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Corresponding Author: Associate Professor David M Fergusson

Christchurch Health and Development Study

Christchurch School of Medicine

Christchurch

New Zealand

Tel: (03) 3720-406

Fax: (03) 3720-405

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Childhood Sexual Abuse and Psychiatric Disorder in Young Adulthood:

Part II: Psychiatric Outcomes of Childhood Sexual Abuse

DAVID M FERGUSSON PH.D., L JOHN HORWOOD, M.SC., B.A.

and MICHAEL T LYNSKEY, M.SC.

From the Christchurch Health and Development Study, Christchurch School of Medicine, Christchurch, New Zealand

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ABSTRACT

Objective: This is the second in a series of papers that describe the prevalence, correlates and consequences of childhood sexual abuse (CSA) in a birth cohort of over 1000 New Zealand children studied to the age of 18 years. This paper examines the associations between reports of CSA at age 18 and DSM-IV diagnostic classifications at age 18.

Method: A birth cohort of New Zealand children was studied at annual intervals from birth to age 16 years. At age 18 years retrospective reports of CSA prior to 16 and concurrently measured psychiatric symptoms were obtained.

Results: Those reporting CSA had higher rates of major depression, anxiety disorder, conduct disorder, substance use disorder and suicidal behaviors than those not reporting CSA ($p<.002$). There were consistent relationships between the extent of CSA and risks of disorder with those reporting CSA involving intercourse having the highest risks of disorder. These results persisted when findings were adjusted for prospectively measured childhood family and related factors. Similar but less marked relationships between CSA and non-concurrently measured disorders were found.

Conclusions: The findings suggest that CSA, and particularly severe CSA, was associated with increased risks of psychiatric disorder in young adults even when due allowance was made for prospectively measured confounding factors.

Key Words: Childhood sexual abuse; DSM-IV disorders; childhood and family factors.

The previous paper in this series described the prevalence, characteristics and childhood correlates of reports of childhood sexual abuse (CSA) in a large cohort of New Zealand children studied to the age of 18. This paper addresses the question of the extent to which exposure to CSA increased risks of psychiatric disorder in young adulthood.

There has been increasing interest in the extent to which exposure to CSA may increase later vulnerability to psychiatric disorder and there has been growing evidence to suggest that individuals who report CSA are at increased risks of a range of outcomes including depression (Bifulco et al., 1991; Burnam et al., 1988; Mullen et al., 1993; Sedney and Brooks, 1984; Winfield et al., 1990), anxiety and fears (Briere and Runtz, 1988; Burnam et al., 1988; Mullen et al., 1993; Murphy et al., 1988; Sedney and Brooks, 1984; Winfield et al., 1990), substance abuse (Burnam et al., 1988; Bushnell et al., 1992; Briere and Runtz, 1988; Mullen et al., 1993; Sedney and Brooks, 1984; Winfield et al., 1990), suicidal behaviors (Briere and Runtz, 1986; 1987; Mullen et al., 1993; Peters and Range, 1995; Sedney and Brooks, 1984) and other psychiatric symptoms (Briere and Runtz, 1988; Chu and Dill, 1990; Runtz and Briere, 1986; Rowan et al., 1994; Sanders and Giolas, 1991; Winfield et al., 1990).

While there is consistent evidence to suggest that reports of CSA are associated with higher rates of psychiatric disorder and symptoms, it is open to debate whether these associations reflect direct cause and effect relationships. In particular, there are two potentially major threats to validity in drawing causal conclusions about the associations between CSA and psychiatric disorder.

Confounding. First, it could be proposed that the associations between CSA and psychiatric adjustment may not arise from a direct cause and effect association in which CSA exposure leads to greater vulnerability to psychiatric disorder but rather, may arise from

confounding social, family and contextual factors that are associated with both CSA and increased risks of disorder (Green, 1993; Paradise et al., 1994; Plunket and Oates, 1991; Stern et al., 1995). There is some evidence to support this conjecture. First, risks of CSA do not appear to be randomly distributed across the childhood population and appear highest in children reared in dysfunctional family environments characterised by parental conflict, impaired parent child relationships and parental adjustment problems (Fergusson et al, submitted; Fergusson et al, in press; Mullen et al, 1993; Paradise et al, 1994; Stern et al, 1995). In general, it would appear children exposed to CSA, and particularly severe CSA involving intercourse, are exposed to a more adverse childhood ecology and it could be suggested that, in part, the associations between CSA and risks of psychiatric disorder arise because of the childhood and family context within which CSA occurs rather than as a result of the direct traumatic effects of CSA (Mullen et al, 1993; Paradise et al, 1994).

