Early sexual abuse and lifetime psychopathology: a co-twin–control study

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ABSTRACT

Background. This study was designed to determine lifetime prevalence of psychiatric disorders among twins who reported childhood sexual abuse (CSA), and to compare these rates with those among non-abused co-twins. The contribution of familial and individual-specific factors to reported sexual abuse was also examined.

Method. Information about lifetime psychopathology and substance use was obtained by structured telephone interviews with 5995 Australian twins. Twins who reported a history of childhood sexual abuse (CSA) were contrasted on lifetime psychopathology with subjects without such a history; in addition, comparisons were made between same-sex twin pairs discordant for CSA.

Results. A history of CSA was reported by 5·9% of the women and 2·5% of the men. In the sample as a whole, those reporting CSA were more likely to receive lifetime diagnoses of major depression, conduct disorder, panic disorder and alcoholism, and were more likely to report suicidal ideation and a history of suicide attempt. Abused women, but not men, were also more likely to report social phobia. When comparisons were restricted to non-abused co-twins, no differences in psychopathology were seen. However, rates of major depression, conduct disorder and suicidal ideation were higher if both co-twins were abused than if the respondent alone reported CSA. Model-fitting indicated that shared environmental factors influenced risk for reported CSA in women, but not in men.

Conclusion. The association between CSA and psychopathology arises at least in part through the influence of shared familial factors on both risk of victimization and risk of psychopathology.

INTRODUCTION

Awareness of the extent and sequelae of child sexual abuse (CSA) has increased dramatically over the last two decades. However, because of substantial differences in ascertainment of samples, phrasing of questions and variability of definition of CSA, lifetime prevalence estimates of CSA vary considerably, with reported figures

ranging from 7 to 45% of women (Wyatt, 1985; Siegal *et al.* 1987; Stein *et al.* 1988). However, if significant (> 5 years) disparity in age between victim and perpetrator and behaviours short of physical contact (e.g. exhibitionism) are included in the definition of CSA (Wyatt & Peters, 1986), recent estimates suggest that 15–20% of women may have experienced some form of CSA (Finkelhor, 1979, 1984; Bagley, 1989; Mullen *et al.* 1993).

Depressed feelings, anxiety, guilt and anger appear to be common among victims (Bryer *et al.* 1987; Stein *et al.* 1988; Hussey & Singer,

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1993; Kendall-Tackett *et al.* 1993; McCauley *et al.* 1997) and rates of psychiatric syndromes including mood and anxiety disorders, eating disorders, substance abuse disorders, somatization disorder and borderline personality disorder also have been reported to be elevated in this population (Putnam *et al.* 1986; Stein *et al.* 1988; Ogata *et al.* 1990; Bifulco *et al.* 1991; Brown & Anderson, 1991; Palmer *et al.* 1992; Pribor & Dinwiddie, 1992; Pribor *et al.* 1993; Roesler & McKenzie, 1994; Wonderlich *et al.* 1996). Less is known about male victims, though symptoms and sequelae of abuse appear to be similar (Briere *et al.* 1988; Myers, 1989; Swett *et al.* 1990; Schulte *et al.* 1995).

If the relationship between CSA and psychopathology is directly causal, the apparent base-rate of CSA would imply that it is a substantial contributor to psychiatric dysfunction in the community. However, studies of psychopathology and CSA to data suffer from several methodological limitations. Most studies have been retrospective, usually based on interview of psychiatric in-patients or other treatment-seeking individuals. Indeed, when comparison is made with children referred for psychiatric treatment but without a history of CSA, victimized children as a group may actually demonstrate fewer symptoms of psychopathology (Hussey & Singer, 1993; Kendall-Tackett *et al.* 1993). Additionally, rates of psychiatric illness among victims of CSA tend to be lower (though still elevated) in samples ascertained through the community rather than through treatment (Stein *et al.* 1988).

Perhaps more importantly, environmental factors associated with increased risk for CSA, such as significant family conflict or violence, extended institutional placement, and presence of a stepfather are known to influence the risk of later psychological maladjustment and psychopathology even in the absence of CSA (Russell, 1984; Alexander & Lupfer, 1987; Bifulco *et al.* 1991), given the likely interrelationships between such manifestations of family dysfunction and CSA, any specific effect on subsequent psychiatric difficulty may be difficult to identify (Briere, 1986; Fleming *et al.* 1999).

Finally, because many psychiatric illnesses have a significant genetic component, any impact of CSA may also be confounded by constitutional factors. The hypothesis that the relationship between CSA and later psychopathology could be traced to shared familial or genetic vulnerability factors has not been tested.

One way of doing so is to use the twin-study method. By comparing the similarity between identical and fraternal twins in reporting of CSA, the degree to which risk aggregates in families can be estimated. Moreover, by using non-abused co-twins (monozygotic or dizygotic) as controls, the direct effect of CSA can be separated from shared environment factors also predisposing to subsequent behavioural difficulties and psychiatric symptomatology. In this study, we first explore the overall relationship between reported CSA and selected lifetime psychopathology, then use the twin study paradigm to examine the influence of shared environmental factors on risk for CSA and risk of psychopathology.

