Childhood Sexual Abuse and Adult Psychiatric and Substance Use Disorders in Women

An Epidemiological and Cotwin Control Analysis

Kenneth S. Kendler, MD; Cynthia M. Bulik, PhD; Judy Silberg, PhD; John M. Hettema, PhD, MD; John Myers, MS; Carol A. Prescott, PhD

Background: Women who report childhood sexual abuse (CSA) are at increased risk for developing psychiatric disorders in adulthood. What is the diagnostic specificity and cause of this association?

Methods: In a population-based sample of 1411 female adult twins, 3 levels of CSA were assessed by self-report and cotwin report: nongenital, genital, and intercourse. Interviews with twins and parents assessed family background and diagnoses of psychiatric and substance dependence disorders. Odds ratios (ORs) were calculated by logistic regression.

Results: By self-report, 30.4% reported any CSA and 8.4% reported intercourse. Self-reported CSA was positively associated with all disorders, the highest ORs being seen with bulimia and alcohol and other drug dependence. The ORs were modest and often nonsignificant with nongenital CSA and increased with genital CSA and especially intercourse, where most ORs exceeded 3.0. A similar pattern of findings was seen with CSA as reported by the cotwin, although many ORs were smaller. Controlling for family background factors and parental psychopathology produced a small to modest reduction in ORs. In twin pairs discordant for CSA, the exposed twin was at consistently higher risk of illness.

Conclusions: Women with CSA have a substantially increased risk for developing a wide range of psychopathology. Most of this association is due to more severe forms of CSA and cannot be explained by background familial factors. Although other biases cannot be ruled out, these results are consistent with the hypothesis that CSA is causally related to an increased risk for psychiatric and substance abuse disorders.

Arch Gen Psychiatry. 2000;57:953-959

Although women who report a history of childhood sexual abuse (CSA) are clearly at increased risk for psychiatric disorders in adulthood,1-6 4 critical issues about this association remain unclear. First, what is the magnitude of the association given that the impact of CSA on current adjustment may be modest?7-10 Second, CSA often occurs along with multiple other risk factors that reflect disturbed family and parent-child relationships.4,5,9,10 Is the CSA-psychopathology association causal or is it due to these confounded risk factors?11 Third, CSA is a sensitive subject. Could the CSA-psychopathology association arise through reporting bias wherein persons with psychiatric illness are more likely to recall and report abuse experiences?12,13 Fourth, how specific are the effects of CSA? Does CSA increase risk for only 1 or 2 psychiatric disorders or for a wide range of psychopathological disorders, including psychoactive substance use disorders? We examine these questions in 1411 female twins ascertained from a population-based registry.

RESULTS

DESCRIPTION

Of 1942 individual twins completing our fourth-wave interview, 1411 (72.7%) returned the mailed questionnaire with CSA information. Sexual invitation, sexual kissing, fondling, and exposing had similar prevalences (17%-21%) (Table 1). Sexual touching and intercourse were less common. Of 427 individuals (30.3%) who reported 1 or more episodes of any form of CSA, age at the first incident was 10.2±3.5 years; multiple perpetrators and female perpetrators were reported by 32.9% and 9.9%, respectively, and 35.2% reported being forced or threatened by the perpetrator(s), the ages of whom were most commonly 25 to 49 years (29.5%), followed by 15 to 18 years (21.8%), younger than 15 years...
PARTICIPANTS AND METHODS

SAMPLE

Participating twins came from an ongoing investigation of female-female twin pairs from the population-based Virginia Twin Registry, the details of which have been outlined elsewhere.14-16 The pairs derive from 2 related samples, born between 1934 and 1974, who became eligible to participate if both members responded to a mailed questionnaire (response rate was about 64%). Eighty-eight percent of our sample was first interviewed face to face in 1987-1989 (at which time their mean ± SD age was 30.1 ± 7.6 years [range, 17.0-55.0 years]) and has been the subject of 3 additional telephone interview waves. The last of these waves was completed in 1995 through 1997, a mean ± SD of 92 ± 7 months after their first assessment. The remaining 12% were first interviewed face to face in 1992 through 1994 and assessed a second time (with the same interview given to the rest of the sample during the fourth wave) by telephone in 1996 through 1997. As approved by the institutional review board of Virginia Commonwealth University, Richmond, written consent was obtained in this study for all face-to-face interviews, and assent was obtained for telephone interviews and mailed questionnaires.

