Behavior Genetics, Vol. 33, No. 4, July 2003 (© 2003)

Direction of Causation Modeling Between Cross-Sectional Measures of Parenting and Psychological Distress in Female Twins

Nathan A. Gillespie,[1,4] Gu Zhu,[1] Michael C. Neale,[2] Andrew C. Heath,[3] and Nicolas G. Martin[1]

Received 24 Apr. 2001—Final 27 Jan. 2003

Under certain conditions, cross-sectional analysis of cross-twin intertrait correlations can provide important information about the direction of causation (DOC) between two variables. A community-based sample of Australian female twins aged 18 to 45 years was mailed an extensive Health and Lifestyle Questionnaire (HLQ) that covered a wide range of personality and behavioral measures. Included were self-report measures of recent psychological distress and perceived childhood environment (PBI). Factor analysis of the PBI yielded three interpretable dimensions: Coldness, Overprotection, and Autonomy. Univariate analysis revealed that parental Overprotection and Autonomy were best explained by additive genetic, shared, and nonshared environmental effects (ACE), whereas the best-fitting model for PBI Coldness and the three measures of psychological distress (Depression, Phobic Anxiety, and Somatic Distress) included only additive genetic and nonshared environmental effects (AE). A common pathway model best explained the covariation between (1) the three PBI dimensions and (2) the three measures of psychological distress. DOC modeling between latent constructs of parenting and psychological distress revealed that a model which specified recollected parental behavior as the cause of psychological distress provided a better fit than a model which specified psychological distress as the cause of recollected parental behavior. Power analyses and limitations of the findings are discussed.

KEY WORDS: Direction of causation (DOC) modeling; PBI; parenting; depression; anxiety; somatic distress; twins; genes.

INTRODUCTION

Does the quality of parenting influence the likelihood of developing psychiatric disorders in adult life? Bowlby has argued that maladaptive behavior including mood and anxiety disorders can be attributed to deviations in the development of attachment behavior and patterns of parental bonding (Bowlby, 1977). In order to identify and quantify the dimensions of parenting that may contribute to an increased liability to psychiatric disorders, Parker designed the Parental Bonding Instrument (PBI), which measures recollections of parental behavior along the dimensions of Care and Overprotection (Parker *et al.,* 1979).

The PBI has good internal reliability for both the long and short versions (Todd *et al.,* 1994). The measure is also stable over time and unaffected by the age, social class, or sex of the respondent (Parker, 1989). In terms of concurrent validity, there is a modest correlation between mothers' self-reports and offspring ratings of maternal Care (0.44) and also between mothers'

[1] Queensland Institute of Medical Research, Brisbane, Australia.
[2] Department of Psychiatry, Virginia Institute for Psychiatric and Behavioral Genetics, Richmond, Virginia, USA.
[3] Missouri Alcohol Research Center, Department of Psychiatry, Washington University School of Medicine, St Louis, Missouri, USA.
[4] To whom correspondence should be addressed at Genetic Epidemiology Unit, Queensland Institute of Medical Research, Post Office, Royal Brisbane Hospital, Brisbane, Queensland, 4029, Australia. Tel: +61+7+3362 0228. Fax: +61+7+3362 0101. e-mail; nathanG@qimr.edu.au

0001-8244/03/0700-0383/0

self-reports and offspring ratings of Overprotection (0.55) (Parker, 1981a). Studies using larger population-based samples have found higher sibling correlations (0.37 to 0.63) compared to parent–offspring correlations (0.10 to 0.29) (Kendler, 1996; Kendler *et al.,* 2000). These modest correlations have led Kendler to conclude that the PBI represents at least a "partial reflection of true parenting" (Kendler, 1996). Although Parker has consistently found that the combination of low Care and high Overprotection (termed "affectionless control") best predicts liability to symptoms of depression and anxiety (Parker, 1979a, b, 1981b), more recent studies have found no interaction between Care and Overprotection in terms of increasing the liability to depression and anxiety (Kendler *et al.,* 2000; Mackinnon *et al.,* 1993). Instead, more attention is now given to the parental Coldness items alone as better predictors of mood and anxiety symptoms including other forms of psychopathology (Duggan *et al.,* 1998; Kendler *et al.,* 2000; Mackinnon *et al.,* 1993).

A possible explanation for the PBI–mood/anxiety association is that an increased liability to depression and anxiety distorts memory of parenting received. In addition to the PBI scores being stable over time, Parker has demonstrated that PBI scores of depressives are unaffected by changes in the number of self-reported depression symptoms (Parker, 1981a). Duggan *et al.,* using a small sample of relatives of depressed probands, claimed that the significant differences in parental Care scores between "never-depressed" versus " recovered-depressives" could not be attributable to biased recall among the recovered depressives (Duggan *et al.,* 1998). Although one study found that recollection of parental behaviour was affected by depression (Lewinsohn & Rosenbaum, 1987), overall the above findings suggest that a scar hypothesis, i.e., distorted perception or memory of parental behavior that persists beyond recovery from an episode of major depression is unlikely and that the elevated PBI scores are not an artifact of previous depression.

Correlations between the PBI Care dimension and Neuroticism (Eysenck and Eysenck, 1964) are typically higher in "recovered" depressives compared to "never-depressed" subjects (−0.35 vs. −0.08) (Duggan *et al.,* 1998). Given the significant covariation between neuroticism with symptoms of mood and anxiety (Jardine *et al.,* 1984), the PBI–mood/anxiety association might therefore be attributable to a common personality diathesis such as neuroticism. Dugan *et al.* (1998) tested this hypothesis and found that the significant association between PBI Care and depression still remained even after the effect of neuroticism was removed. The PBI Coldnessdimension is also significantly associated with a history of parental major depression, phobia, and generalized anxiety disorders for which familial aggregation is almost entirely genetic (Kendler *et al.,* 2000). Kendler therefore suggested that the liability for offspring to develop the same disorders might be related to inherited genetic influences rather than the effects of parental Coldness per se (Kendler *et al.,* 2000). Yet once the history of parental psychopathology was controlled for, there was only a "modest diminution" in the parenting and psychiatric symptoms relationship, which led the authors to speculate that the link between parental behavior and psychopathology was in part causal.

