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Clarifying the causal relationship in women between childhood sexual abuse and lifetime major depression

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Abstract

Background—Childhood sexual abuse (CSA) is strongly associated with risk for major depression (MD) but the degree to which this association is causal remains uncertain.

Method—We applied structural equation modeling using the Mplus program to 1493 longitudinally assessed female twins from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders.

Results—Our model included (i) retrospective self- and co-twin reports on CSA, (ii) major potentially confounding covariates, (iii) assessment of lifetime history of MD at two separate interviews, and (iv) mood-congruent recall (implemented by allowing current depressive symptoms to predict reporting of CSA). In a model with only measurement error, CSA explained 9.6% of MD. Including four key covariates reduced the variance explained to 5.3%, with the largest effects found for parental loss and low parental warmth. Adding the effect of mood-congruent recall to a final well-fitting model reduced the percentage of variance explained in lifetime MD (LTMD) by CSA to 4.4%. In this model, current depressive symptoms significantly predicted recall of CSA.

Conclusions—In a model correcting for measurement error, confounding and the impact of mood-congruent recall, CSA remains substantially associated with the risk for LTMD in women. These findings strongly suggest, but do not prove, that this association is causal, and are consistent with previous results in this sample using a co-twin control design, but also indicate that more than half of the uncorrected CSA–MD association is probably not causal. Traumatic life experiences contribute substantially to the risk for LTMD.

Keywords

Causal relationship; childhood sexual abuse; major depression; mood-congruent recall

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Declaration of Interest
None.

Introduction

No problem in psychiatric epidemiology is more important or more difficult than that of clarifying the causal relationship between disorders and the experience of adversity. Such relationships cannot be studied in a controlled manner. Of the various adversities, childhood sexual abuse (CSA) is especially problematic. Only a small percentage of cases are reported to legal authorities, so relying on official records produces an unrepresentative sample (Ammerman, 1998). Legal reporting requirements for suspected CSA in many countries make it challenging to study using prospective designs. Therefore, an important tool in understanding the potential pathogenic effects of CSA has been to examine its effect using retrospective designs from adult samples. Using this approach, a strong association has consistently been shown with a wide range of psychiatric and substance use disorders and particularly major depression (MD) (Fergusson & Mullen, 1999; Kendler *et al*. 2000; Cong *et al*. 2011).

However, accurately estimating the direct effect of CSA on lifetime MD (LTMD) using a retrospective design is challenging and requires examining biasing infiuences that might upwardly and downwardly alter the true causal relationship. Downward biases are probably more easily corrected. Lifetime histories of both MD and CSA are reported with only moderate reliability (Aneshensel *et al*. 1987; Fergusson *et al*. 2000). This unreliability can be corrected for with multiple reports.

More challenging is to correct for non-causal sources that upwardly bias the CSA–MD association. Two are noteworthy. Potential confounders such as low social class, parental psychopathology or poor parenting behaviors could independently increase risk for both CSA and MD. If well measured, these confounders can be corrected with standard statistical approaches and co-relative designs. A previous analysis in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD) showed that rates of psychiatric disorders including MD were elevated in twins exposed to CSA compared to their unexposed co-twins (Kendler *et al.* 2000).

A more problematic source of non-causal association between CSA and MD arises from reporting bias. A substantial proportion of women with officially recorded CSA fail to report abuse when interviewed in adulthood (Widom & Shepard, 1996; Maughan & Rutter, 1997; Widom & Morris, 1997). Of particular concern is that, for individuals with CSA, their psychological state at the time of interview, particularly their level of depressive affect, could impact recall and reporting (Lewinsohn & Rosenbaum, 1987). A dysphoric mood can, through a psychological process termed mood-congruent recall (or mood-congruent memory), increase the salience of prior negative experiences leading to improved recall (Bower, 1981; Blaney, 1986; Ellis & Moore, 1999). Mood-congruent recall is defined as ‘a phenomenon in which emotional material is remembered more reliably in moods that match the emotional content of the memories.’ (Lewis & Critchley, 2003, p. 431). A good example of this process arose when, in the VATSPSUD, we examined individuals who had reported a lifetime history of MD at an earlier interview. In accord with prior research (Aneshensel *et al*. 1987), their probability of reporting a depressive episode at a subsequent interview was strongly infiuenced by their level of current depressive symptoms (Kendler *et al*. 2001). The

more depressed they were at second interview, the more likely they were to recall the depressive episode they had reported previously.