PSYCHIATRIC DIAGNOSES

Psychiatric and substance use disorders were diagnosed via personal interview by clinically experienced interviewers using an adaptation of the Structured Clinical Interview for DSM-III-R17 and DSM-III-R18 criteria, with 2 exceptions. First, a 1-month duration of illness for generalized anxiety disorder (GAD) was used.19 Second, diagnostic hierarchies were ignored.

A number of psychiatric and substance use disorders were assessed multiple times in these twins. We used lifetime diagnoses of major depression (MD), GAD, and alcohol and other drug dependence as assessed at the fourth interview. Lifetime panic disorder and bulimia nervosa (BN) were assessed at earlier interviews only (waves 1 and 3), and so those diagnoses were used here. For further details of the diagnostic algorithms and diagnostic reliability, see Kendler et al.14,19-22 No other disorders were assessed except for phobic disorders, which we do not report because their early onset14 makes a causal interpretation of their associations with CSA problematic.

ASSESSMENT OF CHILD ABUSE

During our second-wave interview, we examined the willingness of twins to answer questions about CSA and their preferred method of assessment. 0.8% of women said they would not cooperate with such a survey. Of the 1337 twins who preferred 1 mode of assessment, 108 preferred face-to-face interview, 229 preferred telephone interview, and 1000 preferred mailed questionnaire. Childhood sexual abuse was assessed by mailed questionnaire using items developed by Martin et al.23 Our initial item was as follows:

Before you were 16, did any adult, or any other person older than yourself, involve you in any unwanted incidents like (1) inviting or requesting you to do something sexual, (2) kissing or hugging you in a sexual way, (3) touching or fondling your private parts, (4) showing their sex organs to you, (5) making you touch them in a sexual way, or (6) attempting or having sexual intercourse.

We term these forms of CSA sexual invitation, sexual kissing, fondling, exposing, sexual touching, and intercourse, respectively. The possible responses were “never,” “once,” and “more than once.” We also asked each twin the same questions about CSA in her cotwin, adding the response option “not sure.”

FAMILY ENVIRONMENT AND PARENTAL PSYCHOPATHOLOGY

Twins provided the addresses of their living biological parents. Of 1632 parents who were identified and eligible, 1472 (90.2%) (mean ± SD age, 58.6 ± 9.3 years) were successfully interviewed, including 855 mothers and 617 fathers.

We assessed lifetime history of MD, GAD, panic disorder, and alcohol dependence. In using parental diagnoses as covariates, we used the same diagnosis in parents that we were examining in their daughters. Lacking parental diagnoses of BN and drug dependence, we used, as proxies, MD and alcohol dependence, respectively.

We assessed retrospectively, for the time the twins were growing up, a range of measures of family disruption. For some variables, we had assessments from parents (P) and twins (T): (1) 7 items on quality of the parental relationship (P)26; (2) 3 factors on quality of the parent-child relationship from 16 items from the Parental Bonding Instrument (warmth, protectiveness, and authoritarianism)27,28 (P, T); (3) 4 items reflecting family financial status (P); (4) 2 factors derived from 14 items from the Family Environment Scale29 reflecting discordant vs harmonious qualities of the home environment (P, T); (5) frequency of family church attendance (P); (6) years of parental education (P, T); (7) 2 factors of parental disciplinary practices from 8 items, including spanking, slapping, and hitting, adapted from the Home Environment Interview30 (P, T); and (8) prolonged parent-child separations (P, T).

STATISTICAL ANALYSIS

We examined the association between CSA and psychiatric and drug abuse disorders using logistic regression, correcting for the correlated structure of the data with the General Estimating Equation (GEE) option in the SAS routine GENMOD.31 Odds ratios (ORs) and their associated 95% confidence intervals (CIs) were obtained from logistic regression. To examine the relationship between CSA and comorbidity, we predicted the risk of having 2 or more of the 6 individual diagnoses.

