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ORIGINAL ARTICLE

Childhood abuse and psychotic experiences – evidence for mediation by adulthood adverse life events

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Aims. We have previously reported an association between childhood abuse and psychotic experiences (PEs) in survey data from South East London. Childhood abuse is related to subsequent adulthood adversity, which could form one pathway to PEs. We aimed to investigate evidence of mediation of the association between childhood abuse and PEs by adverse life events.

Methods. Data were analysed from the South East London Community Health Study (SELCoH, $n = 1698$). Estimates of the total effects on PEs of any physical or sexual abuse while growing up were partitioned into direct (i.e. unmediated) and indirect (total and specific) effects, mediated *via* violent and non-violent life events.

Results. There was strong statistical evidence for direct (OR 1.58, 95% CI: 1.19–2.1) and indirect (OR 1.51, 95% CI: 1.32–1.72) effects of childhood abuse on PEs after adjustment for potential confounders, indicating partial mediation of this effect *via* violent and non-violent life events. An estimated 47% of the total effect of abuse on PEs was mediated *via* adulthood adverse life events, of which violent life events made up 33% and non-violent life events the remaining 14%.

Conclusions. The association between childhood abuse and PEs is partly mediated through the experience of adverse life events in adulthood. There is some evidence that a larger proportion of this effect was mediated through violent life events than non-violent life events.

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Introduction

Childhood adversity, in the form of sexual or physical trauma, is associated with the development, maintenance and recurrence of a range of psychiatric disorders later in life (Carr *et al.* 2013). In particular, there is a widely reported association between the experience of childhood abuse, such as physical and sexual trauma, and the occurrence of both psychotic disorder (Mäkikyrö *et al.* 1998; Schreier *et al.* 2009; Arseneault *et al.* 2011*a*; summarised and reviewed in Varese *et al.* 2012) and low-level psychotic experiences (PEs) (Alemany *et al.* 2011; Morgan *et al.* 2013; Van Nierop *et al.* 2014). The mechanism for this relationship remains unclear (Sheinbaum *et al.* 2012; Kelleher *et al.* 2013*b*; Van Nierop *et al.* 2014), but does not appear to be explained by confounding by genetic (Arseneault *et al.* 2011*b*; Alemany *et al.* 2013) or socioeconomic factors (Varese *et al.* 2012), or by recall bias (Bonoldi *et al.* 2013). Furthermore, there are likely to be multiple causal pathways to psychosis, involving an array of factors acting over the life course (Krieger, 1994; Morgan *et al.* 2010).

We have previously reported: (a) associations between childhood abuse, and life events and PEs (Morgan *et al.* 2014*a*); (b) that associations are strongest for those experiences involving severe threat/violence (Morgan *et al.* 2014*b*) and (c) that exposure to both childhood abuse and life events combines to increase risk the beyond effect of each alone (Morgan *et al.* 2014*b*). That is, we found evidence that one way in which early and later stress combines is by compounding risk, such that the influence of these two groups of factors together was greater than the sum of the separate effects. This may be because both operate on similar mechanisms, for example, the stress response (Collip *et al.* 2011).

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However, this may not be the only way in which early and later stress may be involved in the development of psychosis. It is well documented that early adversity increases likelihood of subsequent adversity (Kuijpers *et al.* 2012; Iverson *et al.* 2013), possibly through social processes whereby the negative effects of early adversity persist over time, restricting subsequent opportunities and increases likelihood of poverty and exposure to adverse events (Pantazis *et al.* 2006). It is therefore possible that part of the effect of childhood adversity on risk of psychosis is mediated *via* increased likelihood of exposure to adult stresses. In other words, childhood adversity and life events may *both* combine synergistically to increase risk (through effects on similar biological and psychological mechanisms) *and* be on a causal path, such that some of effect of childhood adversity on psychosis is mediated through increased likelihood (due to social processes) of subsequent adverse life events. This would imply a contributory environmental mechanism for the effect of childhood abuse on psychosis risk, and the possibility that intervening on adulthood negative experiences at a population level could reduce occurrence of psychosis.