METHOD

Sample and measures

Subjects were ascertained from the Australian National Health and Medical Research Council (NH & MRC) Twin Register, a volunteer panel established in 1978 and described in detail elsewhere (Heath *et al.* 1997; Statham *et al.* 1998). Eligible for inclusion in the sample were: (*i*) twins from pairs where both had responded to a mailed questionnaire in 1980–82 ('1981 survey') and where at least one twin had responded to a follow-up survey by mailed questionnaire or brief telephone interview in 1988–9 ('1989 survey'); and (*ii*) twins not already included in (*i*) who had participated in an alcohol challenge study in 1978–9. Telephone interviews were completed in 1992–3 with 3787 women and 1973 men from (*i*) (response rates of 88·4% and 82·8% respectively) and 68 women and 61 men from (*ii*) (72·3% and 78·2% response rates). An additional 60 women and 46 men have been included in the sample from pairs who had both returned questionnaires in 1981 only. The final total included 1341 monozygotic (MZ) pairs (940 female, 401 male), 776 dizygotic (DZ) pairs (540 female and 236 male), and 604 opposite-sex twin pairs, plus data on 553 'singleton' twins (i.e. where data were available on only one member of a twin pair).

A verbal consent procedure was used, with the elements of informed consent reviewed and

formal consent given before proceeding with the interview. (Such procedures have been approved by the Office of Protection of Research Risks.)

Subjects were interviewed by trained lay interviewers using a telephone adaptation of the SSAGA (Bucholz *et al.* 1994) to obtain lifetime history of psychiatric symptoms and generate DSM-III-R diagnoses of alcohol abuse/dependence, major depression, anxiety disorders (including panic disorder and social phobia) and conduct disorder, though the algorithm used to diagnosed conduct disorder did not require temporal clustering of conduct problems.

Family history of symptoms of specific disorders was also obtained, with such symptoms were considered present if reported by either twin. History of suicidal ideation and attempts was also assessed, with a serious suicide attempt defined as an attempt where the respondent reported either a definite intent to die, or a method classified as of at least moderate lethality.

Included in this survey was one question which asked 'Before age 18, were you ever forced into sexual activity, including intercourse?'. Subjects responding affirmatively to this item were considered to have been sexually abused.

Statistical analysis

Statistical tests were performed using SAS version 6.09 (SAS Institute, 1989). Comparisons were first made by treating twins as having been individually ascertained, to allow comparison to other population-based studies of sexual abuse. For these analyses, univariate tests included χ^2 tests with continuity adjustment for categorical data; where cell sizes were fewer than five, alpha values using Fisher's exact test are reported. For continuous data, *t* tests were used and where sample variances were found to be unequal, the approximate *t* statistic and approximation of degrees of freedom are reported. For these preliminary statistical tests, we did not correct for the non-independence of observations on twin pairs.

The hypothesis of genetic influence on risk of reported abuse was examined by fitting genetic models using the MX program (Neale, 1997) to the set of five twin pair contingency tables (MZ male and female pairs, DZ male and female pairs, and unlike-sex pairs) (Neale & Cardon, 1992). The assumption underlying this modelling is that resemblance between twins can be accounted for by additive genetic factors (which will therefore contribute twice as much to any correlation between MZ, as compared to DZ twins); environmental factors shared by both twins ('shared environment'); and non-shared environmental influences. Measurement error is also included in the latter term. Models can be further modified by constraining parameters, and by sequentially eliminating certain terms from models. Models were fitted by maximum likelihood, yielding a χ^2 test of goodness-of-fit for each model. Various submodels were compared to the most general model by likelihood ratio (χ^2 difference) test to determine the contribution of the eliminated variable, as reviewed elsewhere (Neale & Cardon, 1992). While the appropriateness of the liability threshold model used in these analyses has been questioned (Kraemer, 1997), the likelihood-ratio χ^2 test for the significance of genetic effects will give results that are very close to a comparable likelihood-ratio statistic which uses comparison of logistic regression models to test for genetic effects (and indeed identical when the prevalence of CSA does not vary as a function of zygosity (Heath *et al.* 1998)). Logistic regression was used to calculate odds ratios for psychiatric diagnoses. This was initially performed without reference to status as twins, to allow for comparison to other studies of psychopathology among victims of CSA. Confidence intervals, adjusted for the non-independence of observations on twin pairs, were estimated by bootstrapping (Effram & Tibshirani, 1986).