Our cotwin control analyses examined only pairs discordant for CSA and the relevant disorder, in which the OR is defined as the ratio of the number of pairs where the abused twin vs the nonabused twin was affected. Because of the small numbers involved, we calculated the exact 1-tailed P value from the binomial distribution, predicting higher risk in the exposed twin. Although previous literature suggesting an association between CSA and psychopathologic disorders justifies 1-tailed tests, we report 2-tailed P values to reduce the risk of type I errors.
VALIDITY OF MEASURE OF CSA

We subdivided CSA into 3 exclusive, hierarchical categories: (1) no genital contact (sexual invitation, sexual kissing, and exposing) (n = 110, 7.8%), (2) genital contact but no intercourse (fondling and sexual touching) (n = 199, 14.1%), and (3) intercourse (n = 118, 8.4%). We refer to these as nongenital CSA, genital CSA, and intercourse, respectively.

In the 506 pairs for whom reports were available on both members, the level of resemblance of self-reported CSA was highly significant ($\chi^2=123.7$, $P<.001$; contingency coefficient = 0.44, weighted $k = 0.36$ [95% CI, 0.29-0.44]). In examining the relationship between self-reported and cotwin-reported CSA, we had self-reports and cotwin reports from each twin, resulting in 903 twin-cotwin pairs. Although the relative frequencies of the types of CSA were similar in the self-report and cotwin report, the absolute rates of CSA, when reported by cotwins, were about half those reported by the twin herself (Table 1).

ASSOCIATION OF SELF-REPORTED CSA AND PSYCHIATIC AND SUBSTANCE USE DISORDERS

Table 2 presents the association between 6 lifetime psychiatric and substance use disorders and any self-reported CSA as well as these associations across the 3 levels of CSA, in each case being compared with twins who reported no CSA. All disorders, except BN, were significantly associated with any CSA. The significant ORs for the 3 psychiatric disorders (MD, GAD, and panic disorder) were all approximately 1.9, whereas the ORs for alcohol and other drug dependence were 2.8 to 3.1.

Nongenital CSA was associated with significantly increased risk for developing alcohol and other drug dependence only. Genital CSA was significantly associated with every disorder except panic disorder and BN. All disorders were significantly associated with intercourse, with ORs for the psychiatric disorders ranging from 2.6 to 3.3 and exceeding 4.0 for alcohol and other drug dependence. Comorbidity was significantly predicted by all forms of CSA.

ASSOCIATION WITH COTWIN REPORT

The pattern of association between cotwin-reported CSA and lifetime disorders is different (Table 3). For GAD and drug dependence, the ORs with cotwin report are substantially lower than seen with self-report and no longer significant. For MD, panic disorder, and alcohol dependence, the pattern of ORs is modestly attenuated, and many comparisons remain significant. For BN, the association with CSA is stronger with cotwin report. Using cotwin report, comorbidity is significantly predicted by any CSA, genital CSA, and intercourse.

Table 1. Rates of Specific Forms of Childhood Sexual Abuse by Self-report and Cotwin Report

<table>
<thead>
<tr>
<th>Childhood Sexual Abuse</th>
<th>Self-report (n = 1411)</th>
<th>Cotwin Report (n = 1219-1259)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexual invitation</td>
<td>263 (18.6)</td>
<td>120 (9.5)</td>
</tr>
<tr>
<td>Sexual kissing</td>
<td>240 (17.0)</td>
<td>89 (7.2)</td>
</tr>
<tr>
<td>Fondling</td>
<td>297 (21.0)</td>
<td>128 (10.3)</td>
</tr>
<tr>
<td>Exposing</td>
<td>236 (16.8)</td>
<td>98 (8.1)</td>
</tr>
<tr>
<td>Sexual touching</td>
<td>149 (10.6)</td>
<td>55 (4.5)</td>
</tr>
<tr>
<td>Intercourse</td>
<td>118 (8.4)</td>
<td>38 (3.1)</td>
</tr>
</tbody>
</table>

Table 2. Childhood Sexual Abuse (CSA) by Self-report and the Odds Ratios for Psychiatric and Substance Use Disorders