As noted, we have previously found and reported evidence of synergistic (combined) effects (Morgan *et al.* 2014*b*). In this paper, we extend the analysis to consider whether there is evidence of mediation. Our aim was to assess the extent to which the association between childhood abuse and PEs, demonstrated previously (Morgan *et al.* 2014*b*) might be explained by the experience of both violent and non-violent adulthood life events, using mediation modelling. Using a large representative household survey conducted in South East London (UK) adult residents, we investigated the direct and indirect effects of childhood abuse on low-level PEs, hypothesising that the previously reported association between childhood abuse and psychosis would be mediated through adulthood violent life events. Lastly, we tested the specificity of any mediation *via* violent life events, by comparing it with mediation *via* non-violent life events. In this way, we aimed to assess to what extent these cross-sectional data were consistent with a pathway from childhood abuse to PEs *via* adulthood life events.

Methods

Participants

Data for this analysis were taken from SELCoH-1. The South East London Community Health study (SELCoH; full details of methods available elsewhere (Hatch *et al.* 2011)) is a representative household survey of South East London residents collected between 2008 and 2010. The analytic sample was composed of 1698 adult (16+) residents of Lambeth and Southwark, two London boroughs, residing in 1075 households selected through random sampling of the small user residential postcode address file. Sampled units were weighted in the analysis to account for non-response within households. Ethical approval was received from the King's College London Research Ethics Committee (CREC/07/08-152) and all participants provided informed consent and were interviewed by researchers.

Data collection

Exposure

Trained research workers asked study participants about any experiences of sexual abuse ('Did anyone who was responsible for your care ever sexually abuse you?'), or physical abuse ('did anyone ever hit you so hard that it left bruises or marks?'), before the age of 16 years. Based on these two binary items (reflecting any childhood physical abuse and any childhood sexual abuse), a binary variable reflecting 'any physical or sexual abuse during childhood' was derived.

Outcome

The Psychosis Screening Questionnaire (Bebbington & Nayani, 1995) was used to assess PEs. This is a five-item questionnaire that assesses different psychotic symptom domains experienced in the previous year. These comprise: hypomania, strange experiences, paranoia, hallucinations and thought disorder. Each domain contains an initial 'probe' item, which is followed by secondary questions. Because the present study was focused on non-affective psychosis, responses to the hypomania item were not examined. Individuals were considered have PEs if they endorsed one or more secondary items in the four remaining domains. This approach was consistent with a previous analysis of PEs originating from this data (Morgan *et al.* 2014*b*). The PSQ displays good correspondence with psychosis items on the Schedules for Clinical Assessment in Neuropsychiatry (Bebbington & Nayani, 1995), and has seen frequent use in population studies (Johns *et al.* 2002, 2004; Bebbington *et al.* 2004*a*).

Covariates

Age was grouped into 10-year categories. Employment was categorised into unemployed *v.* not unemployed (containing those who were employed, students, or retired). Ethnicity was operationalised as a five-category variable including White, Black Caribbean,

Black African, Asian and Other groups. Highest educational attainment was categorised into 'no qualifications', 'GCSE', 'A-Level', and 'degree level and above'.

Potential mediators

Adverse life events collected in SELCoH were, in the last year: separation, death of a loved one, serious accident/injury, homelessness, witnessing violence, exposure to a war zone in the last year, victim of a crime in the previous year, injury with a weapon, or being attacked. Scores were generated for each individual based on the number of different violent (witnessing violence, exposure to a war zone, victim of a crime, injury with a weapon, or being attacked) and non-violent life events (separation, death of a loved one, serious accident/injury, homelessness) experienced in the previous year.

Mediation analysis

Mediation analyses were carried out in MPlus version 7 (Muthén & Muthén, 1998) and took account of non-response weights and clustering of responses within households. To test our hypotheses, we examined whether the effects of childhood physical and sexual abuse on PEs were mediated through (a) violent life events in the previous year and (b) non-violent life events in the previous year using multiple mediation analysis. Total effects were apportioned into direct and indirect effects for violent and non-violent adverse life events. Logit coefficients and odds ratios for total and specific indirect effects of violent and non-violent adverse life events were estimated using maximum likelihood estimation in Mplus (Muthén & Muthén, 1998).

Results

Description

Among the total survey sample of 1698, 55 participants with missing data on PE ($n = 8$), childhood abuse (15), educational attainment (19), ethnicity (2), unemployment (9), violent events (20) and non-violent events (16) were excluded. The overall analytic sample therefore consisted of 1643 SELCoH participants interviewed between 2008 and 2010, of whom 306 reported PEs and 1337 did not. Basic socio-demographic characteristics and data on adulthood adverse life events by PE status are shown in Table 1. Associations were found between PEs and reporting childhood abuse, younger age, lower overall educational attainment, non-White ethnicity, being unemployed, and recent attack, crime victimisation, war exposure, witnessing of violence, experience of an accident, death of a partner and separation. There were strong linear trends for the odds of reporting PEs across scores for both non-violent and violent life events. There were also strong linear trends in odds of reporting either physical or sexual abuse during childhood, across score for both non-violent and violent life events (displayed in Table 2).

