Genetic and environmental risk factors for depression assessed by subject-rated Symptom Check List versus Structured Clinical Interview

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ABSTRACT

Background. It is not known if a subject’s characteristic level of self-rated depression symptoms index their genetic or environmental liability to major depressive disorder when measurement error and other occasion-specific influences are taken into account.

Method. Monozygotic (N = 408) and dizygotic (N = 295) adult female twin pairs from a population-based registry were surveyed twice with an average follow-up interval of 61 months. At each occasion subjects completed a structured clinical interview (SCID) to assess lifetime history of major depression and the subject-rated Symptom Check List (SCL) to assess current level of depressive symptomatology. A bivariate measurement model was used to estimate the genetic and environmental correlations between liability to reliably diagnosed lifetime history of major depression and the characteristic or temporally stable SCL depression score.

Results. The genetic and non-familial environmental correlation between liability to reliably diagnosed major depression and the characteristic level of SCL depression symptoms (and the proportion of variance shared between measures) is \( > 0.70 \) and \( > 0.24 \) respectively.

Conclusions. When allowance is made for diagnostic unreliability and temporal fluctuations in the level of subject-rated symptoms, 70% of the variance in genetic risk factors and 24% of the variance in environmental risk factors is shared by a diagnosis of lifetime major depression and total SCL depression symptom score. SCL depression scores may therefore be a useful screening measure for many of the genetic risk factors which influence liability to major depression.

INTRODUCTION

Subject-rated symptom check lists and structured clinical interviews are both widely used in epidemiological studies of depression. The degree to which these two measurement constructs index similar or distinct risk factors remains unclear. If different genetic or environmental risk factors underlie variation in symptom check list ratings and risk for depressive disorders then attempts to integrate findings from a wide range of depression research initiatives will be confounded by the unique variance indexed by different measures of depression.

Several genetically informative analyses of subject-rated depression check lists (Clifford et al. 1984; Jardine et al. 1984; Kendler et al. 1986, 1987, 1994a; Eaves et al. 1987; MacKinnon et al. 1990; Silberg et al. 1990) and interviewer-rated histories of depressive disorder (Torgersen, 1986; McGuffin et al. 1991, 1996; Kendler et al. 1992a, 1993a; Foley et al. 1998; Kendler & Prescott, 1999) have been conducted. The findings of these studies converge in showing that both genetic and individual-specific environmental risk factors contribute to liability for depression measured in different ways. These
studies do not address, however, if the same genetic and environmental risk factors influence liability to interviewer-rated depressive disorder and subject-rated depression symptoms. How do these different depression constructs relate to each other?

Three findings suggest that symptom check lists may usefully screen for liability to depressive disorder. Symptom check list ratings demonstrate high sensitivity (but relatively low specificity) for depressive disorder in cross-sectional surveys (Hammen, 1980; Myers & Weissman, 1980; Lewinsohn & Teri, 1982; Wetzler, 1989; Wetzler & Marlow, 1993). Symptom check list ratings are relatively stable over time despite being conceptualized as ‘state’ measures. Duncan-Jones et al. (1990) estimated that between 40–76% of the variance in subjects’ ratings reflects characteristic levels of symptomatology. Variation in symptom check list ratings over time is not influenced by genetic or other familial risk factors (MacKinnon et al. 1990). Occasion-specific influences on LTH of MD do not index familial risk factors for MD (Foley et al. 1998). An individual’s characteristic level of self-rated symptoms (the variance that is stable over time) may therefore index familial risk factors for depressive disorder.

We aim to characterize the covariance between subject-rated depression symptoms and the clinical syndrome of major depression, correcting for errors of measurement and other occasion-specific influences by modelling longitudinal data from a population-based sample of adult female twins.

**METHOD**

**Sample**

The data analysed for this report were collected as part of a longitudinal study of the genetic and environmental risk factors for common psychiatric disorders in a population-based sample of adult female twins from the state of Virginia, USA (Kendler et al. 1992b). The first contact with these twins was a mailed survey to which 64% of individuals responded. The true cooperation rate is likely to be higher because an unknown proportion of non-respondents never received the mailed survey due to incorrect mailing addresses, incorrect forwarding of mail, etc. Of the 1176 twin pairs who returned the mailed survey, 1033 pairs completed a psychiatric interview at home about their lifetime history of major depression (LTH of MD) and a subject-rated questionnaire an average of 12.3 months later (s.d. = 4.0) (Time 1). Of these, 849 pairs completed a second psychiatric interview about their LTH of MD over the telephone and another subject-rated questionnaire an average of 61.3 months later (s.d. = 25.8) (Time 2).