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Any CSA (n = 1403-1410)</th>
<th>Nongenital CSA (n = 1089-1094)</th>
<th>Genital CSA (n = 1179-1183)</th>
<th>Intercourse (n = 1097-1101)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depression</td>
<td>1.93† (1.52-2.44)</td>
<td>1.37 (0.93-2.03)</td>
<td>1.73† (1.27-2.36)</td>
<td>3.14† (2.08-4.75)</td>
</tr>
<tr>
<td>GAD</td>
<td>1.89† (1.41-2.53)</td>
<td>1.48 (0.92-2.40)</td>
<td>1.59† (1.08-2.35)</td>
<td>2.94† (1.90-4.55)</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>1.89§ (1.19-3.00)</td>
<td>1.45 (0.63-3.32)</td>
<td>1.75 (0.97-3.27)</td>
<td>2.55§ (1.29-5.02)</td>
</tr>
<tr>
<td>Bulimia nervosa</td>
<td>1.61 (0.78-3.34)</td>
<td>1.80 (0.52-6.22)</td>
<td>0.53 (0.13-2.13)</td>
<td>3.30§ (1.37-7.96)</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>2.80† (1.88-4.16)</td>
<td>2.42§ (1.27-4.61)</td>
<td>2.39§ (1.42-4.01)</td>
<td>4.01§ (2.33-6.91)</td>
</tr>
<tr>
<td>Drug dependence</td>
<td>3.09† (1.89-5.03)</td>
<td>2.93§ (1.39-6.16)</td>
<td>1.97§ (1.01-3.85)</td>
<td>5.70† (3.04-10.69)</td>
</tr>
<tr>
<td>≥2 Disorders</td>
<td>2.58† (1.96-3.40)</td>
<td>1.61‡ (1.02-2.56)</td>
<td>2.04† (1.43-2.92)</td>
<td>5.47‡ (3.56-8.40)</td>
</tr>
</tbody>
</table>

*CI indicates confidence interval; GAD, generalized anxiety disorder.
†$P<.001$.
‡$P<.05$.
§$P<.1$.
We examined the degree to which the association between self-reported CSA and psychiatric and substance use disorders changed with the inclusion of variables that might reflect the family context in which CSA is likely to emerge. Our main analyses relied on the report of these covariates by parents (Table 4). Comparing Tables 2 and 4, the addition of these covariates resulted in a modest attenuation of the relationship between CSA and most psychiatric and drug use disorders. However, for other disorders, such as panic disorder and BN, the association with CSA was strengthened. We repeated these analyses including covariates reported by the twins with similar, although somewhat more attenuated, results.

**ASSOCIATION WITH SELF-REPORT CONTROLLING FOR BACKGROUND FAMILIAL FACTORS**

We examined the degree to which the association between self-reported CSA and psychiatric and substance use disorders changed with the inclusion of variables that might reflect the family context in which CSA is likely to emerge. Our main analyses relied on the report of these covariates by parents (Table 4). Comparing Tables 2 and 4, the addition of these covariates resulted in a modest attenuation of the relationship between CSA and most psychiatric and drug use disorders. However, for other disorders, such as panic disorder and BN, the association with CSA was strengthened. We repeated these analyses including covariates reported by the twins with similar, although somewhat more attenuated, results.

**ASSOCIATION WITH SELF-REPORT CONTROLLING FOR BACKGROUND FAMILIAL FACTORS AND PARENTAL PSYCHOPATHOLOGY**

We next examined the association between self-reported CSA and psychiatric and substance use disorders controlling for possible confounding familial factors and for parental psychopathologic disorders as assessed at personal interview (Table 5). For each disorder examined in the twins, we controlled for the history of that specific disorder in the parents. These results are based on a substantially smaller number of families because they required personal interviews with both parents. Overall, the results were relatively similar to those seen in Tables 2 and 4, suggesting that little of the association between CSA and psychopathology is accounted for by parental psychopathologic disorders. We repeated these analyses using family history reports by the twin in the full sample and obtained similar results.

**COTWIN CONTROL ANALYSES**

In examining twin pairs discordant for self-reported CSA (Table 6), we examined narrow discordance (one twin reported CSA and the cotwin reported no CSA) and broad discordant (one twin reported CSA and her cotwin reported no or a less deviant form of CSA). Qualitatively, the magnitude of the ORs was similar to that seen in Tables 3 to 5. However, only a modest number of them were statistically significant. In 19 of 22 twin pairs only the abused twin reported CSA.
had the comorbidity. This was unlikely to occur by chance ($P<.001$).

