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The Structure of Perfectionism: A Twin Study

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Background: Perfectionism may be a premorbid risk factor for eating disorders. Evidence of familial transmission suggests features of perfectionism may be genetically determined. This study examines the structure of perfectionism using classical twin design models. *Methods*: Independent (IP) and common (CP) pathway models are used to investigate the extent to which genetic and environmental factors can help to identify and differentiate three behavioral domains of Perfectionism as measured by a shortened version of the Multidimensional Perfectionism Scale (MPS) [Frost *et al.* (1990). Cognit. Ther. Res. **14:** 449–468]. Three of the original subscales were included: Personal standards (PS), Doubts about actions (DA), Concern over mistakes (CM). We studied a sample of 1022 paired and unpaired female twins from the Mid-Atlantic Twin Registry. *Results*: MZ correlations were consistently higher than DZ twin correlations for all three composite subscales. The multivariate independent pathway model provided a better fit to the twin correlations then did the more parsimonious common pathway model suggesting the pattern of familial resemblance for the three subscales is not well characterized by a unidimensional perfectionism factor. CM phenotypic variance was completely accounted for by common heritability influences in both the IP and CP models. Based on the IP model results, there was evidence that PS and CM but not DA shared some common genetic effects, with DA and CM sharing some common environmental factors. *Conclusions*: These multivariate twin modeling results support conceptualizations of perfectionism as a multidimensional construct. The biometric structural results for the three subscales examined here suggest CM is the core feature of Perfectionism with DA and PS serving as indicators of CM. Although not the best fitting model, the common pathway model estimated this behavioral domain to be isomorphic with the construct of perfectionism. The better fitting independent pathway model provided evidence of non-trivial differences in the pattern of heritability for CM, DA, and PS.

KEY WORDS: Dimensionality; eating disorders; heritability; perfectionism; twin models.

INTRODUCTION

> "One of the most important distinctions between the efforts of the true masters of their craft and those of the perfectionistic person is that the striving of the first group brings them solid satisfaction. They are happy with the results. Their efforts enhance their self-esteem. They rejoice in their mastery. This is not true for the perfectionistic person. His striving is accompanied by the corrosive feeling that "I am not good enough. I must do better." (Missildine, 1963)

Perfectionism has been recognized as a debilitating problem (Pacht, 1984) that is associated with several psychiatric disorders and symptoms such as anxiety disorders (Antony *et al.*, 1998; Frost and Steketee, 1997), eating disorders (Bastiani *et al.*, 1995; Bulik *et al.*, 2003; Halmi *et al.*, 2000; Lilenfeld *et al.*, 2000; Stein *et al.*, 2002; Sutandar-Pinnock *et al*, 2003; Woodside *et al.*, 2002), depression

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and suicidal ideation (Hamilton and Schweitzer, 2000; Hewitt and Flett, 1991a; Hewitt *et al.*, 1996, 1997). The association between perfectionism and eating disorders (EDs) appears to be particularly robust (Bulik *et al.*, 2003). A growing body of literature suggests that perfectionism may be a premorbid risk factor for EDs. It has been shown to predate the onset of eating disorders (Fairburn *et al.*, 1999), characterize the acute phase of the illness (Halmi *et al.*, 2000; Lilenfeld *et al.*, 2000) and persist after recovery (Bastiani *et al.*, 1995; Kaye *et al.*, 1998; Srinivasagam *et al.*, 1995; Sullivan *et al.*, 1998; Sutandar-Pinnock *et al.*, 2003). Moreover, perfectionism appears to run in families (Lilenfeld *et al.*, 2000; Woodside *et al.*, 2002).

Anorexia and bulimia nervosa are complex illnesses influenced by genetic and environmental factors. Identifying traits that relate to anorexia and bulimia is a first step in the attempt to uncover their etiologies. Investigating genetic and environmental influences on such traits will allow us to identify potential endophenotypes for genetic studies of eating disorders. Perfectionism being a core feature of individuals with eating disorders, constitutes a candidate trait. However, no studies have investigated the extent to which this trait is influenced by genetic factors. Moreover, investigating the role of perfectionism in psychopathology is particularly complex because of its loose definition. Different definitions and measures of this construct have been used in the literature, and several questions about its structure and definition remain (Burns, 1980; Frost *et al.*, 1990: Hewitt and Flett, 1991b; Hewitt *et al.*, 2003; Rheaume *et al.*, 1995; Shafran *et al.,* 2003).

There are three problematic areas in the definition of perfectionism: (1) What are the core features of perfectionism? (2) Is perfectionism best conceptualized as including both healthy and pathological features? (3) Should perfectionism be defined as a unitary construct or it is multidimensional?

Since the 1970s (Hamachek, 1978; Hollender, 1965), perfectionism has received substantial clinical and research attention, and varying definitions of perfectionism have been proposed. Only few relevant aspects will be presented here; however, extensive reviews of the concept can be found in (Frost *et al.*, 1990; Hewitt *et al.*, 1991b, 2003; Shafran and Mansell, 2001).