Mediation

To assess pathways from childhood abuse to psychosis *via* violent and non-violent life events, estimates of the total effects of any physical or sexual abuse while growing up were partitioned into direct (i.e. unmediated) and indirect (total and specific) effects using multiple mediation analyses. There was strong evidence for unadjusted direct (OR 1.7, 95% CI: 1.3–2.23) and total indirect effects (OR 1.54, 95% CI: 1.36–1.75) of abuse on PEs status, indicating partial mediation of this effect *via* violent and non-violent life events. This evidence for an association remained strong upon adjustment for age, gender, unemployment, ethnicity and educational attainment (direct effect: OR 1.58, 95% CI: 1.19–2.1; total indirect effect: OR 1.51, 95% CI: 1.32–1.72; see Table 2). In the fully adjusted model, an estimated 47% of the total effect of abuse on PEs was mediated *via* adulthood adverse life events, of which violent life events made up 33% and non-violent life events the remaining 14%.

In other words, the overall association between childhood abuse and PEs was explained partially by indirect effects *via* the experience both of adulthood violent and non-violent adverse life events. However, the proportion of the total indirect effect mediated through violent life events was around twice as large as that mediated through non-violent life events (see Table 3).

Discussion

Summary of main findings

Previous research from our group has found evidence for effect moderation, or synergy between adverse life events and childhood abuse on risk for PEs, such that the combined effect of abuse and life events was greater than the effect of each alone. Given that a variable can be both a moderator and mediator (Baron & Kenny, 1986), the present analysis assessed hypotheses involving mediation, to assess if adulthood violent life events could be on the causal pathway between childhood abuse and PEs, alongside their role as moderators.

We found evidence that the association between childhood abuse and PEs was partially explained by

Table 1. *Sociodemographic characteristics of sample analysed*

	No PEs (%)	PEs (%)	Total (%)	Degrees of freedom	X^2	*p* value
Age						
16–24	269 (76.20)	84 (23.80)	353 (100)			
25–34	329 (82.66)	69 (17.34)	398 (100)			
35–44	266 (81.35)	61 (18.65)	327 (100)			
45–54	197 (79.44)	51 (20.56)	248 (100)			
55–64	131 (84.52)	24 (15.48)	155 (100)			
65–	145 (89.51)	17 (10.49)	162 (100)	5	15.36	0.009
Gender						
Male	570 (79.50)	147 (20.50)	717 (100)			
Female	767 (82.83)	159 (17.17)	926 (100)	1	2.96	0.085
Education						
No qualifications	166 (77.21)	49 (22.79)	215 (100)			
GCSE/O Level	257 (80.06)	64 (19.94)	321 (100)			
A Levels	320 (76.37)	99 (23.63)	419 (100)			
Degree level or above	594 (86.34)	94 (13.62)	688 (100)	3	20.92	<0.001
Ethnicity						
White	864 (83.97)	165 (16.03)	1029 (100)			
Black Caribbean	95 (68.84)	43 (31.16)	138 (100)			
Black African	171 (78.08)	48 (21.92)	219 (100)			
Asian	53 (88.33)	7 (11.67)	60 (100)			
Other	154 (78.17)	43 (21.83)	197 (100)	4	23.68	<0.001
Unemployment						
No	1225 (82.88)	253 (17.12)	1478 (100)			
Yes	112 (67.88)	53 (32.12)	165 (100)	1	22.05	<0.001
Any childhood physical or sexual abuse						
No	1038 (85.50)	176 (14.50)	1214 (100)			
Yes	299 (69.70)	130 (30.30)	429 (100)	1	52.25	<0.001
Adulthood physical attack						
No	1302 (82.30)	280 (17.70)	1582 (100)			
Yes	35 (57.38)	26 (42.62)	61 (100)	1	24.07	<0.001
Adulthood injury with a weapon						
No	1327 (81.56)	300 (18.44)	1627 (100)			
Yes	10 (62.50)	6 (37.50)	16 (100)	1	3.8	0.051
Adulthood victim of crime						
No	1284 (81.89)	284 (18.11)	1568 (100)			
Yes	53 (70.67)	22 (7.19)	75 (100)	1	5.95	0.015
Adulthood war exposure						
No	1334 (81.49)	303 (18.51)	1637 (100)			
Yes	3 (50.00)	3 (50.00)	6 (100)	1	3.91	0.048
Adulthood witnessed violence						
No	1246 (83.12)	253 (16.88)	1499 (100)			
Yes	91 (63.19)	53 (36.81)	144 (100)	1	34.42	<0.001
No money in last year (adulthood)						
No	1328 (81.42)	303 (18.58)	1631 (100)			
Yes	9 (75.00)	3 (25.00)	12 (100)	1	0.32	0.569
Adulthood accident						
No	1320 (81.73)	295 (18.27)	1615 (100)			
Yes	17 (60.71)	11 (39.29)	28 (100)	1	8.02	0.005
Adulthood bereavement						
No	1226 (81.73)	265 (18.27)	1491 (100)			
Yes	111 (60.71)	41 (39.29)	152 (100)	1	7.7	0.006
Adulthood separation						
No	1291 (82.12)	281 (17.88)	1572 (100)			
Yes	46 (64.79)	25 (35.21)	71 (100)	1	13.47	<0.001
Grand total	1337 (100)	306 (100)	1643 (100)			