The rates in our sample are well within the range reported in comparable studies for any CSA and for intercourse.\(^1\) With respect to CSA, it is likely that this sample is broadly representative of white women in the United States. We found only moderate agreement (weighted $k=0.40$) between self-reported and cotwin-reported CSA. However, more than one third of abused women reported telling no one about their experience. The only comparable results\(^3\) of which we are aware report slightly higher reliability (weighted $k=0.52$) in 87 sister pairs oversampled for those with adverse childhood experiences. In that sample, sibling resemblance for CSA (weighted $k=0.27$) was lower than that observed in this study, consistent with the expectation that twin sisters are more concordant for CSA than nontwin sisters.

Four recent meta-analyses\(^7\) of the impact of CSA on a wide range of continuous measures of adjustment (eg, self-esteem, social adjustment, symptoms of anxiety, and alcohol problems) found a small effect size. In the largest of these reviews,\(^10\) the average effect size (expressed as a Pearson correlation coefficient) was 0.09, meaning that CSA accounted for less than 1% of the variance in their adjustment measures. The means±SDs of the tetrachoric correlations between a history of any CSA and intercourse and risk for the 6 disorders we examined were 0.24±0.08 and 0.35±0.08, suggesting that any CSA and intercourse accounted for up to 6% and 12% of the variance in liability.

### Table 5. Childhood Sexual Abuse (CSA) by Self-report and the Odds Ratios for Psychiatric and Substance Abuse Disorders Adjusted for Measures of Family Functioning as Reported by Parents and Parental Psychopathologic Disorders\(^*\)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>All CSA (n = 713-714)</th>
<th>Nongenital CSA (n = 572)</th>
<th>Genital CSA (n = 613)</th>
<th>Intercourse (n = 562-563)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depression</td>
<td>1.67† (1.01-2.37)</td>
<td>1.08 (0.59-1.95)</td>
<td>1.58‡ (1.00-2.51)</td>
<td>2.79† (1.44-5.40)</td>
</tr>
<tr>
<td>GAD</td>
<td>1.63† (1.00-2.65)</td>
<td>1.19 (0.54-2.02)</td>
<td>1.34 (0.70-2.58)</td>
<td>2.58‡ (1.17-5.69)</td>
</tr>
<tr>
<td>Bulimia nervosa</td>
<td>1.36 (0.48-3.86)</td>
<td>1.30 (0.23-7.33)</td>
<td>0.50 (0.08-3.07)</td>
<td>4.15† (1.13-15.30)</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>2.96§ (1.59-5.51)</td>
<td>3.20† (1.18-8.73)</td>
<td>1.91 (0.85-4.27)</td>
<td>6.48§ (2.36-17.84)</td>
</tr>
<tr>
<td>Drug dependence</td>
<td>2.58§ (1.27-5.23)</td>
<td>3.57† (1.13-11.30)</td>
<td>1.21 (0.48-3.09)</td>
<td>6.55‡ (2.10-20.46)</td>
</tr>
</tbody>
</table>

* CI indicates confidence interval; GAD, generalized anxiety disorder; and NE, not estimable.
† $P<.01$.
‡ $P<.05$.
§ $P<.001$.

### Table 6. Childhood Sexual Abuse (CSA) by Self-report and the Odds Ratios (OR) in Twin Pairs Discordant for Exposure to Psychiatric and Substance Use Disorders\(^*\)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Any CSA (n = 133)</th>
<th>Nongenital (n = 45)</th>
<th>Genital (n = 71)</th>
<th>Intercourse (n = 53)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depression</td>
<td>35/25</td>
<td>8/8</td>
<td>18/13</td>
<td>21/15</td>
</tr>
<tr>
<td></td>
<td>1.40</td>
<td>1.38</td>
<td>1.40</td>
<td>2.25</td>
</tr>
<tr>
<td></td>
<td>2.43†</td>
<td>1.25</td>
<td>2.75</td>
<td></td>
</tr>
<tr>
<td>GAD</td>
<td>22/15</td>
<td>9/6</td>
<td>10/8</td>
<td>7/3</td>
</tr>
<tr>
<td></td>
<td>1.47</td>
<td>1.33</td>
<td>1.40</td>
<td>2.33</td>
</tr>
<tr>
<td></td>
<td>2.75</td>
<td>2.75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulimia nervosa</td>
<td>17/6</td>
<td>4/3</td>
<td>0/0</td>
<td>3/0</td>
</tr>
<tr>
<td></td>
<td>2.83†</td>
<td>2.33</td>
<td>4/0</td>
<td>4/0</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>10/5</td>
<td>4/1†</td>
<td>2/3</td>
<td>3/4</td>
</tr>
<tr>
<td></td>
<td>2.00</td>
<td>0.67</td>
<td>0.75</td>
<td>4.00</td>
</tr>
<tr>
<td></td>
<td>4.00</td>
<td>4.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug dependence</td>
<td>30/13</td>
<td>7/3</td>
<td>11/7</td>
<td>13/8</td>
</tr>
<tr>
<td></td>
<td>2.31†</td>
<td>1.57</td>
<td>1.63</td>
<td>4.00†</td>
</tr>
<tr>
<td></td>
<td>6.33§</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* $n_{12}$ indicates pairs discordant for CSA and diagnosis where the exposed twin is the affected one; $n_{21}$, pairs discordant for CSA and diagnosis where the unexposed twin is the affected one; OR, odds ratio; GAD, generalized anxiety disorder, NE, not estimable; and $+\infty$, value is positive and infinite.
† $P<.05$.
‡ $P<.01$.
§ $P<.001$.
to these disorders, respectively. Childhood sexual abuse seems to be more strongly related to lifetime psychopathologic disorders than to cross-sectional measures of adjustment. This may not be surprising in that diagnoses capture liability over the lifetime, whereas measures of adjustment focus solely on current well-being.

