Childhood abuse and psychotic experiences in adulthood: findings from a 35-year longitudinal study

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Abstract

Importance: The extent to which exposure to sexual and physical abuse in childhood increases the risk of psychotic experiences in adulthood is currently unclear.

Objective: To examine the relationship between childhood sexual abuse, childhood physical abuse and psychotic experiences in adulthood taking into account potential confounding and time-dynamic covariate factors.

Design, Setting, Participants: This longitudinal birth cohort study used data from the Christchurch Health and Development Study which consisted of 1265 participants studied periodically from birth (1977) to 35 years.

Exposure: At ages 18 and 21 years cohort members were questioned about exposure to childhood sexual and physical abuse (<age of 16).

Main outcomes and measures: At ages 30 and 35 years, cohort members were questioned about psychotic experiences (symptoms of abnormal thought and abnormal perception). Generalized Estimating Equation models were used to investigate covariation of the association between abuse exposure and psychotic experiences. These included both potential confounding factors in childhood (socioeconomic disadvantage, adverse family functioning) and time-dynamic covariate factors (mental health and substance use, along with life stress and unemployment).

Results: Data were available for 962 participants (463 males, 499 females); 7.1% had been exposed to severe sexual abuse and 6.3% to severe physical abuse in childhood. Adjustment for confounding factors and time-dynamic covariate factors reduced the magnitude of associations between exposure to severe childhood sexual abuse and psychotic experiences in adulthood although they remained significant. Those exposed to severe sexual abuse had rates of abnormal thought symptoms that were 2.24 times higher and rates of abnormal perception symptoms that were 4.08 times higher than those in the “no exposure” group. There was no statistically significant association between exposure to severe childhood physical abuse and psychotic experiences after controlling for confounding and time-dynamic covariate factors.

Conclusion and Relevance: Findings from this study indicate that exposure to severe levels of childhood sexual abuse (but not physical abuse) is independently associated with an increased risk of

psychotic experiences in adulthood (particularly symptoms of abnormal perception) and this association could not be fully accounted for by either confounding or time-dynamic covariate factors.

Key points

Question: Is exposure to childhood sexual and physical abuse independently associated with psychotic experiences in adulthood, or is this association explained by other factors such as socioeconomic disadvantage, adverse family functioning, poor mental health, substance use or stressful life events?

Finding: Using data from a longitudinal birth cohort, those who had experienced severe childhood sexual abuse (but not physical abuse) had significantly increased psychotic symptoms as adults after controlling for a wide range of confounding and time-dynamic covariate factors.

Meaning: Exposure to severe childhood sexual abuse is independently associated with psychotic experiences in adulthood.

Psychotic experiences (PEs) of hallucinations and delusional experiences are common in the general population with lifetime prevalence rates of 5.8-12.5% being consistently reported by meta-analysis[1] and large epidemiological surveys.[2] These rates are considerably higher than those for psychotic disorders and there has been increasing recognition that PEs should no longer be viewed solely as risk indicators for psychotic illnesses.[3]

Over recent years the relationship between childhood adversity (CA) and both PEs and psychosis has been increasingly studied.[4, 5] Meta-analyses have reported that exposure to CA is associated with a 76% increased risk of PEs[6] and an increased risk for psychosis **(**OR=2.78; CI 2.34-3.31).[7] Although different forms of CA often co-occur, some specificity has been reported, with exposure to childhood sexual abuse (CSA) being particularly associated with symptoms of abnormal perception and childhood physical abuse (CPA) with symptoms of abnormal thought.[8, 9]

The relationship between CSA, CPA and PEs is complex. CSA and CPA often co-occur[10] and are associated with confounding risk factors that reflect other environmental exposure, such as childhood socioeconomic disadvantage and adverse family functioning.[11] There are also established associations between PEs and mental health disorders (such as anxiety, depression),[12] cannabis use,[13] exposure to stressful life events [14] and unemployment [2] occurring contemporaneously with PEs suggesting that these factors may mediate the association between CA and PEs. This complexity has been a major limitation of many previous studies which have not accounted for all of these factors.