Table 2. *Categorical distributions of violent and non-violent adulthood life events by PE status and by childhood abuse status, for the total sample*

Total sample, $n = 1643$	Any abuse (%)	No abuse (%)	Total (%)	X^2 (d.f)	p (trend)	Odds ratio for any abuse and PE	No PEs (%)	PEs (%)	Total (%)	X^2 (d.f)	p (trend)
Number of different violent life events in the previous year											
0	1076 (76.10)	338 (23.90)	1414 (100)			2.21 (1.64–2.96)[a]	1182 (83.59)	232 (16.41)	1414 (100)		
1	114 (64.04)	64 (35.96)	178 (100)				128 (71.91)	50 (28.09)	178 (100)		
2	15 (44.12)	19 (55.88)	34 (100)				19 (55.88)	15 (44.12)	34 (100)		
3	8 (66.67)	4 (33.33)	12 (100)				6 (50.00)	6 (50.00)	12 (100)		
4	1 (20.00)	4 (80.0)	5 (100)	35.98 (4)	<0.001	2.76 (1.55–4.90)[b]	2 (40.00)	3 (60.00)	5 (100)	43.13 (4)	<0.001
Number of different non-violent life events in the previous year											
0	1060 (75.77)	339 (24.23)	1399 (100)			2.48 (1.84–3.35)[c]	1163 (83.13)	236 (16.87)	1399 (100)		
1	148 (65.49)	78 (34.51)	226 (100)				165 (73.01)	61 (26.99)	226 (100)		
2	5 (29.41)	12 (70.59)	17 (100)				9 (52.94)	8 (47.06)	17 (100)		
3	1 (100)	0 (0)	1 (100)	28.62 (3)	<0.001	2.14 (1.20–3.80)[d]	0 (0)	1 (100)	1 (100)	26.72 (3)	<0.001
Total	1214 (73.89)	26.11 (100)	1643 (100)				1337 (81.38)	306 (18.62)	1643 (100)		

Footnotes refer to the association between abuse and PEs among those with:
[a]No violent life event in the previous year.
[b]At least one type of violent life event in the previous year.
[c]No non-violent life event in the previous year.
[d]At least one type of non-violent life event in the previous year.

Table 3. *Total, direct, total indirect and specific indirect effects (odds ratios) for childhood abuse, violent life events in the previous year, and non-violent life events in the previous year, on the presence of PEs*