Recall bias. A second threat to validity arises from recall bias. It might be suggested that associations between CSA and psychiatric disorder reflect a recall bias in which those prone to psychiatric disorder are more prone to report or disclose CSA than those not prone to psychiatric disorder. This would result in an artefactual association between reports of CSA and psychiatric disorder in which some component of this association reflected the effects of mental state on the reporting of CSA.

The present study provided opportunities to address both of these issues. Since the study is based on a longitudinal design in which childhood and family circumstances have been assessed prospectively and prior to the disclosure of CSA, it was possible to control associations between CSA and psychiatric adjustment for prospectively measured risk factors.

It was more difficult to address threats to validity arising from recall bias in CSA reports

since, as we have noted, the only practical method of gathering CSA data on this cohort was through the use of retrospective reports of CSA when sample members became young adults. The use of retrospective measures of CSA raises the possibility that the reporting of CSA may have been contaminated by the individual's mental state at the time CSA was assessed. Nonetheless, the longitudinal nature of the design permits some exploration of the extent to which mental state variables may have led to contamination of reports of CSA. In particular, if the reporting of CSA was influenced by current mental state, it would be expected that the associations between CSA and psychiatric disorder would be stronger when CSA and disorder were measured concurrently than they would be when CSA and disorder were measured non-concurrently.

Against this general background the aims of this paper were as follows:

i) To document the extent to which reports of CSA made at age 18 were related to concurrently assessed measures of psychiatric disorder and psychiatric symptoms.

ii) To examine the extent to which associations between reports of CSA at age 18 and concurrently measured psychiatric symptoms could be explained by childhood and family factors that were likely to be associated with both increased risks of CSA and with increased vulnerability to psychiatric disorders and symptoms.

iii) To examine the extent to which associations between CSA and psychiatric symptoms varied when: a) CSA reports and symptoms were assessed concurrently and b) when CSA reports and symptoms were assessed non-concurrently.

METHOD

The study was based on a sample of 1019 eighteen year olds who were participants in the Christchurch Health and Development Study. The selection of this sample and study

response rates have been described in a previous paper in this series (Fergusson et al, in press). At age 18 all subjects were questioned in private on a structured interview which lasted between 1.5 to 2 hours and assessed a range of mental health and related topics. All interviewing was conducted by trained and experienced staff recruited for the study.

The following measures were used in the analysis.

Exposure to CSA. As explained previously, at age 18 subjects were questioned about their exposure to CSA prior to the age of 16 years. On the basis of this questioning subjects were assigned to one of four groups reflecting the severity of exposure to CSA. These groups were: a) those not reporting CSA; b) those reporting non-contact CSA only; c) those reporting contact CSA not involving attempted or completed intercourse; d) those reporting contact CSA involving attempted or completed vaginal, oral or anal intercourse. This assignment was based on detailed inspection of the history of CSA given by each subject on the basis of both responses to screening items and narrative material about the subject's CSA experiences.

Psychiatric disorder (16-18 years). Concurrently with the measurement of CSA, subjects were questioned about their psychiatric symptoms over the period from 16-18 years using a questionnaire based upon the Composite International Diagnostic Interview (CIDI, World Health Organisation, 1993) supplemented by an instrument based on the Self-Report Delinquency Instrument (SRDI, Elliott and Huizinga, 1989). The CIDI items were used to assess mood disorders, anxiety disorders and substance use disorders in the sample while the SRDI was used to assess conduct disorder.

Using this information DSM-IV criteria (American Psychiatric Association, 1994) were used to construct a series of diagnoses of psychiatric disorder for each subject over the period from 16-18 years. These diagnoses included:

i) Major depression. This was assessed using CIDI items. Subjects were classified as having a major depressive disorder if they met DSM-IV criteria for at least one major depressive episode over the period from 16-18 years. Overall, 21.8% of the sample reported at least one major depressive episode during the two year period, with 7.0% meeting DSM-IV criteria for current major depression.