The twin pairs who are the subject of the current study completed a psychiatric interview and the subject-rated questionnaire at both Time 1 and Time 2. The questionnaire was completed immediately after the interview at both Time 1 and Time 2. The sample comprises 408 monozygotic (MZ) female–female twin pairs and 295 dizygotic (DZ) female–female twin pairs. Informed consent was obtained in writing prior to the Time 1 interview and over the telephone prior to the Time 2 interview.

Zygosity determination (Kendler et al. 1992a) is based on the subject’s ratings of their physical similarity to their co-twin, the frequency with which they were confused as children (Eaves et al. 1989), photographs and DNA typing (Spence et al. 1988).

**Measures**

**Symptom Check List**

The 90-item Symptom Check List (SCL-90) is a self-report clinical rating scale designed to capture nine primary symptom dimensions observed in psychiatric out-patients (Derogatis et al. 1970). One of these dimensions is depressive symptomatology, and the SCL depression subscale has been empirically validated in a series of clinical investigations involving over 2500 patients (Derogatis et al. 1970). This subscale is designed to assess a broad range of concomitants of clinical depression, including some cognitive correlates of depression that are not included in the DSM criteria for major depression (e.g. #22, feeling of being ‘trapped’ or ‘caught’, and #31 worrying too much about things). Ten SCL-90 depression items were included in the questionnaire completed by subjects immediately following the Time 1 and Time 2 interview. These items were chosen based on regression analysis that indicated that these items accounted for > 95% of the variance in the
summary score derived from the complete set of (13) SCL depression items. The items are: #5 loss of sexual interest or pleasure; #14 feeling low in energy or slowed down; #22 feeling of being ‘trapped’ or ‘caught’; #26 blaming yourself for things; #30 feeling blue; #31 worrying too much about things; #32 feeling no interest in things; #54 feeling hopeless about the future; #71 feeling everything is an effort; and, #79 feelings of worthlessness. Subjects were instructed to rate the discomfort associated with each symptom during the previous 30 days on a five-point scale: ‘not at all’, ‘a little bit’, ‘moderately’, ‘quite a bit’ or ‘extremely’. A total SCL depression score was derived by adding the ratings made for each SCL depression item.

Structured Clinical Interview
Each twin in a pair was interviewed by a different field worker blind to the lifetime history of the co-twin. Interviewers held a Master’s degree in social work or had at least 2 years of clinical experience. LTH of MD was assessed
using an adapted version of the SCID (Spitzer et al. 1987) following the DSM-III-R criteria (American Psychiatric Association, 1987). At Time 1, LTH of MD refers to the subject’s LTH of MD prior to the Time 1 interview. At Time 2, LTH of MD also refers to the subject’s LTH of MD prior to the Time 1 interview. This approach may underestimate diagnostic reliability over time because subjects who recall previously denied episodes of MD at Time 2 may erroneously move their onset forward (Angst et al. 1984; Rubio-Stipec et al. 1992).

**Statistical analysis**

The twin model used here is based on a liability threshold model (Falconer, 1960; Kendler & Kidd, 1986), which divides variation in liability into three classes: (1) additive genetic (A), which contributes twice as much to the correlation in MZ twins as DZ twins because MZ twins share all their genes identical by descent, while DZ twins share half their genes on average; (2) family or ‘common’ environment (C), those familial factors that make twins similar in
Risk factors for depression assessed by SCL v. SCID

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Figure 1. The model is drawn for one half of a twin pair as model parameters are constrained to be equal for each twin within a pair. A detailed description of this model, and the fit of alternate models, is given in the accompanying text. (a) A bivariate twin model that partitions the covariance between liability for a lifetime history of major depression (LTH of MD) and the characteristic or temporally stable level of subject-rated Symptom Check List (SCL) depression symptoms. (b) Parameter estimates and 95% confidence intervals obtained for the full model. (c) Parameter estimates and 95% confidence intervals obtained for the best-fitting model. (A, Additive genetic effects; C, Common or familial environment effects; E, Individual-specific or non-familial environment effects.)