The association observed between CSA and psychopathology could in theory be due to a direct causal effect, an indirect effect (e.g. family background factors increasing both risk of psychopathology and risk of CSA), reverse causation (psychopathology increasing risk of CSA), or some mixture of these effects. To gain further evidence for a direct effect of CSA, comparisons were first made on same-sex twin pairs discordant for abuse, using matched pairs odds ratios with the non-abused co-twin as the referent. If there were an important direct effect on risk of psychopathology, significant matched-pairs odds ratios would be observed, though low statistical power due to small numbers of discordant pairs might be a limiting factor.

Secondly, in a re-analysis of data from the entire sample, odds of psychopathology were compared in respondents from twin pairs where: (*a*) both twins reported CSA; (*b*) only the respondent reported CSA; or (*c*) only the respondent's co-twin reported CSA, using as a comparison group (*d*) pairs where neither twin reported a history of CSA. If the association with CSA were entirely due to a direct causal effect of CSA, we would expect to see significant odds ratios for groups (*a*) and (*b*), but not (*c*); and also to find that the odds ratio for group (*b*) would be similar in magnitude to the conditional odds ratio in the matched-pairs comparison. (The odds ratio for group (*a*) might be higher if cases of CSA in respondents whose co-twin was also abused were on average more serious.)

In the case of reverse causation, a significant odds ratio for group (*c*) also should be seen: Since there is significant familial aggregation of the psychopathology measures, siblings of CSA victims should report increased rates of psychopathology compared to respondents from pairs where neither reported CSA, though rates would not necessarily be as high as among their abused siblings. In the case of an indirect association, we would also expect to observe a significant odds ratio for group (*c*) – who shared family background risk factors in common with a CSA victim – as well as for groups (*a*) and (*b*). Under these two latter hypotheses, we also would expect that the odds ratio estimate for group (*b*) would be higher than that obtained in the conditional logistic regression, since it would be partly explained by family background effects (or correlated phenotypes) which would be controlled for in the matched-pairs comparison.

RESULTS

Demographics and family background

The prevalence of reported CSA in this sample was 5·9% among women, 2·5% among men. Prevalence did not vary as a function of birth cohort in men. However, prevalence of reported CSA was highest among women born after 1940 (7·0%) and significantly lower both in those born 1930–1939 (Odds Ratio = 0·47, 95% confidence interval (CI) 0·28–0·79) and those born prior to 1930 (OR = 0·23, 95% CI 0·11–0·48). Women reporting CSA were more likely than other women to be separated or divorced at the time of interview, were less likely to have had children, and were more likely to report a low personal income (Table 1). When birth cohort was controlled women were also significantly less likely to have completed more than 12 years of education (OR = 0·69, 95% CI 0·52–0·91). None of these effects was significant among men.

Individuals reporting CSA did not differ in socio-economic background, as indexed by reported paternal educational level (Table 1), but were more likely to report a parental history of alcohol problems or depression. The association with parental depression appeared to be a consequence of the association between parental alcoholism and depression. In a multiple logistic regression analysis, reported CSA in women was significantly associated with alcoholism in both parents (OR = 2·57, 95% CI 1·23–5·36), in the mother only (OR = 1·99, 95% CI 1·03–3·86), and in the father only (OR = 1·77, 95% CI 1·23–5·36), but with neither maternal (OR = 1·13, 95% CI 0·83–1·55) nor paternal (OR = 1·40, 95% CI 1·00–1·96) history of depression. Reported CSA in men was significantly associated with paternal alcoholism (OR = 2·32, 95% CI 1·22–4·41) and, marginally, with paternal history of depression (OR 2·00, 95% CI 1·03–3·87).

Twin pair concordance for reported CSA

Table 2 summarizes twin pair concordances and tetrachoric correlations for CSA. These estimates were more precise for women, where 21% of female like-sex twin pairs were concordant for CSA, than for men, where only a single male like-sex pair was concordant for abuse. Concordance for CSA was not significantly greater for female MZ than DZ twins. The most parsimonious model was one that allowed for significant shared environmental effects in women (63% of the variance in liability), with no familial correlation for liability to CSA in men ($\chi^2 = 11·19$, df = 8, $P = 0·19$, AIC = 4·81). Genetic effects did not play a significant role in either men or women (improvement in model fit for women, $\chi^2 = 1·84$, df = 1, $P = 0·17$; for men, $\chi^2 = 0·50$, df = 1, $P = 0·48$). Thus, the data are consistent with either an important shared environmental (e.g. family background) effect on risk of reported CSA in women, or joint abuse of both twin sisters in some cases.

Table 1. *Demographic and family history characteristics of the sample*

	Women		Men	
	Abused (N = 228) %	Non-abused (N = 3639) %	Abused (N = 52) %	Non-abused (N = 2027) %
Respondent characteristics				
Education > 12 years	42·1[NS]	48·2	69·2[NS]	70·5
Personal income > $25000†	11·4***	21·2	62·8[NS]	65·3
Currently separated/divorced	17·5***	9·5	11·5[NS]	6·7
No children	10·0**	5·3	26·9[NS]	25·7
Age	41·4±9·7***	45·0±12·7	43·3±10·3[NS]	42·7±11·8
Family background				
Paternal education ≤ 12 years	64·9[NS]	67·7	51·1[NS]	62·9
Paternal alcoholism	29·0***	16·1	38·5***	15·8
Maternal alcoholism	6·1*	3·5	5·8[NS]	2·8
Paternal depression	25·4***	16·5	26·9*	15·5
Maternal depression	31·1*	25·0	36·5*	22·7

NS Difference between abused and non-abused respondents not significant; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.
† At time of mailed questionnaire survey in 1989.