Mood-congruent recall might upwardly bias the association observed between CSA and MD problems because individuals with LTMD, on average, have higher levels of current depressive symptoms (Foley *et al*. 2001). Thus we might have a non-causal pathway that runs from LTMD → current depressive symptoms → increased recall of CSA.

In this study, we developed a sequence of latent variable structural models using data from VATSPSUD to investigate the relationship between CSA and LTMD. First, we accounted for errors of measurement in CSA and MD variables using a common factor model from two reports for each measure. Second, we included key background covariates to determine the degree to which these sources altered the magnitude of the estimated CSA → MD effect. Third, we examined the potentially biasing infiuence of current mood state on recall of self-reported CSA.

Method

Sample

Participating twins came from female–female pairs from the VATSPSUD, the details of which have been outlined elsewhere (Kendler & Prescott, 2006). The twin pairs, born from 1934 to 1974, became eligible to participate if both members responded to a mailed questionnaire, the response rate to which was around 64%. Of the eligible sample, 92% were first interviewed face to face in 1987–1989, at which time their mean age was 30.1 (S.D. =7.6, range 17–55) years. These participants were the subjects of three additional telephone interview waves, with participation rates of 88–92%. The last of these waves was completed in 1995–1997, an average of 92±7 months after their first assessment. As approved by the Virginia Commonwealth University Institutional Review Board (VCU IRB), written consent was obtained in this study for all face-to-face interviews and assent was obtained for telephone interviews and mailed questionnaires.

LTMD was diagnosed based on personal interview by clinically experienced interviewers using an adaptation of the SCID (Magee *et al*. 1996) that included DSM-III-R criteria (Fawcett, 1997) at the first and fourth waves. At each interview, the risk for MD was assessed separately for the past year and for time periods prior to the past year, and added together to produce a lifetime prevalence. We also used multiple prompts to encourage ‘effortful recall’.

During our third-wave interview, we examined the willingness of twins to answer questions about CSA and their preferred method of assessment. A large majority of women (75%) said they would be happy to participate and preferred to respond using a self-report questionnaire. Therefore, we assessed CSA by questionnaire at our fourth-wave interview using items developed by Martin *et al*. (1993). Our initial question was: ‘Before you were 16, did any adult, or any other person older than yourself, involve you in any unwanted incidents like (i) inviting or requesting you to do something sexual, (ii) kissing or hugging you in a sexual way, (iii) touching or fondling your private parts, (iv) showing their sex

organs to you, (v) making you touch them in a sexual way, or (vi) attempting or having sexual intercourse?' We term these forms of CSA sexual invitation, sexual kissing, fondling, exposing, sexual touching, and intercourse respectively. We subdivided CSA into three exclusive, hierarchical categories: (i) no genital contact (sexual invitation, sexual kissing and/or exposing), (ii) genital contact but no intercourse (fondling and sexual touching) and (iii) intercourse. We refer to these as non-genital CSA, genital CSA and intercourse, respectively. We also asked each twin to respond to the same questions about CSA in her co-twin, adding the response option 'Not sure'.

We used four covariates in our analyses. Parental loss was assessed at the first interview and recorded dichotomously according to whether or not the twin lived with both biological parents continuously up to the age of 16. Parental socio-economic status (SES) was operationalized at the first interview by years of education of the breadwinner and ranged from 2 to 17. Parental warmth was defined from the Parental Bonding Instrument (PBI; Parker *et al*. 1979) using items outlined previously (Kendler, 1996), as reported by the twins at the second interview. An average of the maternal and paternal warmth sum scores was created. Lifetime parental MD was assessed at personal interview of parents using the same interview as that used with the twins for those who were alive and among the 87% who were cooperative (Kendler & Prescott, 2006) and by family history from the twins using Family History Research Diagnostic Criteria (FH-RDC) criteria (Endicott *et al*. 1975) for those who were not interviewed. This was a binary variable that refiected whether one parent or both parents *versus* neither parent had a lifetime history of MD.

Current depressive symptoms (CDS) were assessed by nine items from the Symptom Checklist 90 (SCL-90; Derogatis *et al*. 1973) completed by the twins as part of their fourth-wave interview. These items describe the intensity of depression-related symptoms experienced over the past 30 days. Representative items were 'Loss of sexual interest or pleasure', 'Feeling low in energy or slowed down', 'Feeling blue' and 'Feelings of worthlessness'.

