Shared genetic and environmental risk factors between undue influence of body shape and weight on self-evaluation and dimensions of perfectionism

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ABSTRACT

Background. Theory and evidence strongly suggest that perfectionism may be a risk factor for eating disorders. The purpose of the current study was to investigate a model that would explain the relationship between the cognitive diagnostic criterion for both anorexia nervosa and bulimia nervosa, namely undue influence of body weight or shape on self-evaluation, and dimensions of perfectionism. The model of particular interest was the common cause model, which hypothesizes that the phenotypes are caused by the same underlying genetic and environmental risk factors.

Method. Female twins ($n = 1002$) from the Australian Twin Registry (ATR), aged 28 to 39 years, were interviewed using the Eating Disorder Examination (EDE). In addition, questions relating to the Equal Environment Assumption (EEA) and the Frost Multidimensional Perfectionism Scale, namely concern over mistakes (CM), personal standards (PS) and doubts about actions (DA), were assessed.

Results. There was no evidence of violation of the EEA for any of the four phenotypes. Univariate models showed all phenotypes to be influenced by both genetic and non-shared environmental action, where genetic estimates ranged from 25% to 39% of the variance. Multivariate analyses suggested the best explanation of covariation among the phenotypes was an independent pathways, rather than a common pathways, model.

Conclusions. Undue influence of body weight or shape on self-evaluation shared about 10% of its sources of genetic and environmental variance with perfectionism, thus suggesting that a common cause model does not represent the best explanation of the relationship between perfectionism and this cognitive diagnostic criterion for eating disorders.

INTRODUCTION

The nature of the relationship between perfectionism and eating disorders is unclear (Shafran & Mansell, 2001). Perfectionism has been defined as self-evaluation that relies on the continual pursuit of personally demanding standards (Shafran *et al.* 2002). It has been suggested that, when an eating disorder is present,

these standards are singularly focused on control over eating, shape and weight (Shafran *et al.* 2002). Vigorous debate exists regarding whether perfectionism is best conceptualized as a unidimensional or a multidimensional construct (e.g. Shafran *et al.* 2002; Dunkley *et al.* 2006).

Perfectionism has been hypothesized to precede the development of eating disorder symptoms in the transdiagnostic theory of eating disorders (Fairburn *et al.* 2003). This theory postulates that perfectionism is a proximal risk factor for the development of overvaluation of the importance of weight and shape, considered

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to be the core cognitive psychopathology of eating disorders as opposed to body image disturbance (Cooper & Fairburn, 1993), and included as a DSM diagnostic criterion for both anorexia and bulimia nervosa (i.e. 'undue influence of body weight or shape on self-evaluation'). Evidence strongly suggests that this core cognitive substrate leads to the development of eating disordered behaviour (Killen *et al.* 1996; Cooper & Goodyer, 1997; McKnight Investigators, 2003), that it is a proximal risk factor for eating disorders (Jacobi *et al.* 2004), and that it remains significantly elevated even after the eating disorder behaviour is no longer present (Wade *et al.* 2006*a*).

As yet, longitudinal studies have not clearly determined the causal role of perfectionism with respect to eating disorder diagnostic criteria (Stice, 2002). A recent review concluded that perfectionism could only be considered a retrospective correlate (Jacobi *et al.* 2004), where both women recovered from anorexia and bulimia nervosa report higher levels of pre-morbid perfectionism than psychiatric or healthy controls (Fairburn *et al.* 1999). A more recent review (Lilenfeld *et al.* 2006) concluded that evidence from quasi-prospective, retrospective and family study research strongly suggests that perfectionism may be a predisposing factor for eating disorders.