ii) Anxiety disorders. These were assessed from CIDI items for generalized anxiety disorder, social phobia, specific phobia, panic disorder and agoraphobia. Subjects were classified as having an anxiety disorder if they met DSM-IV criteria for at least one of the above. Overall 17.1% of the sample were classified as having an anxiety disorder during the period from 16-18 years, with 2.9% reporting generalized anxiety disorder, 7.8% reporting social phobia, 9.6% specific phobia, 2.9% a panic disorder and 1.5% agoraphobia.

iii) Conduct disorder. This was assessed using the SRDI items. Subjects were classified as having conduct disorder (CD) if they reported 3 or more of the 13 age appropriate DSM-IV criteria for CD. The criteria relating to "staying out at night despite parental prohibition" and "often truants" were not included on the grounds that these items were not appropriate for the assessment of CD in 18 year olds. The prevalence of CD in the cohort was 4.8%.

iv) Alcohol abuse/dependence. These diagnoses were assessed using CIDI items. Subjects were classified as showing alcohol dependence if they reported experiencing at least three of the following: increasing tolerance for alcohol; withdrawal symptoms when alcohol was ceased; heavy drinking and overuse of alcohol; unsuccessful attempts to quit or cut down on drinking; large amounts of time spent in alcohol related activities; restriction of social and other activities as a result of drinking; physical or psychological problems caused by heavy

and prolonged drinking. Subjects were classified as showing alcohol abuse if they did not meet criteria for alcohol dependence and reported at least one of the following: alcohol misuse had led to repeated difficulties at school or neglect of schoolwork, difficulties at work or failure to attend work; use of alcohol had placed them at physical hazard from drink driving, accident or they had been injured in a fall or accident as a result of drinking; they had been arrested or stopped by police for alcohol related offending on at least two occasions; they had continued alcohol use despite objections from family or friends or after alcohol use had caused legal, financial or personal problems. Overall 13.8% of the sample met criteria for alcohol abuse and a further 5.7% met criteria for alcohol dependence during the period from 16-18 years.

v) Other substance abuse/dependence. This was based on CIDI items using DSM-IV criteria and was assessed using similar criteria to those used for alcohol abuse/dependence. Overall 9.1% of the sample met criteria for other substance abuse including cannabis abuse (7.2%) and abuse of other substances (excluding nicotine) (2.7%), and 4.0% of the sample met criteria for other substance dependence including cannabis dependence (3.8%) and other substance dependence (excluding nicotine) (0.8%).

Suicide attempt. Subjects were questioned about suicidal behaviors over the period from 16-18 years. On the basis of this information a measure was constructed reflecting whether the subject had ever made a suicide attempt during this period. These estimates were combined with reports of suicide attempts prior to 16 (Fergusson and Lynskey, 1995) to produce an estimate of suicide attempts made prior to age 18. Using this definition 5.4% of the cohort reported making a suicide attempt before 18.

Psychiatric disorder (14-16). Parallel to the diagnostic classification made at 18 years,

diagnoses of major depression, anxiety disorder, conduct disorder, alcohol abuse/dependence and other substance use disorders were obtained from measures of symptoms gathered at ages 15, 16 years. The methods used to construct these diagnostic groupings have been described previously (Fergusson, Horwood and Lynskey, 1993).

5. Confounding factors. A previous paper identified a series of prospectively measured childhood and family factors that were associated with increased risks of CSA (Fergusson et al, in press). These factors included: measures of family socio-demographic background (gender, maternal age, education, ethnicity, family socio-economic status); family stability (changes of parents, family conflict, step-parenthood); parent child relationships (extent of childhood adversity, parental bonding, parental attachment) and parental adjustment (parental substance abuse, psychiatric disorder and criminal offending). These prospectively measured risk factors were used to control associations between CSA and risks of disorder for possible confounding.

To explore the resilience of associations between CSA and risks of disorder to control for childhood factors, additional covariate factors were included in the analysis. The measurement of these additional factors has been described in previous papers and these factors included: measures of conduct problems and anxiety/withdrawal by age 8 years (Fergusson and Horwood, 1993); measures of child intelligence at age 8 years and scholastic ability at age 13 years (Fergusson and Horwood, 1995); measures of self-esteem based on the Coopersmith Self-esteem Inventory obtained at 10 years and 15 years (Lawton, Fergusson and Horwood, 1989); a measure of child neuroticism based on a short form version of the Eysenck Personality Inventory (Eysenck and Eysenck, 1964); measures of adolescent life events during the period from 14-16 years based on the Feeling Bad Scale (Lewis et al., 1984); and measures of affiliations with delinquent peers in adolescence (Fergusson and Horwood, 1996).