liability and contribute equally to the correlation between MZ and DZ twins; and (3) individual-specific or non-familial environment (E), which reflects experiences unshared by co-twins and which therefore contribute to differences in liability between both MZ and DZ co-twins. The equal environment assumption or EEA (that exposure to relevant familial environmental risk factors for depression are approximately equal in MZ and DZ twins) has previously been examined in this sample of twins and no evidence has been found to reject it (Kendler et al. 1992a; 1993b, c, 1994b; Hettema et al. 1995; Kendler & Prescott, 1999).

As pictured in the ellipses drawn in Fig. 1 (a), we assume a true latent liability to LTH of
MD and a characteristic or temporally stable component to SCL depression ratings. The Time 1 and Time 2 interview are considered to be fallible indices of the liability to LTH of MD. The paths $\lambda_1$ and $\lambda_2$ represent the degree to which the assessment of LTH of MD at Time 1 and Time 2 reflects the true liability. The square of the $\lambda_1$ and $\lambda_2$ paths may also be used to estimate the reliability of LTH of MD diagnosed at Time 1 and Time 2. The Time 1 and Time 2 SCL depression ratings are considered to be fallible indices of the subject's characteristic level of self-rated depression symptoms. The paths $\lambda_3$ and $\lambda_4$ estimate the degree to which SCL depression ratings at Time 1 and Time 2 reflect the individual's characteristic (or temporally stable) level of self-rated depression symptoms.

The other paths to liability for LTH of MD and SCL depression symptoms represent occasion-specific influences at Time 1 and Time 2, including measurement error. The paths $\beta_1$ and $\beta_2$ represent occasion-specific influences on the recall and diagnosis of a LTH of MD at Time 1 and Time 2, and the paths $\beta_3$ and $\beta_4$ represent occasion-specific influences or transient state effects on SCL depression symptoms at Time 1 and Time 2. For each depression measure at each time $\lambda^2 + \beta^2 = 1$. The liability to LTH of MD and the liability to a characteristic level of SCL depression symptoms, and the covariance between them, is then modelled in a standard twin design, as outlined above. The sources of variance in liability to LTH of MD and SCL depression symptoms measured on two occasions, and the covariance between them (expressed here in terms of the cross-measure correlation), are partitioned into additive genetic, familial environmental and individual-specific environmental factors. This covariance between liability for LTH of MD and SCL depression symptoms that is stable across occasions of measurement is expressed in terms of the additive genetic ($r_g$), familial environmental ($r_e$) and non-familial environmental correlation ($r_o$).

A LTH of MD and subject-rated SCL depression symptoms is expected to index different occasion-specific effects. A diagnosis of MD at Time 1 or Time 2 requires $\geq 5$ depression symptoms for at least 2 weeks across two occasions of measurement that refer to the same time period (the lifetime preceding the Time 1 interview). Occasion-specific variance in the recall and diagnosis of LTH of MD will therefore reflect unreliability due to variable recall by the subject, denial of symptoms at one occasion, or variable evaluation of reliably reported histories by different interviewers. The SCL surveys symptoms experienced in the past 30 days. The Time 1 and Time 2 SCL depression symptom ratings are therefore completed in reference to symptoms experienced during the month preceding the Time 1 interview and the month preceding the Time 2 interview respectively – two non-overlapping time periods separated by an average of 5 years. Occasion specific influences on SCL depression symptoms will therefore reflect errors of measurement and true symptom change (or other transient characteristics).

Although psychiatric symptom scales like the SCL are designed to measure current symptom levels, these scales demonstrate substantial stability over time (Duncan-Jones et al. 1990; MacKinnon et al. 1990). We are therefore interested here in modelling the extent to which the variation in SCL depression symptoms that is stable across measurement occasions indexes liability to a reliably diagnosed LTH of MD. In other words, does a subject’s characteristic level of self-rated depression symptoms index their liability to major depressive disorder when diagnostic reliability is taken into account?