Any childhood physical or sexual abuse on PEs	Model I	95% CI	p[a]	Model II	95% CI	p[a]	Model III	95% CI	p[a]	Model IV	95% CI	p[a]
Total effect (TE)	2.64	2.02–3.45	<0.001	2.59	1.98–3.39	<0.001	2.61	1.98–3.45	<0.001	2.39	1.8–3.18	<0.001
Direct effect	1.7	1.3–2.23	<0.001	1.7	1.29–2.24	<0.001	1.7	1.29–2.24	<0.001	1.58	1.19–2.1	0.002
Total indirect effect	1.54	1.36–1.75	<0.001	1.52	1.3–1.78	<0.001	1.54	1.35–1.76	<0.001	1.51	1.32–1.72	<0.001
Indirect *via* violent events[b]	1.34	1.17–1.54	<0.001	1.32	1.15–1.52	<0.001	1.34	1.17–1.54	<0.001	1.34	1.17–1.54	<0.001
% of TE mediated *via* violent indirect effect[c]	30%	22–34%		29%	20–34%		30%	23–35%		33%	27–37%	
Indirect *via* non-violent[d] events	1.16	1.07–1.26	<0.001	1.15	1.06–1.25	0.001	1.15	1.06–1.25	0.001	1.13	1.04–1.23	0.004
% of TE mediated *via* non-violent indirect effect[c]	15%	10–19%		15%	9–18%		15%	9–18%		14%	7–18%	

[a]p values are from Wald tests.
[b]Violent life events comprised adulthood witnessed violence, being exposed to a war zone, being victim to a crime, injury with a weapon, or being attacked.
[c]Percentages based on logit coefficients.
[d]Non-violent life events comprised adulthood separation, death of a loved one, serious accident/injury, and homelessness. Model I was unadjusted for covariates. Model II was adjusted for age only. Model III was adjusted for age and gender, and Model IV was adjusted for age, gender, unemployment, educational attainment and ethnicity.

a pathway through the experience of adverse life events in the previous year. In particular, there was evidence for mediation *via* both violent and non-violent life events, with weak evidence suggesting that the indirect effect *via* adulthood violent life events was stronger than that *via* non-violent adulthood life events.

Previous literature

To our knowledge, no previous studies have assessed a mediating role for adverse life events in PEs. Morgan *et al.* (2008) found a linear relationship between markers of social disadvantage and the odds of psychosis in the AESOP study, suggesting that the interplay of environmental adversities could be important in the aetiology of psychosis. A further study, based on the same data, suggested that the effects of parental separation on psychosis risk were partly mediated through later educational and social disadvantage (Morgan *et al.* 2014*a*). Although less burdensome than psychotic disorders, low-level PEs are important because these experiences are associated with an elevated risk of both psychotic disorder (Kaymaz *et al.* 2012; Werbeloff *et al.* 2012; Fisher *et al.* 2013*a*) and other adverse mental health outcomes, including suicidality (Kelleher *et al.* 2012, 2013*a*) and the use of mental health services (DeVylder *et al.* 2014; Bhavsar *et al.* 2017).

Methodological limitations

Reports of PEs and life events were collected at the same time point, limiting inference about the temporal relationship between the variables. In some subjects, PEs could have preceded life events. Prospective studies are necessary to clarify the temporal association between adverse events over the life course and the occurrence of PEs (Fisher *et al.* 2011; Fisher *et al.* 2013*b*). In total 55 records (3%) were dropped because of missing data on one or more of the modelled variables. However, this compared favourably to other studies of this type (Bebbington *et al.* 2004*b*; McManus *et al.* 2009; Hatch *et al.* 2012). Furthermore, the crude association between abuse and PEs, and between life events and PEs, was similar in those with missing data, suggesting that important selection bias due to missing data was unlikely. The dichotomous measure of childhood abuse was based on two binary items (physical, and sexual abuse in childhood), rather than a more extensive questionnaire, limiting our ability to evaluate the severity, extent, or personal significance of childhood trauma exposure in detail. Given that the outcome for this study was PEs occurring in a general population sample, any generalisation of our findings should be limited to general, non-clinical populations, rather than to patients. We have recently demonstrated, however, that those reporting PEs in this study do experience greater use of mental healthcare over time (Bhavsar *et al.* 2017), implying that PEs could be a significant marker for clinically important morbidity at a general population level.

There is a possibility that people with PEs could have over-reported exposure to childhood abuse, or to adverse life events. However, in relation to childhood abuse, misclassification has been found to be limited in previous studies (Bonoldi *et al.* 2013). We included only life events in the previous year, which limited the influence of recall bias. Nevertheless, some evidence indicates that higher levels of neuroticism, with which PEs are correlated, increase misreporting of childhood traumatic experiences in health research (Reininghaus *et al.* 2013; Reuben *et al.* 2016). Data on number of specific life events, or severity of life events, would have been informative for this analysis, but this data was not available on this sample. Although we adjusted for a large range of possible confounders, unmeasured or unknown confounding, and residual confounding for inaccurately measured confounding variables, remained possible sources of bias.