Modeling Causation

Experimental manipulation is not an option when investigating direction of causation (DOC) between PBI dimensions and psychiatric symptoms, so alternative statistical approaches are needed. Longitudinal or two-wave data designs, while potentially informative, are not without their disadvantages. These include stringent methodological requirements (Heath *et al.,* 1993; Neale *et al.,* 1994a) in addition to the cost and time required for data collection. An expedient and novel approach is to model direction of causation based on pairs of relatives measured on a single occasion (Duffy and Martin, 1994; Heath *et al.,* 1993; Neale *et al.,* 1994a). When modeled using genetically informative data such as twins, the pattern of cross-twin cross-trait correlations can under certain conditions falsify strong hypotheses about the direction of causation between two variables measured on one occasion provided several assumptions are satisfied: (1) that members of a twin pair are not having any mutual effect on one another, i.e., sibling cooperation/rivalry, either within or across variables; (2) the relationship between the two target variables is equivalent for twin 1 and twin 2; (3) twin pair correlations are different between target variables (Neale *et al.,* 1994a); and (4) there are no unmeasured variables that influence both measures and thereby inflate the correlations arising through the causal influence of one variable on the other. Assumption (3) is critical since the power to detect DOC will be greatest when the target variables have very different modes of inheritance (Heath *et al.,* 1993).

Figure 1 provides an illustrative example of DOC modeling based on cross-sectional data. Let us assume that variable *A* is best explained by shared (C) and

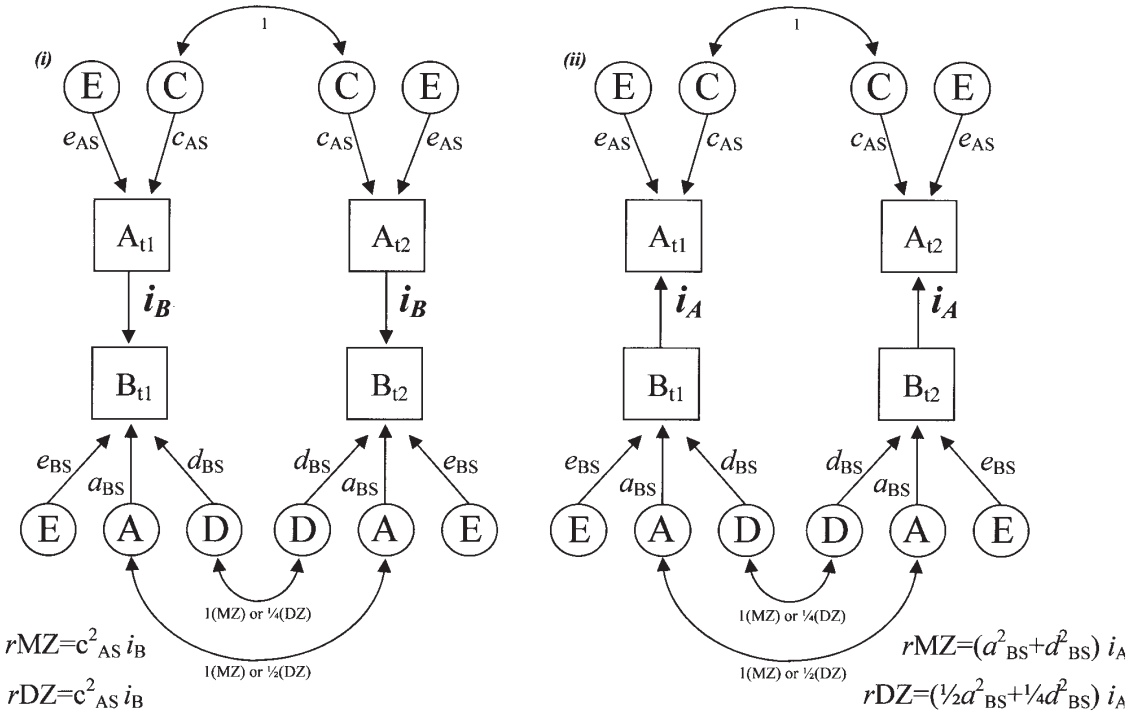

Fig. 1. Unidirectional causation hypotheses between two variables *A* and *B*, measured on a pair of twins. (i) Trait *A* causes Trait *B* and (ii) Trait *B* causes Trait *A*. Example based on simplified model of causes of twin pair resemblance in Neale and Cardon (1992, p. 266). In the boxes are given the expected cross-twin cross-trait correlations for MZ and DZ twins under each unidirectional hypothesis.

nonshared (E) environmental effects, while variable *B* is best explained by additive genetic (A), dominant genetic (D), and nonshared (E) environment effects. Under the "*A* causes *B*" hypothesis (i), the cross-twin cross-trait correlation is $c^2_{AS}i_B$ for MZ and DZ twin pairs alike. However, under the "*B* causes *A*" hypothesis (ii), the cross-twin cross-trait correlation would be $(a^2_{BS} + d^2_{BS})\, i_A$ for MZ and $(^1/_2 a^2_{BS} + {}^1/_4 d^2_{BS})\, i_A$ for DZ twin pairs. It is apparent that if variables *A* and *B* have identical modes of inheritance, then the cross-twin cross-trait correlations will be equivalent for MZ and DZ twin pairs alike, regardless of the direction of causation, and the power to detect direction of causation will vanish.

The PBI dimensions demonstrate a strong shared environmental component (Heath *et al.,* 1993), while family resemblance for measures of psychiatric symptomatology is largely determined by genetic effects (Kendler *et al.,* 1995b) so there is an *a priori* expectation, given sufficient statistical power, that the causal relationship between the PBI and psychiatric symptoms can be resolved by DOC modeling. Previously, Neale *et al.* modeled direction of causation based on cross-sectional data between symptoms of depression and PBI Care and Overprotection (Neale *et al.,* 1994b). They found that models that specified parental rearing as the cause of depression (parenting→depression) fitted the data significantly better than did a model that specified depression as causing parental rearing behavior (depression→parenting). Yet when a term for error of measurement (omission of which is known to produce biased estimates of the causal parameters (Neale and Cardon, 1992) was included, the fit of the "parenting→depression" model improved, but no longer explained the data as parsimoniously as a common additive genetic effects model alone (i.e., implying indirect causation).

