

Editorials

Childhood sexual abuse and psychosis: aetiology and mechanism

PAUL BEBBINGTON

Abstract. The aim of this Editorial is to describe the association between child sexual abuse and psychosis, and to consider potential mechanisms for the association. The association is strongly supported by the literature. Evidence from a variety of sources is triangulated in order to create a plausible model of the link, which can then be used to direct future research. The mechanisms are certainly complex, and there is evidence of interacting contributions at genetic, neurophysiological, behavioural, cognitive and emotional levels. Child sexual abuse is an important antecedent in psychosis, both theoretically and clinically. At the theoretical level, it potentially illuminates mechanisms by which psychotic symptoms are generated, at the clinical level it opens possibilities for improving cognitive-behavioural approaches to treatment.

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INTRODUCTION

The place of stress in the emergence of psychotic symptoms has been the subject of clinical contention in psychiatry for over a century (Bebbington *et al.*, 1993). Empirical studies of recent stressful events date back to the 1960s, the events being seen as triggers, the last element in the aetiological chain (Brown & Birley, 1968). It has never been clear over what period events exert their effects, whether the few weeks before onset or a more prolonged period of months (Day *et al.*, 1987; Ventura *et al.*, 1989; Hultman *et al.*, 1997; Dohrenwend *et al.*, 1987; Bebbington *et al.*, 1993; Hirsch *et al.*, 1996). Indeed some studies found no association between life events and onset (Jacobs & Myers, 1976; Malzacher *et al.*, 1981; Al Khani *et al.*, 1986; Chung *et al.*, 1986; Gureje & Adewumni, 1988; Malla *et al.*, 1990; Hirsch *et al.*, 1996). Nevertheless the research does suggest overall that in some cases stressful social circumstances are closely associated with the emergence of schizophrenia.

Over the last decade or so, the focus has moved away from the effect of recent stress to the study of much earlier social adversity in people who develop schizophrenia. This has been tied conceptually to the stress-vulnerability paradigm. As originally framed, this carried the implicit view that vulnerability in schizophrenia is genetic, or perhaps related to the early physical environment. There was considerable reluctance to attribute to social factors effects of a greater persistence than those implied by mere triggering. However, genetic and biological indicators of vulnerability are far from universal in individuals with schizophrenia (Tosato & Lasalvia, 2009), and there is certainly no logical reason why vulnerability should not in some cases be psychosocial, arising from adverse experiences early in life (Read *et al.*, 2001).

Childhood adversity takes many forms (Morgan & Fearon, 2007). It may include actual abuse, whether physical, sexual or emotional. Such abuse seems to be common. Thus, a recent UK survey found a prevalence of 24% for childhood physical abuse and of 11% for sexual abuse (May-Chahal & Cawson, 2005); even higher estimates have been reported from the USA (Friedman *et al.*, 2002). The estimates depend on the definition, and on how many types of events are used as prompts. Increasing attention is also being paid to the serious effects of bullying, whether inside or outside school. Approximately 17% of children from a nationally repre-

Address for correspondence: Professor P. Bebbington, UCL Department of Mental Health Sciences, 2nd Floor, Charles Bell House, 67-73 Riding House Street, London W1W 7EJ (United Kingdom).

Fax: 020-7679.9426

E-mail: P.Bebbington@ucl.ac.uk

sentative birth cohort in the UK reported being bullied by the age of seven years (Arseneault *et al.*, 2006).

While the deleterious effects of childhood trauma are now well established, they seem relatively non-specific. Thus there is an enhanced risk of adult depression, personality disorder, PTSD, drug and alcohol abuse, and suicidality (Bebbington *et al.*, 2004; 2009). Nevertheless, there is now increasing evidence of strong, perhaps particularly strong, links with psychosis. Set against this there have been concerns about the reliability of reports of abuse in people whose mental state is affected by schizophrenia. However, Goodman *et al.* (1999) showed that their accounts are consistent over time, and concluded that the information obtained is sufficiently reliable to allow research in this area.

Childhood abuse in people with psychosis

Ellason & Ross (1997) found a significant association of child physical and sexual abuse with psychotic symptoms, confirmed by Mueser *et al.* (1998). Lysaker *et al.* (2001) found 40% of a sample of people with schizophrenia acknowledged child sexual abuse. In the British National Psychiatric Morbidity Survey, the relative odds of psychosis were around 12 in people who had experienced sexual abuse (Bebbington *et al.*, 2004).