### Statistical software

The correlation between total SCL depression score and LTH of MD at Time 1 and Time 2 was estimated using the data summary program PRELIS version 2.06 (Jöreskog & Sörbom, 1991). The twin models are specified in Mx (Neale, 1997) using the method of asymptotic weighted least squares (Browne, 1984). The best fitting model is selected by Akaike’s Information Criterion (AIC, Akaike, 1987), which reflects both fit and parsimony (Williams & Holahan, 1994).

### RESULTS

#### Prevalence of MD and SCL depression symptoms

The estimated lifetime prevalence of MD with an onset reported to precede the Time 1 interview is 32.9% (507/1540) at Time 1 and 24.1% at Time 2.
Correlation between MD and SCL depression symptoms

The within-person and cross-twin correlations for MZ and DZ twin pairs are given in Table 1. Time 1 and Time 2 SCL depression symptom ratings and Time 1 and Time 2 LTH of MD will be referred to as SCL1, SCL2, MD1, and MD2 respectively.

The within-person cross-measure correlations are higher than the corresponding MZ and DZ cross-twin correlations at Time 1 (SCL1-MD1, 0.32 > 0.20 > 0.13) and Time 2 (SCL2-MD2, 0.28 > 0.24 > 0.09). This suggests that individual-specific environmental influences contribute to the within-time covariance between SCL depression symptom ratings and LTH of MD. The cross-twin cross-measure correlations are approximately twice as high in MZ pairs versus DZ pairs at Time 1 (SCL1-MD1, 0.20 > 0.13) and Time 2 (SCL2-MD2, 0.24 > 0.09). This suggests that genetic influences contribute to the within-time cross-measure covariance. The within-person cross-measure correlation across time is slightly higher than the corresponding correlation for MZ twins for SCL1-MD2 (0.27 > 0.21) but not for SCL2-MD1 (0.23 < 0.25). This suggests that individual-specific environmental influences have a relatively small impact on the cross-measure covariance over time. The cross-twin cross-measure correlations are approximately twice as high in MZ pairs versus DZ pairs for SCL2-MD1 (0.25 > 0.12), and more than twice as high in MZ pairs versus DZ pairs for SCL1-MD2 (0.21 > 0.04). This suggests that genetic influences account for the majority of the cross-time cross-measure covariance.

Model fitting

The full model, Model 1, provides an adequate fit to the data (Fig. 1, (b), $\chi^2 = 53 \pm 21$, df = 47, $P = 0.25$, AIC = 40-78). The additive genetic correlation ($r_a$) is estimated at +0.70 and the individual-specific environmental correlation ($r_e$) is estimated at +0.24. Three parameters are estimated at zero: the familial environmental paths to liability for MD and temporally stable SCL ratings, and the correlation between them ($r_c$). These three parameters could therefore be dropped from Model 2 with no change in model fit ($\chi^2 = 53 \pm 21$, df = 50, $P = 0.35$, AIC = 45-52). Working from Model 2 we alternately set the two remaining cross-measure correlations to zero. In Model 3 the non-familial environmental correlation ($r_e$) is set to zero, and the decrease in model fit is just sufficient to produce a deterioration in the AIC value ($\chi^2 = 56 \pm 48$, df = 51, $P = 0.28$, AIC = 45-52). This parameter is therefore retained in the model. In model 4 the genetic correlation ($r_g$) is set to zero, and the model fit decreased substantially ($\chi^2 = 234 \pm 62$, df = 51, $P < 0.0001$, AIC = 132-62). This parameter is also retained in the model. Working from Model 2 we then tested if the Time 1 and Time 2 assessments were statistically equivalent indices of liability to MD and temporal stability in SCL ratings by equating $\lambda_1$ and $\lambda_2$, and $\lambda_3$ and $\lambda_4$ respectively. In model 5 both pairs of $\lambda$ parameters could be equated with no significant decrease in model fit ($\chi^2 = 53 \pm 68$, df = 52, $P = 0.41$, AIC = 50-32). The results for model 5, the best-fitting model, are seen in Fig. 1 (c), and the 95% confidence interval (CI) for each parameter estimate is given in parentheses. The individual parameter estimates obtained for models 2, 3 and 4 are available upon request.