As mentioned, measurement error greatly reduces the statistical power for resolving alternative causal hypotheses. One remedy would be to use multiple

indicators and model DOC between latent constructs for the PBI dimensions and psychological distress (Heath *et al.,* 1993; McArdle, 1994; Neale and Cardon, 1992). This approach assumes that measurement error occurs, not at the latent variable level but at the level of the indicator variables, and is uncorrelated across the indicator variables (Neale and Cardon, 1992). Here we model direction of causation, using cross-sectional data, between latent constructs of (1) parenting as measured by the PBI and (2) psychological distress in a female twin population.

METHOD

Sample

Twins were drawn from the Australian National Health and Medical Research Council Twin Register (ATR). The ATR is a volunteer register founded in 1978 with approximately 25,000 pairs of all types and all ages enrolled and in various stages of active contact. We estimate that this represents 10–20% of living twins in Australia. Numerous analyses have shown that the ATR is typical of the Australian population in many respects, including the prevalence of psychiatric symptoms (Kendler *et al.,* 1986), although the ATR sample tends to be slightly more middle class and educated than average, particularly for males (Baker *et al.,* 1996).

The current project was based on two ATR cohorts: an older cohort of 3808 twin pairs born before 1964, referred to as "Cohort 1" (Jardine *et al.,* 1984); and a younger cohort of 4269 twin pairs born 1964–1974, referred to as "Cohort 2." We chose to analyze female twins aged 18–45 years at the time of completing the questionnaire. The advantage of using young to middle age cohorts is that while the total sample is old enough to report symptoms of psychopathology, twins should still be young enough to recollect parental rearing style. The advantage of using data from females is that they report higher rates of affective and anxiety symptoms and are more cooperative in terms of returning completed questionnaires (Kendler, 1996).

As part of a study sponsored by the National Health and Medical Research Council (NHMRC), Cohort 1, first surveyed in 1980–1982, was followed up from 1988–1990 to investigate persistence and changes in drinking habits. This follow-up, described here as the Alcohol Cohort 1 Study, contained a lengthy self-report Health and Lifestyle Questionnaire (HLQ), which incorporated many of the questions sent out to the same twins eight years previously. The HLQ assessments included age, sex, zygosity, tobacco use, alcohol consumption, personality, sociodemographic variables, psychiatric symptoms, and numerous other behavioral measures (Heath *et al.,* 1994). A new section pertaining to women's health (premenstrual symptoms, postnatal depression, perceived control, interpersonal dependency, self-esteem, coping strategies, and life events) was also included (Treloar *et al.,* 1999). In order to reduce postage cost and maximize response, considerable effort was made to verify the addresses of twins prior to mailing. However, after an eight-year gap since completing the first questionnaire, large numbers of twins were lost and extensive efforts were made to locate these twins in 1988–1990. This involved telephoning nonresponding twins, their co-twins, or the parents who had initially enrolled them.

Twins from Cohort 2 were first assessed in 1989 using a self-report questionnaire that included many of the same assessments as the follow-up questionnaire used with the older cohort (Heath *et al.,* 2001). This study was designed to investigate drinking behavior in a younger cohort of Australian twins born between 1964 and 1971. Cohort 2 had been recruited when at school some ten years earlier as part of the earlier study, so it was not surprising that, despite extensive follow-up efforts, we were unable to re-establish contact with 1000 pairs. Twins who failed to return a completed questionnaire were followed up by telephone up to five times, at which point they were asked to complete an abbreviated telephone interview to obtain basic demographic information only.

Measures

Apart from assessing general demographic information, the HLQs sent to both cohorts included the same measures of personality, social behavior and attitudes, psychiatric symptoms, general health/illness, and the occurrence of life stressors. Zygosity was determined based on twins' responses to standard questions about similarity and the degree to which others confused them. Such procedures have previously demonstrated at least 95% agreement with diagnoses based on extensive blood analyses (Martin, 1975; Ooki *et al.,* 1990).

Psychiatric symptoms were assessed using 14 anxiety and depression items from the Delusion Symptoms States Inventory (DSSI/sAD) (Bedford and Deary, 1997; Foulds and Bedford, 1975) and 19 items from the 90-item Symptom Checklist (SCL-90) (Derogatis *et al.,*

1973). Eighteen items were chosen from four SCL subscales: Anxiety (4 items), Depression (5 items), Phobic Anxiety (PHOB) (5 items), and Somatic Distress (SOMAT) (4 items). One item, dealing with early morning awakening or insomnia, was chosen from additional items available in the SCL-90. All items were rephrased to conform to the DDSI/sAD format of inquiry, "*Recently I have had. . .*" rather than the SCL-90 format, "*In the past two weeks. . .*". The response set was also changed from a 5-point scale of distress from "*not at all*" (0) to "*extremely*" (4) (Derogatis *et al.,* 1973) to the DSSI/sAD four-point distress scale: (1) "*not-at-all,*" (2) "*a little*," (3) "*a lot*," (4) "*unbearably*."

The HLQ also contained a 14-item reduced version of the Parental Bonding Instrument (PBI) derived from Parker *et al.*'s original 25-item version (Parker *et al.,* 1979) designed to measure parental treatment along the dimensions of Care (4 items) and Overprotection (10 items) (Parker, 1989, 1990). Two Care and five Overprotection items tap subjects' recollections of the maternal parenting they received as adolescents. The remaining 7 items were identical but reworded to measure paternal parenting instead. The original 4-point response set was retained but rephrased from "*very unlike*" (1) to "*very like*" (4) to (1) "*not-at-all*" to (4) "*a lot*."

Imputation of Missing Item Responses

The imputation option in PRELIS 2.20 (Jöreskog and Sörbom, 1998) was used to impute missing values for the psychiatric symptoms using the full 33 items as matching variables. The same procedure was repeated for the PBI items using the 14 items as matching variables. This approach substitutes values for the missing values from other cases with similar response patterns and no missing values in the matching variables from other cases, provided that the variance in the values from the other cases is acceptable (Jöreskog and Sörbom, 1993). In order to avoid the possibility of artifactual inflation of twin correlations, imputation was carried out on an individual basis ignoring the paired structure of the data. Although no more than 0.1% of the total number of psychiatric and PBI items in both cohorts were imputed, imputation of missing values increased the total effective female sample size for the psychiatric items by 4.5% ($N = 3756$) in Cohort 1 and by 5.9% ($N = 2219$) in Cohort 2. The same procedure increased the total number of completed PBI responses from female twins by 2.3% ($N = 3512$) in Cohort 1 and by 0.7% ($N = 2107$) in Cohort 2.