Janssen *et al.* (2004) showed that childhood abuse predicted the development *de novo* of positive psychotic symptoms in members of the general population over a two-year period. Ukok & Bikmaz (2007) found child sexual abuse is especially associated with the *positive* symptoms of psychosis in patients with first episodes of schizophrenia. If true, this might indicate some specificity of mechanism for positive symptoms.

Reviewing the literature linking childhood trauma to psychosis, Read *et al.* (2005) felt able to conclude that the link was now well established. Morgan & Fisher (2007) have been more cautious, acknowledging that recent population-based studies provide robust evidence of an association, but pointing out several conceptual and methodological issues.

In addition to its effect on the *emergence* of psychotic symptoms, an adverse early environment may contribute to their *maintenance*. Thus it may lead to chronicity and poor outcome in people who do develop psychosis. Fowler (1999) found that histories of severe early trauma were more common in people with chronic psychosis than in those in their first episode (who include a proportion heading for a good outcome). In a German study, traumatic experiences and adverse circumstances in

childhood were related to outcome in the form of relapse and rehospitalization (Doering *et al.*, 1998). Sub-clinical symptoms of psychosis may also be abnormally persistent in people with early trauma (Cougnard *et al.*, 2007).

PTSD and psychotic symptoms

Evidence is also accumulating of a close relationship between post-traumatic stress disorder (PTSD) and psychotic symptoms. This is worth examining in some detail, as it has obvious implications for persistent effects of stress in the aetiology of schizophrenia.

One place to search for such links is in military personnel. Psychotic symptoms in response to combat trauma were reported by Paster as long ago as 1948, and are not uncommon. Forty per cent of one sample of Vietnam veterans had experienced psychotic symptoms in the 6 months before assessment (David *et al.*, 1999). Calhoun *et al.* (2007) provide evidence that, although commonly under-diagnosed, PTSD is highly prevalent in war veterans with schizophrenia. Hamner *et al.* (2000) compared veterans displaying long-standing PTSD and psychotic features with a sample of patients with schizophrenia without PTSD. They found very few differences in the form and intensity of the psychotic features, either positive or negative, suggesting that schizophrenia associated with PTSD is indistinguishable clinically from the condition in general.

Rates of PTSD in people with psychosis are also relevant here. Mueser *et al.* (1998) studied 275 patients with a range of severe mental disorders, largely psychosis. Forty-three per cent had PTSD as a result of a severe trauma at some stage of their lives, often in childhood. However, only in 2% was the diagnosis recorded in their notes. Ninety-eight per cent of the sample had been exposed to at least one severe trauma. Seedat *et al.* (2003) systematically reviewed the association between PTSD and psychosis. Rates of serious trauma were generally very high in people with psychosis, while rates of actual PTSD were lower, although substantial, ranging from 13% to over 50%. The figures vary with the method of assessment (Priebe *et al.*, 1998; Neria *et al.*, 2002; Resnick *et al.*, 2003; Kilcommons & Morrison, 2005; Shevlin *et al.*, 2007).

The severity of trauma is associated with the severity both of PTSD and of psychotic symptoms, and also with current levels of distress and poor psychosocial functioning (Resnick *et al.*, 2003; Lysaker *et al.*, 2001; Kilcommons & Morrison, 2005). Thus early trauma appears to create a plethora of interacting adverse conse-

quences, leaving the mechanism linking traumatic and psychotic symptoms open to interpretation.

Mueser *et al.* (2002) argue that PTSD may mediate the negative effects of trauma on the course of severe mental illness. This may operate both directly, through the capacity of PTSD to affect symptom generation in psychosis, and indirectly, through re-traumatization, substance abuse and social difficulties. Both channels are probably important. Traumatic early life experiences predispose individuals both to psychosis and to substance abuse. The substance abuse often makes outcome worse: childhood trauma and PTSD both seem especially frequent in dual diagnosis patients (Scheller-Gilkey *et al.*, 2004). Gearon *et al.* (2003) found particularly high rates of traumatic life events in women with psychosis and comorbid substance abuse: revictimization was common, and rates of concurrent PTSD were especially high.

PTSD is a condition defined essentially by an intimate relationship between the experience of strongly traumatic events and the content of symptoms, in particular the phenomenon of re-experiencing. One possibility is that some people exposed to extreme trauma develop psychotic symptoms (delusions, hallucinations) whose content is also closely related to the details of the traumatic experience. If this happens, it may represent a totally different process from that responsible for symptoms in the majority of cases of schizophrenia, or it may not.