In the best-fitting model, as in the full model, the additive genetic correlation ($r_g$) is estimated at +0.70 (95% CI = 0.57, 0.84) and the individual-specific environmental correlation ($r_e$) is estimated at +0.24 (95% CI = 0.10, 0.51). The phenotypic correlation between liability to LTH of MD and the temporally stable component of SCL depression symptom ratings can be decomposed using the path estimates from the best fitting model, with 83% due to common genetic factors and 17% due to common individual-specific environmental risk factors.

The square of the parameter estimates for the A and E paths shown in Fig. 1 (c) represent the variance attributable to additive genetic and individual-specific environmental risk factors in the best fitting model. Additive genetic effects account for 65% ($a^2_{SCL}$) and individual-specific environmental factors account for 35% ($e^2_{SCL}$) of the variance in liability to LTH of MD. Occasion-specific factors (including measurement error) account for 30% ($c^2_{SCL}$) of the
Table 1. Correlations between a lifetime history of major depression and SCL depression symptom ratings in adult female twins

<table>
<thead>
<tr>
<th></th>
<th>Correlation</th>
<th>Time 1 MD</th>
<th>Time 2 MD</th>
<th>Time 1 SCL</th>
<th>Time 2 SCL</th>
</tr>
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<tbody>
<tr>
<td>Time 1 MD</td>
<td>Within individuals</td>
<td></td>
<td>0.43</td>
<td></td>
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<tr>
<td></td>
<td>Between MZ twins</td>
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<td></td>
<td>Between DZ twins</td>
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<td></td>
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<tr>
<td>Time 2 MD</td>
<td>Within individuals</td>
<td></td>
<td>0.66</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Between MZ twins</td>
<td></td>
<td>0.44</td>
<td>0.45</td>
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<tr>
<td></td>
<td>Between DZ twins</td>
<td></td>
<td>0.17</td>
<td>0.15</td>
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</tr>
<tr>
<td>Time 1 SCL</td>
<td>Within individuals</td>
<td></td>
<td>0.32</td>
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<tr>
<td></td>
<td>Between MZ twins</td>
<td></td>
<td>0.20</td>
<td>0.21</td>
<td>0.28</td>
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<tr>
<td></td>
<td>Between DZ twins</td>
<td></td>
<td>0.13</td>
<td>0.04</td>
<td>0.09</td>
</tr>
<tr>
<td>Time 2 SCL</td>
<td>Within individuals</td>
<td></td>
<td>0.23</td>
<td></td>
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<tr>
<td></td>
<td>Between MZ twins</td>
<td></td>
<td>0.25</td>
<td>0.24</td>
<td>0.26</td>
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<tr>
<td></td>
<td>Between DZ twins</td>
<td></td>
<td>0.12</td>
<td>0.09</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Correlations within individuals are for all individual MZ and DZ twins combined.
MD is a lifetime history of DSM-III-R major depression with an onset reported to precede the Time 1 interview.
SCL is the total Symptom Check List Depression subscale score.
The Time 1 and Time 2 assessments are separated by an average of 5 years.

Almost half of the within-subject variance in SCL depression symptom ratings is stable across two measurement occasions separated by an average of 5 years. This is consistent with findings reported for other minor psychiatric symptom-rating scales, and supports the contention that such ratings partly index factors which act to sustain subjects at a stable or characteristic symptom level (Duncan-Jones et al. 1990).

The genetic correlation between liability to reliably diagnosed LTH of MD and the temporally stable component of SCL depression symptom ratings is +0.24. This suggests that the SCL and a history of MD index mostly unique environmental risk factors when measurement error and other state effects are accounted for. Environmental risk factors for MD will accrue over the lifetime of the subject whereas environmental risk factors for subject-rated depression symptoms are likely to be those that are proximal to both the Time 1 and Time 2 administration of the SCL. Although the characteristic level of subject-rated SCL depression symptoms could conceivably index an enduring indirect effect of past experiences, the relatively low environmental correlation between LTH of MD and SCL ratings suggests that repeated administrations of the SCL index mostly proximal environmental risk factors that represent only a subset of the risk factors for MD (e.g. persistent interpersonal difficulties or recurrent stressful life events, Kendler & Gardner, 2001).