If perfectionism is a risk factor for undue influence of body weight or shape on self-evaluation and consequent eating disordered behaviour, the nature of this relationship has important implications for treatment approaches, as well as having the ability to guide future risk factor and genetic studies. For example, two basic risk factor models have been proposed to explain the co-occurrence between personality and eating disorders (Lilenfeld *et al.* 2006). The first, a pre-dispositional model, where the two conditions are independent and the aetiology distinct, would suggest treating both conditions. A common cause model, where both conditions are caused by the same underlying factor or factors, would suggest treating the underlying condition, or alternatively that a focus on one condition should impact on the other. Support of this latter model would also implicate perfectionism as an endophenotype of undue influence of body weight or shape on

self-evaluation along the pathway to eating disorder vulnerability, which would support the idea that perfectionism could act as a candidate trait for weight and shape concern.

Multivariate twin studies provide a powerful test of the common cause model, as they yield information about the extent to which perfectionism and undue influence of body weight or shape on self-evaluation share correlated liabilities with respect to genetic and environmental influences. While a twin study has shown that different dimensions of perfectionism are influenced to a small degree by shared genetic and environmental influences and that perfectionism is likely to be a multidimensional construct (Tozzi *et al.* 2004), no twin studies have examined the relationship between dimensions of perfectionism and eating disorder symptomatology. Our goal was to use a female twin population to examine the multivariate relationships among dimensions of perfectionism and undue influence of weight and shape on self-evaluation. We address two questions: (1) To what degree do genetic and the environmental factors influence these phenotypes? (2) To what degree are the genetic and environmental liabilities for these phenotypes correlated or overlapping? A high level of correlation would provide support for the common cause model.

METHOD

Participants

Participating twins were derived from a cohort of 8536 twins (4268 pairs) born 1964–1971, who were registered as children with the Australian Twin Registry (ATR) during 1980–1982, in response to media and systematic appeals through schools. Female–female twins who had participated in at least one of two waves of data collection (Heath *et al.* 2001), one during 1989–1992 when the twins were aged 18–25 years and the other during 1996–2000 when the median age of the sample was 30 years, were approached during 2001–2003 to participate in a third wave of data collection ($n = 2320$), of whom 1083 individual twins (47%) approached actively consented to participate (Wade *et al.* 2006*b*). Of those consenting, 1002 (43%) completed a semi-structured interview over the telephone relating to current and lifetime eating and 1016 (44%) completed a mailed self-report

questionnaire assessing various aspects of personality (Wade *et al.* 2006*a*), with 962 women completing both (42%). In all, 1056 females (46%) participated in at least one of the data collection components.

The sample included 348 complete sister–sister pairs who completed Wave 3 data collection, 226 monozygotic (MZ) pairs and 122 dizygotic (DZ) pairs, and 360 incomplete pairs (170 MZ and 190 DZ), where only one twin participated. Both interview and questionnaire were completed by 293 complete pairs. Zygosity was determined on the basis of responses to standard questions about physical similarity and confusion of twins by parents, teachers and strangers, methods that give better than 95% agreement with genotyping (Eaves *et al.* 1989). The mean age of the women at the time of the data collection was 35 years (s.d. = 2·11, range 28–40). The Flinders University Clinical Research Ethics Committee approved the study and written informed consent was obtained.

Measures

Equal Environment Assumption (EEA)

Given concern about the robustness of the EEA (Jacobi *et al.* 2004), we assessed the EEA using 12 items measuring environmental similarity used in previous twin research (Bulik *et al.* 1998).

Undue influence of body weight or shape on self-evaluation

The Eating Disorder Examination (EDE) 14th edition (Fairburn & Cooper, 1993) is a semi-structured interview where all diagnostic questions address occurrence over a 1-month time-frame for each of the previous 3 months. In addition, the EDE was revised to address lifetime occurrence of behavioural diagnostic questions (Wade *et al.* 2006*c*). The assessment also included questions relating to dietary restraint (five items, $\alpha = 0.60$), eating concern (five items, $\alpha = 0.72$), weight concern (five items, $\alpha = 0.74$) and shape concern (seven items, $\alpha = 0.86$) over the past 28 days. Every participant was asked each question in the interview. All interviewers were postgraduate clinical psychology trainees ($n = 10$) who were trained in use of the EDE. Interviews were taped and corrective feedback was provided until the

interviewer reached the criterion. Monthly meetings addressed the interview process and ensured interview fidelity.