After combining the imputed data from both cohorts, there were 1030 monozygotic (MZ) and 661 dizygotic (DZ) same-sex female twin pairs with complete responses to the psychiatric symptoms plus an additional 200 MZ and 204 DZ singletons, while for the PBI items there were 969 MZ and 608 DZ complete twin pairs and a further 222 MZ and 233 DZ singletons. For the joint analysis of psychological distress and PBI measures, there were 944 MZ and 595 DZ twin pairs.

It is important to note that the Cohort 1 individual response rates were 73% and 70% for the psychiatric and PBI items, respectively, whereas for Cohort 2 they were 53% for the psychiatric and PBI items alike. Although these rates improved after imputation, the response rate for Cohort 2 indicated possible systematic differences between responders and nonresponders for the psychiatric symptoms and PBI items alike (for a more complete discussion of predictors of questionnaire nonresponse in Cohort 2, see Heath *et al.,* 2001). During the Cohort 2 data collection phase, measures of social class and educational attainment were obtained for both mail and telephone respondents, whereas telephone respondents to the abbreviated questionnaire were asked neither the psychiatric symptom checklist nor the PBI items. Additionally, there were mail respondents who failed to complete the psychiatric symptom checklist ($N = 165$) and PBI items ($N = 289$). Within the total sample of respondents we could therefore test whether the distribution of self-reported social class and education differed between those responding versus not responding to the psychiatric symptoms and the PBI items. Significantly higher education levels were found in female twins who completed the psychiatric symptom checklist ($\chi^2 = 41.35$, $df = 6$, $p < .001$) and PBI ($\chi^2 = 65.25$, $df = 11$, $p < .001$). PBI respondents also tended to be more likely to report themselves as middle class ($\chi^2 = 11.11$, $df = 2$, $p < .01$). Although significant, the correlation between the psychiatric items and education was modest ($r = 0.10$). The correlation between the PBI responses and education was also significant but small ($r = 0.06$), whereas the correlation between PBI and education was significant and modest ($r = 0.14$).

Factor Structure

A principal axis factor analysis of the PBI items based on 4514 female twin individuals (including females from opposite-sex DZ twin pairs) was performed followed by Promax rotation. Initially, ratings of maternal and paternal PBI items were analyzed

separately and very similar factor structures were extracted. We therefore combined the 7 maternal and 7 paternal PBI items in a joint factor analysis from which three interpretable factors were extracted: Autonomy, Coldness, and Overprotection. Correlations between the maternal and paternal dimensions within the PBI dimensions of Autonomy, Overprotection, and Coldness were 0.76, 0.78 and 0.70, respectively. Bivariate genetic analyses also revealed that additive genetic, shared, and nonshared environment correlations between latent maternal and paternal factors nearly all exceed 0.80. The first factor, Autonomy, included such items as "My mother/father let me do the things that I like doing" and "My mother/father liked me to make my own decisions." The second factor, labeled Coldness, was reflected by items such as "My mother/father seemed emotionally cold to me" and "My mother/father made me feel not wanted." The final factor, Overprotection, included items such as "My mother/father tried to make me dependent" and "My mother/father was overprotective of me."

Cronbach alphas for Autonomy, Coldness, and Overprotection were 0.74, 0.81, and 0.77, respectively. Previously, factor analysis of the psychiatric symptoms based on item responses from the Alcohol Cohort 1 yielded a four-factor structure: Depression, Anxiety (with Phobia), Somatic Distress, and Sleep Disturbance (Gillespie *et al.,* 1999). Largely equivalent dimensions were again extracted using the combined data but only the dimensions of Depression (6 items), Anxiety with Phobia (7 items), and Somatic Distress (6 items) were retained for the current analyses. Cronbach alphas for the psychiatric dimensions of Depression, Anxiety, and Somatic Distress were 0.87, 0.83, and 0.70, respectively.

Test-Retest Reliability

Approximately two years after completing the 1988 questionnaire, a sample of 500 females from the Cohort 1 were resent the same questionnaire. Complete psychiatric symptom and PBI data were available from 430 and 405 female respondents, respectively, and since these twins were surveyed before all of the original survey responses had been returned in 1988, uncooperative twins were therefore undersampled. Test-retest correlations for the factor-analytic-derived psychiatric symptom measures and PBI dimensions were Depression (0.64), Anxiety (0.67), Somatic Distress (0.70), Autonomy (0.67), Coldness (0.64), and Overprotection (0.68).

Analysis of Ordinal Data

The application of raw data methods to ordinal data, based on multivariate normal theory, enables the preliminary testing of basic assumptions concerning the equality of response (threshold) distributions within twin pairs, across sex and zygosity, as well as tests of hypotheses about the equality and structure of correlations, which is directly analogous to testing the equality of means and covariance structure when analyzing raw continuous data (Lange *et al.,* 1976). Since the approach uses both complete and incomplete twin pair data, it has the added advantage of increasing the accuracy of the estimation of the thresholds, thereby improving estimation of the polychoric correlations. The major limitation is that computational demands are proportional to the number of categories. Contingency tables with zero-frequency cells will also cause optimization to fail. In order to overcome these limitations as well as deal with the significant positive skew observed on each variable, we reduced the number of response categories for Depression, Anxiety, Somatic Distress, and Coldness to three, while Autonomy and Overprotection were reduced to four categories. This was only done after ensuring that this did not lead to an appreciable loss of information nor to more than minimal changes in the estimated polychoric correlations and their variances.

Genetic Analysis

Standard biometrical genetic model-fitting methods were used (Neale, 1999). The total variance in each observed variable is decomposed into additive (A) and nonadditive (D) (dominance or epistasis) genetic variance plus shared (C) and unique (E) environmental variance. MZ twins are genetically identical, therefore correlations for additive and nonadditive genetic effects between MZ twins are both 1.0. For DZ twins, the correlations for additive and nonadditive genetic effects are 0.5 and 0.25, respectively. An important assumption of the biometrical model is that shared environmental effects correlate to an equal extent in MZ and DZ twin pairs. Nonshared environmental effects are by definition uncorrelated and also reflect measurement error, including short-term fluctuations.

