or partial regression, coefficients have been squared to calculate the proportion of variance accounted for by the latent predictor variables. Self-esteem, emotional reliance on others and perceptions of parental protectiveness had the highest genetic contribution, at around 40%, followed closely by the disordered eating variable (34%). Perceptions of parental care were heavily influenced by environmental influences.

## 4. Discussion

The aim of this study was to examine the relationships between disordered eating,

Table 2
Results of multivariate model fitting, investigating sources and structure of covariation between the six variables[a]

| Model | Specific factors | Common factors | Pathway | Overall fit function | df | $\chi^2$diff | d.f. | $p$ |
|---|---|---|---|---|---|---|---|---|
| 1 | ACE | ACE | independent | 18 072.11 | 9573 | | | |
| 2 | ACE | ACE | common | 18 102.89 | 9583 | 30.78 | (10) | < 0.01 |
| 3 | ACE | AE | independent | 18 073.53 | 9579 | 1.4 | (6) | > 0.95 |
| 4 | ACE | CE | independent | 18 162.16 | 9579 | 90.05 | (6) | < 0.01 |
| 5 | AE | AE | independent | 18 086.54 | 9585 | 14.43 | (12) | > 0.2 |

[a] $\chi^2$ and d.f. are calculated by subtracting the fit function (d.f.) from the full and best fitting model (in this case the full ACE independent pathway model) from the fit function (d.f.) of interest.
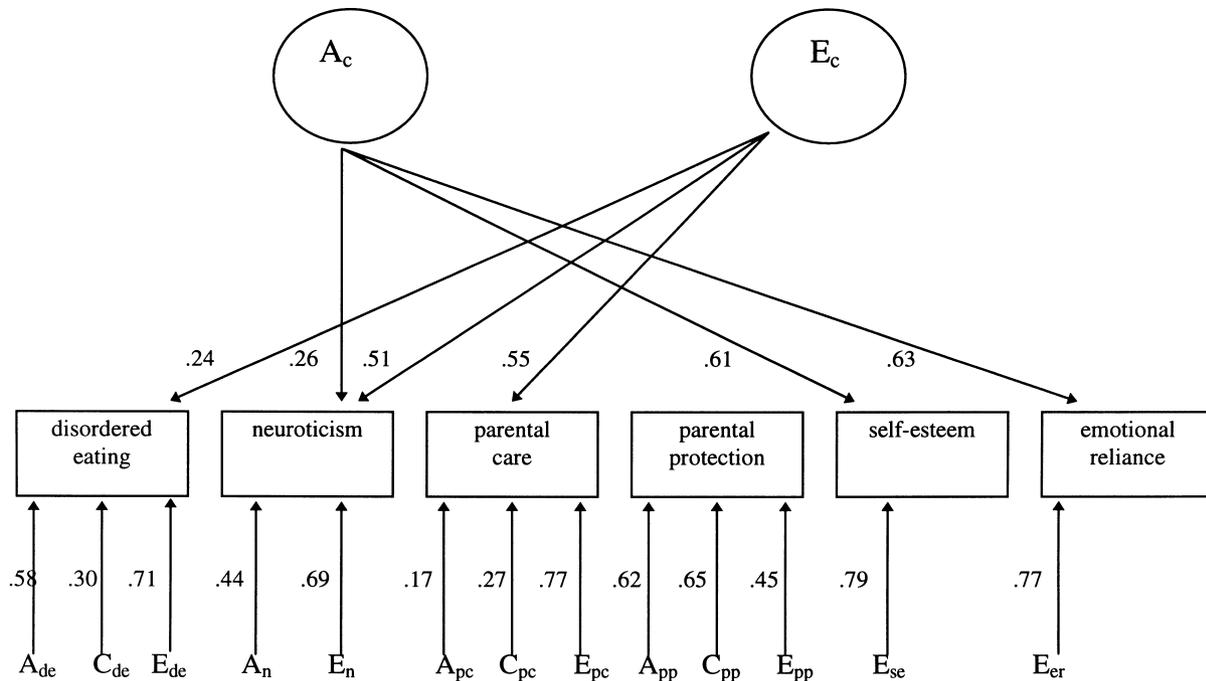
Fig. 1. Results from the best fitting submodel (model 3). A indicates additive genetic influences, E indicates unique environmental influences and C indicates shared environmental influences. The subscript C refers to those influences common to the traits and specific effects are notated using the initials of the trait. The magnitude of each path (path estimates) is shown in the figure.

psychological and family variables, using biometrical modeling and a multivariate analysis of data from twin pairs. The self-report measures included an index of disordered eating, neuroticism, self-esteem, interpersonal dependency (representing a measure of locus of control) and perceived parental care and protectiveness in the first 15 years. In order to clarify the