Table 2. *Twin-pair concordance for being victims of childhood sexual abuse*

Zygosity group	N pairs	Both abused	One abused	Neither abused	Probandwise concordance	Tetrachoric correlation	Standard error
MZ female pairs	923	20	63	840	(38·8)	0·68	0·07
DZ female pairs	525	8	44	473	(26·7)	0·52	0·11
MZ male pairs	398	0	14	384	(0)	—	—*
DZ male pairs	233	1	11	221	(15·4)	0·43	0·27
DZ unlike-sex pairs	600	3	F†: 41 M: 16	540	F†: (6·8) M: (15·8)	0·21	0·16

The probandwise concordance is the probability that the co-twin of an abused twin also reports childhood sexual abuse.
† Gender of abused twin.
* Not estimable.

Reported CSA and lifetime psychopathology

Fig. 1 compares rates of lifetime psychiatric illness and history of suicidal ideation and serious suicide attempts as a function of reported CSA. Elevated rates among abused individuals were seen for all disorders in women, and for all disorders except social phobia in men. The high lifetime rates of psychopathology in men as compared with women are a consequence of the broad-band operationalizations of DSM-III-R Alcohol Dependence and of Conduct Disorder used in this study.

Table 3 summarizes the association between lifetime psychiatric illness and reported history of CSA, using data from the entire sample. The association between CSA and reporting a serious suicide attempt was strong in both genders, with odds ratios > 7 for each, while lifetime major depression and any suicidal ideation were even more strongly associated with reported CSA in men than in women, as was the case also for panic disorder. Both history of conduct disorder and alcohol dependence were strongly associated with reported CSA, particularly among women, while an association with social phobia was seen only among women.

Table 4 shows odds ratios estimated from twin pairs discordant for CSA. Because model-fitting did not demonstrate an effect of genetic factors and no systematic differences were found in MZ *versus* DZ female like-sex pairs, results from all male and female like-sex discordant pairs are tabulated. While only a single odds ratio reached statistical significance (the association between CSA and suicidal ideation in men), with the exception of alcohol dependence in men, all were in the predicted direction of increased psychopathology in the abused twin compared to the non-abused twin. In women,
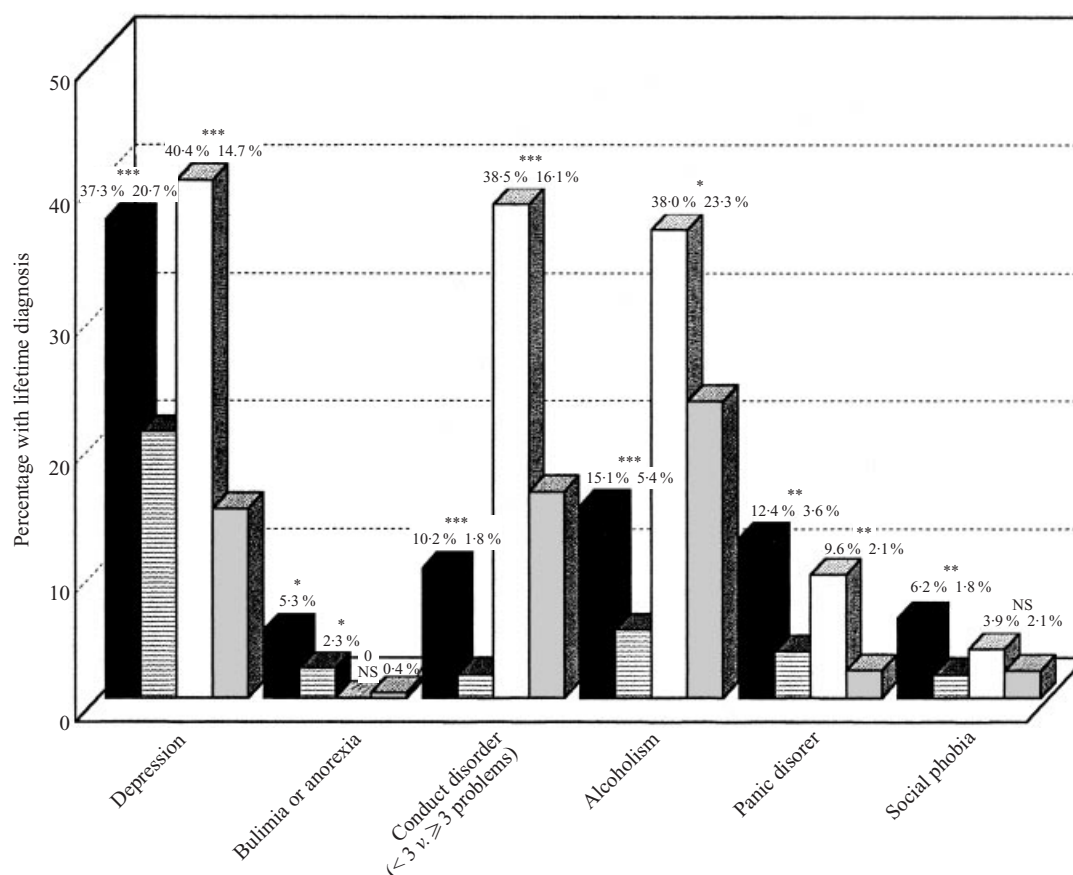
FIG. 1. Comparison of rates of lifetime psychiatric illness and history of suicidal ideation and serious suicide attempts as a function of reported childhood sexual abuse. (■, Female abused ($N = 228$); ▤, female non-abused ($N = 3640$); □, male abused ($N = 52$); ▨, male non-abused ($N = 2039$). * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; NS not significant.)

