

Review article

Childhood trauma, psychosis and schizophrenia: a literature review with theoretical and clinical implications

Read J, van Os J, Morrison AP, Ross CA. Childhood trauma, psychosis and schizophrenia: a literature review with theoretical and clinical implications.

Acta Psychiatr Scand 2005; 112: 330–350. © 2005 Blackwell Munksgaard.

Objective: To review the research addressing the relationship of childhood trauma to psychosis and schizophrenia, and to discuss the theoretical and clinical implications.

Method: Relevant studies and previous review papers were identified via computer literature searches.

Results: Symptoms considered indicative of psychosis and schizophrenia, particularly hallucinations, are at least as strongly related to childhood abuse and neglect as many other mental health problems. Recent large-scale general population studies indicate the relationship is a causal one, with a dose-effect.

Conclusion: Several psychological and biological mechanisms by which childhood trauma increases risk for psychosis merit attention. Integration of these different levels of analysis may stimulate a more genuinely integrated bio-psycho-social model of psychosis than currently prevails. Clinical implications include the need for staff training in asking about abuse and the need to offer appropriate psychosocial treatments to patients who have been abused or neglected as children. Prevention issues are also identified.

**J. Read¹, J. van Os^{2,3},
A. P. Morrison^{4,5}, C. A. Ross⁶**

¹Department of Psychology, The University of Auckland, Auckland, New Zealand, ²Department of Psychiatry and Neuropsychology, Maastricht University, Maastricht, The Netherlands, ³Division of Psychological Medicine, Institute of Psychiatry, London, ⁴Department of Psychology, University of Manchester, Manchester, ⁵Psychology Services, Bolton Stafford and Trafford Mental Health Partnership, Manchester, UK and ⁶Colin A. Ross Institute for Psychological Trauma, Richardson, TX, USA

Key words: child abuse; trauma; psychosis; schizophrenia; hallucinations; delusions; literature review

John Read, Department of Psychology, The University of Auckland, Private Bag 92019, Auckland, New Zealand.
E-mail: j.read@auckland.ac.nz

Accepted for publication August 19, 2005

Summations

- Child abuse is a causal factor for psychosis and ‘schizophrenia’ and, more specifically, for hallucinations, particularly voices commenting and command hallucinations.
- Understanding the mechanisms by which child abuse leads to psychosis requires a genuine integration of biological and psychosocial paradigms which acknowledges that adverse events can alter brain functioning.
- Researchers and clinicians should routinely ask about childhood trauma when trying to understand or assist people diagnosed as psychotic or schizophrenic.

Considerations

- There are still only a small number of well-designed studies, controlling for possible confounding variables, with large samples.
- The study of the mechanisms linking child abuse and psychosis is still in its infancy and requires more research to evaluate, and integrate, the theories that have recently been proposed.
- Space prohibited discussion of the ideological, political and economic barriers to the social causes of psychosis being adequately addressed by clinicians, researchers and policy makers (218).

Introduction

Although attention to trauma increased dramatically in the mental health community after the arrival of the post-traumatic stress disorder (PTSD) diagnosis, researchers have, until recently, focussed predominantly on the relationship of trauma to non-psychotic disorders. Meanwhile, the possibility of a relationship with psychosis has been minimized, denied or ignored. The possible reasons for this selective attention have been discussed elsewhere (1), and include rigid adherence to a rather simplistic biological paradigm, inappropriate fear of being accused of 'family-blaming', avoidance of vicarious traumatization on the part of clinicians and researchers, and re-diagnosing from psychosis to PTSD, dissociative disorders and other non-psychotic diagnoses once abuse is discovered.

There is now substantial evidence linking child sexual abuse (CSA) and child physical abuse (CPA) to a range of mental health problems in childhood (2). Child abuse has also been shown to have a causal role in most adult disorders, including: depression, anxiety disorders, PTSD, eating disorders, substance abuse, sexual dysfunction, personality disorders and dissociative disorders, as well as suicidality (3–10). Some of these problems are common in people diagnosed as schizophrenic.