Statistical analysis

All structural equation models were fit to ordinal data using the limited information robust weighted least-squares mean- and variance-adjusted estimator in Mplus version 6.12 (Muthén & Muthén, 2007). Fit indexes and standard errors were adjusted for the non-independence of twin members using the complex sampling approach in Mplus. An initial exploratory factor analysis was performed on the self/co-twin items coding the three forms of CSA.

Three fit indices and the Mplus robust χ^2 difference test were used to evaluate model fit and comparison among restricted models. The Tucker–Lewis Index (TLI; Tucker & Lewis, 1973) and the Comparative Fit Index (CFI; Bentler, 1990) are relative fit indices ranging between 0 and 1, with values ≥0.95 considered to indicate good-fitting models. The root mean square error of approximation (RMSEA; Steiger, 1990) was developed from the understanding that no model is an exact representation of data. RMSEA values of ≥0.05 are considered to be good approximations.

Three different models were developed to examine the relationship between CSA and LTMD. The models progressively estimated the CSA → LTMD structural effect (1) with no background covariates, (2) including covariates, and (3) adding congruent recall bias effects. Additional hypotheses were tested by placing restrictive constraints on specific pathways and testing the misfit incurred using robust χ^2 difference testing.

Results

Our sample included 1493 women who had completed information on CSA and LTMD and had reports from their co-twins. The rate of total CSA from self- and co-twin reports in this sample was 26% and 10% respectively, broken down as follows: self-report: non-genital 5%, genital 13% and intercourse 8%; co-twin report: non-genital 3%, genital 5% and intercourse 2%. The prevalences of LTMD reported at the first and fourth wave interviews were 30% and 29% respectively.

Model 1

Our first model, as depicted in Fig. 1, has three note-worthy features. First, the latent construct of experienced CSA displayed high loadings for both self-report (SR) and co-twin report (CR) and of non-genital (n), genital (g) and intercourse (i) levels of abuse. For both self- and co-twin report, genital-level sexual abuse (both +0.99) was the strongest indicator of the CSA factor. Second, LTMD had estimated equated factor loadings of +0.70. Third, the standardized estimated path from the CSA factor to the LTMD factor was +0.31, indicating that CSA accounted for 9.6% of the variance in liability to LTMD. This model fits these data well according to the relative fits (CFI =0.99, TLI=0.99) but the approximate fit was marginal (RMSEA=0.08).

Model 2

The second model, depicted in Fig. 2, adds to our first model four potentially important background variables that might predict both CSA and LTMD and hence bias upward our estimates of the CSA–LTMD association. The strongest associations with CSA and LTMD are negative from parental warmth (P warm) and positive from a history of parental loss (P loss). Weaker effects are seen for a parental history of MD (P MD) and parental SES (P SES). Including these covariates reduces the direct path from CSA to LTMD to +0.23 so that, in this model, CSA is explaining 5.3% of the variance in liability to LTMD. Overall model fit indexes were again very good (CFI =0.99, TLI=0.99), with some improvement in approximate fit (RMSEA=0.07).

Model 3

In the third model, shown in Fig. 3, a factor representing the potential impact of mood-congruent recall was added. Reporting bias was modeled as direct pathways from the CDS factor defined by nine SCL items assessing depressive symptoms at the time that the self-report history of CSA was obtained. This model includes both mediated and direct paths between CSA, LTMD and CDS. The mediational pathway (LTMD → CDS) point estimate was + 0.45 whereas the direct effect (CSA → CDS) was −0.03 (and not significantly different from zero). Most importantly, the pathways from the CDS factor to the self-report

of having experienced the three forms of CSA were all positive and significant. That is, this model suggests that individuals with higher levels on the CDS factor are more likely to recall and report CSA. Although the CDS bias parameters differed in their effect size across the three forms of experienced CSA, a test constraining the parameters to be equal was non-significant ($\Delta\chi^2$=3.1, df =2, p=0.21). Including the potential impact of mood-congruent recall further reduced the direct path coefficient from CSA to LTMD to + 0.21, accounting for 4.4% of the variance in liability to LTMD. This model maintained good relative fit (CFI =0.99, TLI=0.99) and there was a clear improvement in the approximate fit (RMSEA =0.04).