Some might argue that florid symptoms in PTSD merely mimic psychotic symptoms. Thus, re-experiencing may have a compelling visual or auditory quality capable of being mistaken for hallucinations. However, even in veterans exposed to extreme combat stress, the distinction between flashbacks and psychotic symptoms can be clearly made (Ivezic *et al.*, 1999).

Trauma and hallucinations

Hallucinations seem to be particularly frequent in psychoses associated with trauma, thus, in all but one case of Vietnam veterans with psychosis studied by David *et al.* (1999), their symptoms included auditory hallucinations. Kilcommons & Morrison (2005) found that sexual abuse was related specifically to hallucinations. This may be particularly true of *child* sexual abuse, while adult sexual assault may be related more generally to the full range of schizophrenic psychopathology (Read *et al.*, 2003). This trauma/hallucination link is not restricted to schizophrenia: trauma appears just as strongly associated with auditory hallucinations in bipolar disorder (Hammersley *et al.*, 2003). Again the link seems particularly strong for child sexual abuse.

However, Hardy *et al.* (2005) found that, in many cases where trauma preceded hallucinations, the hallucinations were only *thematically* related to the trauma, and did not often involve actual recapitulation of the traumatic event. Thus women with a history of rape often hear voices calling them obscene and denigrating names. In other studies as well, meaningful connections between the characteristics of trauma and the content of symptoms are not always apparent. Butler *et al.* (1996) felt that, in their series, the psychotic symptoms associated with PTSD were not themselves linked to re-experiencing the trauma. Likewise, in a military study, the severity of psychotic symptoms associated with combat-related PTSD was correlated with the severity of PTSD symptoms, but there was no apparent link between psychotic symptoms and re-experiencing the traumatic event per se (Hamner *et al.*, 1999). From this, it appears almost as though psychotic symptoms are an alternative way of re-experiencing.

Trauma, dissociation and psychosis

Another aspect of the trauma/PTSD/psychosis triad concerns the salience of *dissociative processes*. Glaslova *et al.* (2004) have suggested that traumatic stress exerts its influence on schizophrenia precisely by increasing the tendency to dissociation. Holowka *et al.* (2003) certainly found that childhood trauma was associated with significant dissociative symptoms in people with schizophrenia. Kilcommons & Morrison (2005) likewise found that dissociative processes consequent upon trauma were linked to psychotic experiences, and with hallucinations in particular. Irwin (2001) suggested that dissociative experiences and PTSD were connected because both were associated with childhood trauma, although controlling for childhood trauma did not remove the association. However, there are difficulties in defining and identifying dissociative processes, and the association with schizophrenia is not universally accepted (Brunner *et al.*, 2004).

Mechanisms of the stress-psychosis link

In the origination of psychosis, stress almost certainly operates at more than one level. It appears to trigger the emergence of psychotic symptoms, and people with psychosis may in any case have a particular vulnerability to stress. However, this vulnerability in turn may be the consequence of earlier stresses. There has been considerable speculation (and some research) about the mechanisms by which triggering, vulnerability and early adver-

sity exert their effects. This triangulates the stress/psychosis relationship in a way that increases its plausibility.

There are several levels at which vulnerability to stress can be characterized in psychosis. Genetic, neurophysiological, affective, cognitive and behavioral abnormalities may be demonstrable, and may interact. Current models of vulnerability are speculative but nonetheless valuable if they direct and drive future research.

The emergence of psychotic symptoms is a relatively uncommon response to stress: the more usual response involves affective symptoms. Thus, explaining the triggering of psychosis without adducing a pre-existing personal vulnerability would rely on the postulation of normal individuals facing an abnormal concatenation of circumstances, perhaps invoking affective changes and specific appraisals. The current cognitive models of psychosis emphasize both the person's appraisal of the impact of stress and the ensuing cognitions and emotions, and how they might interact with perceptual abnormalities (Fowler, 2000; Garety *et al.*, 2001; 2007; Bentall *et al.*, 2001; Morrison, 2001; Birchwood, 2003; Broome *et al.*, 2005).

There is appreciable evidence that individuals with a prior history of psychotic symptoms do respond to stressful circumstances with normal anxiety and depression. Such affective changes are also seen in the prodromal period before the emergence or re-emergence of psychotic symptoms. Ventura *et al.* (2000) showed that the recurrence of psychotic symptoms is not the only response to stressful life events in people who have experienced prior episodes of schizophrenia. In many instances, the exacerbation does not proceed beyond non-psychotic depressive symptoms. This links into the literature on affective prodromes of relapse (Birchwood *et al.*, 1992), not all of which lead to the re-emergence of psychotic symptoms (Yung & McGorry, 1996). Myin-Germeys *et al.* (2005) were able to show that the clear affective response to small stressors was paralleled by moment-to-moment variation in subtle positive psychotic experiences.