Moreover, child abuse is related to severity of disturbance whichever way one defines severity. Patients subjected to CSA or CPA have earlier first admissions, longer and more frequent hospitalizations, spend longer in seclusion, receive more medication, are more likely to self-mutilate and to try to kill themselves, and have higher global symptom severity (3, 10–19). In one study, suicidality in adult out-patients was better predicted by childhood abuse than by a current diagnosis of depression (17). Studies of bipolar disorder have found child abuse and neglect to be related to earlier onset, severity of mania, number of manic episodes, clinical course and, again, higher rates of suicide attempts (13, 20, 21).

Aims of the study

Despite the research summarized above, it was not until 2004 that sophisticated, large-scale studies addressed the still contentious issue of whether childhood trauma can cause psychosis. It seemed timely, therefore, to summarize and evaluate the pre-2004 literature, examine these important 2004 additions, and to identify the various theoretical and clinical implications.

Material and methods

Review strategy

The literature review began with a search of the electronic database PsycINFO for the period 1872 to week 4 November 2004. Of the 13946 articles under 'Child Abuse' and the 42048 under 'Schizophrenia' 23 resulted when these two classifiers were entered together. Thus only 0.05% of articles on schizophrenia concerned child abuse. (This compares to 4.1% for 'Genetics', 5.0% for 'Neurotransmitters', 8.0% for 'Brain' and 21.5% for 'Drug Therapy'.) Repeating the exercise for 'Child Abuse' and 'Psychosis' (53971) produced 52 articles. Expanding the search to include 'Sexual Abuse', 'Physical Abuse', 'Child Neglect' and 'Emotional Abuse' elicited totals of 42 in relation to 'Schizophrenia' and 90 in relation to 'Psychosis'. Just over half of all the articles found were research studies. Just before this paper was resubmitted, in July 2005, the searches were repeated and five additional research studies were identified. Three previous reviews (1, 22, 23), and the bibliographies of recent studies, were also utilized to try and insure comprehensive coverage.

Almost all the research articles directly assessing the relationship between child abuse and schizophrenia or psychosis are included in the review. In relation to Tables 1 and 2 (concerning abuse prevalence rates in severely disturbed psychiatric samples) studies were excluded if patients were not asked about abuse, via either interview protocols or questionnaires, by giving specific examples of abusive acts; thus chart reviews, and studies merely asking 'were you abused', were not included. Also excluded from these two tables were studies of in-patient alcohol services (24) and military in-patient units (25) with low proportions of psychotic patients. Also excluded were two studies of in-patient units serving populations known to have particularly high rates of child abuse. The rates of CSA were 87% in a PTSD unit (26) and 92% in a unit treating dissociative identity disorder (DID) (27). For some of the studies where the published data were not analysed by gender or type of child abuse researchers generously provided the necessary data when contacted.

Psychosis and schizophrenia

This review focuses on what Kapur, in a paper presented later (28), calls 'psychosis-in-schizophrenia'. We pay particular attention to hallucinations

Table 1. Percentages of reported child abuse among female psychiatric in-patients, or out-patients of whom at least half were diagnosed psychotic