Univariate and Multivariate Analysis

Genetic models were fitted to the ordinal data by the method of Maximum Likelihood (ML) using Mx (Neale, 1999). In the absence of data from separated

twin pairs, half siblings, or similar pairs of relatives, nonadditive and shared environmental effects are confounded. Nonadditive effects tend to produce DZ correlations less than one-half the corresponding MZ correlations, while shared environmental effects tend to produce DZ correlations greater than one-half the MZ correlations. Since much larger sample sizes are required for detecting genetic nonadditivity (Martin and Eaves, 1977), our analyses focused on fitting models allowing for additive genetic plus shared and nonshared environmental effects. The goodness-of-fit of the full ACE model was then compared to that of the submodels by likelihood-ratio chi-squared tests. Best-fitting models are chosen on the basis of parsimony, i.e., nonsignificant changes in the chi-square and the smallest number of parameters. To this end, the Akaike's Information Criterion (AIC) is calculated for each model and the model with the lowest value of this index is chosen as the best fitting.

Since our central aim was to model direction of causation between latent constructs of parenting as measured by the PBI and psychological distress items, we needed to determine *a priori* how well multiple indicators of parenting (Coldness, Overprotection, and Autonomy) and psychological distress (Depression, Anxiety, and Somatic Distress) each loaded onto single, latent constructs. This was achieved by comparing the fit of common and independent pathway genetic models. A common pathway model assumes that both genetic and environmental effects contribute to one or more latent intervening variables, which in turn are responsible for the observed patterns of covariance between symptoms, whereas the independent pathway model predicts that genes and environment have different effects on the covariance between symptoms. It can be shown algebraically that the common pathway is nested within the independent pathway model (Kendler *et al.,* 1987), so the two models can be compared using a likelihood-ratio chi-squared statistic.

We then began DOC modeling by fitting (1) the full bivariate, (2) reciprocal, (3) unidirectional, and (4) noncausal multivariate models to the distress and parenting data. The full bivariate model predicts that a common source of additive genetic as well as shared and nonshared environmental effects account for the covariance between the latent constructs of parenting and distress, whereas under the reciprocal causation and unidirectional models, the parenting and distress constructs are each determined by independent genetic and environmental effects, with the covariance between the two latent variables explained by either reciprocal interaction or unidirectional parameters. Heath *et al.* have shown that the unidirectional and reciprocal causation models are in fact nested within the full bivariate model, which permits model comparisons using goodness-of-fit statistics (Heath *et al.,* 1993). Modeling three sources of variance for each of the latent constructs also enables us to compare the unidirectional against the reciprocal and full bivariate models (Heath *et al.,* 1993). Goodness-of-fit of the full bivariate model can therefore also be compared to that of the submodels by likelihood-ratio chi-squared tests.

RESULTS

Tests of Threshold Homogeneity and Polychoric Correlations

Descriptive statistics for the sample of female twins based on the full-score distributions prior to recoding are shown in Table I. No differences in thresholds (i.e., response distributions) were observed between first and second twins or across zygosity for any of the variables. Polychoric correlations appear in Table II. Age correlated negatively with Depression (−0.20) and Anxiety (−0.18), suggesting that symptoms of depression and anxiety decrease over time. Correlations between age and the remaining variables did not exceed 0.10. Correlations between the parenting dimensions and distress measures were modest (0.13 to 0.24). Overprotection correlated moderately with Autonomy (0.27) and Coldness (0.20), whereas the correlation between Coldness and Autonomy was much higher (0.47).

Estimating Twin Pair Correlations

Age-corrected maximum-likelihood twin pair polychoric correlations together with 95% confidence intervals for the six target variables are shown in Table III. For every variable, MZ correlations were larger than their

Table I. Descriptive Statistics for the Three Dimensions of Psychological Distress and Three PBI Dimensions

	AGE	DEP	ANX	SOM	AUTO	OVERP	COLD
N	5816	3786	3786	3786	3609	3609	3609
Range	18–45	0–18	0–21	0–18	0–18	0–12	0–12
Mean	27.76	1.44	1.02	2.10	6.85	3.22	1.66
Variance	52.85	7.00	5.02	6.20	16.56	8.91	6.07

Based on female twins from same-sex zygosity groups; DEP = depression, ANX = anxiety, SOM = somatic distress, AUTO = autonomy, OVERP = overprotection, COLD = coldness.

Table II. Polychoric Correlations Between the Three Dimensions of Psychological Distress and Three PBI Dimensions

	AGE	1.	2.	3.	4.	5.	6.
1. DEP	−0.20	1.00					
2. ANX	−0.18	0.65	1.00				
3. SOM	−0.07	0.51	0.54	1.00			
4. AUTO	0.08	0.22	0.16	0.16	1.00		
5. OVER	−0.07	0.17	0.16	0.13	0.27	1.00	
6. COLD	0.09	0.24	0.22	0.20	0.47	0.20	1.00

$N = 3562$ females

DZ counterparts. For Depression, the MZ correlation was greater than twice the DZ correlation, suggesting genetic dominance. The three PBI DZ correlations were clearly larger than half their MZ counterparts, suggesting common environmental effects. A model that fixed MZ and DZ correlations to zero gave a very poor fit in every case, indicating significant twin pair resemblance for each variable.

Univariate Analysis

The most parsimonious univariate model for the psychological distress variables was an additive genetic and nonshared environment (AE) model. Overprotection and Autonomy were best explained by a combination of additive genetic, shared, and nonshared environmental effects (ACE), whereas an AE model provided a marginally better fit for Coldness. Despite the sample's age range, age accounted for no more than 3–4% of the total variance in Depression and Anxiety and made only negligible contributions (0–1%) to the remaining variables. Maximum-likelihood point estimates of the additive genetic, shared, and nonshared environmental parameters also appear in Table III.

Multivariate Analysis

Fitting the multivariate genetic models to the ordinal data by ML proved too computationally demanding. We therefore fitted these models using weighted least square (WLS) in Mx. Polychoric and asymptotic weight matrices were calculated in PRELIS (Jöreskog *et al.,* 1998). When compared to an independent pathway model, a common-pathway genetic model provided a more parsimonious fit for both the psychological distress ($\chi^2_4 = 4.41$) and PBI dimensions ($\chi^2_4 = 3.53$). Models are shown in Fig. 2.

DOC Modeling

Direction of causation model fitting results are shown in Table IV. The fit of the reciprocal causation model "distress⇆parenting" was not significantly worse than that of the full bivariate ($\chi^2_1 = 0.47$, $p = .49$). The unidirectional "distress→parenting" model fitted poorly ($\chi^2_2 = 10.63$, $p < .01$). The "parenting→distress" model provided a marginally better fit ($\chi^2_2 = 1.48$, $p = .48$) when compared to the reciprocal causation and full bivariate. We also tested the model of no relationship between distress and parenting, which fitted very poorly ($\chi^2_3 = 230.63$, $p < .001$).