We are aware of 3 general population samples that present ORs between CSA and diagnosed psychiatric disorders. Their results are generally similar to those in this study, although they reported somewhat higher ORs for MD and lower ORs for alcohol and other drug dependence. Also consistent with 2 of these studies, which reported similar data, we found a “dose-response” relationship between CSA and risk for developing psychiatric illness. Risk increased monotonically from nongenital CSA to genital CSA to intercourse.

We were particularly concerned with evaluating noncausal mechanisms of association between CSA and psychiatric and drug use disorders. This association might be noncausal because familial factors such as family discord and conflict predispose to CSA and psychopathology. Our results suggest that little of the CSA-psychopathology association could be explained by this association because the ORs declined only slightly when we controlled for a range of such potential covariates when reported either by the parents or by the twins. These results are broadly consistent with similar analyses reported by 3 other groups. Inclusion of measures of family discord as covariates could, however, represent “overcorrection” because some proportion of family dysfunction might arise from rather than contribute to CSA.

Childhood sexual abuse could be also be a direct or indirect manifestation of parental psychopathologic disorder, the liability to which could then be genetically transmitted to their offspring. Based on our analyses, this is an unlikely explanation because the association between CSA and psychiatric disorders in the daughters declined only slightly when we controlled for parental psychopathology. We are unaware of previous studies that have involved personal diagnostic interviews of parents in a population-based sample in which CSA was assessed.

Perhaps the most rigorous method in which to evaluate the impact of these 2 possible confounding variables (family dysfunction and parental psychopathology) on the CSA-psychopathology association is a cotwin control design. Both members of a twin pair would be highly correlated in their exposure to family conflict and have an equal genetic relationship with their parents. The interpretation of our cotwin control analyses is limited because of small sample size. However, the pattern of the findings for most disorders supported a causal interpretation of the association between CSA and psychiatric and drug abuse disorders because, despite both being raised in the same family environment, the twin exposed to CSA had a consistently elevated risk for psychopathologic disorders compared with her unexposed cotwin.

A recent study from a large Australian volunteer twin registry found ORs between CSA in women—as assessed by a single global question—and MD, panic disorder, and alcohol dependence in the range of those in this study. In 107 pairs of female-female twins discordant for any CSA, the ORs between CSA and MD and alcohol dependence were increased (albeit nonsignificantly) and similar in magnitude to those found in our sample (ORs, 1.43 and 2.50, respectively). These similar findings from 2 independent twin samples provide support for the hypothesis that part of the CSA-psychopathology association is causal.

More difficult to evaluate is the possibility that the association between CSA and psychopathologic disorder arises through reporting bias. For example, a spurious association would occur if individuals with a history of psychiatric illness, in an effort to understand their disorder, were more likely to recall and report CSA. We were able to evaluate this bias by examining the association between self-reported psychiatric and drug abuse disorders and CSA as reported by the cotwin. The results of these analyses were not entirely consistent. For some disorders, the association was substantially lower with cotwin vs self-reported CSA, while for others it was only slightly attenuated, and for one (BN) it was even stronger. Power was reduced because of lower cotwin report vs self-report rates of CSA and because a range of factors are likely to affect when a cotwin would know about, recall, and be comfortable reporting CSA in her cotwin. Although far from definitive, a conservative conclusion from these analyses would be that it is implausible that all or most of the CSA-psychopathology association is due to reporting bias.