This paper addresses these issues by using data from a 35-year longitudinal birth cohort study (the Christchurch Health and Development Study; CHDS) to examine the relationship between exposure to CSA, CPA and PEs in adulthood taking into account potential confounding and time-dynamic covariate factors.

Methods

Participants

Data were gathered from the CHDS, a birth cohort of 1265 individuals (635 males, 630 females) born in the Christchurch (New Zealand) urban region in mid-1977 and studied at birth, 4 months, 1 year and annually to age 16, and again at 18, 21, 25, 30 and 35 years.[15, 16] All information was confidential

and collected with signed consent. The study is approved by the Canterbury Ethics Committee. Sample sizes, based on outcome measures used in the study, were 984 (age 30) and 959 (age 35), representing 79-80% of the surviving sample at each observation. As in previous epidemiological studies of PEs,[2] an *a priori* decision was made to exclude participants with a formal diagnosis of psychotic disorder (by self-report at age 35). This resulted in exclusion of 3 participants. We did not have access to other clinical measures and it is possible that a small number of participants with psychotic illness, who had not reported these diagnoses to us, were not excluded.

Measures

Exposure to CSA and CPA (ages 0-16)

*CSA***.** At ages 18 and 21, sample members were questioned about their experience of CSA (<16 years). Those who reported this were questioned further and classified on a 3-level scale reflecting the most extreme form of CSA reported at either age; "no exposure" (85.9%), "some exposure" (non-contact abuse or contact CSA not involving attempted or completed intercourse) (7.8%) and "severe exposure" (attempted/completed oral, anal, or vaginal intercourse) (6.3%).

CPA. At ages 18 and 21, sample members were asked to describe the extent to which their parents used physical punishment during childhood.[17] This information was classified on a 3-level scale reflecting the most severe form of physical punishment; "no or rare exposure" (82.5%), "some exposure" (at least one parent used physical punishment on a regular basis) (11.2%), and "severe exposure" (at least one parent used physical punishment too often or too severely, or treated the respondent in a harsh or abusive manner) (6.4%).

Psychotic experiences (PEs)

At ages 30 and 35, sample members were questioned about their experience of PEs during the 5-year period since the previous assessment. Measurement of PEs was derived from the Diagnostic Interview Schedule for DSM IV,[18] assessing two classes of PEs: symptoms of abnormal thought (delusions of persecution or guilt, bizarre delusions, delusions of reference, passivity and thought control) and symptoms of abnormal perception (auditory, visual, olfactory, gustatory and tactile hallucinations). Cohort members were asked which PEs they had experienced, using a 3-point scale labeled "no",

"maybe" and "yes". A total symptom score for each class of symptoms for each participant was obtained by summing the number of items to which participants indicated "yes". For this investigation, the measures of abnormal thought and abnormal perception were used as separate outcome variables. Full details of the questions are given in the Online Supplement.

Potential confounders

A number of potential confounding factors were abstracted from the study database, on the basis that they have been shown to be related to both abuse exposure and PEs in adolescence and adulthood.[2]

Time-dynamic covariate factors (ages 25-30, 30-35)

A series of measures of mental health and substance use, along with life stress and unemployment (as a significant stress) were selected from the study database.

Details of the measurement of these potential confounding and time-dynamic factors are given in Online Supplement.

Statistical analysis

The data analyses took place over four steps.

In the first step, bivariate associations between the classification of exposure to CSA and CPA and the repeated measures of PEs in adulthood were obtained by fitting a series of negative binomial Generalized Estimating Equation (GEE) models to the data using Stata.

In the second step, bivariate associations between the classification of exposure to CSA and CPA and the potential confounding factors noted above were obtained via Spearman's rank-order correlations, estimated using SAS 9.4.

In the third step, the associations between: a) abnormal thought symptoms and abnormal perception symptoms at ages 30 and 35; and b) mental health, substance use, life stress and unemployment at ages 30 and 35; were obtained via Spearman's rank-order correlations, estimated using SAS 9.4.

In the final step, the GEE models described above were extended to include the potential confounding factors noted above. In order to investigate possible mediators of the association between abuse exposure and PEs, the adjusted models were further extended to include the time-dynamic covariate factors described above, entered simultaneously.

Full details are given in Online Supplement.