Originally, perfectionism was viewed as a unitary construct (Burns, 1980; Garner *et al.*, 1983; Pacht, 1984). The unidimensional definition of perfectionism focused exclusively on self-directed cognitions, and included aspects such as setting unrealistic standards, selective attention to failure, all or nothing thinking (total success *versus* total failure), and "the belief that a perfect state exists that one should try to attain" (Burns, 1980; Hamachek, 1978; Hollender, 1965; Pacht, 1984). Further developments conceptualized perfectionism in a more complex dimensional structure: it was no longer considered as only having a unidimensional self-directed nature, and additional aspects were included as core features of the construct (Frost *et al.*, 1990; Hewitt *et al.*, 1991b). Yet, recent critics have argued that the multidimesionalization of perfectionism may have been ill-advised as it merges the concept of perfectionism with other features peripherally related to it, such as the interpersonal component of perfectionism ("other-oriented" and "socially-prescribed" perfectionism in the Hewitt scale, and "parental criticism" and "parental expectations" in the Frost scale) (Shafran *et al.*, 2001, 2002, 2003). Structural analyses, such as those conducted in this study, could help clarify this issue by providing empirical evidence regarding the dimensionality of perfectionism.

Another controversy in the perfectionism literature involves its association with psychopathology. Hamachek (1978) first described the dual nature of perfectionism: healthy striving for high standards *versus* "neurotic" perfectionism. He viewed "neurotic" perfectionists as individuals who "never seem good enough, at least in their own eyes...They are unable to feel satisfaction because in their own eyes they never seem to do things well enough to warrant that feeling, (p. 27)". In contrast, he viewed healthy perfectionists as individuals who set high goals, are able to enjoy their successes, and are able to accept less-than-perfect performances. Several authors (Enns *et al.*, 2001; Frost *et al.*, 1990; Hamachek, 1978; Slade and Dewey, 1986; Terry-Short *et al.*, 1995) concur that the trait of perfectionism comprises both negative (maladaptive or pathological) and positive (adaptive or sound) facets, although their definitions and measures of these facets diverge (Burns, 2001; Frost *et al.*, 1990; Garner *et al.*, 1983; Hewitt *et al.*, 1991b; Slade and Dewey, 1986). To support the hypothesis of a pathological and an adaptive facet of perfectionism, several studies showed that only select dimensions are strongly associated with psychopathology (Antony *et al.*, 1998; Bulik *et al.*,

2003; Coles *et al*., 2003), in contrast other facets are not (Antony *et al*., 1998; Bulik *et al*., 2003; Frost *et al*., 1990). Shafran *et al*. (Frost *et al*., 1990; Hewitt *et al*., 1991b; Shafran *et al*., 2002) claim that viewing perfectionism as having both healthy and pathological facets is problematic and advocate reserving the term *perfectionism* for the pathological features. They note that the current definition of perfectionism is erroneously based on the two most widely used multidimensional scales (Frost *et al*., 1990; Hewitt *et al*., 1991b), rather than on theoretical or clinical evidence. They also defined a concept of "clinical perfectionism" to distinguish a "circumscribed clinical construct" (Shafran *et al*., 2003) from a "general personality orientation" [cited in Shafran *et al*., 2003 from (Flett and Hewitt, 2002)]. This concept is defined as "the overdependence of self-evaluation on the determined pursuit of personally demanding, self-imposed, standards in at least one highly salient domain, despite adverse consequences" (Shafran *et al*., 2002).

The definition of perfectionism used in this study is the one proposed by Frost *et al*. (1990). Frost and colleagues (Frost *et al*.. 1990; Slade and Dewey, 1986) underscored that the central aspect of perfectionism that differentiates perfectionists from high achievers is a tendency to be overcritical of one's own performance. Referring to Hamachek (1978), they noted that perfectionistic individuals are driven by fear of failure rather than a drive to achieve. They also viewed negative self-evaluation and a need for external approval as integral to the construct of perfectionism. Excessive concern over making mistakes is also a central characteristic of perfectionism, which has been related to psychopathological symptoms (Frost *et al*., 1990).

In sum, as perfectionism has become a trait of critical interest in eating disorders research, the need for a clarification of its dimensionality and an accurate definition of the construct is critical. The lack of clear agreement on its definition and the use of different measures (based on different conceptualizations of the construct), has contributed to the current controversy and lack of consistency in findings.

The present study aims to investigate the structure of perfectionism, as measured by the MPS (Frost *et al*., 1990), and explore the contribution of genetic and environmental factors to liability to perfectionism. This study represents the first twin study on perfectionism and an attempt to contribute to the further refinement of the construct.

METHODS

Participants

Twins in this study derive from two interrelated projects utilizing the population-based Virginia twin registry (Kendler and Prescott, 1999), which now constitutes part of the Mid-Atlantic Twin Registry. *Female–female* twin pairs, born between the years of 1934–1974, were eligible if both members had previously responded to a mailed questionnaire, the response rate to which was $\sim$64%. They have been approached for four waves of personal interviews from 1988 to 1997. In late 1999, we mailed questionnaires to all prior participants in these two studies ($n = 7230$). Only modest resources were available for follow-up, which was largely limited to phone calls to non-responding twins whose co-twin had responded. We received a total of 2616 questionnaires, representing 36.2% individual response rate. The present study focuses only *on female–female twins* ($N = 1022$), for two reasons: first, data on perfectionism as a risk factor for eating disorders are from female samples; second, there are no data available about sex differences in perfectionism. The sample was composed of 246 MZ female twin pairs and 122 MZ female unpaired twins, 158 DZ female twin pairs and 92 DZ female unpaired twins.