A final noncausal explanation would be that some individuals possess a trait that increases probability of exposure to CSA and independently their risk for developing psychiatric disorders. Given that twins are likely to share this trait, our cotwin control analyses have power to address this difficult issue. This model predicts that the nonabused member of a pair discordant for CSA (who is likely to have this trait) should also be at high risk for developing future psychopathologic disorders. Therefore, within these twin pairs, there should be minimal association between CSA and future risk. We do not observe this pattern for most disorders examined. Our sample of discordant pairs is too small to divide meaningfully into monozygotic and dizygotic pairs, which would give us greater power at examining this hypothesis.

We found little evidence for diagnostic specificity of CSA. In some analyses, the association was slightly weaker with anxiety disorders and stronger with BN. However, the 95% CIs were usually overlapping. Overall, CSA was more strongly associated with alcohol and other drug abuse than with the psychiatric disorders. Further research is needed to clarify whether different features of the CSA experience (eg, age, nature of perpetrator, and use of force) provide evidence for differential risk across these psychiatric and substance use disorders.

These results should be interpreted in the context of potentially significant methodological limitations. First, the sample was entirely female. The experience of CSA might differ substantially between the sexes. Second, we assessed CSA retrospectively from adults. In addition, we found only modest reliability in our CSA between self-report and twin report. If the errors introduced into our assessment were random, this would attenuate the true associations. However, biases that would exaggerate the true associations are also possible. Third, an assessment of reliability usually assumes that the critical information was available to all informants. This will not always be the case.
for CSA in which the cotwin might not be aware of the abuse. Our reliability estimates are therefore likely to be biased downward. Fourth, our assessments of potential confounding variables were also retrospective. However, the pattern of findings was relatively similar when parental or twin reports of these variables were used, and one might expect the biases of these 2 reporters to be rather different. Fifth, twins with CSA may have been underrepresented in our sample. Two analyses of this question yielded contradictory evidence (results not shown). However, our rates of reported CSA were similar to those found in other population surveys. Sixth, CSA was assessed by mailed questionnaire. This, however, was the clear preference of this sample. Survey data suggest that more “anonymous” response modes might obtain better cooperation and reporting in highly sensitive material such as CSA.36-38 Furthermore, by using a mailed questionnaire we were able to separate our assessments of CSA and psychopathologic disorder in time and in mode, thereby reducing the chances of correlated errors. Seventh, we have not attempted to address the temporal relationship between CSA and the onset of psychiatric and substance use disorders. The utility of such an analysis with retrospective data is uncertain. However, in twins who reported both CSA and a lifetime history of MD or alcohol dependence, we found that 95.8% and 100% of them, respectively, reported that the CSA began before or during the same year as their psychiatric condition. It is unlikely that much of the CSA-psycho pathologic association could be explained by previous psychopathologic disorder increasing the risk for subsequent CSA.

Accepted for publication May 15, 2000.

This work was supported by grants MH-40828 and AA-09095 from the National Institutes of Health, Bethesda, Md; Research Scientist Award MH-01277 from the National Institutes of Health (Dr Kendler); and the Rachel Brown Banks Endowment Fund.

We acknowledge the contribution of the Virginia Twin Registry, now part of the Mid-Atlantic Twin Registry (MATR), to the database of twins for this study. The MATR, directed by Linda Corey, PhD, and Lenn Murrelle, PhD, has received support from the National Institutes of Health; the Carman Trust, Richmond, Va; the W. M. Keck Foundation, Los Angeles, Calif; the John Templeton Foundation, Radnor, Pa; and the Robert Wood Johnson Foundation, Princeton, NJ. These data were collected under the direction of Patsy Waring, Lisa Halberstadt, MS, and Barbara Brooke, MSW.

Reprints: Kenneth S. Kendler, MD, Department of Psychiatry, Medical College of Virginia/Virginia Commonwealth University, PO Box 980126, Richmond, VA 23298-0126.

REFERENCES