Only two items are used to assess the diagnostic criterion of 'undue influence of body weight or shape on self-evaluation', namely those relating to the importance of weight and of shape over the previous 3 months, each measured on a seven-point Likert scale for each month in that 3-month time-frame. Therefore, the scores on the weight diagnostic items were added together and the same procedure was carried out for the shape diagnostic items. The mean of these six scores was then calculated and used in all of the analyses. Internal reliability for these six combined items was high (Cronbach's $\alpha = 0.97$).

Perfectionism

The Frost Multidimensional Perfectionism Scale (MPS-F; Frost *et al.* 1990) is a 35-item self-report measure rated on a five-point Likert scale. The MPS-F contains six subscales but only three of these are considered to measure the current personality dimensions related to perfectionism (Frost *et al.* 1990): concern over mistakes (CM), personal standards (PS), and doubts about actions (DA). This latter subscale is a partial index of obsessionality given that three of the four items are adapted from the Maudsley Obsessional Compulsive Inventory (Hodgson & Rachman, 1977). Participants were asked to indicate their agreement to statements on a scale ranging from 1 (strongly disagree) to 4 (strongly agree), where higher scores are indicative of greater perfectionism. Originally based on a five-point Likert scale, we used a four-point scale to force choice. Internal reliability was good [Cronbach's $\alpha = 0.90$ (CM), 0·85 (PS) and 0·82 (DA)].

Statistical analyses

EEA

To examine the factor structure of the EEA questions, a product–moment correlation matrix for all items was submitted to a factor analysis with varimax rotation, where factors with an eigenvalue greater than unity were extracted. A factor analysis was conducted separately for each twin. To assess the similarity of the factors across the different reporters,

Tucker's congruency coefficients were calculated (Tucker, 1951). Polychotomous linear regression was used to evaluate the EEA for each of the four phenotypes. There were two regressions for each phenotype, for each of the twin's reports. In each case, the dependent variable was the absolute value of the difference between the eating disorder phenotype for Twin 1 and Twin 2. The difference score was transformed ($\log_{10} x + 1$) because of the positive skew of the data. The independent variables were zygosity and, in turn, the two measures of EEA.

Univariate twin analyses

Maximum likelihood estimate correlations for each phenotype between the MZ pairs and DZ pairs were calculated, along with 95% confidence intervals (CIs). To examine the sources of individual difference in the four phenotypes (CM, PS, DA and the weight and shape measure), PRELIS2 (Joreskög & Sörbom, 1996) was used to produce two 2×2 variance–covariance matrices (one each for MZ and DZ twin pairs) for each phenotype. In each case, the phenotype scores were transformed ($\log_{10} x + 1$) because of the positive skew of the data. Such matrices only include cases where complete data for all measures are available from both twins in the pair. These matrices were subjected to structural equation model fitting using the statistical package Mx (Neale, 1997). In the traditional univariate twin model, the sources of variance in liability to a disorder are divided into that proportion accounted for by three different influences: additive genetic (A), common or shared environmental (C), and non-shared or unique environmental (E). This latter factor also contains the variance of any error measurement. Each factor is latent and not directly observed. Initially, a full model (ACE) was fit to the data, followed by an AE model, a CE model, and a model containing only non-shared environment (E).

The goal of model fitting is to explain the observed data as an optimal combination of goodness-of-fit and parsimony. Akaike's Information Criterion (AIC; Akaike, 1987) reflects these criteria, where the more negative the value, the better the fit of the model. Finally, we estimated the proportion of variance contributed by genetic (a^2), shared environmental (c^2), and

non-shared environmental (e^2) factors to the four phenotypes, along with the 95% CI.