Results

Data were available for 962 participants; 7.1% had been exposed to severe CSA and 6.3% to severe CPA. 5.4% of the cohort reported one or more abnormal thought symptom at age 30, and 2.4% at age 35. At age 30, 3.7% reported at least one symptom of abnormal perception, while at age 35 the rate was 2.6% (changes in rates were due in part to losses to follow-up).

Associations between abuse exposure (0-16) and PEs (ages 25-30, 30-35)

As shown in Table 1, for CSA exposure, rates of PEs generally increased with increasing levels of CSA. Those in the "severe exposure" category reported significantly ($p < .05$) greater levels of both abnormal thought and abnormal perception symptoms over the periods 25-30 and 30-35 years than those in the "not exposed" or "some exposure" groups. The pooled rates (per 100) of PEs showed that the "severe exposure" group had rates of PEs that were 3.2 to 4.3 times higher than in the "not exposed" group.

Table 1 also shows that a similar pattern was observed for CPA, except that individuals in both the "some exposure" and "severe exposure" groups had pooled rates of PEs over the periods 25-30 and 30-35 years that were significantly ($p < .05$) higher than individuals in the "not exposed" group. The pooled rates (per 100) of PEs showed that the "some exposure" group had rates of symptoms that were 2.2 to 2.5 times higher than the "not exposed" group, and that the "severe exposure" group had rates of symptoms that were 2.4 to 3.7 times higher than the "not exposed" group.

Insert Table 1 Approx here

Associations between abuse exposure (0-16) and potential confounding factors in childhood

As noted above, the associations in Table 1 could, at least partially, be accounted for by the influence of childhood factors that increased the likelihood of exposure to either CSA or CPA. Table 2 shows that both CSA and CPA were significantly ($p < .05$) correlated with a series of sociodemograpic factors reflecting childhood disadvantage and a series of measures of adverse family functioning in childhood. The pattern of correlations shows that individuals with higher levels of abuse exposure were also more likely to have been exposed to higher levels of family dysfunction and parental

maladaptive behaviour during childhood. It also shows that a series of individual factors measured in childhood were also significantly ($p < .05$) associated with CSA and CPA. For example, those exposed to higher levels of CSA (but not CPA) were more likely to be female. Also, higher levels of CSA and CPA were related to: lower IQ; lower parental attachment; higher rates of conduct, attention problems, anxious/withdrawn behaviours in childhood; and higher rates of major depression, anxiety disorder, and suicidal ideation in mid-adolescence.

Insert Table 2 Approx here

Associations between PEs (ages 25-30, 30-35) and potential time-dynamic covariate factors related to mental health, substance use, life stress and unemployment

As noted above, the associations between abuse exposure in childhood and adult PEs could, in part, be explained by the effects of time-dynamic covariation. Table 3 shows that, with a few exceptions, there was a general pattern of moderate to strong statistically significant ($p < .05$) correlations between PEs and each of the mental health, substance use, life stress and unemployment measures, at ages 25-30 and 30-35 years. Those reporting higher rates of PEs were also more likely to meet criteria for mental health and substance use disorders, and to report higher levels of life stress and unemployment.

Insert Table 3 Approx here

Adjustment of associations between abuse exposure (ages 0-16) and PEs (ages 25-30, 30-35) for potential confounding factors, and time-dynamic covariate factors

The final step in the analyses involved fitting two pairs of GEE models (one pair for abnormal thought symptoms, and the other for abnormal perception symptoms) to the data. In the first model, for each symptom class, the three level indicators of CSA and CPA were entered simultaneously, followed by a series of potential confounding factors. In the second model, for each symptom class, the fitted model was augmented by a series of time-dynamic covariate factors measured at ages 30 and 35 years. Table 4 shows that for CSA, those in the "severe exposure" group had significantly ($p < .05$) higher rates of both abnormal thought and abnormal perception symptoms, after controlling for confounding and time-dynamic covariate factors. After controlling for confounding factors (Model 1), those in the "severe exposure" group had rates of abnormal thought symptoms that were 4.41 times higher than those in the "no exposure" group, and rates of abnormal perception symptoms that were

4.74 times higher than those in the “no exposure” group. Further adjustment for time-dynamic covariate factors (Model 2) reduced the magnitude of these associations, with those in the “severe exposure” group having rates of abnormal thought symptoms that were 2.24 times higher than those in the “no exposure” group, and rates of abnormal perception symptoms that were 4.08 times higher than those in the “no exposure” group. On the other hand, there was no evidence of a statistically significant difference in rates between the “some exposure” and the “no exposure” group.