Discussion

The goal of this study was to clarify the relationship between CSA and LTMD by examining their association in three different models. In a model accounting for measurement error, background covariates and mood-congruent recall bias, CSA predicted risk for LTMD accounting for 4.4% of the population variance in liability. However, background covariates and reporting bias together accounted for more than half of the uncorrected association between CSA and LTMD. Thus, not considering these additional sources of covariance would lead to a substantial overestimate of the magnitude of the impact of CSA on depression.

Five more detailed results are also noteworthy. First, the latent liability to experience CSA was strongly indexed by reports of both the responding twin and her co-twin. As expected, retrospective reports by relatives provide useful information about a history of CSA.

Second, as shown previously (Bromet *et al*. 1986; Prusoff *et al*. 1988; Kendler *et al*. 1993), the reliability of the assessment of LTMD is moderate. Considerable additional information about the underlying risk for MD is obtained in longitudinal studies by interviewing subjects twice.

Third, model 3 had a non-significant direct effect of CSA on the current depressive symptom factor but showed a significant mediated effect (+0.45) going through LTMD. That is, allowing for an indirect effect of CSA on CDS through LTMD, CSA had no additional prediction on current depressive status. Our results are consistent with prior studies suggesting that individuals with CSA are, on average, somewhat more symptomatic than unexposed individuals (Kendall-Tackett *et al*. 1993), although the magnitude of this effect may be modest (Rind & Tromovitch, 1997).

Fourth, of our four covariates, parental loss and low parental warmth each predicted non-trivial proportions of variance of the risk for both CSA and LTMD, and thus refiected important confounders of the CSA–MD association. By comparison, parental SES and parental history of MD both had much smaller impacts on the relationship between CSA and LTMD.

Fifth, we found positive and significant paths from current depressive symptoms to reporting a history of CSA. That is, controlling for all other variables in our model, subjects with high levels of depressive symptoms were significantly more likely to report CSA than those with

few depressive symptoms. However, the magnitude of these effects overall is relatively modest. By comparing models, we can estimate that reporting bias accounts for only about 17% of the observed covariance between CSA and LTMD, controlling for the effects of the confounders included in the second model. Our results are consistent with the predictions of mood-congruent recall. They are also in accordance with our prior finding that the association between CSA and LTMD was moderately stronger using self-report measures of CSA [odds ratio (OR) of 1.93 for any CSA and 3.14 for intercourse] than using co-twin reports (OR of 1.58 for any CSA and 2.65 for intercourse) (Kendler *et al*. 2000). The association between current depressive symptoms and CSA reporting could arise in two different ways that cannot be discriminated definitively in our study. High levels of depressive symptoms could be associated with an increased rate of true positive reporting of CSA so that the overall accuracy of CSA assessment is higher in symptomatic than in asymptomatic individuals. Alternatively, high levels of depression could increase false-positive reporting of CSA so that the overall accuracy of reporting CSA is higher in asymptomatic than in symptomatic individuals. We consider the first mechanism to be considerably more plausible than the second for three reasons.

First, throughout survey research, false-negative reporting is much more commonly biased that false-positive reporting (e.g. Coughlin, 1990; Brown & Adams, 1992; Wu *et al*. 2000). This is especially true for traits or behaviors that are socially stigmatizing and potentially shame inducing such as drug use, sexual behaviors or personal and family histories of psychiatric illness (Siemiatycki, 1979; Orvaschel *et al*. 1982; ACSF, 1992; Roy *et al*. 1996; Hser, 1997; Moffitt *et al*. 2010). Second, applying sophisticated modeling to assessments of CSA at two time points in a population-based cohort, Fergusson *et al*. (2000) found that reporting of experienced CSA in adulthood is relatively unreliable, with approximately 50% of subjects who report CSA at one wave denying it at the next wave. Importantly, their modeling suggested that nearly all this unreliability resulted from false-negative and not false-positive reporting. Third, a considerable body of empirical work on mood-congruent recall supports the plausibility of this hypothesis. An earlier review of this area concluded that ‘the support for claims of such a phenomenon is impressive in its size, consistency, and diversity’ (Blaney, 1986). For example, individuals with depressive symptoms tend to display an even-handed recall of positively and negatively valenced stimuli whereas asymptomatic individuals are typically biased toward the recall of positive stimuli (Bradley & Mathews, 1983; Mathews & Bradley, 1983; Gilboa *et al*. 1997; Geerlings *et al*. 1998). In more naturalistic settings, depressed mood has been associated with enhanced recall of negative information including parenting behavior (Lewinsohn & Rosenbaum, 1987). A distinct body of work on cognitive theories of depression suggests that, as individuals enter into a depressed state, negative memories gain in salience (Dalgleish & Watts, 1990; Lemogne *et al*. 2006). We hypothesize that the paths in our model from current symptoms to recall of CSA refiect the process of mood-congruent recall whereby individuals in a depressed state are better able to recall and report prior CSA.