	Sample type	<i>n</i>	Child sexual abuse (CSA)	Incest	Child physical abuse (CPA)	Either CSA or CPA	Both CSA and CPA
Friedman and Harrison 1984 (54)	sc	20	60				
Bryer et al. 1987 (55)		66	44	23	38	59	23
Jacobson and Richardson 1987 (56)		50	22		44	56	10
Sansonnet-Hayden et al. 1987 (19)	ad	29	38		23		
Craine et al. 1988 (57)		105	51	42	35	61	26
Goodwin et al. 1988 (58)		40	50				
Hart et al. 1989 (59)	ad	16	75		69	81	62
Chu and Dill 1990 (60)		98	36		51	63	23
Jacobson and Herald 1990 (61)		50	54				
Shearer et al. 1990 (62)		40	40		25		
Goff et al. 1991 (14)	ps	21				48	
Lanktree et al. 1991 (63)	ch	18	50	44			
Margo and McLees 1991 (64)		38	58		66	76	47
Rose et al. 1991 (18)	op	39	50	41	38		
Carlin and Ward 1992 (65)		149	51				
Lobel 1992 (66)		50	60				
Ito et al. 1993 (67)	ch	51				73	
Muenzenmaier et al. 1993 (68)	op	78	45	22	51	64	32
Mullen et al. 1993 (3)	ex	27	85				
Greenfield et al. 1994 (69)	ps	19	42		42	53	32
Ross et al. 1994 (70)	sc	25	32		32	48	16
Swett and Halpert 1994 (71)		88	61	40	57	76	50
Trojan 1994 (72)	ps	48	25				
Darves-Bornoz et al. 1995 (50)	ps	89	34	18			
Goodman et al. 1995 (73)	op	99	65		87	92	60
Cohen et al. 1996 (74)	ad	73	51		52	68	34
Davies-Netzley et al. 1996 (75)	op	120	56	26	59	77	38
Miller and Finnerty 1996 (76)	sc	44	36				
Wurr and Partridge 1996 (77)		63	52	17			
Briere et al. 1997 (12)	op	93	53		42		
Mueser et al. 1998 (78)	op	153	52		33		
Goodman et al. 1999 (47)	ps	29	78*				
Lipschitz et al. 1999 (79, 80)	ad	38	77		47	90	34
Lipschitz et al. 2000 (81)	ad	57	39		30	65	
Fehon et al. 2001 (82)	ad	71	55		51		
Goodman et al. 2001 (83)	op	321	49		54	67	36
Friedman et al. 2002 (84)	sc	9	78				
Chandra et al. 2003 (85)		146	12				
Holowka et al. 2003 (86)	sc	7	57		17	57	17
Offen et al. 2003 (87)	ps	7	71				
Resnick et al. 2003 (88)	sc	30	47				
Compton et al. 2004 (89)	sc	2	100		100	100	100
Kilcommons and Morrison 2005 (92)	sc	7	14		14		
Schenkel et al. 2005 (90)	sc	15	47		47	53	40
Shafer et al. (91)	sc	30	37		20	41	13
Bowe et al. (93)	sc	8	62		75	75	62
Weighted average (%)			47.7	29.0	47.8	68.8	35.5
Total n			1243/2604	193/666	853/1785	960/1395	450/1266

sc = all diagnosed schizophrenic or schizophrenia spectrum; ps = all diagnosed psychotic; op = out-patients with at least 50% diagnosed psychotic; ad = adolescent in-patients; ch = child in-patients; ex = ex-in-patients.

*Midpoint of two measures.

and delusions, and therefore our paper may, like Kapur's, 'have more implications for understanding the occurrence of psychosis in other illnesses (e.g. manic psychosis) than it does for understanding the non-psychotic (i.e. negative and cognitive) symptoms in schizophrenia' (28, p. 18). Although we shall mention studies concerning childhood trauma and these other symptoms, they are not our focus.

Results

Adult trauma and psychosis

Although not the subject of this review it is necessary to briefly acknowledge, first, the research literature concerning psychosis and post-childhood trauma. Apart from being an important issue in itself, trauma in adulthood is a potential medi-

Table 2. Percentages of reported child abuse among male psychiatric in-patients, or out-patients of whom at least half were diagnosed psychotic

	Sample type	n	Child sexual abuse (CSA)	Incest	Child physical abuse (CPA)	Either CSA or CPA	Both CSA and CPA
Jacobson and Richardson 1987 (56)		50	16		54	58	12
Sansonnet-Hayden et al. 1987 (19)	ad	25	24		52		
Metcalfe et al. 1990 (94)	op	100	34*				
Jacobson and Herald 1990 (61)		50	26				
Goff et al. 1991 (14)	ps	40				42	
Lanktree et al. 1991 (63)	ch	17	12				
Rose et al. 1991 (18)	op	50	22	9	38		
Ito et al. 1993 (67)	ad	53				34	
Greenfield et al. 1994 (69)	ps	19	16		47	53	11
Palmer et al. 1994 (95)	op	100		6			
Ross et al. 1994 (70)	sc	56	30		23	43	11
Trojan 1994 (72)	ps	48	27				
Cohen et al. 1996 (74)	ad	32	34		47	62	19
Wurr and Partridge 1996 (77)		57	39	7			
Mueser et al. 1998 (78)	op	122	36		38		
Goodman et al. 1999 (47)	op	21	45*				
Lipschitz et al. 1999 (79, 80)	ad	33	33		45	66	12
Lipschitz et al. 1999 (81)	ad	38	16		55	71	
Fehon et al. 2001 (82)	ad	59	12		68		
Goodman et al. 2001 (83)	op	461	29		58	65	22
Lysaker et al. 2001 (96)	sc	52	35				
Friedman et al. 1999 (79)	sc	13	0				
Holowka et al. 2000 (81)	sc	19	47		21	53	16
Offen et al. 2001 (82)	ps	19	26				
Resnick et al. 2001 (83)	sc	17	18				
Compton et al. 2002 (84)	sc	16	31		75	75	31
Lysaker et al. 2004 (97)	sc	37	38		57	63	32
Kilcommons and Morrison 2005 (92)	sc	25	12		32		
Schenkel et al. 2005 (90)	sc	25	16		24	40	0
Bowe et al. (93)	sc	14	36		57	57	36
Lysaker et al. (98)	sc	65	28				
Weighted average (%)			28.3	6.8	50.1	59.1	19.4
Total n			435/1536	14/207	542/1081	518/877	145/746