The relationship between trauma, psychosis and other trauma-related psychopathology is likely to be complex. For example, Morrison *et al.* (2003) a variety of potential pathways linking post-traumatic stress disorder (PTSD) and psychosis to trauma, including the possibility that the type of trauma-related psychopathology is determined by mood, physiology and attributional style, driving either the presentation of psychotic or PTSD, which are postulated to manifest a common post-traumatic response. On the other hand, Mueser *et al.* (2002) suggest that PTSD symptoms might themselves influence the symptomatic severity of psychosis, and its response to treatment. Our study did not aim to examine the role of PTSD symptoms as a modifier or mediator of the effect of adulthood trauma on PEs. Therefore, although unlikely to have introduced bias into the results reported here, it remains possible that PTSD acts as an intermediate factor in the relationship between adulthood violence exposure and the development of PEs, a proposition that requires further study. More generally, it is possible that trauma over the life course influences risk for PEs *via* changes in sensitivity to stress, which has been conceptualised as an 'affective pathway' to psychosis (Myin-Germeys & Van Os, 2007). Although the outcome analysed in the present report was sub-clinical symptoms of psychosis, our results are consistent with the explanation that childhood traumatic events influence stress-reactivity and affect the expression of psychotic symptoms later in life, in the context of stressful events, such as further exposure to violence.

PEs and childhood abuse

Although there is now relatively strong evidence that childhood abuse is related to the occurrence of psychosis and PEs, exact mechanisms remain unclear. However, causal pathways to psychosis are likely to be complex and probably involve the interplay of genetic risk with an array of social environmental factors, including experiences of abuse and trauma, over the life course (Morgan *et al.* 2010). In keeping with this complexity, childhood abuse could plausibly exert its influence on PEs in a variety of ways; for example, through effects on the stress sensitivity of the mesolimbic dopamine system (Howes & Kapur, 2009), regulation of the hypothalamic-pituitary-adrenal axis (Borges *et al.* 2013), the developmental of maladaptive cognitive schema (Garety *et al.* 2001, 2007), and changes to affective and cognitive processing (Fisher *et al.* 2011, 2013*b*; Rottenberg *et al.* 2014).

Childhood abuse and adulthood adverse life events

Some pathways from childhood abuse to PEs could involve the experience of adversity in adulthood. Although a number of separate socio-environmental risk factors (e.g. deprivation, migration, urban residence and ethnicity) have been linked to PEs (Das-Munshi *et al.* 2012; Linscott & Van Os, 2013; Schofield *et al.* 2016), including exposure to trauma (Morgan *et al.* 2014*a*), it is also widely accepted that markers of social adversity tend to cluster in individuals over time (Pantazis *et al.* 2006). For example, physical or sexual abuse during childhood is associated with sexual (Russell, 1983; Siegel *et al.* 1987; Mayall & Gold, 1995) and physical (Briere & Runtz, 1987; Chu & Dill, 1990) violence exposure later on in life. There are various possible explanations for this relationship- for example, childhood victimisation may be accompanied by residential instability and institutionalisation, which in turn may result in greater opportunities for other adversities, including violence exposure, in adulthood (Finkelhor, 1979). A likely explanation for re-victimisation is the effect of the original victimisation on psychological vulnerabilities. People exposed to violence as children may feel powerless to defend themselves from such exposures as adults, leading to increased vulnerability (Russell, 1983).

Conclusions

Our findings suggest tentatively that a putative socio-developmental pathway between childhood abuse and PEs might involve the experience of adversity in adulthood. This is potentially important for public health, because intervening on adulthood adversity in those exposed to childhood trauma might be a more efficient and practicable strategy for the prevention of psychoses than intervening on childhood trauma *per se*. The distinction between violent and non-violent adulthood life events in possibly mediating the association between abuse and PEs could, if replicated, be important in the targeting, design and implementation of early intervention services. Further research on the inter-relationships of modifiable social environmental risk factors involved in the occurrence of psychoses could identify ways of intervening to reduce risk. Our results emphasise the potential clinical relevance of repeated traumatic experiences, including those occurring during adulthood, in influencing mental health and underline the importance of understanding the mechanisms of recurrent victimisation in future research on mental disorders.

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Conflict of Interest

The authors have no conflicts of interest to declare.

Availability of Data and Materials

The data that support the findings of this study are available from Professor Matthew Hotopf but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are however available from the authors upon reasonable request and with permission of Professor Matthew Hotopf.

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