Measure

The original MPS includes six subscales: personal standards (PS), doubts about actions (DA), concern over mistakes (CM), and organization (O), and two parental subscales [parental criticism (PC) and parental expectations (PE)]. Based on prior MPS research and communication with the scale developers, we included items that focused on individual (rather than parental) characteristics. We chose four items each from the CM and PS subscales that had shown reliable factor loadings in previous investigations (Frost *et al*., 1990; Rheume *et al*., 1995) (the original DA subscale only includes four items). Items were not included from the O subscale, as this scale is not believed to capture a

core component of perfectionism (Frost *et al.*, 1990). (See Table I).

The PS subscale contains items referring to the setting of high standards and the importance placed on these standards for self-evaluation. This scale has been associated with positive achievement striving (Frost *et al.*, 1990). The DA subscale refers to the tendency to doubt about the ability to accomplish tasks. Finally, the CM subscale contains items referring to the negative reaction to mistakes and the tendency to interpret mistakes as equivalent of failure (Frost *et al.*, 1990).

Statistical Analyses

Analyses were carried out in several steps. First, a confirmatory factor analysis was performed to examine whether the subset of MPS items selected maintained the structure of the original full scale. Given the categorical nature of the data and their non-normal distribution, factor analyses were performed using the MPLUS software and the weighted least squares estimator with robust standard errors and chi-square fit statistics (Muthen and Muthen, 1998). Factor analysis showed that items selected from the original questionnaire followed the structure proposed by Frost (data not presented here). For all further statistical analysis, composite scores were created as the sum of the individual items for each MPS subscales. A structural equation modeling (SEM) approach is used to analyze the twin data (Eaves, 1977; Heath *et al.*, 1989; Neale and Cardon, 1992).

Univariate and two multivariate (independent and common pathway) twin model analyses were performed. The independent pathway model provides a relatively saturated model for the investigation of the covariance structure of each of the three components of variance (additive genetic, shared environment, and specific environment). This model is just identified when there are three scales being modeled per twin, as in this analysis. The second model, the common pathway model, is a natural extension of a simple phenotypic factor analysis model, in which variation in a single common factor is partitioned into three higher order factors (A, C and E). This model uses fewer parameters and therefore, is more parsimonious if it fits as well as the independent pathway model. If the common pathway fits as well as the independent pathway model, it is to be preferred on account of its relative simplicity (three fewer parameters).

Table I. Multidimensional Perfectionism Scale (Frost *et al.*, 1990). Subscales and Items

Multidimensional Perfectionism Scale (MPS) Frost *et al.* (1990)
CM: Negative reactions to mistakes, tendency to interpret mistakes as equivalent to failure, tendency to believe that one will lose the respect of others following failure
If I fail at work/school, I am a failure as a person
I should be upset if I make a mistake
If someone does a task at work/school better than me, then I feel like I failed the whole task
If I fail partly, it is as bad as a complete failure
I hate being less than the best at things
People will probably think less of me if I make a mistake
If I do not do as well as other people, it means I am an inferior human being
If I do not do well all the time, people will not respect me
The fewer mistakes I make, the more people will like me
PS: Setting of very high standards and the excessive importance placed on these high standards for self-evaluation
If I do not set the highest standards for myself, I am likely to end up a second rate person
It is important to me that I be thoroughly competent in everything I do
I set higher goals for myself than most people
I am very good at focusing my efforts on attaining a goal
I have extremely high goals
Other people seem to accept lower standards from themselves than I do
I expect higher performance in my daily tasks than most people
DA: Doubt their ability to accomplish tasks
Even when I do something very carefully, I often feel that is not quite done right
I usually have doubts about the simple everyday things I do
I tend to get behind in my work because I repeat things over and over
It takes me a long time to do something "right"
PE: Tendency to believe that one's parents set very high goals
My parents set very high standards for me
My parents wanted me to be the best at everything
Only outstanding performance is good enough in my family
My parents have expected excellence from me
My parents have always had higher expectations for my future than I have
PC: Perception that one's parents are/were overly critical
As a child, I was punished for doing thing less than perfectly
My parents never tried to understand my mistakes
I never felt like I could meet parents' expectations
I never felt like I could meet my parents' standards
O: Tendency to be organized, emphasis on order and orderliness
Organization is very important to me
I am a neat person
I try to be an organized person
I try to be a neat person
Neatness is very important to me
I am an organized person

Note: Items in bold are included in our questionnaire. CM: Concern over mistakes; PS: Personal standards; DA: Doubts about actions; PE: parental expectations; PC: Parental criticism; O: Organization.

For all analyses, models were fit to raw ordinal data for paired and unpaired twins. The Mx statistical program was used to fit the twin models (Neale, 1997).

The multivariate twin models were fit to investigate the pattern of covariation among the three subscales and their relation to the construct of perfectionism. The initial model fit was the independent pathway model. It specifies a structure for the subscale relationships allowing for separate A, C, and E decompositions of each observed subscale. To investigate whether the three subscales define a single construct of perfectionism, a common pathway model is fit. This model assumes that all three subscales are indicators of a single latent (unobserved) trait. This is a more restricted model than the independent pathway model providing a testable hypothesis about the structure of the three perfectionism subscales. The relative effect of the latent construct on the observed subscales is indicated by the absolute value of the factor loadings. In addition to the A, C, and E decomposition of the latent trait. A, C, E parameters are also estimated for the unique (specific plus error) variance for each subscale. For a more formal and detailed discussion of the IP and CP models see (McArdle and Goldsmith, 1990).