DISCUSSION

Factor Structure

The three-factor structure of Depression, Anxiety, and Somatic Distress based on factor analysis of the psychological distress items was largely equivalent to the factor structure derived in our earlier analyses based on the younger female and male twin data from Cohort 2 (Gillespie *et al.,* 1999). Principal axis factoring of the

Table III. Age-Corrected Twin Pair Correlations, 95% Confidence Intervals (CIs) and Best-Fitting Univariate Models with Maximum-Likelihood Point Estimates for the Proportions of Variance Attributable to A, C, and E

	Age-corrected twin pair correlations and 95% CIs		Age-corrected proportions of variance attributable to best-fitting model parameters				
	MZ twin pairs	DZ twin pairs	A	C	E	−2LL	df
Depression	.38 (.30–.45)	.12 (.02–.22)	.33	—	.67	7399.99	3780
Anxiety	.45 (.37–.52)	.24 (.14–.34)	.43	—	.57	6455.72	3780
Somatic Distress	.34 (.27–.41)	.19 (.09–.29)	.34	—	.66	7634.86	3780
Coldness	.60 (.53–.66)	.41 (.30–.51)	.61	—	.39	5979.01	3603
Overprotection	.46 (.39–.52)	.35 (.25–.43)	.22	.24	.54	7438.84	3602
Autonomy	.49 (.44–.55)	.33 (.25–.41)	.33	.17	.51	9216.17	3599

A = additive genetic, C = common environment, E = nonshared environment.

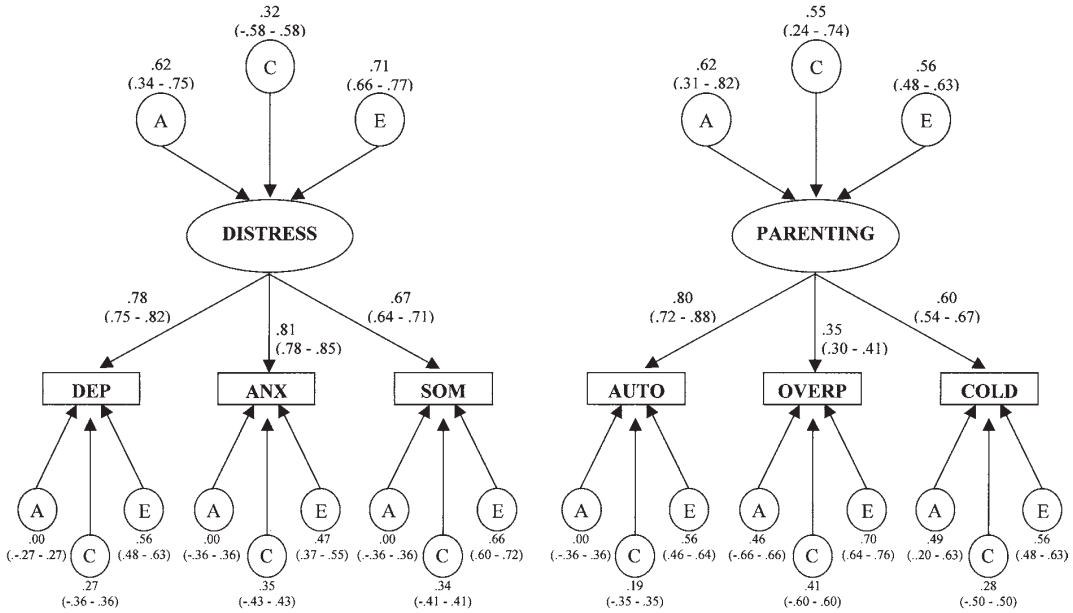

Fig. 2. Best-fitting common-pathway multivariate genetic models for the psychological distress and PBI parenting dimensions with standardized path coefficients and 95% confidence intervals.

14 PBI items did not replicate Parker's two-factor structure of Care and Overprotection. Instead, we extracted three factors, which we labeled Coldness, Overprotection, and Autonomy. Coldness was more or less equivalent (after reversing the signs of the factor loadings) to the original PBI Care factor (Parker and Lipscombe, 1979) with the exception of item 3 ("Mother appeared to understand my problems"), which loaded on Autonomy.

Table IV. Results of Fitting Direction of Causation Models to the Psychological Distress and Parenting Variables

	Goodness of fit					
Model	χ^2	df	$\Delta\chi^2$	Δdf	p	AIC
Full bivariate	141.65	105				−68.35
Reciprocal Causation	142.12	106	0.47	1	0.49	−69.88
Distress[a]→ Parenting[b]	152.28	107	10.63	2	**	−61.72
Parenting→ Distress	143.13	107	1.48	2	0.48	−70.87
No correlation	350.60	108	208.95	3	***	134.6

* $p < .05$, ** $p < .01$, *** $p < .001$, results based on 944 female MZ twin pairs and 595 DZ twin pairs aged 18 to 45.

[a] Distress as measured by 3 indicators: Depression, Anxiety, and Somatic Distress.

[b] Parenting as measured by 3 indicators: Coldness, Overprotection, and Autonomy.

In our analysis, Parker's original Overprotection factor split into two factors, which we have labeled Overprotection and Autonomy. Overall, our parenting factors validate the solutions found by previous studies using longer versions of the PBI (Cubis *et al.,* 1989; Murphy *et al.,* 1997; Sato *et al.,* 1998), two of which were also based on a large, population-based twin registry (Kendler, 1996; *et al.,* 2000). Although based on a smaller number of PBI items, our factor-analytic dimensions of Coldness, Overprotection, and Autonomy are very similar to Kendler's dimensions of warmth, protectiveness, and authoritarianism (Kendler, 1996).

Our decision to combine the maternal and paternal items was supported by the findings of Kendler and colleagues, who found no significant differences between maternal and paternal ratings for psychiatric disorders (Kendler *et al.,* 2000). In another study, Reiss and colleagues investigated the degree to which parents treated their offspring differently (Reiss *et al.,* 1995). Based on parental self-reports and child and observer ratings, the authors found that the correlations between latent maternal and paternal first-order factors reflecting parental conflict, warmth, and control were 0.85, 0.50, and 0.75, respectively (Reiss *et al.,* 1995), indicating a high degree of congruency.