Mood and cognitive attributes seem to have independent associations with psychotic and quasi-psychotic symptoms in clinical and non-clinical populations (Garety *et al.*, 2005; Fowler *et al.*, 2006; Smith *et al.*, 2006; Bentall *et al.*, 2009). In these studies, a combination of anxiety and schematic views of the self as weak, vulnerable, and inadequate, and of other people as devious, threatening, and bad, was found to be specifically associated with paranoia. Such cognitions have been adduced to explain the excess of psychosis in some immigrant groups (Cooper *et al.*, 2008).

What of the biological correlates of stress in schizophrenia? At one level these correspond to the stress

responses of normal people, although, as we shall see, there is always a tendency to invoke a modifying vulnerability in people with schizophrenia. In patients newly admitted with an acute episode of psychosis, the severity of recent life stressors was proportionate to the increase in serum cortisol (Mazure *et al.*, 1997). People with first episode psychosis may display increased HPA axis activation (Ryan *et al.*, 2004). There is even evidence that the onset of psychosis may be associated with actual pituitary enlargement (Pariante *et al.*, 2005). Jones & Fernyhough (2007) incorporated the modulation of cortisol production by stress into their neural diathesis-stress model of schizophrenia. They suggested, further, that cortisol is most strongly produced in response to situations that imply uncontrollable threats to social standing or to important goals.

While cortisol metabolism is central to the normal stress response, the predominant explanation of anomalous experiences in schizophrenia has been couched in terms of the dopamine theory. There is converging evidence from animal research and clinical studies supporting a role for dopamine dysregulation in the prefrontal cortex in schizophrenia (e.g. Goldman-Rakic *et al.*, 2004; Kapur *et al.*, 2005). However, dopamine release is also a recognized response to stress in non-psychotic individuals (Adler *et al.*, 2000; Pruessner *et al.*, 2004). For this reason it remains possible that schizophrenic symptoms might arise in the absence of vulnerability, for instance if the degree of stress were overwhelming, as in psychoses following war trauma.

Vulnerability may operate at the behavioural level, involving, for instance, impaired coping mechanisms and ineffective ways of accessing support (Hultman *et al.*, 1997; MacDonald *et al.*, 1998). Pallanti *et al.* (1997) provide evidence of sensitivity to events in terms of coping deficits. Patients whose relapse did not follow a life event in the month before relapse showed less effective coping and poorer information processing capacity. The implication is that patients who have good coping resources (including cognitive capacity) will only be unsettled by events of considerable threat, in contrast to those without.

Horan *et al.* (2007) compared responses to the 1994 Californian earthquake in people with schizophrenia, those with bipolar disorder and healthy controls (Horan *et al.*, 2007). Both patient groups reported a high level of avoidant symptoms on the Impact of Events Scale. Moreover, avoidant coping was associated with higher residual stress symptoms in the schizophrenia group at follow-up. Horan & Blanchard (2003) showed that patients with schizophrenia responded with more negative mood if they had maladaptive coping styles and trait negative affectivity.

Horan *et al.* (2005) reported that, compared with controls, patients with schizophrenia appraised events as less controllable, and thought they had handled them less well. They also down-rated positive events. The psychological mediation of vulnerability to stress may also involve hopelessness. This is very common in people who have newly developed schizophrenia, and appears to be linked to poor outcome (Aguilar *et al.*, 1997).

The ability to access social support may represent a behavioral component of vulnerability. There is an extensive literature in the study of depression directed at the stress-buffering function of social support (Alloway & Bebbington, 1987). Hultman *et al.* (1997) have investigated schizophrenia in similar terms: they found that the time between events and relapse was increased in people with better social support and a coping strategy characterized by active support seeking. Penn and his colleagues (2004) have also suggested a role for social support in maintaining recovery.

Vulnerability to stress may vary over time, and there have been suggestions that the experience of an episode of psychosis may itself alter sensitivity, a sort of scarring process. In bipolar disorder, it has been suggested that life events may only be in excess in early episodes, and that later episodes appear less associated with events because of the process of kindling (Ramana & Bebbington, 1995). There has been little examination of this possibility in schizophrenia: in a small study ($n = 32$) of American veterans, life events were more likely to be associated with earlier episodes of schizophrenia (Castine *et al.*, 1998), although others have not found this (Bebbington *et al.*, 1996).