Table 3
Sources of variance in liability to the six traits from the best fitting multivariate model

| Trait | Proportion of variance | | | | |
| | Common | | Specific | | |
| | A | E | A | C | E |
|---|---|---|---|---|---|
| Disordered eating | 0.00 | 0.06 | 0.34 | 0.09 | 0.51 |
| Neuroticism | 0.07 | 0.26 | 0.20 | 0.00 | 0.47 |
| Parental care | 0.00 | 0.30 | 0.03 | 0.07 | 0.60 |
| Parental protection | 0.00 | 0.00 | 0.38 | 0.42 | 0.20 |
| Self-esteem | 0.37 | 0.00 | 0.00 | 0.00 | 0.63 |
| Emotional reliance | 0.40 | 0.00 | 0.00 | 0.00 | 0.60 |

relationship between the six measured phenotypes in terms of genetic and environmental risk factors, we examined models which allowed for both common risk factors (a risk factor shared among traits) and specific risk factors (a risk factor specific to only one trait). The first finding to note was that only one variable, parental protectiveness, did not share either genetic or environmental risk factors with any of the measured variables. Perception of parental care and protectiveness while growing up appear to be unrelated in terms of genetic and environmental risk factors. This is not an unexpected finding, as these measures were designed to be orthogonal and a different genetic and environmental structure for these two measures has been found previously (Kendler, 1996).

The relationship between the six traits was not close, as would be observed if the traits were a variation of a single disorder. Those traits which share unique environmental risk factors include disordered eating (6%), neuroticism (26%) and parental care (30%), confirming a previous finding that neuroticism and perceptions of parental care are risk factors for eating disorders (Kendler et al., 1991). This suggests that the some of the same unique environmental factors that shape a wide variety of eating problems in women also shapes neuroticism and perceptions of parental care. The interactions between these three phenotypes may reflect person-environment correlations, where the characteristics of individuals affect their interactions with others (Scarr & McCartney, 1983). For example, children at genetic risk of antisocial behavior were more likely to evoke and receive negative parenting from their adoptive parents than other adopted children, in turn increasing the likelihood of behavioral problems (O'Connor, Deater-Deckard, Fulker, Rutter & Plomin, 1998). Similarly, children at genetic risk of developing an eating disorder may create an environment in which they evoke, or perceive less warmth from parents and are more likely to express neurotic traits. This environment, in turn, may reciprocally act on increasing the possibility that disordered eating is expressed at a later stage in life.

Disordered eating did not share any genetic risk factors with the other five traits. Given the genetic link between neuroticism, depression and bulimia nervosa (Kendler et al., 1993; Walters et al., 1992), one might expect to find shared genetic risk factors between disordered eating and neuroticism. However, while neuroticism, self-esteem and emotional reliance on others shared genetic risk factors (7, 37 and 40% respectively) and moderate phenotypic correlations, we find a low phenotypic correlation between the neuroticism and disordered eating (around 0.2), and no shared genetic risk factors. A recent study (Lilenfeld et al., 1998) has found that obsessional personality traits may be a specific risk factor for anorexia nervosa, whereas cluster B personality disorders (reflecting affective instability and impulsivity) may be a specific risk factor for bulimia nervosa. Thus it seems unlikely that a general measure of disordered eating, that included problems with obesity, bulimia nervosa, anorexia nervosa and binge-eating, will have a strong relationship with one measure of personality.

It is of interest to note that the best fitting model suggested that 9% of the variance in the measure of disordered eating was due to a specific shared environmental influence. This is only the third study, all of which use different measures of disordered eating, to find that the shared environment can contribute to the development of an eating disorder (Kendler et al., 1995; Wade, Martin & Tiggemann, 1998). The specific nature of this common environmental influence is unclear, and this study does not find that disordered eating shares common environmental risk factors with the parenting variables. Thus we may be looking outside of the

family for this source of common environment. The most likely candidate would be the western sociocultural environment, which can potentially be experienced similarly among women. This suggestion would be consistent with the small amount of variance accounted for by the common environment, as we know that all women in western culture are exposed to this environment, but not all develop eating disorders, suggesting that many other factors work together as the major risk factors for an eating disorder.

There are three important limitations of this study. First, as the study relied on single measures of traits (as opposed to multiple measures), we were not in a position to distinguish between measurement error and the unique environment, leaving open the possibility that the estimations of unique environment are inflated. This problem may have been exacerbated as some of the measures used in this study were based on subsets of items of the full measure. Second, the use of a general measure of disordered eating in a randomly selected sample means that the results are applicable to a range of problems, and can not be related to one particular eating disorder. Finally, the modeling process is unable to inform us of the specific aspect of environment risk factor that is shared between eating problems, neuroticism and parental care, and how this works to produce disordered eating.

In summary, this study has presented evidence that unique environmental risk factors are shared among a wide variety of eating problems in women, neuroticism and perceptions of parental care. The common variance shared between the measure of eating and the other two variables was small, which may indicate that the heterogeneity observed in our measure of disordered eating does not allow for strong relationships between specific risk factors. Further research in this area should utilise specific types of disordered eating, or measures that focus on styles of thinking common across different eating disorders, such as the overvalued ideas associated with bulimia nervosa and anorexia nervosa (Cooper & Fairburn, 1993) or the styles of cognition which have been shown to be highly associated with eating disorders (Butow, Beumont & Touyz, 1993).

## Acknowledgements

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