Univariate Results

Familial aggregation for all three psychological distress variables including parental Coldness was best explained by additive genetic effects alone. Neale and colleagues also found no evidence of shared environmental effects on an equivalent Coldness dimension (Neale *et al.,* 1994b). Age accounted for 3–4% of the variance in Depression and Anxiety, which is in general agreement with previous findings showing a decline in symptoms of depression and anxiety with increasing age (Henderson *et al.,* 1998). Age accounted for negligible variance in Somatic Distress and the three PBI dimensions. In addition to additive genetic effects, there was a significant contribution of shared environment to the sources of familial aggregation in Overprotection and Autonomy. Kendler has attributed the increased twin pair resemblance in MZs on the PBI dimensions to greater behavioral similarity in MZ twin pairs, which elicits more similar behavioral responses from parents toward MZ co-twins (Kendler, 1996). To a lesser extent he has suggested increased MZ twin pair similarity in temperament means that they perceive their treatment as being more similar, despite being objectively treated no more similarly than DZ co-twins. The effect of parents' social preconceptions and expectations about MZ versus DZ twins are likely to exert only a very minor influence on parental behavior (Kendler, 1996). In any event, differential treatment of MZ and DZ twins by their parents is unlikely to represent a significant bias in twin studies of these major psychiatric disorders (Kendler *et al.,* 1994).

Multivariate Results

The latent distress factor in the common pathway model explained approximately two thirds of the variance in Depression and Anxiety, but less than half of the variance in Somatic Distress. The remaining proportion of variance in Somatic Distress was attributable to nonshared (42%) and additive genetic (11%) effects unique to that phenotype. We also found when analyzing data from the younger Cohort 2 that somatic symptoms, although correlated with anxiety and depression, show some specificity of genetic influence (Gillespie *et al.,* 2000). The common-factor model for parenting provided the most parsimonious fit to the data when explaining the sources of covariance between the three PBI dimensions. The latent parenting factor accounted for 42%, 12%, and 67% of the variance in Coldness, Overprotection, and Autonomy, respectively, and was explained by additive genetic, shared, and nonshared environmental effects. Although there were specific genetic contributions to the variance in Coldness and Overprotection, there was no evidence of shared environmental effects specific to Coldness, Overprotection, and Autonomy.

The model that specified the ratings of parents as the cause of psychological distress in the female twins fit the data significantly better than the model that specified psychological distress as the cause of parental ratings. In other words, recalling one's parents as having been cold, overprotective, and not granting enough autonomy tended to increase levels of psychological distress. Since both the "distress→parenting" and no causation models deteriorated significantly from the full bivariate model, the correlations between the PBI scores and distress measures cannot be explained by the hypothesis that PBI scores were altered by symptoms of psychological distress.

Estimates of the magnitude of association between our measures of parenting and distress vary widely among studies. Parker reported that 9 to 10% of the variance in trait depression and anxiety scores could be explained by PBI factor scores (Parker, 1979b). Kendler found that despite significant associations between the PBI dimensions and clinical measures of psychopathology, the aggregate effect of parenting (summed across all three PBI dimensions as well as the maternal and paternal ratings) could only explain 1–4% of the correlation in liability between siblings for clinical measures of major depression, phobia, and generalized anxiety disorder (Kendler *et al.,* 2000). Larger estimates of the magnitude of association have been reported. For instance, Reiss and colleagues found that between 31 and 41% of variance in the symptoms of adolescent depressive behavior could be explained by parental conflict and negativity (Reiss *et al.,* 1995). Interestingly, the same authors also reported that certain aspects of parental monitoring and control when directed toward a child were associated with a slight reduction in the symptoms of antisocial behavior and depression in the child's sibling. Our own estimate of the magnitude of association was more modest; when based on the unidirectional "parenting→distress" model we estimated that 18% of the variance in psychological distress is attributable to the parental behavior.

Limitations

Although the "parenting→distress" model provided the best fit to the data as judged by the lowest

AIC value, the improvement was marginal when compared to the full bivariate or reciprocal causation models. Subsequent analyses revealed that rejection of the full bivariate model with 80% power (0.05% significance level) in favor of the reciprocal "distress⇆ parenting" and unidirectional "parenting→distress" models would require samples sizes of 22,799 and 7242 twin pairs, respectively. So while there was there was sufficient statistical power to reject the "distress→ parenting" model, we cannot rule out the possibility that the PBI and psychological distress measures were correlated because of shared genetic or environmental effects or simply arose via a reciprocal interaction between parental recollections and psychological distress.

The fact that there was insufficient power to reject the full bivariate and reciprocal causation models allows us to explore other means by which an association between parenting and psychological adjustment can arise. For instance, it is also possible that the association between psychological distress and the PBI measures may be better explained by other unmeasured variables. Therefore, in addition to tests of social class and education, we investigated the effect of being raised by both natural parents. Most of the current sample of female twins also participated in another project called the Semi-Structured Assessment for the Genetics of Alcoholism Study (Bierut *et al.,* 1999). One item asked twins whether or not they had been reared by both natural parents until the age of 16 years and complete responses from 3111 same-sex female twins were obtained. Female twins who indicated that they had been raised by only one natural parent until the age of 16 reported significantly higher means for parental Autonomy and Coldness, yet the effect explained no more than 2% of the variance in the PBI dimensions and the correlation with any of the psychological distress measures did not exceed 0.12. With regard to personality, we found that correlations between EPQ Neuroticism (Eysenck & Eysenck, 1991) and the three psychological distress measures were high and ranged from 0.43 to 0.60, while the correlations between Neuroticism and the three PBI dimensions ranged from 0.13 to 0.19. Previous reports have shown that partialling out Neuroticism fails to remove the association between PBI scores and depression (Duggan *et al.,* 1998; Parker, 1979c, 1981a). As mentioned previously, age accounted for no more than 3–4% of the total variance in Depression and Anxiety and made only negligible contributions (0–1%) to the remaining variables. When we re-ran our analyses by using a maximum-likelihood model-fitting approach to partial out the contribution of age, almost exactly the same pattern of results was obtained.