Life events themselves may modulate the increased sensitivity of patients with psychosis to day-to-day stress (Read *et al.*, 2001; Myin-Germeys *et al.*, 2003). Moreover, the risk may accumulate over time, as more adverse experiences occur (Myin-Germeys & van Os, 2007). Myin-Germeys *et al.* (2003) found that the increased sensitivity of patients with psychosis to day-to-day stress assessed using the Experience Sampling Method (ESM) was markedly modulated by the background life event rate. They took this as evidence for a separate, affective, route to symptom formation in psychosis, characterized by a more episodic reactive type of psychosis with a relatively good outcome.

The mechanisms of vulnerability could in part be gene-environmental. Thus Kinderman & Cooke (2000) interpreted the Finnish study of Tienari *et al.* (1994) as indicating that the family environment was playing a crucial part in moderating genetic risk. Read *et al.* (2001), however, argued for a direct biological effect of adverse

life events in early life on the diathesis underlying psychosis, thus positing a traumagenic neurodevelopmental model of schizophrenia. There is some suggestion that patients with a history of childhood trauma have reduced hippocampal volume (Driessen *et al.*, 2000); this is also seen in people experiencing a first episode of psychosis (Shenton *et al.*, 2001; Lappin *et al.*, 2006). Childhood adversity may also induce lasting effects on the main hormonal stress response system, the hypothalamico-pituitary-adrenal (HPA) axis (Read *et al.*, 2005; Spauwen *et al.*, 2006). Women physically or sexually abused in childhood show HPA dysregulation (Heim *et al.*, 2000).

Trauma may exert its effect on vulnerability through the induction of persistent affective changes. Mood may have persistent as well as temporary effects on the propensity to develop schizophrenic symptoms. Dinzeo *et al.* (2004) suggest that trait arousability is increased in people with schizophrenia, and that this may be related to stress responsiveness and symptom presentation.

The role of affect in the production of psychotic symptoms in war veterans is clear, and a majority of such cases meet criteria for major depressive disorder (David *et al.*, 1999; Hamner *et al.*, 1999).

Anxiety is also likely to be an important link. It is of interest that people with PTSD and those with schizophrenia both display abnormal startle responses (Howard & Ford, 1992). Priebe *et al.* (1998) found that, in community patients with schizophrenia, PTSD was related to levels of neurotic symptoms. Crittendon & Ainsworth (1989) demonstrated that children who have been abused or bullied are hypervigilant to hostile cues in their environment. This may lead such children to make hostile attributions about the intentions of others, and they may, not unreasonably, have more general negative beliefs about the behaviour of other people. Lysaker & Salyers (2007) used cluster analysis to examine links between trauma history and symptoms in schizophrenia. They found that self-report of sexual trauma predicted high levels of anxiety, and severe anxiety was particularly associated with severe hallucinations.

However, the effect of a stressful early environment may equally be mediated through enduring cognitive predispositions, that is to say, a mechanism at the psychological level. Bak *et al.* (2005) examined the link between childhood trauma and incident psychosis. Trauma was associated with a greater degree of distress in response to symptoms and poorer coping responses (Bak *et al.*, 2005). The authors conclude that the early experience of trauma may create lasting cognitive and affective vulnerabilities for the development of clinical symptoms. Gracie *et al.* (2007) found support for two separate routes

linking trauma and the predisposition to psychosis. One involved mediation by negative beliefs about the self and others. However, there also appeared to be a second, direct, association between re-experiencing symptoms and hallucinations.

We now have a considerable amount of suggestive evidence about the nature of vulnerability to psychosis. The picture is complex, with interacting contributions at genetic, neurophysiological, behavioural, cognitive and emotional levels. The mechanisms of vulnerability may overlap extensively the mechanisms involved in the actual generation of psychotic symptoms, and vulnerability may sometimes be a repetitive rather than an enduring state. The idea of a repetitive state lends itself to a dynamic rather than a static interpretation, and is reflected in the idea of a continuum propensity to psychosis. While we have hints of how sexual abuse may be tied into the various mechanisms of vulnerability, this seems a topic ripe for research.