RESULTS

Representativeness of the Sample

To examine possible sample selection issues, we predicted participation in the female-female twin sample using major demographic variables. Participation was not significantly predicted by age, zygosity, or financial status. Only years of education predicted survey participation (odds ratio (OR) = 1.23 [per year], $\chi^2 = 16.62$, df = 1, $p < 0.0001$). Predictors of response in this sample (female-female only) differed from samples where males and females were both included (Kendler *et al*., 2003). However, we note that educational level in general has been found to be a common predictor of participation in twin studies (Lykken *et al*., 1978, 1987).

Polychoric Correlations

All three subscales showed higher MZ than DZ correlations. Polychoric correlations for PS were 0.40 (MZ) (CIs: 0.30–0.52) and 0.21 (DZ) (CIs: 0.15–0.28), for CM 0.38 (MZ) (CIs: 0.27–0.49) and 0.27 (DZ) (CIs: 0.16–0.42) and for DA 0.30 (MZ) (0.20–0.43) and 0.18 (DZ) (CIs: 0.10–0.24).

Table II. Univariate Twin Model Results

	a^2	c^2	e^2
PS	0.42	0	0.58
95% CIs	0.22–0.52	0–0.15	0.49–0.70
DA	0.32	0	0.68
95% CIs	0.11–0.43	0–0.16	0.57–0.80
CM	0.29	0.19	0.60
95% CIs	0–0.50	0–0.40	0.49–0.71

Note: PS: Personal standards; DA: Doubts about actions; CM: Concern over mistakes; a^2: proportion of variance attributed to additive genetic influences: c^2: proportion of variance for shared environment; e^2: proportion of variance for nonshared environment and error; CIs: 95% confidence intervals.

Univariate Twin Model

Table II presents results for fitting separate univariate ACE models to each subscale. Although nested models were examined (i.e., AE and CE restricted versions), only full ACE model results are reported. Statistical precision precluded selecting among restricted model fits. Statistical power for univariate twin analyses can become an issue in cases where effect sizes, sample sizes, or trait prevalence are low (Neale *et al*., 1994). Also, reliance on the use of global fit indices such as Akiake's information criterion to select among nested models can be problematic (Sullivan and Eaves, 2002).

A few univariate results are noted. The point estimate for the proportion of variance accounted for by additive genetic effects was the largest for PS. However, the upper confidence intervals were inclusive for all three a^2 subscale estimates. Only the a^2 for CM was not statistically different from zero. CM was the only subscale for which a shared environmental effect was detected, although the 95% CI was not statistically different from zero. All unique environmental proportion of variance estimates (e^2) for the three subscales were over 0.5 and statistically significant.

Independent Pathway Model

The independent pathway (IP) model simultaneously considers the covariation among the