Multivariate twin analyses

Given that more missing data are introduced when using multivariate analysis, a different approach was used in the analysis of the multivariate data, one that does not provide the advantage of an AIC statistic but does optimize the data set, namely the method of maximum likelihood using Mx (Neale, 1997), where models are fit to raw data from all twins, including those with missing data and those pairs where only one twin participated. Given that previous analyses showed no relationship between participation in the current wave of data collection and variables from the previous two waves, including the number of eating problems, personality variables or lifetime depression (Wade *et al.* 2006*b*), this statistical approach can reduce the impact of any respondent bias when the data are missing at random (Little & Rubin, 1987). An advantage of using these different approaches is that parameter estimates derived from the first method, which only includes data from complete twin pairs, can be compared to those derived from the second method, which contains data from all twins, to see if any bias in response patterns makes a substantial difference to these estimates.

The transformed phenotype scores were used in the multivariate analysis. Given that the univariate estimates of C were zero for each of the four phenotypes and that the most parsimonious univariate models for the four phenotypes were the AE model, only this model was examined in subsequent analyses.

Pearson correlations were used to investigate the predicted associations among the four variables. Cholesky decomposition was used to calculate the genetic and environmental correlations across the four phenotypes. These correlations represent the extent to which the same genes or environmental factors contribute to the observed phenotypic correlation among the variables. Both an AE independent pathways (IP) model and a common pathways (CP) model were examined to select the best model to explain covariation among the phenotypes. In the IP model, each of the two common latent factors (A and E) has their own paths to each of the four phenotypes. In other words, the

four phenotypes have these influences in common. The sources of unique variance to each phenotype from A and E sources are also estimated – that is, those sources unshared with the other phenotypes, known as specific pathways. The CP model is more stringent than the IP model and is considered to be a submodel (McArdle & Goldsmith, 1990), as it hypothesizes that the covariation among the four phenotypes is determined by a single phenotypic latent variable, which is itself determined by latent sources of variance from A and E. In addition, this model allows for unique or specific sources of variance to contribute to each phenotype. The two models were compared by subtracting the fit function (-2 log-likelihood of raw data) and the degrees of freedom (df) of the IP model from the fit function and df of the CP model, yielding a χ^2 value and an associated df.

RESULTS

Descriptives

The mean item score for the weight and shape measure was 2·77 (s.d. = 1·27), with a median of 2·50, and a range from 0·50 to 6·00. The mean item (s.d.) scores for CM, PS and DA were 1·81 (0·49), 2·46 (0·50) and 2·00 (0·54) respectively. The respective median values were 1·94, 2·43 and 2·00.

The women were divided into two groups to examine the relationship between lifetime eating disorder behaviour and the weight and shape measure. The first comprised women ($n = 294$) who had reported the presence of any of six lifetime eating disorder behaviours, namely objective binge eating, self-induced vomiting, laxative misuse, diuretic misuse, fasting, and self-reported low body weight [body mass index (BMI) $\leqslant 17\cdot5$]. In each case, the behaviour had to meet the frequency and duration thresholds in the specific DSM-IV eating disorder diagnoses (e.g. low body weight was sustained over a 3-month period, objective binge eating occurred at least twice a week over a 3-month period). The second group consisted of women who had not reported any of these behaviours ($n = 708$). The respective weight and shape scores were 3·01 (s.d. = 1·38) and 2·66 (s.d. = 1·19), where the women with lifetime eating behaviours had significantly higher scores with an odds ratio

of 1·24 (95% CI 1·10–1·41). Consistent with previous research (Wade *et al.* 2006*a*), the experience of lifetime eating disorder behaviours can be seen to have a permanent 'scarring' effect on the women in terms of its ongoing impact on the undue influence of weight and shape on self-evaluation.