The overall impact of CSA on MD, correcting for errors of measurement, confounders and effect of mood congruent recall, was moderate, explaining slightly less than 5% of the population variance in liability. At a population level, CSA therefore explains about one-

eighth as much of the overall risk for MD as total genetic effects (given a best estimate of heritability of 37%; Sullivan *et al*. 2000). From the path estimates and our prevalence figures for CSA and MD from our best-fit final model, the OR between CSA and MD can be estimated to equal 1.83. This substantial effect reinforces the importance of traumatic environmental experiences as risk factors for MD. In comparison, all of the genome-wide significant genetic variants found in the largest such study to date for MD (which emerged when including bipolar illness) had ORs for the pathogenic allele of around 1.10 (Major Depressive Disorder Working Group of the Psychiatric GWAS Consortium, 2013).

Our results can be usefully compared with a recent study with similar aims by Fergusson *et al*. (2011). The authors constructed a path model examining retrospective reports of CSA and current mental health in a population-based cohort assessed at ages 18 and 21 years. They modeled recall bias for CSA and found it to be present but small in magnitude, accounting for less than 1% of recall bias. Consistent with our own findings, they suggest that retrospective recall bias for childhood abuse may not be large enough to pose a major threat to study validity.

Although we should be most concerned about aspects of our analyses that upwardly bias the CSA– MD association, it is worth considering the reverse possibility. In particular, in a proportion of cases, parental separation or MD could be a result of, rather than contributing to, CSA. If this is the case, then we have overcorrected and are at risk of underestimating the direct component of the CSA–MD relationship.

Our results suggest that CSA has a direct effect on increasing risk for MD in women. How confident should we be about this conclusion based solely on observational data? The greatest concern is that we have inadequately captured the confounding variables, and that had a larger set of covariates been considered, the association between CSA and MD would have disappeared. Two prior studies in the VATSPSUD argue against this concern. First, using more traditional regression analysis, we have previously examined a more complete set of potential covariates and shown that they had very little additional impact on the association between CSA and MD (Kendler *et al*. 2000). Second, confounding due to unknown and unmeasured variables is always a possibility in observational data. We also conducted a previous co-twin control analysis looking at the 53 twin pairs in our sample discordant for exposure to CSA (Kendler *et al*. 2000). Within those pairs, who were matched for all features of their rearing environment and partly or entirely for their genotype, the exposed twin was significantly more likely to report MD with an OR (2.43) only moderately lower than that observed in the entire population. These results, which were broadly replicated in an Australian twin sample (Nelson *et al*. 2002), are inconsistent with the hypothesis that, with proper correction for confounding variables, the association between CSA and MD would disappear. In summary, although caution is always indicated in attempting to infer causal processes from non-experimental data, we would argue that our analysis provides substantial evidence that the experience of CSA in women causally increases risk for the subsequent experience of LTMD.

Limitations

Our findings should be considered in the light of five potential methodological limitations. First, our subjects are all white twins born in Virginia. Although the VATSPSUD has been shown to be broadly representative of the USA for a range of relevant traits (Kendler & Prescott, 2006), the degree to which these findings extrapolate to other samples is uncertain. Second, our subjects were all female. The experience of CSA may differ considerably between the sexes (Fergusson & Mullen, 1999) so our results may not extrapolate to males. Third, our rates for LTMD are higher than those found in many epidemiological surveys. However, we had trained mental health professionals as interviewers, asked in two separate interview sections about LTMD and used several prompts to encourage effortful reporting. Given that false-negative reporting is so much more of a problem than false-positive reporting for lifetime psychopathology (Moffitt *et al*. 2010), we are inclined to think that our assessment of LTMD is in general fairly accurate. Fourth, rates of CSA based on co-twin reports were considerably lower than those based on self-report. However, this discrepancy can be largely explained by the fact that more than one-third of women in our sample reported that they had never told anyone about their CSA (Kendler *et al*. 2000). Of note is that, in our models, co-twin report of CSA was almost as strong an index of the latent liability to CSA as was self-report. Fifth, we assessed CSA by mailed questionnaire. This, however, was the clear preference of this sample. Survey data suggest that more ‘anonymous’ response modes may obtain better cooperation and reporting of highly sensitive material such as CSA (Siemiatycki, 1979).