RESULTS

The Associations Between Risks of Disorder and the Extent of CSA.

Table 1 shows the sample of 1019 subjects classified into four groups according to reports of the extent of CSA exposure prior to the age of 16. The four groups are: a) those who did not report CSA; b) those who reported non-contact CSA only; c) those who reported contact CSA that did not involve attempted or completed intercourse; d) those reporting CSA involving attempted or completed vaginal, anal or oral intercourse. For each group the Table shows the rate of major depression, anxiety disorders, conduct disorder, substance use disorders and attempted suicide over the period from 16 to 18 years. Each comparison is tested for statistical significance using the chi square test. The associations between CSA exposure and risks of each outcome are described by three odds ratio estimates corresponding to contrasts between: a) those exposed to non-contact abuse only and those not subject to abuse; b) those subject to contact abuse not involving intercourse and those not subject to abuse and c) those subject to CSA involving intercourse or attempted intercourse and those not reporting abuse.

The Table shows the presence of consistent dose/response relationships between the extent of reported CSA and rates of disorder at age 18: in all cases those reporting severe CSA involving attempted or completed intercourse had the highest rates of disorder and in most cases those not reporting CSA had the lowest ($p<.002$). The odds ratio contrasts show:

i) Those exposed to non-contact abuse only had significantly ($p<.05$) higher rates of anxiety and depression. However rates of alcohol abuse, other substance use and suicide attempts were not significantly greater ($p>.05$) amongst those reporting non-contact abuse.

ii) Those reporting contact abuse not involving intercourse had significantly higher

rates ($p<.05$) of depression, anxiety, alcohol abuse/dependence and suicide attempts.

iii) In all cases those reporting severe CSA involving attempted/completed intercourse had significantly higher odds of disorder ($p<.001$) with the odds ratio between severe CSA and disorder ranging from 3.3 to 11.8.

INSERT TABLE 1. HERE

Control for Confounding Factors.

Whilst the results in Table 1 suggest that those reporting CSA, were at increased risk of disorder at age 18, it could be suggested that these associations were spurious and arose from other social, family and related factors that were associated with both increased risks of CSA and increased risks of later adjustment problems.

To take into account these factors, the associations between CSA and disorder were adjusted (using logistic regression methods) for a series of prospectively measured childhood and family factors that included: gender; measures of family social background (parental age, education, family socio-economic status, ethnicity); measures of family stability (changes of parents, parental conflict, adverse family life events); measures of parenting and parent-child relationships (parental attachment, parental bonding, overall family functioning); measures of parental adjustment (substance use/abuse, offending, psychiatric illness). The results of these analyses are summarised in Tables 2 and 3.

Table 2 shows rates of disorder adjusted for confounding factors. The Table also shows significance levels for log likelihood ratio chi squared tests of the association between CSA and risks of disorder after adjustment for confounding factors and the confounding factors that were found to be significant. The Table shows:

i) In all cases associations between CSA and rates of disorder remained statistically

significant ($p<.01$) after adjustment for potentially confounding childhood and family factors.

ii) For four out of the six outcomes (major depression, anxiety disorders, alcohol abuse/dependence, suicide attempt) adjustment for confounding factors reduced the strength of association between CSA and risks of disorder. The association between other substance abuse/dependence and CSA remained almost unaffected by adjustment for confounders. However, for the conduct disorder measure, the adjusted association with CSA appeared to be stronger than the unadjusted association. The reasons for this were that control for gender differences led to an increase in the size of the association.

INSERT TABLE 2. HERE

Table 3 gives adjusted odds ratio estimates for three contrasts: a) the comparison between those reporting non-contact CSA only and those not reporting CSA; b) the comparison between those reporting contact CSA not involving intercourse and those not reporting CSA; c) those reporting CSA involving attempted/completed intercourse and those not reporting CSA. The Table also gives estimates of the population attributable risk (PAR). The PAR estimates the reduction in rates of disorder that would have occurred had all subjects not been exposed to CSA. Table 3 shows:

i) For three of the five comparisons given (alcohol, other substance abuse/dependence, suicide attempt) there was no significant association between non-contact abuse and risks of disorder. However, even after adjustment for childhood and family factors those reporting non-contact CSA at age 18 had significantly ($p<.05$) higher rates of major depression and anxiety disorders.