Table 3.   *Associations (odds ratio [OR] and 95% confidence interval [CI]) between childhood sexual abuse and lifetime psychopathology in the total sample*

| | Total sample | | | |
| | Women (N = 3868) | | Men (N = 2078) | |
	Adjusted OR*	95% CI	OR	95% CI
Major depression	2·20	1·66–2·92	3·93	2·23–6·93
Any suicidal thoughts	3·05	2·32–4·01	4·59	2·62–8·03
Serious suicide attempt	7·74	4·70–12·75	7·07	2·63–19·04
Panic disorder	3·54	2·29–5·47	5·02	1·90–13·25
Social phobia	3·41	1·75–6·66	1·02[NS]	0·14–7·57
Alcohol dependence	2·81	1·89–4·17	1·91	1·08–3·39
Conduct disorder†	5·47	3·32–8·99	3·27	1·85–5·79
Any psychopathology‡	2·47	1·89–3·24	3·79	2·04–7·05

    * Controlling for birth cohort (women only).
    † Excludes pre-1930 birth cohort in women, which had no conduct disorder cases.
    ‡ Including serious suicide attempt.
    [NS] Not statistically significant.

Table 4. *Associations between CSA and psychopathology in twin pairs discordant for CSA*

| | Discordant-pair analyses | | | |
| | Women ($N = 107$) | | Men ($N = 25$) | |
	OR	95% CI	OR	95% CI
Major depression	1·43[NS]	0·72–2·83	1·50[NS]	0·42–5·32
Any suicidal thoughts	1·58[NS]	0·89–2·81	5·50	1·22–24·81
Serious suicide attempt	2·33[NS]	0·60–9·02	*	*
Panic disorder	2·00[NS]	0·68–5·85	*	*
Social phobia	1·50[NS]	0·42–5·32	*	*
Alcohol dependence	2·50[NS]	0·97–6·44	1·00[NS]	0·20–4·96
Conduct disorder†	1·25[NS]	0·34–4·66	2·00[NS]	0·50–8·00
Any psychopathology‡	1·55[NS]	0·88–2·72	2·00[NS]	0·60–6·64

* Could not be computed.
[NS] Not statistically significant.
†, ‡ See footnote to Table 3.

the association between reported CSA and alcohol dependence fell marginally short of statistical significance ($P = 0.06$). Additional analyses of sociodemographic variables in discordant female like-sex pairs (not shown) failed to find a direct association between reported CSA and marital separation or divorce (OR = 1·00, 95% CI 0·81–1·24), but did confirm that abused twins were less likely to have children (OR = 0·42, 95% CI 0·31–0·56), and were more likely to report low income than their non-abused co-twins (OR = 1·65, 95% CI 1·36–1·99).

Beyond co-twin–control comparisons

In the case of lifetime psychopathology, with only one exception (suicidal ideation in men), odds ratios estimated from the discordant pair analyses were systematically lower than the corresponding odds ratios derived from the entire sample, consistent with an influence of shared (e.g. family background) risk factors both on risk of CSA and separately on risk of other psychopathology, or (less plausibly), reverse causation.

However, one further check was needed. Despite its intuitive appeal, the co-twin–control design has an important limitation, in that it discards information from pairs in which both twins reported abuse, and data from discordant unlike-sex pairs. Table 5 summarizes results of analyses using data from the entire sample of female respondents, which compared odds of psychopathology, controlling for birth cohort,

in twins from pairs where neither had been abused, twins whose co-twin only had been abused, twins who had themselves been abused but whose co-twin reported no abuse, and twins from pairs in which both twins reported CSA. Two examples were examined: lifetime major depression and conduct disorder. In both cases, the highest odds of psychopathology were seen in those women reporting CSA whose twin also reported CSA. The lower odds ratios reported for the discordant-pair analyses in Table 4 must in part result from women from CSA-discordant pairs being at lower risk of psychopathology than women from CSA-concordant pairs. A similar pattern was observed for all other measures of psychopathology in women and for all measures except history of conduct disorder in men (data not shown).