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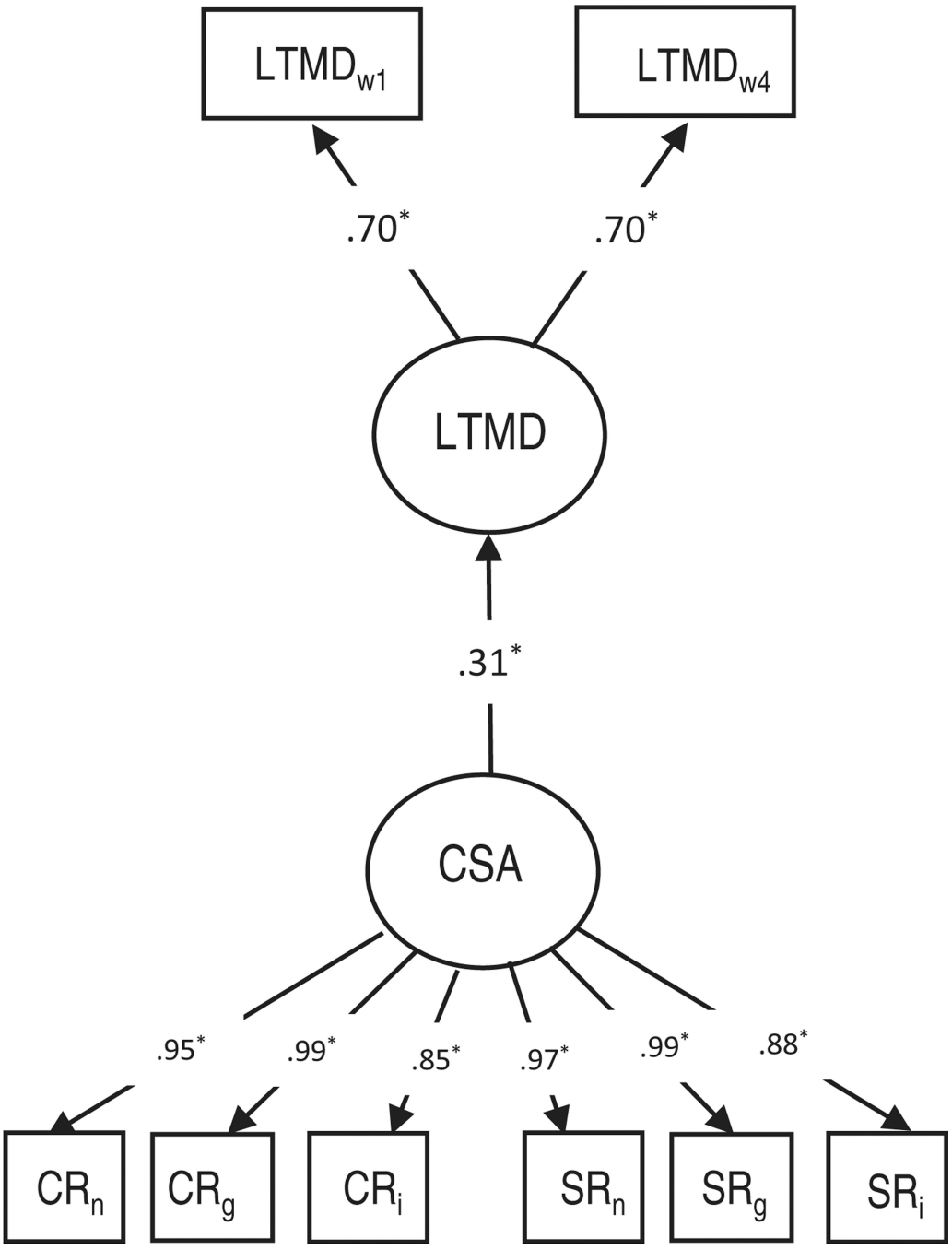

Fig. 1.