ii) For four of the six analyses comparing those exposed to contact CSA not involving intercourse with those not reporting CSA there was evidence of increased risk amongst those reporting CSA. The comparisons involved major depression, anxiety disorders, conduct

disorder and alcohol abuse/dependence. However, contact CSA not involving intercourse was not significantly associated with risks of suicide attempts and other substance abuse behaviors.

iii) For all comparisons the results show that even after adjustment, CSA involving attempted/completed intercourse was associated with increased risks of disorder. Those reporting severe CSA involving intercourse had adjusted odds of disorder that were between 2.7 to 11.9 times higher than those not reporting CSA.

iv) The population attributable risk estimates suggest that, if all CSA in this cohort had been eliminated, risks of disorder for cohort members would have been reduced by between 9.3% to 19.5%.

INSERT TABLE 3. HERE

Threats to Validity

Tables 2 and 3 suggest that even when due allowance was made for confounding factors, CSA (and particularly severe CSA involving intercourse) was associated with increased risks of adolescent disorders. There are two major threats to the validity of these conclusions:

i) Failure to control confounding factors. It may be suggested that the analysis failed to control a number of relevant confounding factors and that, as a consequence, the estimates between CSA and risks of disorder shown in Tables 2 and 3 may over-estimate the impact of CSA on adolescent adjustment. To address this issue the analysis was extended to include a further series of factors that may have been related to risks of CSA but which may also have been a consequence of CSA. These factors included: early conduct disorder, child intelligence, school achievement, self-esteem during childhood, adolescent life events,

neuroticism and affiliations with delinquent peers. It was found that, even after control for these additional confounding factors, the associations between CSA and risks of disorder were similar to the association shown in Tables 2 and 3. This suggests that the estimates shown in these Tables are resilient to the control for a large number of potentially confounding childhood and family factors.

ii) Recall bias. It could be proposed that the associations between reports of CSA made at age 18 and risks of disorder at age 18 may arise from the effects of a recall bias in which those prone to psychiatric disorder were more prone to report or recall CSA.

It was possible to examine this issue by estimating the associations between CSA and risks of disorder at the age of 14 to 16 years. This analysis is shown in Table 4 which gives odds ratio estimates corresponding to contrasts between a) those reporting non-contact abuse and those not reporting abuse; b) those reporting contact abuse not involving intercourse and those not reporting abuse; c) those reporting CSA involving attempted/completed intercourse and those not reporting abuse. The Table shows these estimates before and after adjustment for the confounding factors used in the adjustments in Table 3.

Comparison of the results in Tables 3 and 4 suggest that analysis of the relationships between CSA and concurrently and non-concurrently measured disorder produced generally similar conclusions.

i) For both analyses there was clear evidence to suggest that CSA involving intercourse was associated with increased risks of disorder even after adjustment for confounding factors. The results in Table 3 suggest CSA involving intercourse was associated with ORs of between 2.7 to 11.9 using concurrently measured disorder whereas the ORs for non-concurrently measured disorder ranged from 3.0 to 8.7.

ii) Both analyses lead to similar conclusions about the relationships between contact

CSA not involving intercourse and risks of disorder. However, the concurrently measured disorder variables show slightly stronger associations with CSA reports. In particular the data in Table 3 show that four of the five associations between contact CSA not involving intercourse and disorder were significant whereas the results for non-concurrently measured disorder show that only one of these associations was significant after adjustment for covariates.

iii) Both sets of analyses lead to the general conclusion that, as a rule, non-contact abuse was not strongly associated with increased risks of disorder: for concurrently measured disorder two associations remained significant after control for covariates in comparison to only one of the five associations for non-concurrently measured disorder.

INSERT TABLE 4. HERE

DISCUSSION

This study has used data gathered over the course of an 18 year longitudinal study to examine the extent to which exposure to CSA was associated with increased risks of psychiatric disorders in young adulthood. The major findings of this analysis are reviewed below.