EEA analysis

The factor structure obtained across both twins was very similar, identifying two robust factors: (1) co-socialization, indicating the tendency to socialize together in childhood and adolescence, with respective internal consistencies of 0·80 and 0·81, and (2) similitude, indicating the degree to which the twins' similarities were emphasized by themselves and others, with respective α values of 0·70 and 0·71. The factor structure across Twins 1 and 2 was very similar, with Tucker congruency coefficients of 0·936 and 0·941 for the first and second factors respectively, indicating a highly stable structure. Factor scores from a combined factor analysis were used to derive scales for use in further analyses. A series of regression analyses was performed with each of the phenotypes as an outcome variable (Table 1). None of the phenotypes was associated with co-socialization or similitude (i.e. no violations of the EEA were evident).

Univariate twin analyses

Univariate twin model results are summarized in Table 2. For each phenotype, correlations between the MZ twin pairs were higher than the DZ twin pairs, where all 95% CIs for the DZ pairs and none for the MZ pairs included 0. This pattern of results indicated an influence of genetic factors on all four phenotypes. With respect to each measured phenotype, the AIC indicated that the AE model was the most parsimonious, and did not significantly differ from the full ACE model (χ^2 as calculated by the difference in AIC values). However, the parameter estimates of the full model were also calculated because use of the AIC to select among nested models in univariate analyses can be problematic (Sullivan & Eaves, 2002). Across all of the full models the c^2 estimate was 0. With respect to CM, both the CE ($\chi^2 = 8\cdot45$, df = 1, $p < 0\cdot05$) and E ($\chi^2 = 34\cdot99$, df = 2, $p < 0\cdot01$) models fit significantly worse than the full model. The same held for PS (CE: $\chi^2 = 4\cdot13$, df = 1, $p < 0\cdot05$; E: $\chi^2 = 28\cdot14$, df = 2,

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Table 1. *Regression analyses examining differences with respect to the phenotypes as the outcome variable, and zygosity and the EEA as the independent variables*

| | Co-socialization | | | | Similitude | | | |
| | Twin 1 | | Twin 2 | | Twin 1 | | Twin 2 | |
Phenotype	Zygosity β (ES)	EEA β (ES)	Zygosity β (ES)	EEA β (ES)	Zygosity β (ES)	EEA β (ES)	Zygosity β (ES)	EEA β (ES)
Weight and shape concern	0·08 (0·15)	0·04 (0·08)	0·10 (0·19)	−0·09 (0·17)	0·13 (0·24)*	−0·11 (0·20)	0·07 (0·12)	0·03 (0·05)
Concern over mistakes	0·23 (0·46)***	−0·03 (0.0.6)	0·19 (0·39)**	0·04 (0·08)	0·23 (0·42)***	−0·02 (0·03)	0·14 (0·26)*	0·12 (0·22)
Personal standards	0·12 (0·24)	0·00 (0·00)	0·07 (0·13)	0·04 (0·07)	0·14 (0·25)*	−0·04 (0·07)	0·04 (0·07)	0·07 (0·13)
Doubt about actions	0·09 (0·18)	0·07 (0·13)	0·07 (0·14)	0·12 (0·23)	0·06 (0·11)	0·09 (0·16)	0·10 (0·17)	−0·01 (0·02)

EEA, Equal Environment Assumption; ES, effect size (Cohen's *d*).
* $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table 2. *Results of univariate model fitting to determine the role of genetic and environmental influences on the phenotypes*