Table 4 also shows that for CPA, those in the “severe exposure” group had significantly higher rates of abnormal perception symptoms after controlling for confounding factors (but not after controlling for time-dynamic covariate factors). After controlling for confounding factors, those in the “severe exposure” group had rates of symptoms that were 2.14 times higher than those in the “no exposure” group. Further control for time-dynamic covariate factors reduced this association to statistical non-significance, suggesting that the observed association between severe levels of physical abuse and PEs were mediated by contemporaneous mental health and substance use disorders, and life events. There was no evidence of a statistically significant association between exposure to CPA and abnormal thought symptoms after controlling for confounding and time-dynamic covariate factors, and no evidence of statistically significant differences between the “some exposure” and “no exposure” groups for either outcome, in either model.

Insert Table 4 Approx here

Discussion

Using data from a 35-year study of a longitudinal birth cohort (the CHDS) we report that those who had been exposed to severe levels of CSA (but not CPA) reported PEs at a higher frequency than those with no, or less severe CSA. This association could not be fully accounted for by either confounding factors or time-dynamic covariate factors.

The association between exposure to severe CSA and PEs was strong. After controlling for confounding and time-dynamic covariate factors, rates of abnormal perception and abnormal thought symptoms were 4.1 and 2.2 times higher respectively, compared to those without CSA exposure. Although there has been increasing evidence from cross-sectional studies linking exposure to CSA

with PEs [19] this study is the first to examine this association using a longitudinal design while investigating the impact of confounding and time-dynamic covariate factors.

As discussed above, the relationship between CSA and the reporting of PEs is complex and the analyses used in this study reflected this. We showed that exposure to CSA was associated with a large number of individual confounding factors and those associated with sociodemographic disadvantage and disturbed family functioning. Although confounding factors have been examined in cross sectional population studies[20] these have often relied on retrospective recall whereas this longitudinal birth cohort design was able to ascertain this information prospectively and over multiple time points. We also examined potential time-dynamic covariate factors and showed that those reporting higher rates of PEs were more likely to meet criteria for mental health and substance use disorders, and to report higher levels of life stress and exposure to unemployment. Previous studies have reported similar findings and noted the bi-directional associations between PEs and mental disorders with the presence of PEs increasing the risk of mental disorders, and most mental disorders increasing the risk of PEs.[21] Our analyses showed that it was important to adjust for these confounding and time-dynamic covariate factors but that doing so only reduced the magnitude of the association between CSA and PEs which remained significant.

The association between the experience of PEs and CSA was strongest when the exposure was severe, involving attempted or completed oral, anal, or vaginal intercourse. In fact there was no evidence of a statistically significant difference in rates of PEs between the "some exposure" group and the "no exposure" group. This dose-response effect has been reported previously,[22, 23] with the association between CSA and PEs being particularly strong when it involved sexual intercourse.[20]

We also examined the impact of another form of childhood adversity, CPA, on PEs. While there were observed associations between CPA and PEs these were explained by confounding factors and time-dynamic covariation arising from co-occurring mental health and substance use disorders, and life events. These findings contradict previous studies which have reported a significant association between exposure to CPA and PEs.[7, 24-26] We suggest that these differences are explained by the quality of our study design which controlled for a wide range of confounding and time dynamic covariate factors, and simultaneously modelled both CSA and CPA on PEs.

We also found some specificity of PEs reported with exposure to CSA particularly increasing the risk of symptoms of abnormal perception. These experiences were four times greater in the severely exposed group than those who had had no exposure (in contrast, abnormal thought symptoms were twice as high in those severely exposed). Previous studies have suggested that exposure to CSA can result in changes to emotional, cognitive and neurobiological processes such as increased emotional reactivity, poor emotion regulation and cognitive control. The mechanisms involved have not been established but may involve impacts on biological systems (dysregulated cortisol,[27] reduced cortical thickness[28] and changes in the dopamine system[29]) and/or psychological processes (source monitoring biases i.e. the ability to differentiate between internal and external stimuli, dissociation, and cognitive schema/thinking styles[30]).