The Role of CSA in Psychiatric Disorder

In confirmation of findings from previous studies (Bifulco et al., 1991; Briere and Runtz, 1986; 1987; 1988; Burnam et al., 1988; Bushnell et al., 1992; Mullen et al., 1993; Murphy et al., 1988; Peters and Range, 1995; Sedney and Brooks, 1984; Winfield et al., 1990), those reporting CSA at age 18 years had increased risks of common psychiatric disorders including mood disorders, anxiety disorders, conduct disorders, substance use disorders and suicidal behaviours both at age 16 years and age 18 years. Odds ratio estimates

suggested that those exposed to severe CSA had odds of these outcomes that were 2.7 to 11.9 times higher than those of young people not exposed to CSA. These findings are generally consistent with a growing body of evidence that suggests that risks of psychiatric disorder are elevated amongst those reporting CSA. Further, in confirmation of a number of recent studies (Collings, 1995; Fergusson et al, submitted; Mullen et al, 1993), the analysis suggested the presence of dose/response relationships between the extent of exposure to CSA and risks of disorder. In general, risks of disorder appear to increase with increasing CSA severity. These results are generally consistent with the view that exposure to CSA may act as a risk factor that increases later vulnerability to psychiatric disorder. There are, however, two major threats to the validity of this conclusion.

First, it may be suggested that the associations between CSA and risks of disorder may reflect confounding social, family and related factors that are associated with exposure to CSA and that contribute independently to risks of later disorder (Beitchman et al, 1992; Browne & Finkelhor, 1986; Finkelhor, 1990; Plunket & Oates, 1990). The present study was in a strong position to control this threat to validity owing to the availability of a wide range of prospectively measured childhood, family and related factors. With one exception, control for childhood factors reduced the associations between CSA and risks of disorder suggesting that, in part, these associations arose from common childhood factors associated with CSA rather than from the direct effects of CSA. Nonetheless, even after extensive attempts to control for antecedent childhood factors, associations between CSA and risks of disorder remained.

The exception to this trend occurred for conduct disorder and it was found that control for antecedent factors tended to increase rather than reduce the associations between CSA and conduct disorder. The reasons for this centred around the associations between gender, sexual abuse and conduct disorder: gender was positively associated with conduct disorder with

males being more prone to these disorders but negatively associated with CSA with females being more prone to CSA. The net effects of these associations were that, when gender was taken into account, the associations between CSA and conduct disorder tended to increase.

A second threat to validity arises from the possibility of recall bias. In particular, it could be suggested that the associations between reports of CSA at the age of 18 and risks of disorder arose because those with disorder were more prone to report CSA. It was not possible to eliminate this possibility using the present design. Nonetheless, the evidence clearly suggests that the associations between CSA and risks of disorder are unlikely to reflect the effects of short term mental state on the reporting of CSA since associations were found between reports of CSA and disorder assessed two years prior to the disclosure of CSA.

On the other hand, there was some evidence to suggest the possibility of some recall bias in this association. In particular, the analysis shows that those reporting non-contact CSA had increased rates of anxiety and depression even after adjustment for confounding factors. The finding that those reporting non-contact abuse were at increased risks mood and anxiety disorders is not consistent with the evidence reported in the previous paper (Fergusson et al, in press) that suggested that most of those reporting non contact abuse did not perceive these events as abusive or distressing. This inconsistency suggests a possible recall bias in which those who were prone to anxiety or depression may have been more prone to report and recall minor episodes of non-contact abuse. This bias, however, appears to be confined to anxiety and mood disorders since non-contact abuse was not associated with increased risks of other disorders.

From the results of the present study it was possible to estimate the extent to which exposure to CSA contributed to risks of disorder in this cohort. Estimates of the population

attributable risks suggested that, if all CSA in this cohort had been eliminated, rates of specific disorders in the cohort would have been reduced by between 9.3% to 19.5% of the current prevalence rates.

Clinical and Related Implications

The topic of childhood sexual abuse has been associated with strong claims about the role that this experience plays in the etiology of psychiatric disorder with these claims sometimes implying that the presence of psychiatric disorder is frequently or invariably symptomatic of sexual abuse and consequent claims about the need to recover lost or repressed memories of such abuse amongst those presenting with psychiatric disorders (Fredrickson, 1992; Herman, 1992). The present study suggest that, while childhood sexual abuse was a significant risk factor for later psychiatric disorder, the impact of this factor on risks of disorder may not be as large and pervasive as has sometimes been claimed. The estimates from this study suggest that in the region of 10% to 20% of the risks of psychiatric disorder in young adults may be accounted for by exposure to CSA.

The major clinical implication of such findings is clearly that there is a need place CSA in a more general clinical perspective as being one of a large class of adverse childhood factors spanning social disadvantage, family dysfunction and childhood adverse life events which individually make relatively small contributions to risks of disorder but which in combination may have a large impact on individual adjustment.