| | Maximum likelihood estimation of correlation | | Goodness-of-fit of model (AIC) Most parsimonious model in bold | | | | Proportion of variance contributed by each parameter – ACE and the most parsimonious model | | |
Phenotype[a] (*n* twin pairs)	MZ (95% CI)	DZ (95% CI)	ACE (df=3)	AE (df=4)	CE (df=4)	E (df=5)	a² (95% CI)	c² (95% CI)	e² (95% CI)
Concern over mistakes (324)	0·42 (0·30–0·52)	0·05 (−0·14 to 0·23)	3·322	**1·322**	11·770	38·316	39 (20–49) 39 (27–49)	0 (0–15) —	61 (51–73) 61 (51–73)
Personal standards (300)	0·37 (0·24–0·49)	0·15 (−0·04 to 0·33)	2·348	**0·348**	6·458	30·487	36 (9–47) 36 (24–47)	0 (0–22) —	64 (54–76) 64 (54–76)
Doubt about actions (301)	0·23 (0·10–0·36)	−0·07 (−0·25 to 0·11)	−0·237	**−2·237**	4·515	13·598	27 (9–38) 27 (14–38)	0 (0–13) —	73 (62–86) 73 (62–86)
Weight and shape concern (348)	0·28 (0·15–0·40)	0·05 (−0·15 to 0·24)	−3·879	**−5·879**	−2·340	9·055	25 (0–36) 25 (14–36)	0 (0–22) —	75 (64–87) 75 (64–87)

AIC, Akaike's Information Criterion; MZ, monozygotic; DZ, dizygotic; CI, confidence interval.
[a] All phenotypes were transformed ($\log_{10} x + 1$) prior to analysis because of positive skew of the data.

Table 3. *Genetic correlations* (*upper diagonal*) *and unique environmental correlations* (*lower diagonal*) *between the perfectionism measures and weight and shape concern* (*95% confidence intervals in parentheses*)

Phenotype	CM	PS	DAA	WSC
Concern over mistakes (CM)	**1·00**	0·47 (0·24–0·70)	0·99 (0·76–1·00)	0·45 (0·18–0·74)
Personal standards (PS)	0·26 (0·13–0·38)	**1·00**	0·49 (0·15–0·78)	0·25 (−0·06 to 0·56)
Doubt about actions (DAA)	0·36 (0·26–0·46)	0·21 (0·09–0·33)	**1·00**	0·53 (0·14–0·89)
Weight and shape concern (WSC)	0·14 (0·02–0·26)	0·08 (−0·05 to 0·21)	0·11 (−0·01 to 0·23)	**1·00**

$p < 0.01$) and DA (CE: $\chi^2 = 4.75$, df $= 1$, $p < 0.05$; E: $\chi^2 = 13.39$, df $= 2$, $p < 0.01$). By contrast, comparison of univariate models for weight and shape measure revealed no significant difference between the full model and the CE model ($\chi^2 = 1.54$, df $= 1$, $p > 0.05$), whereas a significant difference emerged between the full and E only models ($\chi^2 = 12.93$, df $= 2$, $p < 0.01$). Additive genetic estimates ranged from 39% (CM) to 25% (the influence of weight and shape on self-evaluation).

Multivariate twin analyses

First, phenotypic correlations across the four variables were examined. As predicted, all measures were positively and significantly intercorrelated. The weight and shape measure was associated most strongly with CM ($r = 0.32$, $p < 0.001$), followed by DA ($r = 0.28$, $p < 0.001$) and PS ($r = 0.11$, $p = 0.004$). CM and DA were the most strongly associated perfectionism measures ($r = 0.53$, $p < 0.001$), with more moderate correlations between CM and PS ($r = 0.35$, $p < 0.001$), and PS and DA ($r = 0.27$, $p < 0.001$).

Second, results of the Cholesky decomposition are displayed in Table 3. The genetic correlations between the perfectionism measures range from 0·47 to 0·99, while the environmental correlations are somewhat lower, ranging from 0·21 to 0·36. DA and CM share most of their factors in common with 98% of their genetic factors, and 13% of the unique environmental factors. PS shows less overlap of genetic factors, sharing 22% and 24% of it genetic factors with CM and DA respectively, and 7% and 4% of its environmental risk factors with CM and DA respectively. The weight and shape measure shared the highest amount of genetic factors with DA (28%), along with just 1% of the unique environmental factors. The next highest overlap of genetic factors is

with CM (20%), associated with a 2% sharing of environmental factors. The weight and shape measure shared least genetic factors with PS (6%), and 0·6% of shared environmental factors.