The findings from this study have considerable clinical relevance. From a public health perspective they would suggest that much of the disease burden attributable to PEs in adults may be explained by CSA. Efforts to reduce exposure to CSA and to provide effective treatment for those exposed continues to be a vital public health challenge. The findings are consistent with clinical observations, and an increasing number of studies, that in many individuals who report PEs, this can be explained on the basis of CSA exposure and does not necessarily imply an underlying primary psychotic disorder.[31] Furthermore, understanding the etiological basis of these symptoms could help guide treatment, since although antipsychotic medications are an effective treatment for major psychotic disorders[32] their evidence as a treatment for PEs in people who do not have a psychotic disorder is much less clear.

Although there are considerable strengths to the study design there are also some limitations. The number of participants reporting PEs was relatively low (7.1%) however this is similar to other population studies i.e. lifetime prevalence 5.8-12.5%.[2, 33] Similarly the number of participants exposed to severe CSA is also relatively low i.e. 7.1% but also in the range reported by other general population studies i.e. 1.5-8.4%.[2, 33] The reports of CA were retrospective at ages 18 and 21 but we have previously shown that combining reports at ages 18 and 21 led to accurate classification of CA.[34] Other studies have also consistently demonstrated the validity and reliability of retrospective reports of trauma in these populations.[35]

Conclusions

This study provides some of the strongest evidence to date on the relationship between CSA and PEs in adulthood. The results show a robust association between exposure to CSA (but not CPA) and PEs, with a marked increase in risk for symptoms of abnormal perception in particular. While the study is unable to provide conclusive evidence of a causal link between CSA and PEs, these findings add to the large body of evidence showing that reducing exposure to CSA remains a vital public health challenge.

Table 1. Mean (per 100) number of psychotic experiences (PEs), by childhood sexual and physical abuse exposure.

	Exposure to childhood sexual abuse (CSA)		
	Not exposed	**Some exposure**	**Severe exposure**
Abnormal thought symptoms			
Age 30	12.7	12.5	46.9
n	840	80	64
Age 35	5.4	10.4	11.1
n	819	77	63
Pooled mean (per 100)	9.1[a]	11.5[a]	29.1[b]
Abnormal perception symptoms			
Age 30	8.1	11.3	29.7
n	840	80	64
Age 35	4.4	6.5	23.8
n	819	77	63
Pooled mean (per 100)	6.3[a]	8.9[a]	26.8[b]
	Exposure to childhood physical abuse (CPA)		
	Not exposed	**Some exposure**	**Severe exposure**
Abnormal thought symptoms			
Age 30	11.1	21.8	50.0
n	808	110	66
Age 35	4.6	17.5	6.6
n	796	103	60
Pooled mean (per 100)	7.9[a]	19.7[b]	29.4[b]
Abnormal perception symptoms			
Age 30	8.0	15.5	21.2
n	808	110	66
Age 35	4.8	12.6	8.3
n	796	103	60
Pooled mean (per 100)	6.4[a]	14.1[b]	15.1[b]

Note: differing superscripts indicate statistically significant ($p<.05$) difference (LR X^2 difference test).

Table 2. Spearman correlations between measures of childhood sexual abuse (CSA) and childhood physical abuse (CPA) and potential confounding factors.