The specific clinical implications of this conclusion seem clear. First, given the role of sexual abuse in contributing to disorder, it is important that clinical practice includes attempts to intervene with children exposed to CSA to reduce further risks (Green, 1993) and that assessment of childhood experiences of sexual abuse forms an important part of the

assessment of risk factors that may contribute to risks of individual disorder. Second, while the assessment and treatment of CSA forms an important part of clinical practice, this risk factor in isolation should not be elevated to the status of being the primary determinant, cause and explanation of psychiatric disorder. Finally, the results raise important issues about the extent to which the diagnosis and treatment of CSA should be treated in isolation and the extent to which this treatment should be embedded in the more general clinical management of psychiatric disorder. To the extent that the evidence suggests that CSA is only one of many factors that may contribute to risks of psychiatric disorder, the grounds for treating CSA as a specific source of disorder separate from all other causes of disorder are substantially diminished.

The present study raises some further issues that bear on the way in which the role of CSA in the development of psychiatric disorder has sometimes been exaggerated. In particular, what characterises the present study and indeed, all other studies of the relationships between CSA and psychiatric disorder is the presence of methodological uncertainties in establishing the exact nature of the relationships between CSA and risks of disorder. These uncertainties centre around a series of issues relating to: a) the accuracy with which CSA is reported; b) the extent to which the associations between CSA and psychiatric disorder may be contaminated by recall and other biases; c) the extent to which associations between CSA and risks of disorder may reflect the effects of third or confounding factors. It is difficult to address all of these threats to validity in a way that provides a watertight argument about the role of CSA in psychiatric disorder. Under conditions of such uncertainty it is clear that conclusions about the role CSA in risks of disorder should be made cautiously. An unfortunate feature of many public presentations of CSA and risks of psychiatric disorder has been a tendency for strong conclusions to be drawn about the role of CSA in the etiology

of disorder with little regard for the very real uncertainties that surround evidence in this area.

Within the limitations that apply to studies of CSA based on information from self reports, the present study suggests that exposure to CSA, and particularly severe CSA involving intercourse, acts as a factor that increases individual vulnerability to psychiatric disorder. This association cannot be explained adequately by confounding childhood and related factors but the possibility that the association between CSA and disorder may be contaminated by recall bias in which those prone to disorder (and particularly anxiety and mood disorders) are more prone to report or recall CSA cannot be discounted entirely. The weight of the evidence points to the conclusion that CSA may play a significant, but not overwhelmingly strong, role in determining individual vulnerability to psychiatric disorder.

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Table 1. Rates (%) of disorder (16-18 years) by extent of CSA and odds ratios (95% confidence intervals) for disorder for each type of CSA in comparison to non-abused group

	Extent of CSA					Odds Ratios (95% Confidence Intervals) for outcome (in comparison to non-abused group)		
Outcome	None (N=913)	Non-contact (N=24)	Contact (N=46)	Intercourse (N=36)	p	Non-contact Abuse only	Contact Abuse/Not Intercourse	Intercourse
Major depression	18.0	50.0	50.0	63.9	<.0001	4.6 (2.0-10.3)	4.6 (2.5-8.3)	8.1 (4.0-16.3)
Anxiety disorder	14.2	41.7	39.1	44.4	<.0001	4.3 (1.9-9.9)	3.9 (2.1-7.2)	4.8 (2.4-9.5)
Conduct disorder	4.3	0.0	8.7	16.7	<.002	-[a]	2.1[b] (0.7-6.3)	4.5 (1.8-11.4)
Alcohol abuse/dependence	17.6	29.2	34.8	41.7	<.0001	1.9[b] (0.8-4.7)	2.5 (1.3-4.7)	3.3 (1.7-6.6)
Other substance abuse/dependence	11.2	8.3	15.2	38.9	<.0001	0.7[b] (0.2-3.1)	1.4[b] (0.6-3.3)	5.1 (2.5-10.2)
Suicide attempt (ever)	4.1	4.2	10.9	33.3	<.0001	1.0[b] (0.1-7.8)	2.9 (1.1-7.7)	11.8 (5.5-25.5)

[a] Unable to estimate OR because of 0% prevalence of conduct disorder in non-contact abuse group

[b] OR not significantly different from 1 (p>.05).