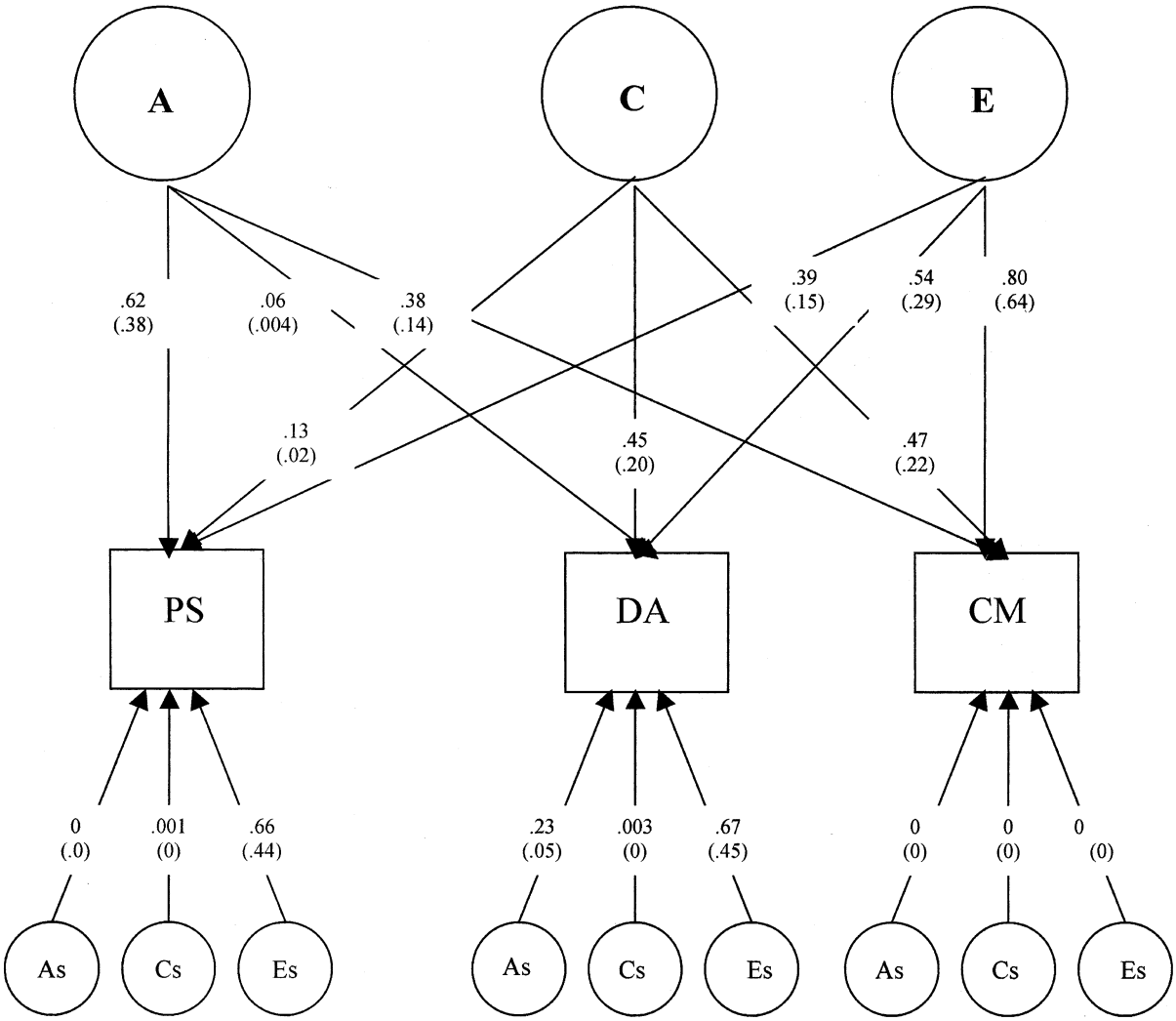

Fig. 1. Path diagram of Independent Pathway Model. PS:Personal standards, DA:Doubts about actions, CM:Concern over mistakes, A:additive genetic, C:shared environment, E:nonshared environment, As:residual specific additive genetic, Cs:residual specific shared environment, ES:residual specific nonshared environment. (): common and unique A, C, and E proportions of variance. Total variance constrained to be 1.

subscales when optimally estimating parameters for the different sources of familial resemblance (i.e., genetic and environmental influences). When fitting models to ordinal data using a threshold approach, a constraint on the total latent biometric variance is needed. Here we constrain the independent a^2, c^2, and e^2 proportions to equal 1. The IP model and standardized parameter estimates for common and specific genetic and environmental influences are shown in Figure 1. Table Ia of the Appendix gives the 95% confidence intervals for these path coefficients. Of the three common additive genetic effects, PS was the only effect statistically different from zero. DA did not share much common additive genetic effects with PS or CM. There is evidence that DA and CM have some common shared environmental effects (significant path coefficient estimates of 0.45 and 0.47, respectively) but not PS. All three subscales had significant common unique environmental effects with CM being the largest. One rather striking result coming from the IP model is that the CM subscale was entirely accounted for by the common A, C, and E latent sources as indicated by the zero estimates for all ACE CM uniqueness estimates.

Table III expresses the parameter estimates of the IP model as sources of covariance. For example,

Table III. Independent Pathway Model Results Expressed as Components of Subscale Covariance

	Independent path (relative influence)			
	Phenotypic	A	C	E
PS/DA	0.31	0.04 (0.13)	0.06 (0.19)	0.21 (.68)
95% CIs		0–0.78	0–0.64	0.48–0.94
PS/CM	0.61	0.24 (0.39)	0.06 (0.10)	0.31 (0.51)
95% CIs		0.28–0.65	0–0.44	0.40–0.67
DA/CM	0.66	0.02 (0.03)	0.21 (0.32)	0.43 (0.65)
95% CIs		0–0.34	0.07–0.47	0.52–0.77

Note: PS/DA: covariance between Personal standards and Doubts about actions; PS/CM: covariance between Personal standards and Concern over mistakes; DA/CM: covariance between Doubts about actions and Concern over mistakes; A: additive genetic effect; C: shared environmental effect; E: nonshared environmental effect; CIs: 95% confidence intervals are for proportions of covariance. (): proportion of covariance attributed to respective A, C, and E effects.

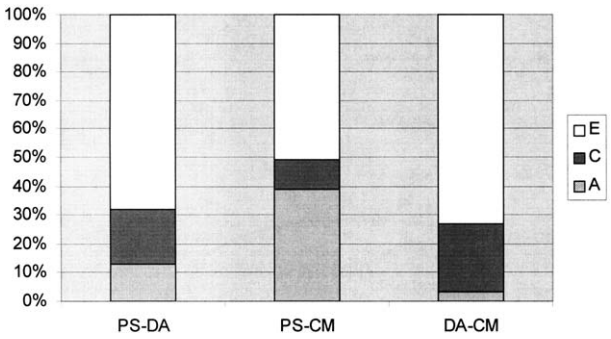

Fig. 2. Graphical representation of relative influence of genes and environment on covariance. PS–DA: covariance between Personal standards and Doubts about actions, PS–CM: covariance between Personal standards and Concern over mistakes, DA–CM: covariance between Doubts about actions and Concern over mistakes, A: additive genetic, C: shared environment, E: nonshared environment.

the phenotypic polychoric correlation of 0.61 between PS and CM was decomposed by the twin IP model into 39% due to additive genetic and 51% to unshared environmental influences—both statistically significant. The A estimate of 0.24 is that portion of the 0.61 correlation attributed to additive genetic effects. The 0.24 value is obtained by taking the product of all possible structural pathways connecting PS and CM in the IP model in Figure 1 (i.e., $0.24 = 0.62 \times 0.38$). Of the correlations across the three subscales, all had significant contributions from unshared environmental sources. Only the correlation between DA and CM showed a significant contribution from shared environment. It is important to note that the composite variable correlations between CM and the other two subscales PS and DA were both greater than 0.6 (item factor analyses estimated these correlations to be even higher, >0.75) whereas the correlation between PS and DA was $\sim$0.3. The high correlations of CM with DA and PS appear to be a prominent feature contributing to the twin modeling results and their departure from those obtained in the univariate models. Figure 2 graphically summarizes these IP components of covariance findings.