The prediction of lifetime major depression (LTMD) in women from the experiences of childhood sexual abuse (CSA). The CSA is a latent variable indexed by co-twin report (CR) and self-report (SR) of three levels of CSA: non-genital (n), genital (g) and intercourse (i). LTMD is a latent variable indexed by the lifetime history of MD reported at the first and fourth waves of interviews (w1 and w4 respectively) in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD). The path values are standardized partial regression coefficients and so need to be squared to equal the proportion of variance

in the dependent variable accounted for by the independent variable. Path coefficients with an asterisk (*) are individually statistically significant.

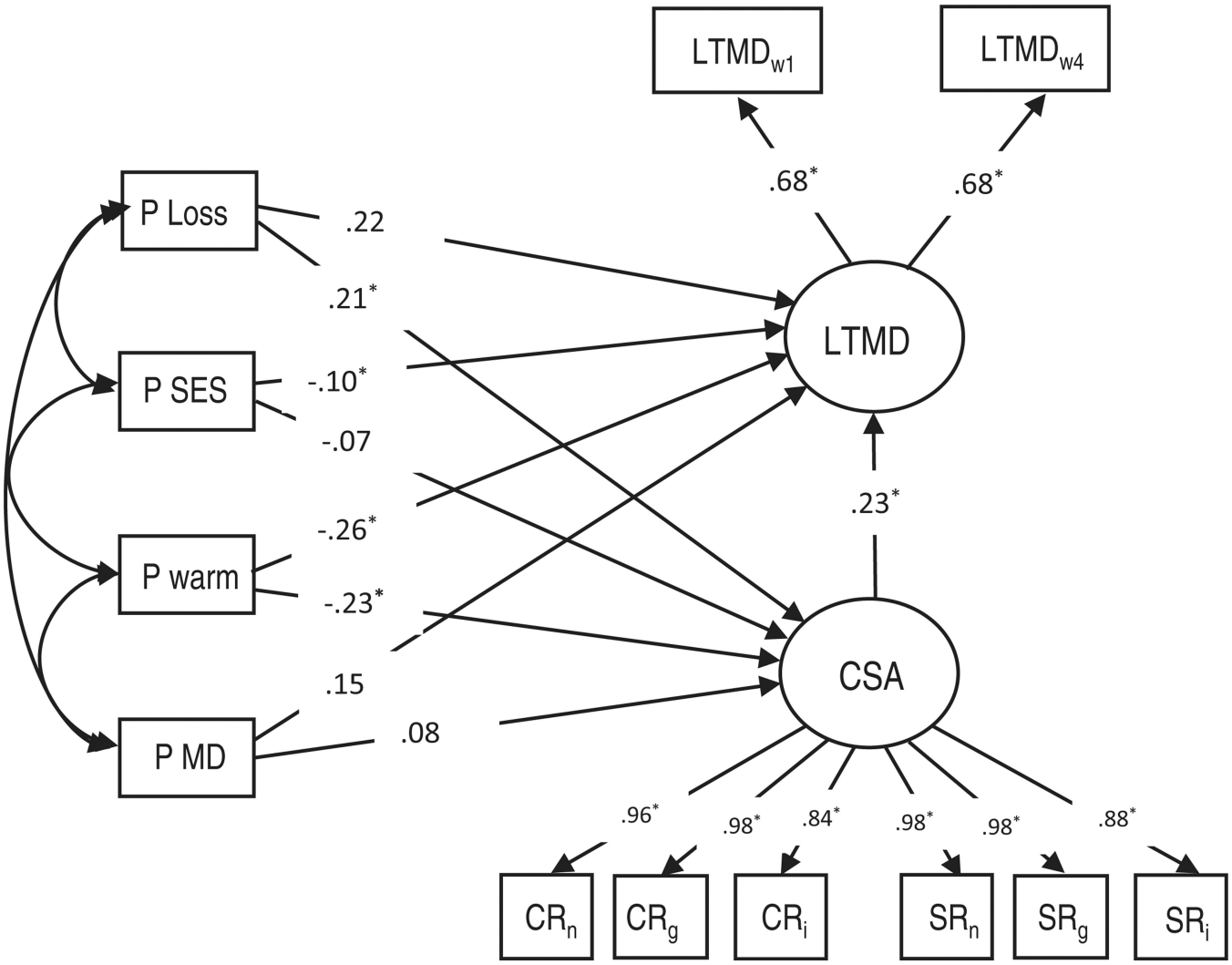

Fig. 2.