In the case of conduct disorder, the observed data were not consistent with a simple cause-and-effect relationship. In pairs discordant for reported CSA, rates of conduct disorder were significantly elevated both in abused twins and in non-abused twin sisters of abused twins, compared with twins from pairs where neither reported CSA, and the odds ratio was not significantly higher in the former than in the latter comparison.

In the case of major depression, in families with only one sister reporting CSA, odds of a history of depression given CSA were somewhat, but not significantly, greater than the odds of depression given CSA in the twin's sister – but only in the former case was the risk of depression significantly elevated compared to pairs where neither sister had been abused. The magnitude of the odds ratio for abused respondents from CSA-discordant pairs was comparable to that observed in the discordant-pair analyses, so that we could not reject the simple cause-and-effect hypothesis.

Indirect associations with CSA

In 48·4% of families where both twin sisters had been abused, at least one parent had a history of alcohol-related problems. Maternal alcohol problems were strongly associated with elevated likelihood of abuse of both sisters, with odds ratios of 8·04 (95% CI 2·19–29·37) for female like-sex pairs from families where both parents had alcohol-related problems and 4·43 (95% CI 1·24–15·70) for families where the mother only

Table 5. *Association in women between history of major depression, and conduct disorder, and own and co-twin's CSA*

	Major depression		Conduct disorder*	
	OR†	95% CI	OR†	95% CI
Neither twin abused	1·00	—	1·00	—
Co-twin only abused	1·34[NS]	0·89–2·03	2·69	1·11–6·44
Respondent only abused	1·90	1·33–2·70	3·73	1·85–7·54
Both twins abused	3·69	2·19–6·21	10·90	5·03–23·71

* Excludes the oldest cohort born prior to 1930, which had no cases of female conduct disorder.
† Adjusted for birth cohort.

Table 6. *Association between psychopathology and history of childhood sexual abuse of respondent and respondent's co-twin, controlling for birth cohort and history of parental alcohol problems* (comparison group are twins from pairs where neither twin was sexually abused)

Respondent abused?: Co-twin abused?:	No Yes		Yes No		Yes Yes	
	OR	95% CI	OR	95% CI	OR	95% CI
Women						
Major depression	1·30[NS]	0·88–1·97	1·84	1·24–2·63	3·39	2·00–5·73
Suicidal thoughts	1·40[NS]	0·93–2·10	2·64	1·88–3·71	3·77	2·23–6·39
Serious suicide attempt	1·62[NS]	0·49–5·34	6·39	3·32–12·29	12·31	5·65–26·85
Panic disorder	1·30[NS]	0·58–3·18	3·23	1·84–5·67	4·14	1·94–8·85
Social phobia	2·88	1·10–7·52	2·95	1·22–7·14	5·88	2·12–16·35
Conduct problems*	2·40[NS]	0·99–5·80	3·38	1·65–6·91	7·58	3·42–16·80
Alcohol dependence	1·02[NS]	0·48–2·14	2·32	1·40–3·86	3·28	1·62–6·68
Any psychopathology	1·48	1·01–2·15	2·01	1·43–2·81	3·43	2·01–5·85
Men						
Major depression	1·49[NS]	0·81–2·75	3·50	1·83–6·70	7·27	1·19–44·58
Suicidal thoughts	1·06[NS]	0·60–1·88	4·47	2·34–8·50	11·95	1·30–109·84
Serious suicide attempt	0·98[NS]	0·13–7·62	7·41	2·31–23·83	21·12	2·01–221·84
Panic disorder	1·28[NS]	0·29–5·57	4·77	1·54–14·71	9·22[NS]	0·92–92·19
Social phobia	0·70[NS]	0·09–5·25	—†	—	12·83	1·26–131·04
Conduct problems*	2·05	1·16–3·63	3·23	1·65–6·32	1·03[NS]	1·11–9·67
Alcohol dependence	0·61[NS]	0·32–1·18	1·28[NS]	1·64–2·56	3·79[NS]	0·60–23·83
Any psychopathology	1·43[NS]	0·86–2·38	3·14	1·56–6·31	—‡	—

* Excludes the oldest birth cohort born prior to 1930, which had no cases of female conduct disorder.
† Could not be computed; no social phobics in this group.
‡ Could not be computed; all five cases reported psychopathology or serious suicide attempt.

had alcohol problems, compared with 2·04 (95% CI 0·89–4·67) for families where only the father had alcohol problems.