The findings of our study accord with reports describing the limited utility of self-report depression scales for identifying clinical depressive syndromes, especially when these scales are administered on only one occasion (Myers & Weissman, 1980; Lewinsohn & Teri, 1982; Wetzler, 1989; Duncan-Jones et al. 1990; Wetzler & Marlow, 1993). Our findings also underscore how the unique variance indexed by different depression measures can confound attempts to integrate aetiological theories across a disparate array of research paradigms (Radloff, 1977; Lewinsohn et al. 1988). Environmental
risk factors for symptoms of depression are clearly complex and heterogeneous (Kendler & Gardner, 2001; Kendler et al. 1987; Silberg et al. 1990) and researchers aiming to characterize environmental risk factors for depressive disorders should be cautious in their attempts to integrate their findings with those obtained for subject-rated checklists.

It is important to emphasize that not all of the estimated genetic risk factors for liability to reliably diagnosed MD are indexed by the SCL. The unique genetic variance in liability to MD that remains (39%) is, however, unlikely to be fully indexed by other subject-rated scales and checklists with properties reminiscent of the SCL. The variation in Neuroticism that is stable across measurement occasions, for example, is highly correlated (0.79–0.93) with the variation in other subject-rated symptom checklists that is stable across measurement occasions (Duncan–Jones et al. 1990). Moreover, like the SCL, temporal variation in Neuroticism ratings is not influenced by genetic or other familial factors (Eaves & Eysenck, 1976; MacKinnon et al. 1990). It is noteworthy that melancholic depression is associated with lower levels of Neuroticism, a greater number of depressive episodes, greater impairment and an increased risk for MD (more so in MZ than in DZ pairs) in this sample of twins (Kendler, 1997). Taken together, these findings suggest that the genetic risk factors for reliably diagnosed MD that are not indexed by an individual’s characteristic level of subject-rated SCL depression symptoms may be responsible for the episodicity and/or severity of major depression. It may therefore be the genetic risk factors that are not indexed by self-ratings of characteristic affective or neurotic state that will ultimately delineate the heritable basis of severe depressive disorders.

Limitations
These findings should be interpreted in light of the following limitations and caveats. First, our estimate of the prevalence of LTH of MD in women is higher than the prevalence reported for the National Comorbidity Survey (NCS) (21.3%, Kessler et al. 1994) but similar to or lower than the prevalence reported for other population-based studies (25.8% Weissman & Myers, 1978; 32.4% Rorsman et al. 1990). History of MD in the Virginia Twin Study (VTS) was assessed separately for the last-year and the lifetime prior to the last year and this protocol provides two ‘chances’ for an individual to meet criteria for MD. While the NCS utilized lay-interviewers and a highly structured psychiatric interview, an approach which may underestimate the population rates of illness (Helzer et al. 1985), the VTS employed experienced clinicians and a semi-structured interview. Like the NCS, the VTS utilized methods to encourage ‘effortful responding’. VTS subjects were somewhat younger than those assessed in the NCS which may also contribute to prevalence differences (Klerman et al. 1985). Recent studies have suggested that, compared to clinician assessments, lay interviewers using highly structured instruments may underestimate the rates of depressive illness in community samples (Eaton et al. 2000; Murphy et al. 2000). Secondly, the data analysed are for women only. Given sex differences in the prevalence and familial transmission of depression (Rice et al. 1984; Weissman et al. 1991; Wilhelm et al. 1997), the nature of the covariance between MD and SCL ratings in men and women may differ. Thirdly, we have modelled correlations of liability using the total SCL depression score and categorical diagnostic assignments. Multivariate analyses (Silberg et al. 1990) and analyses which examine and quantify individual item characteristics (Eaves et al. 1987; Gibbons et al. 1993), may shed additional light on the nature of the covariance between these different depression measures. Fourthly, the twin model used here makes a number of simplifying assumptions (Neale & Cardon, 1992), and it has relatively low power for identifying any but very sizeable effects of environmental risk factors shared by twins (Neale et al. 1994) unless they are measured directly (e.g. Kendler et al. 1992c). The findings reported here are therefore informative in so far as the model assumptions are supportable, or their violation has a negligible effect on the results obtained.

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