Table 2. Rates (%) of disorder (16-18 years) after adjustment for covariates

Outcome	EXTENT OF CSA				p	Significant Covariates
	None	Non-Contact	Contact	Intercourse		
Major depression	17.1	39.5	35.5	48.6	<.001	1,5,8
Anxiety disorder	14.5	30.6	32.0	32.9	<.001	1,2,8,9
Conduct disorder	3.9	3.9[a]	14.7	26.0	<.001	1,4,8
Alcohol abuse/dependence	17.9	28.1	39.2	35.7	<.01	1,8,9
Other substance abuse/dependence	10.6	8.2	17.0	39.0	<.001	1,2,6,8
Suicide attempt (ever)	4.1	3.3	8.3	15.2	<.01	3,4,7,8,10

[a] For analytic purposes rate assumed to be the same as for non-abused group. See footnote to Table 1.

[b] Covariates: 1 = gender; 2 = maternal age; 3 = family socio-economic status; 4 = ethnicity; 5 = changes of parents; 6 = childhood adversity; 7 = maternal over-protection; 8 = parental attachment; 9 = parental history of offending; 10 = parental use of illicit drugs

Table 3. Odds ratios (95% confidence intervals) for disorder (16-18 years) for each type of CSA after adjustment for covariates, and estimated population attributable risk (PAR) for any CSA

	ADJUSTED ODDS RATIO (95% CONFIDENCE INTERVAL)			
Outcome	Non-Contact Abuse Only	Contact Abuse/ Not Intercourse	Intercourse	Estimated PAR for any CSA
Major depression	3.6 (1.3-9.6)	3.0 (1.4-6.1)	5.4 (2.4-12.3)	14.0%
Anxiety disorder	2.8 (1.1-7.1)	3.0 (1.5-6.1)	3.2 (1.5-6.7)	13.3%
Conduct disorder	--[a]	5.0 (1.4-17.9)	11.9 (3.7-38.3)	18.5%
Alcohol abuse/dependence	1.9[b] (0.7-5.0)	3.2 (1.6-6.5)	2.7 (1.2-6.0)	9.3%
Other substance abuse/dependence	0.7[b] (0.1-5.8)	1.8[b] (0.7-4.8)	6.6 (2.9-15.2)	10.8%
Suicide attempt (ever)	0.8[b] (0.1-6.5)	2.3[b] (0.7-7.1)	5.0 (1.9-13.2)	19.5%

[a] Unable to estimate adjusted OR because of 0% prevalence of conduct disorder in non-contact abuse group. For calculation of PAR adjusted rate of disorder assumed to be same as for non-abused group.

[b] OR not significantly different from 1 ($p>.05$)

Table 4. Odds ratios (95% confidence intervals) for disorder 14-16 years for each type of CSA in comparison to non-abused group, before and after adjustment for covariates

Outcome	ODDS RATIO (95% CONFIDENCE INTERVAL)		
	Non-Contact Abuse Only	Contact Abuse/ Not Intercourse	Intercourse
Major depression - before adjustment	3.8 (1.5-9.7)	3.8 (1.9-7.7)	7.3 (3.6-14.9)
- after adjustment	2.0[a] (0.6-6.1)	1.3[a] (0.5-3.5)	3.1 (1.3-7.7)
Anxiety disorder - before adjustment	3.4 (1.5-8.0)	4.0 (2.1-7.6)	5.2 (2.5-10.7)
- after adjustment	2.2[a] (0.8-6.0)	2.4 (1.1-5.4)	3.0 (1.2-7.2)
Conduct disorder - before adjustment	2.0[a] (0.6-6.8)	0.6[a] (0.1-2.7)	8.6 (4.2-17.9)
- after adjustment	4.8 (1.1-21.0)	0.3[a] (0.03-2.5)	8.7 (2.9-26.3)
Alcohol abuse/dependence - before adjustment	1.6[a] (0.5-5.5)	2.1[a] (0.9-4.8)	6.2 (3.0-12.9)
- after adjustment	2.1[a] (0.5-8.2)	0.8[a] (0.2-3.1)	3.3 (1.3-9.0)
Other substance abuse/dependence - before adjustment	1.4[a] (0.2-10.6)	1.5[a] (0.3-6.5)	8.9 (3.7-21.5)
- after adjustment	2.3[a] (0.3-19.5)	1.7[a] (0.3-7.9)	7.3 (2.6-20.8)

[a] Odds ratio not significantly different from 1 ($p>.05$)