Third, the AE model for the CP model was significantly worse fitting than the AE model for the IP model ($\chi^2 = 31.71$, df $= 7$, $p < 0.01$). We further examined the IP model with the standardized variance estimates for both the common and specific pathways for the genetic and environmental variance (Fig. 1). Summing the additive genetic and unique environmental variance for each phenotype gives approximately the same estimates as those obtained from the univariate analyses. The proportion of A for each phenotype was 39% (CM), 37% (PS), 21% (DA) and 26% (weight and shape). Examining the degree to which variance is attributable to specific sources of genetic and environmental variance (i.e. not shared with the other phenotypes), 37% of the variance of CM is not shared with the other phenotypes, compared to 82% for PS, 56% for DA, and 90% for the weight and shape measure.

DISCUSSION

We addressed two questions, namely to what degree do genes and the environment influence perfectionism and the undue influence of body weight or shape on self-evaluation, and to what degree do the same genetic and environmental risk factors influence these phenotypes. We selected three measures of perfectionism as being representative of the core features of a perfectionistic temperament. We identified no violation of the EEA with regard to each of the three perfectionism measures and the weight and shape measure. Hence we are confident that our estimation of genetic action (A) is unlikely
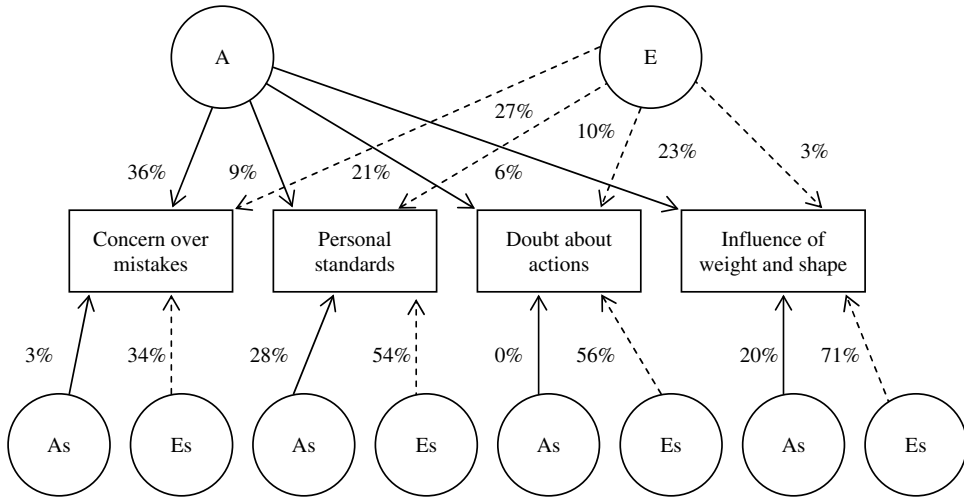
FIG. 1. An AE independent pathways model for one twin showing the percentage of variance contributed to the four phenotypes by each common and specific source of latent phenotype.

to be inflated, and indeed could be underestimated as we only examine one occasion of measurement of each phenotype, which includes error measurement.

We addressed the first question with a series of univariate genetic analyses. Regarding perfectionism, our results show that additive genetic and non-shared environmental influences across all measures of perfectionism confirm previous findings relating to the genetic epidemiology of perfectionism (Kamakura *et al.* 2003). An American sample of similar size using the same measure (Tozzi *et al.* 2004) yielded respective a^2 and e^2 estimates and 95% CI of 29 (0–50) and 60 (49–71) for CM, 42 (22–52) and 58 (49–70) for PS, and 32 (11–43) and 68 (57–80) for DA. These estimates are very similar to the ours, with the exception of CM, where the variance attributed to additive genetic action was reduced because of the inclusion of a small amount of variance for the shared environment. This difference may be a result of the American study using reduced item subscales.