Vulnerability and the continuum theory of psychosis

Schizophrenia (and psychosis in general) is increasingly seen as the extreme end of abnormalities distributed continuously in the general population (Johns *et al.*, 2004; Freeman *et al.*, 2005; Wiles *et al.*, 2006). Paranoid ideation of a mild degree and schizotypy are regarded as minor variants of the full form of the disorder. If so, sexual abuse might also be expected to be linked with sub-clinical symptoms. There is evidence that the determinants of the non-clinical disorders are similar to those of the full form (Johns *et al.*, 2004). For example, Berenbaum *et al.* (2003) found that women with a history of trauma had elevated levels of schizotypal symptoms. The relationship seemed particularly strong for reported childhood neglect. In a large population sample, sub-psychotic delusional ideation was more frequent in people who had experienced trauma (Scott *et al.*, 2007). A diagnosis of PTSD further increased the endorsement of delusional ideation. In adolescent populations childhood bullying and sexual trauma have been associated with sub-clinical quasi-psychotic experiences, and these might indicate risk of later conversion to frank psychotic disorder (Lataster *et al.*, 2006; Schreier *et al.*, 2009).

Thus, people can be conceived as moving along continuous distributions at different times, moving far enough on occasion to develop the full syndrome. This makes it appropriate to regard schizotypy itself as a vulnerability state, and therefore likely to yield insights into the causation of schizophrenia.

Marzillier & Steel (2007) reported that people seeking help to deal with a traumatic event who scored high on schizotypy had more frequent trauma-related intrusions and worse symptoms of PTSD in general. Certain information processing styles associated with schizotypy may account for vulnerability to trauma-related intrusions (Marzillier & Steel, 2007).

Steel *et al.* (2005) emphasise the possible role of *contextual integration* on the development of trauma-related intrusions in psychosis. Schizotypal personality traits are associated with the degree of contextual integration, and also with trauma-related intrusions. There seemed to be a dose-response relationship between the numbers of traumatic events and the likelihood of delusional experiences. Holmes & Steel (2004) used a trauma video to elicit trauma intrusions in a normal sample. People who scored high on schizotypy reported more intrusive experiences as a result of this. The authors link this propensity with the influence of trauma on psychotic disorders.

Trauma, meaning and psychotic symptoms

Some authors have reported series of cases where the psychotic phenomena are obviously related in a meaningful way to the trauma. Thus, Ivezic *et al.* (1999) emphasize the strong symbolic relationship, the meaningful connection with the experience. Likewise, David *et al.* (1999) reported that in Vietnam veterans, the psychotic symptoms were related to aspects of combat and to guilt, averring that the relationship was 'non-bizarre'. However, the level of psychotic symptoms was not related to the severity of PTSD.

A meaningful connection between an external event or set of events and a psychiatric symptom might be said to exist if the characteristics of the event and the content of the symptom are thematically consistent. This can be conceived as the *demand characteristics* of the event. Early examples of attempt to establish meaningful links in this way include those between loss events and depression, and between danger events and anxiety (Brown & Harris, 1978; Finlay-Jones & Brown, 1981). In psychosis, links may exist between "intrusive" events and psychosis, particularly paranoid ideation (Harris, 1987; Raune *et al.*, 2006; 2009). Such links can plausibly be made between the attributes of events and the context of delusions, whose very nature involves a consideration of the world. It is thus relatively easy to conceive that delusions may actually arise from a distorted process of appraisal. However, as we have seen, there is also work suggesting that there may be meaningful connections

between the nature of events and the content of hallucinations. Could this link also explain the *fact* of hallucination? In other words, could the thrust of the event distort the appraisal to the extent that the *form* of thinking becomes disturbed and externally projected? The current evidence on the effects of sexual abuse makes this idea less implausible.

CONCLUSION

There is now considerable evidence of an association between child sexual abuse and psychosis. This relationship is at least as strong as, and may be stronger than, that with other mental disorders. There may be a specific relationship with hallucinations. A history of severe trauma is common in people who develop psychosis, but the link between child sexual abuse and psychosis may be particularly strong, because of the age of the victim and the especially intrusive nature of the abuse. The mechanism of effect may overlap considerably with the triggering action of life events occurring in close proximity to the onset of psychosis. However, we also need an account of the maintenance of effect of child sexual abuse that can explain the establishment of a vulnerability that persists, sometimes for many years, before the actual onset of disorder. This may result from the impact of a series of intervening events and circumstances, and may be facilitated by processes like impaired coping responses, impaired access to appropriate social support, and revictimisation. Part of the mechanism of vulnerability seems likely to result from disturbances of affect and of cognitive schemas. Appreciation of the frequency and importance of child sexual abuse in individuals with psychosis could amplify their cognitive behavioural treatment.

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