Another important limitation concerns our use of data based on twins' recollections of parental behavior. Retrospective reports of parenting might be influenced by recall bias or cognitive distortion and may be less accurate than those reported by parents or co-twins. Indeed, a large number of studies have shown that child, parental, and observer reports do not correlate highly with one another (Feinberg *et al.,* 2001). Correlations between maternal Care and measures of state and trait depression are typically lower when mothers versus twins are interviewed (Parker, 1981a). Mothers also tend to rate themselves as more caring and less overprotective compared to the maternal ratings provided by their offspring (Parker, 1981a). In addition, the contribution of genetic and environmental effects depends upon who is being questioned (Kendler, 1996; Wade and Kendler, 2000). When based on parental self-reports, familial aggregation for the PBI dimensions is largely explained by common environmental effects, whereas the proportion of genetic variance increases when twin and co-twin data are analyzed (Kendler, 1996).

Low interrater agreement and qualitative differences in the proportions of variance attributable to gene and environment action raise important questions concerning the validity of parental measures. Of course, it is possible that child, parental, and observer reports may all be to some extent accurate, but each measures different aspects of parental behavior, leading to observed perceptual differences. In order to resolve these differences, one method of deriving a more accurate parenting score would be to examine the covariance between parents' self-ratings and ratings by their twin children as well as outside observers (Feinberg *et al.,* 2001; Neale *et al.,* 1994b). Multivariate analysis of familial resemblance would then enable discrimination between parent-to-child transmission, pleiotropic genetic factors, and effects of assortative mating as well as genotype-environmental correlations.

In the case of assortative mating, if a trait is genetically influenced, then any correlation between the additive deviations of spouses would lead to an equal increase in the correlation of both the MZ and DZ twin pairs, thereby mimicking the effect of the shared environment. The shared environmental effects for the PBI factor may be partly indicative of assortative mating, whereas the absence of shared environment on the latent psychological distress factor suggests that assortative mating effects are either minimal or nonexistent.

An important caveat is that genetic dominance, which is negatively confounded with the shared environment in the classical twin design, can also mask or attenuate the effects of assortative mating. Unfortunately, the power to detect either shared environmental or dominant genetic effects is greatly reduced by the current sample size. These effects along with those of assortative mating and cultural transmission can only be resolved by way of larger sample sizes incorporating additional data from parents and siblings (Maes *et al.,* 1999; Truett *et al.,* 1994).

Genotype–environment correlations (CorGE) describe situations in which an individual's environment is unlikely to be entirely random but is partly caused by or correlated with his or her genotype (Neale and Cardon, 1992; Scarr and McCartney, 1983). Many CorGE taxonomies have been proposed, including Scarr and McCartney's passive, evocative, and active models (Scarr and McCartney, 1983). Neale and Cardon (1992) describe two types, the first of which refers to a genotype–environment autocorrelation whereby an individual selects, creates, or evokes environments that are a function of his/her genotype. As an example, O'Connor and colleagues found that adopted children at higher genetic risk for antisocial behavior were consistently more likely to receive negative parenting from their adoptive parents (O'Connor *et al.,* 1998). This model of CorGE is analogous to the unidirectional "distress→parenting" hypothesis, which provided a poor fit to the data; i.e., psychological distressed offspring are *not* more likely to evoke patterns of negative parenting. The second form of CorGE arises because the environment in which individuals develop is also provided by their biological relatives (Neale and Cardon, 1992). For example, a child who inherits the genes for anxiety and depression may live in a neurotic home environment because the behavioral tendency for parents to provide such an environment might be underpinned by the same genes for mood and affect. In other words, an increase in the genetic liability to mood and affect may be correlated with a neurotic home environment because the child's genes and the environment are both derived from the parents.

The possibility of genotype-environmental correlations is consistent with Lytton's work on "parent-initiated" and "parent-responsive" behaviors (Lytton, 1977). Kendler has suggested that parents might be biased to recall only "parent-initiated" action, which has been found to be similar across twin groups (Lytton, 1977), whereas twins are more likely to recollect both "parent-initiated" and "parent-responsive" behavior (Kendler, 1996). In the latter case, insofar as parents are responding to genetic differences in their children, this would explain the considerable genetic variance seen in the PBI dimensions. One interpretation of the variance components that we have estimated for the PBI variables might be that "C" represents the children's perceptions of parent-initiated behaviors, while "A" represents the variance attributable to genetically based child-initiated (parent-responsive) behaviors. If this is right, children are not passive receptors of parental influence but are likely to create a large portion of their own environment.

In attempting to unravel the parenting and psychological distress relationship, another important consideration is the possibility of genotype-environmental interactions (Mather and Jinks, 1982). Unlike CorGE, which reflects a nonrandom distribution of environments among different genotypes, genotype-environmental interactions describe the ways in which genes affect environmental sensitivity, or conversely, environments affect gene expression (Neale and Cardon, 1992). As an example, "at-risk" genotypes of depression, anxiety, and somatic distress may be more vulnerable to the pathogenic effects of maladaptive parenting.

These limitations reveal that the relationship between parenting and psychological distress is clearly complex and while this paper has focused on the association between parental behavioral and psychological distress in offspring, parents are not the only influential relatives in terms of a contribution to the psychological developmental of their offspring. For example, the presence or absence of siblings may contribute to important sibling effects (Neale and Cardon, 1992). Likewise, the influence of peers and teachers or the contribution of role models cannot be discounted. Unfortunately, these issues are beyond the scope of the current paper. Despite the above limitations, the chief advantage of the current model-fitting approach is that it provides a clear means of rejecting the "distress→parenting" hypothesis in favor of the "parenting→distress" hypothesis. One final point concerning the PBI is that it is not an exhaustive measure of parenting and so our findings cannot be extrapolated beyond the dimensions of recollected parental Coldness, Overprotection, and Autonomy. Nor can our findings be generalized beyond those reported by females. Kendler has also suggested that twin samples pose yet another limitation insofar as twins are a unique challenge to parents, making it difficult to generalize results to single child or nontwin siblings (Kendler, 1996). Insofar as this "unique challenge" feeds back as worse parenting,

it is certainly not reflected in higher rates of psychiatric symptoms in twins versus singletons, as previously shown (Kendler *et al.,* 1995a).

ACKNOWLEDGMENTS

This work was supported by NIH grants AA07535, AA07728, AA10249, AA11998, AA13321 and AA13326 and NHMRC (Australia) grants 941177 and 971232. We also thank the twins for their cooperation.

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Edited by Carol Prescott