Common Pathway Model

Although the common pathway (CP) model structure is quite different from the IP model (it introduces a latent variable), it can be formally tested as a nested model. In the CP model, the three subscales are hypothesized to be indicators of a single latent phenotype which is further decomposed into A, C, and E sources. Figure 3 displays the CP model and estimates of the structural parameters. Table IIa of the Appendix provides confidence intervals for the path coefficients. For the latent perfectionism construct defined by PS, DA, and CM, 61% of its variance was attributed to unshared environmental factors, 28% to additive genetic sources, and 11% to shared environment. The CM factor loading was estimated to be 1 indicating this variable is isomorphic with the latent perfectionism construct. PS and DA loadings were proportional to their correlations with CM suggesting these subscales serve as proxy indicators of CM. As was the case with the IP, all of the CM variance was accounted for by the common A, C, and E effects with the same pattern of all A, C, and E uniqueness estimates being zero. The dominance of CM in the CP model appears to be altering the PS and DA heritability patterns from those obtained in the univariate analyses.

Table IV presents the corresponding CP path coefficient estimates expressed as sources of covariance. How these portions of covariance are arrived at in the CP model can help to distinguish this parameterization from the IP model. Consider the A effect for the PS/CM covariance. The A effect value of 0.17 is obtained by taking the product of: (1) the factor loading for PS (2) twice the common additive genetic effect, and (3) the factor loading for CM (i.e., $0.17 = 0.6 \times 0 \ .53 \times 0.53 \times 1$). This highlights a key difference between the CP and IP parameterizations and the expected covariances they imply. In the IP

expectations, separate A, C, and E effects for each of the observed subscales are involved whereas under the CP model the same A, C, and E values are used. For the CP model, the A and C effects were not statistically different from zero whereas all three E effects were. There are two main differences between these CP model results and the IP findings. Although both models show significant E effects for all three covariances, only the IP estimates showed significant effects for A in the PS/CM association and C for the DA/CM association.

We examine the relative fits of the IP and CP models next.

Model Comparisons

A formal comparison of the IP and CP models can be used to evaluate the dimensionality of the perfectionism construct defined by the three subscales. The raw −2 log likelihood for the full ACE IP model was $-211 = 13{,}898.8$ on $df = 2919$. An AE IP submodel was also fit which produced a $-211 = 13{,}922.7$ on $df = 2925$. The difference between these two raw likelihoods is asymptotically distributed as a chi-square variate based on the difference in degrees of freedom between the two models. Dropping the shared environmental effects resulted in a significant deterioration of fit ($\Delta\chi^2 = 23.9$, $\Delta df = 6$, $p < 0.001$). Thus, the full ACE IP model was retained to test the CP model. The fit for the full ACE CP model was $-211 = 13{,}929.5$ on $df = 2922$. Comparing the fit of the CP model to the IP model produced a significant chi-square difference test ($\Delta\chi^2 = 30.7$, $\Delta df = 3$, $p < .0001$). The more parsimonious ACE CP model with its biometric decomposition of a single latent factor does a poorer job of reproducing the pattern of across twin across subscale correlations than does the IP model.

DISCUSSION

The relevance of perfectionism as a psychopathological factor involved in several psychiatric disorders, and especially its prominent role in eating disorders, led to the current interest in and the intense debate about the definition of the construct (Hewitt *et al.*, 2003; Shafran *et al.*. 2003). This study addresses questions about the dimensionality of perfectionism by examining its structure using a subset of the Frost perfectionism scale (Frost *et al.*, 1990).

Previous findings about the relations among the MPS subscales were based primarily on correlations and more traditional factor analysis. We utilized genetically informative twin data to the covariation among the subscales. Estimates of genetic and environmental influences on this trait were used to help clarify the structural organization of perfectionism in a sample of female twins.

The overall pattern of results suggests that perfectionism, as assessed by the three Frost subscales (Frost *et al.*, 1990), is moderately heritable. However, not all facets of perfectionism appear to be equally heritable. Additive genetic factors appear to contribute more strongly to PS. CM appears to be the driving component of the construct: CM is highly correlated with both PS and DA, and seems to behave as a bridge between the other two dimensions. Indeed, the CM variable appears to be an exceedingly good measurement of the latent factor, with PS and DA serving as proxy indicators of CM. Therefore, a succinct summary of the model is that CM represents the core of perfectionism, which DA and PS measure imperfectly.