	Childhood sexual abuse (CSA)	Childhood physical abuse (CPA)
Socio-demographic factors		
Family SES (at birth)	-.00	-.14***
Maternal age	-.11***	-.19***
Maternal education level	-.10**	-.11***
Average family living standards (ages 0-10)	-.12**	-.21***
Family functioning		
Number of changes of parents (to age 15)	.12***	.24***
Parental history of alcohol problems	.09**	.17***
Parental depression/anxiety	.02	.10**
Parental history of offending	.06	.15***
Parental illicit drug use	.11***	.04
Parental intimate partner violence	.19***	.26***
Maternal care	-.13***	-.19***
Maternal over-protection	.14***	.20***
Paternal care	-.14***	-.13***
Paternal over-protection	.14***	.17***
Individual factors		
Gender (female)	.25***	-.03
IQ (ages 8-9)	-.08*	-.11**
Parental attachment (age 15)	-17***	-.18***
Conduct problems (ages 7-9)	.07*	.24***
Attention problems (ages 7-9)	.06	.20***
Anxious/withdrawn behaviour (ages 7-9)	.07*	.08**
Major depression (age 15)	.16***	.12***
Anxiety disorder (age 15)	.20***	.12***
Suicidal ideation (age 15)	.25***	.12***
Neuroticism (age 14)	.19***	.06

Extraversion (age 14)	.08*	.05
Novelty-seeking (age 16)	.13***	.11***

* p<.05
** p<.01
*** p<.001

Table 3. Spearman correlations between psychotic experiences (PEs) (ages 30 and 35) and mental health, substance use, life stress and unemployment factors (ages 30 and 35).

	Abnormal thought symptoms	**Abnormal perception symptoms**
Age 30		
Major depression	.27***	.20***
Anxiety disorder	.22***	.17***
PTSD	.17***	.13***
Alcohol use disorder	.05	.08*
Nicotine dependence	.10***	.12***
Cannabis use disorder	.15***	.14***
Other illicit substance use disorder	.15***	.13***
Life stress	.13***	.13***
Unemployment	.15***	.10**
Age 35		
Major depression	.19***	.07*
Anxiety disorder	.23***	.13***
PTSD	.11**	.00
Alcohol use disorder	.09**	.08*
Nicotine dependence	.14***	.18***
Cannabis use disorder	.21***	.04
Other illicit substance use disorder	.17***	.20***
Life stress	.12***	.06
Unemployment	.09**	.05

* p<.05
** p<.01
*** p<.001

Table 4. Incidence rate ratios (and 95% confidence intervals) for the associations between childhood sexual and physical abuse exposure and psychotic experiences (PEs), after adjustment for: a) potential confounding factors; and b) time-dynamic covariate factors.

	Model 1 **Adjusted for confounding factors[1]**			**Model 2** **Adjusted for confounding and time-dynamic covariate factors[2]**		
	No exposure	**Some exposure**	**Severe exposure**	**No exposure**	**Some exposure**	**Severe exposure**
Exposure to CSA						
Abnormal thought symptoms (IRR; 95% CI)	1 --	0.97 (0.46 – 2.02)	4.41 (2.42 – 8.05)	1 --	1.40 (0.65 – 3.04)	2.25 (1.13 – 4.51)
Abnormal perception symptoms (IRR; 95% CI)	1 --	1.30 (0.63 – 2.68)	4.74 (2.67 – 8.39)	1 --	1.42 (0.67 – 2.99)	4.08 (2.28 – 7.30)
Exposure to CPA						
Abnormal thought symptoms (IRR; 95% CI)	1 --	1.55 (0.90 – 2.65)	1.27 (0.60 – 2.70)	1 --	1.07 (0.59 – 1.96)	0.67 (0.27 – 1.63)
Abnormal perception symptoms (IRR; 95% CI)	1 --	1.70 (0.95 – 3.02)	2.14 (1.08 – 4.22)	1 --	0.98 (0.53 – 1.81)	1.28 (0.62 – 2.63)

[1] Statistically significant ($p < .05$) confounding factors (for abnormal thought symptoms) included: maternal age; parental history of illicit drug use; maternal overprotection; paternal overprotection; parental attachment; gender; IQ; major depression (age 15); neuroticism (age 14). Statistically significant ($p < .05$) confounding factors (for abnormal perception symptoms) included: maternal age; maternal education; parental illicit drug use; gender; neuroticism (age 14); extraversion (age 14); novelty seeking (age 16).

[2] Statistically significant ($p < .05$) covariate factors (for abnormal thought symptoms) included: major depression; anxiety disorder; nicotine dependence; other illicit substance dependence. Statistically significant ($p < .05$) covariate factors (for abnormal perception symptoms) included: major depression; anxiety disorder; cannabis use disorder; life stress

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