The prediction of lifetime major depression (LTMD) in women from the experiences of childhood sexual abuse (CSA) and four potentially important confounder variables. The model is identical to that depicted in Fig. 1 with the addition of a history of parental loss before the age of 16 (P loss), parental socio-economic status (P SES), as indexed by the highest education of the breadwinner in the home of origin, parental warmth (P warm), as measured by the Parental Bonding Instrument (PBI; Parker *et al.* 1979), and a lifetime history of parental major depression (P MD), as assessed by personal interview and DSM-III-R criteria (Fawcett, 1997) or the Family History Research Diagnostic Criteria (FH-RDC; Endicott *et al.* 1975). Path coefficients with an asterisk (*) are individually statistically significant.

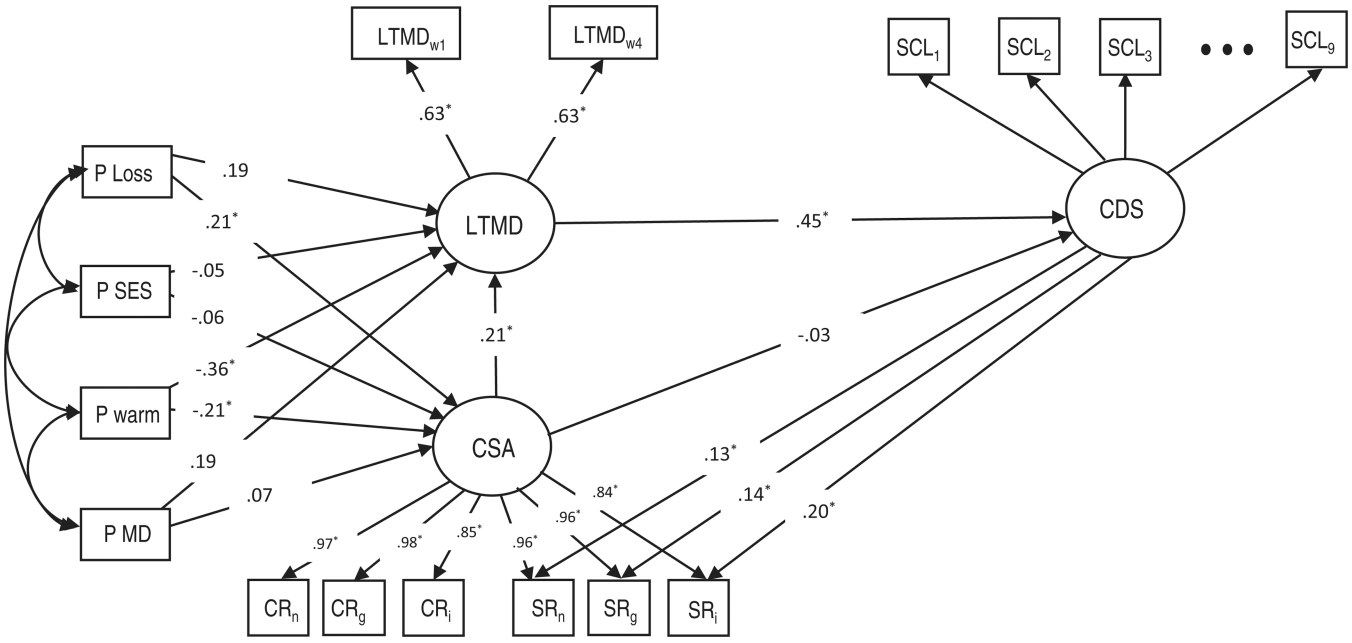

Fig. 3.

The prediction of lifetime major depression (LTMD) in women from the experiences of childhood sexual abuse (CSA), four confounding variables and a correction for the effects of mood-congruent recall. The model is identical to that depicted in Fig. 2 with the addition of current depressive symptoms (CDS) as indexed by nine items from the Symptom Checklist 90 (SCL-90; Derogatis *et al*. 1973) assessing current symptoms of depression with paths representing the effect of mood-congruent recall from CDS to non-genital (n), genital (g) and intercourse (i) forms of CSA. The model also contains paths from LTMD and CSA to CDS. Path coefficients with an asterisk (*) are individually statistically significant.