Table 6 summarizes the results of analyses examining the association between psychopathology and reported CSA in both respondent and co-twin, but controlling for parental alcohol problems as well as for birth cohort. Significantly elevated odds for psychopathology were seen in non-abused respondents whose co-twin reported abuse only for social phobia in women and conduct disorder in men (although in women the association with conduct problems fell just short of significance). In the case of conduct disorder, we noted earlier that the association with CSA appeared to be at least in part indirect; given that odds ratios for psychiatric disorders do not substantially differ once parental alcoholism is controlled for, these additional analyses suggest that such indirect effects are not substantially mediated through parental alcoholism effects.

Conversely, even when parental alcohol problems were controlled for, the pattern of increased risk to abused individuals whose co-twin was also abused, compared to individuals who were only themselves abused, persisted for all measures of psychopathology except conduct

problems in men, although not always reaching statistical significance. In the cases of major depression (in both men and women), and social phobia and panic disorder (in women), once the effect of family history of alcoholism was controlled for, the hypothesis of a simple cause-and-effect relationship could no longer be rejected. The increase in risk to singly abused twins, compared to individuals from families where neither reported abuse, was not greater than the increase in risk estimated from the discordant-pair analyses. (In other words, the discordant-pair analysis odds ratio lies within the 95% confidence interval for the former odds ratio.) In the case of suicidal thoughts, and reporting any serious suicide attempt, in women, the odds ratio estimated from the discordant-pair analyses (ORs = 1·58 and 2·33 respectively) remained much lower than would have been predicted from Table 6, leaving open the possibility that the association with CSA is at least in part indirect, although a 'contagion effect' on suicidal thoughts and behaviour could also explain this pattern of findings.

DISCUSSION

Several methodological limitations to this study should be kept in mind. The assessment instrument we used was primarily designed to assess alcoholism and related characteristics, and identification of sexual abuse was based on self-report, without external validation and without assessment of onset, nature, duration, or severity of abuse, or identity of perpetrator(s). Furthermore, it required the subject to judge that they were forced into this behaviour. In light of reports indicating that psychopathology may be more likely to accompany severe abuse (Mennen, 1993; Fleming *et al.* 1999), an inability to classify severity could potentially lead to an underestimate of any associations. If in our sample the abuse tended to be more severe among twins concordant for CSA, our results are consistent with the hypothesis that a severity effect exists; conversely, however, systematic exclusion of milder forms of CSA might over-estimate such association.

Moreover, given that risk of abuse, like other sexual behaviours (Dunne *et al.* 1997), appears not to have been consistent across age cohorts, it may be that severity of abuse also was not constant across age. It is possible that the nature and emotional impact of the abuse, as well as the identity of the perpetrator, may have varied substantially between age subgroups of victims. Moreover, we were unable to test the hypothesis that some (or all) of the cohort effect may have been related to differential failure to report abuse by members of older cohorts. A related point is that, given the phrasing of the question (specifically the inclusion of sexual intercourse), its salience may have differed between men and women, thus skewing responses based on gender. Also, of course, it is possible that the experience of CSA may have, to an unknown degree, altered the likelihood not of experiencing but of reporting other symptoms of psychopathology, thus potentially distorting the true relationships.

In addition, our definition of CSA required coerced sexual activity, thus raising the concern that some respondents may have reported only CSA involving physical force. This appears to be relatively uncommon: in a national sample of 710 Australian women, Fleming (1997) found that only 7% of CSA victims reported use of physical force by the perpetrator, while an additional 64% reported verbal coercion. While it is likely that in our sample, respondents interpreted being 'forced into sexual activity' in a broad sense rather than restricting it to purely physical compulsion, it should be kept in mind that the psychological salience and sequelae of CSA may differ when, for example, it is the result of physical force by a stranger *versus* emotional duress by a relative – though both may be interpreted as 'force'.

Other limitations should also be noted. The assessment instrument did not specifically evaluate subjects for many forms of psychopathology (e.g. personality disorders, psychotic illnesses, or posttraumatic stress disorder). Additionally, this sample, a volunteer panel of twins, is unlikely to be strictly representative of the population from which it was derived (Heath *et al.* 1997). Finally, our analyses showed that rates of psychopathology were increased among abused respondents from families where the respondent's twin sister had also been abused, thus limiting the generalizability of matched-pairs comparisons of abuse-discordant twin pairs.

These drawbacks must be balanced against the advantages of having the opportunity to study a large group for whom systematic data

on a wide range of psychopathological symptoms were available. This sample avoided potential bias associated with selection through treatment. Use of a twin sample also had the advantage of allowing testing for genetic vulnerability factors as well as allowing the use of a co-twin-control design to account for effects of shared environment. Moreover, data from twin pairs concordant for abuse permitted demonstration of markedly elevated odds for psychopathology – information which would have been missed in conventional discordant-pair analyses.