The PS subscale has been defined as a functional and socially advantageous drive to achieve high standards (Frost *et al.*, 1990). Our data confirm this definition of the PS dimension. Unlike DA and CM (both of which are negatively skewed in this sample), PS appears to be a feature approaching normal distribution in the population (data not shown). Moreover, in a previous investigation we found that whereas CM and DA uniquely predicted psychopathology in the forms of anorexia and bulimia nervosa, PS was unrelated to any diagnostic category (Bulik *et al.*, 2003). However, these analyses also suggest that the favorable aspect of perfectionism (measured by the PS subscale in Frost's MPS) may not be independent from the pathological facet. The correlation between PS and CM is high and has a significant contribution from common genetic factors. Much different is the nature of CM and DA correlation that appears to be significantly influenced by common shared environmental factors. These results suggest that the pattern of interactions across different aspects of perfectionism, as measured by the Frost scale, is complex, and that the pattern of familial resemblance for the three subscales is not well characterized by a unidimensional perfectionism factor.

In light of these findings, we may argue that using a "total" perfectionism score (sum of all subscales) may be misleading, as various aspects of

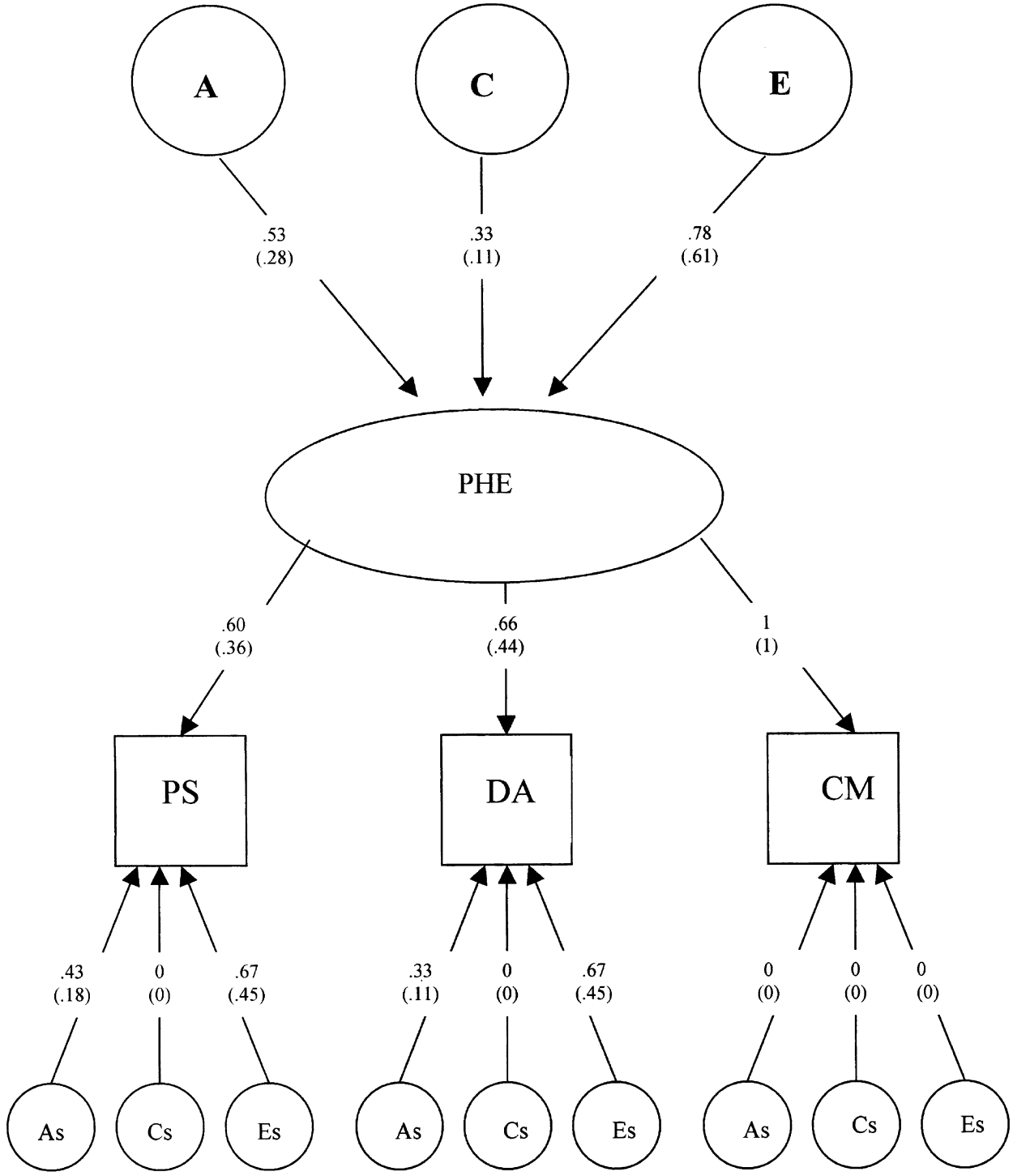

Fig. 3. Path diagram of a Common Pathway Model. PS:Personal standards, DA:Doubts about actions, CM:Concern over mistakes, A:additive genetic, C = shared environment; E = nonshared environment, AS:residual specific additive genetic, Cs:residual specific shared environment, Es:residual specific nonshared environment, PHE:latent perfectionism phenotype. ():common and unique A, C, and E proportions of variance. Total variance constrained to be 1.