Our univariate results for the weight and shape measure are difficult to compare to previous studies, which have tended to examine general measures of weight or shape concern (Wade *et al.* 1998) rather than the diagnostic phenotype used in the current study. One previous study (Reichborn-Kjennerud *et al.* 2004) used a single-item self-report question ('Is it important for your self-evaluation that you keep a certain weight?') answered with a three-point response scale (highly important, somewhat important, not very important) and found no influence of genetic action, only shared and non-shared environmental influences. The measure used in our study was derived from an interview and showed around a quarter of the variance being accounted for by additive genetic influence, considerably less than that estimated for the behavioural manifestations of eating disorders such as binge eating and vomiting (Sullivan *et al.* 1998), where both are moderately heritable (46% and 72% respectively). The remainder of the influence was accounted for by the non-shared environment, although it should be noted that the CE was not significantly worse fitting than the full model.

The multivariate analyses, including the results from the Cholesky decomposition and the pathways models testing, suggest that CM and DA share most in common with respect to genetic and environmental risk factors, whereas both undue influence of body weight or shape on self-evaluation and PS share little in the way of genetic and environmental risk factors with CM or DA. Although there is some indication of shared risk factors among the perfectionism phenotypes, it is clear that none of these phenotypes is completely influenced by the same set of genetic and environmental factors. This latter finding accords with a previous multivariate study of perfectionism (Tozzi *et al.* 2004)

that showed that the CP model was also significantly worse fitting than the IP model, indicating perfectionism as a multidimensional construct, where the different aspects of this construct are somewhat related but also substantially influenced by independent aetiologies.

The weight and shape measure showed substantial independence from the perfectionism phenotypes, with 90% of the risk factors specific to that phenotype and not shared with perfectionism. Thus it can be concluded that the model best explaining the relationship between weight and shape concern and perfectionism is not likely to be one in which there is a large degree of overlap in the aetiological factors. These results provide little support for the common cause model, and may suggest that therapies for eating disorders focus on both phenotypes, as is the current emphasis in transdiagnostic therapy (Fairburn *et al.* 2003).

It can also be predicted that phenotypic correlations between weight and shape concern and CM and DA will be higher than those between weight and shape concern and PS, as was found in the current study. This could explain why previous studies have shown a cross-sectional association between eating pathology and the CM, but not the PS, subscale (Minarik & Ahrens, 1996; Bulik *et al.* 2003). However, given criticisms of the current measurement of perfectionism constructs (Shafran *et al.* 2002), further research is required to investigate the relationships between various dimensions of perfectionism and both cognitive and behavioural dimensions of eating disorders.

Our findings should be interpreted within the context of five limitations. First, the sample size may have limited power to detect some relationships, thus limiting our ability to detect the contribution of shared environment. Second, we had only one occasion of measurement of perfectionism, thus incorporating measurement error into our non-shared environment estimate. Third, while our measure of the current influence of weight and shape on self-evaluation does serve as an indicator for lifetime disordered eating behaviour, the use of this measure in conjunction with a relatively stable personality trait (i.e. perfectionism) may diminish our power to detect shared genetic and environmental risk factors. Fourth, while this study does not address the issue of shared risk

with women who have current eating disorders, it can be used to address what appear to be enduring attitudes and traits that are likely to have been important in the development of eating disorders. Fifth, we had a less than optimal response rate for our Wave 1 and Wave 3 data, and it is uncertain how sample attrition may affect the covariation between weight and shape concern and the perfectionism phenotypes, but ascertainment bias has previously been shown to have negligible effect on estimates of disordered eating (Wade *et al.* 1999).

In conclusion, although our results cannot be taken as evidence for or against the suggestion that perfectionism is a risk factor for the undue influence of body weight or shape on self-evaluation, they do suggest that the observed relationship is not consistent with a common cause model. Future research will need to characterize the nature of that relationship more conclusively. Nonetheless, overvaluation of shape and weight and their control, which has been identified as a risk factor for the development of eating disorders, is a relatively independent construct from perfectionism, and as such, perfectionism cannot be seen to represent an endophenotype of weight and shape concern.

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