Indirect support for the validity of our findings is lent by comparability to other reports. As would be expected given our restrictive definition, our prevalence estimate of CSA is somewhat lower than but consistent with other general-population estimates obtained using structured interviews to assess a wide range of psychiatric symptomatology (Siegel *et al.* 1987; Stein *et al.* 1988). While reported prevalence of CSA in our sample differs substantially from other Australian samples (Goldman & Goldman, 1988; Mazza *et al.* 1996; Fleming, 1997), our restrictive definition accords well with a finding that approximately 6% of women in a sample of undergraduate students from Queensland reported more severe forms of abuse prior to age 17 (Goldman & Padayachi, 1997), and is actually somewhat higher than prevalence of comparable acts reported in a community sample from New Zealand (Mulder *et al.* 1998).

Estimates of risk for psychiatric illness also yielded results similar to other studies (Putnam *et al.* 1986; Siegel *et al.* 1987; Roesler & McKenzie, 1994). Also consistent with other studies, associations were found between CSA and a variety of psychiatric disorders, in our sample including conduct disorder, major depression, suicidal ideation and suicide attempt, panic disorder and alcohol dependence in both men and women, and with social phobia in women.

Notably, these associations were attenuated in the co-twin–control analyses, with non-abused twins appearing not to have substantially differing risk for psychopathology. Nonetheless, in analysis of the total sample, rates of psychopathology were elevated among abused twins, and despite the potential informativeness of the twin pair approach for establishing indirect causation, we were in most cases unable to reject the hypothesis of a direct causal contribution of history of CSA to risk of lifetime psychopathology. Exceptions to this general finding, where psychopathology was also significantly more common in the non-abused twin, were seen in two conditions. It is possible that increased likelihood of social phobia in women may reflect a causal influence of having an abused twin sister. Similarly, in both men and women, conduct disorder appeared to be associated with CSA.

In the case of alcohol dependence in women, even in a matched-pairs comparison, the increased proportion of abused women who were affected as compared to their non-abused sisters approached statistical significance. This result would be predicted if the association between CSA and alcohol dependence at least in part reflected a direct causal influence.

The decrease in odds based on findings from discordant twins may be seen as supporting the hypothesis that a substantial part of the risk for psychopathology might be due to shared vulnerability factors rather than stemming solely from CSA. In fact, among women at least, there was support for the hypothesis of familial aggregation of risk for CSA, with most of the variance accounted for by shared environment, though we did not have sufficient power to exclude the possibility that genetic factors might have had some influence on risk. On the other hand, for men, no influence of shared factors on risk was observed.

What might those shared environmental factors be? One possibility is that the abusers of women were more likely to be family members, whereas for men, abuse may have been more sporadic and perpetrators more likely not to be relatives. Additionally, our data indicate that a family history of alcoholism was an important predictor of risk for reporting CSA, suggesting that this variable might have indexed shared environmental factors, very likely ones relating to family strife and disorganization, in turn increasing the likelihood of concomitant physical and emotional abuse and neglect, which are well known to be associated with parental alcoholism (Steinglass *et al.* 1987; Velleman, 1992). On the other hand, associations between CSA and lifetime psychopathology were not changed significantly when parental alcoholism was con-

trolled for, suggesting that this factor does not play an important role in mediating that relationship, as other alcoholism researchers have also noted (Sher *et al*. 1997).

Our modelling did not indicate a substantial role for genetic factors in risk for CSA, though in women (as would be expected if a substantial proportion of the CSA was intrafamilial), risk aggregated within families. Further support for the importance of experiential factors was the finding that those twins concordant in reporting abuse were also at highest risk for psychopathology. With the exception of conduct disorder in men, in every category for which data were available, the odds ratios for psychiatric illness were lowest among twin pairs in which neither was abused and next lowest among pairs in which the co-twin only was abused. Odds ratios rose further among those in which the respondent only was abused and were consistently highest among those pairs where both twins reported abuse. Thus, our findings give further support to the conclusion that, rather than being a pure cause-and-effect relationship, the association between CSA and subsequent psychopathology in fact reflects a complex interplay of factors, very likely related to parenting. Whether this vulnerability might be mediated through lack of parental supervision and protection (thus exposing the child to outside risk), through failure to adequately equip the child with sufficient social and interpersonal competence to protect against victimization, or via other routes must await further investigation.

Clinically, CSA is a potentially devastating life experience. Its study is important not only because of its deleterious effects, but as a paradigm by which the complex and dynamic interplay between adverse life events and vulnerability factors (environmental and innate) can be studied. The twin study approach, by allowing for estimation not only of genetic but also of shared environmental effects, can deepen our understanding of how multiple factors of varying degrees of specificity can interact to result in mental illness. Our results indicate that study of such life events, in order to most accurately judge their potential impact on mental health, must include appropriate evaluation of relevant familial protective and vulnerability factors. Failure to take these into account may lead to an overly simplistic view of the aetiology of psychiatric illness, as well as minimizing the role of less dramatic but highly significant environmental risk factors.

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