Table IV. Common pathway model results expressed as components of subscale covariance

	Common pathway (relative influence)			
	Phenotypic	A	C	E
PS/DA	0.40	0.11 (0.29)	0.04 (0.11)	0.24 (0.61)
95% CIs		0–0.49	0–0.40	0.51–0.72
PS/CM	0.60	0.17 (0.29)	0.07 (0.11)	0.37 (0.61)
95% CIs		0–0.49	0–0.40	0.51–0.72
DA/CM	0.66	0.19 (0.29)	0.07 (0.11)	0.40 (0.61)
95% CIs		0–0.49	0–0.40	0.51–0.72

Note: PS/DA: covariance between Personal standards and Doubts about actions; PS/CM: covariance between Personal standards and Concern over mistakes; DA/CM: covariance between Doubts about actions and Concern over mistakes; A: additive genetic effect; C: shared environmental effect; E: unshared environmental effect; CIs: 95% confidence intervals are for proportions of covariance; (): proportion of covariance attributed to respective A, C, and E effects.

the construct are only partially overlapping and may reflect different genetic and environmental etiological factors. Although we were not able to address this question using the abbreviated scale, this problem is likely to be greater when the full scale is used which includes parental aspects of perfectionism and organization. This may partially explain contradictory results regarding the familial basis of perfectionism (Devlin *et al*., 2002; Lilenfeld *et al*., 2000; Woodside *et al*., 2002). Previous studies did not identify perfectionism as a valuable covariate in linkage studies of anorexia (Devlin *et al*., 2002). However, in Devlin *et al*., study (2002), a total score of perfectionism was used. The use of a perfectionism total score, which includes interpersonal and intrapersonal aspects of the construct, may be inappropriate because the intrapersonal dimensions of perfectionism show different patterns of genetic and environmental influences and because the interpersonal dimension is of less genetic interest (i.e., parental criticism assesses the respondent's perspective on his/her parents' attitudes).

These results underscore the importance of further investigations into the nature of this phenotype to validate empirically the dimensionality of perfectionism. Further refinement of the trait is particularly relevant for genetic studies. Given that premorbid perfectionism is commonly reported in individuals with anorexia nervosa (Fairburn *et al*., 1999) and it persists after recovery (Srinivasagam *et al*., 1995), it is a prime candidate as an endophenotype or personality covariate for genetic studies.

The strengths of this study include large sample size and the use of a genetically informative sample. To our knowledge, this is the first study to explore the relative contributions of environmental and genetic factors to understand the dimensionality and etiology of perfectionism. Nevertheless, results of this study must be interpreted within the context of several methodological limitations. First, our sample was composed of Caucasian females and the extent to which our results generalize to other groups is unknown. Second, the interpretation of these results is limited to the behavioral domains as defined by the subset of items taken from the Frost scale. Although factor analysis supported the original subscale structure, our instrument did differ from the original full scale. Finally, statistical power may have contributed to our ability to choose the best fitting model from among nested models in the univariate twin analysis.

In summary, the results indicate that a single factor does not satisfactorily account for the familial pattern of correlations for the three purported behavioral domains of perfectionism. The PS, DA, and CM dimensions of perfectionism are partially heritable and differentially linked one to another, which add support to a multidimensional structural interpretation of the construct.

Further iterative process between clinicians and geneticists, and examination of the relation between perfectionism and other personality features (such as obsessionality and self-esteem) will lead to a better understanding of the nature of the construct.

Given the evidence found in this study for differential heritability influences on the components of perfectionism and the established link between perfectionism and eating disorders, the intrapersonal dimensions of perfectionism may become a more central focus of future studies investigating the genetics of eating disorders.

APPENDIX A

Table Ia. Independent Pathway Model Path Coefficient Estimates

	Independent path					
	Common			Specific		
	A	C	E	A	C	E
PS	0.62	0.13	0.39	−0.03	0.001	0.66
95% CIs	0.61–0.71	0–0.45	0.31–0.40	0–0.36	0–0.26	0.61–0.72
DA	0.06	0.45	0.54	0.23	0.003	0.67
95% CIs	0–0.36	0.44–0.54	0.46–0.62	0–0.36	0–0.26	0.61–0.73
CM	0.38	0.47	0.80	0	−0.001	0
95% CIs	0–0.62	0.14–0.63	0.73–0.86	0–0.2	0–0.22	0–0.27

Note: PS: Personal standards; DA: Doubts about actions; CM: Concern over mistakes; A: additive genetic effect; C: shared environmental effect; E: nonshared environmental effect; CIs: 95% confidence intervals for path coefficient point estimates.

Table IIa. Common Pathway Model Path Coefficient Estimates

	Phenotypic					Specific		
	A	C	E	Common pathway		A	C	E
PHE	0.53	0.33	0.78	PS	0.60	0.43	0	0.67
CIs	0–0.70	0–0.63	0.72–0.85	95% CIs	0.56–0.64	0.19–0.52	0–0.33	0.61–0.74
				DA	0.66	0.33	0	0.67
				95% CIs	0.62–0.70	0–0.43	0–0.30	0.61–0.73
				CM	1	0	0	0
				95% CIS	0.99–1	0–0.12	0–0.11	0–0.15

Note: PS: Personal standards; DA: Doubts about actions; CM: Concern over mistakes; A: additive genetic component; C: shared environmental component; E: nonshared environmental component; PHE: latent phenotype; CIs: 95% confidence intervals for path coefficient point estimates.

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