sc = all diagnosed schizophrenic or schizophrenia spectrum; ps = all diagnosed psychotic; op = out-patients with at least 50% diagnosed psychotic; ad = adolescent in-patients; ch = child in-patients; ex = ex-in-patients.

*Midpoint of two measures.

ating factor in the relationship between childhood trauma and psychosis.

War. Combat veterans with PTSD have more schizophrenic symptoms – particularly hallucinations and paranoia – than those without PTSD (29, 30). The literature relating trauma (including child abuse) to psychosis, however, is confounded by the precedence given to a PTSD diagnosis whenever trauma is identified in people who experience psychotic symptoms (1, 22, 31). For example when elevated rates of psychotic symptoms were identified in Vietnam veterans it was automatically assumed that any diagnoses of schizophrenia were misdiagnoses and that these symptoms were therefore somehow non-psychotic (32). Recent research, however, has noted marked similarities between PTSD and schizophrenia (33). Hamner et al. (34) compared patients with PTSD and schizophrenia and concluded that: ‘these two patient populations were remarkably similar with respect to not only positive but also negative symptoms’.

When research scales such as the Minnesota Multiphasic Personality Inventory (MMPI) are used, thereby circumventing diagnostic preferences of clinicians, the highest elevations for combat exposure are on the schizophrenia and depression subscales (35). A recent study identified 55 male war veterans with ‘psychotic combat-related PTSD’ (36). A case study reported delusions of parasites under the skin and around orifices following multiple rapes in the Bosnian war (37). A study of MMPI profiles of CSA survivors and combat veterans found that both had elevated ‘schizophrenia’ scores, and that ‘CSA survivors and combat veterans are much more similar than different in their clinical presentation’ (38, p. 708).

Prisoners of war who had experienced the most severe traumas were found to have a marked increase in schizophrenia (39). The symptoms of psychosis and PTSD have been found to coexist in Cambodians traumatized by the Pol Pot regime (40). One in five Somali refugees in London (most

of whom had fled because of war and risk to their lives) were found to be psychotic (41).

Sexual and physical assault in adulthood. Most psychiatric patients suffer serious physical assaults as adults. One study found that in the year before hospital admission 63% had suffered physical violence by their partners and 46% of those living at home had been assaulted by family members (42). Assaults also occur outside the family, including from mental health staff (43, 44). The majority of women patients have suffered sexual assaults. Approximately a third are raped. About a quarter of male patients are sexually assaulted as adults (22).

Among 409 female in-patients sexual assault was significantly related to schizophrenia, but not to mania, depression, substance abuse or borderline personality disorder (45). Among female patients physical assault (by non-partners) was significantly related to only one diagnostic group: 'non-manic psychotic disorders (e.g. schizophrenia, psychosis not otherwise specified)' (12).

Reliability of self-report

An important issue in the studies reviewed below is the reliability of self-report of abuse. Concern about the accuracy of child abuse disclosures by psychiatric patients is understandable, particularly for those diagnosed psychotic. Such an assumption, however, is not evidence based. Reports of abuse by psychiatric patients, including those diagnosed psychotic, are surprisingly reliable (46, 47). Corroborating evidence for reports of CSA by psychiatric patients has been found in 74% (48) and 82% (49) of cases. One study found that 'the problem of incorrect allegations of sexual assaults was no different for schizophrenics than the general population' (50, p. 82). Psychiatric patients under-report rather than over-report abuse to staff (1, 51–53). A survey of women previously admitted to psychiatric hospital found that 85% reported CSA when interviewed later at home (2), a rate far higher than any found in studies of women still in hospital (Table 1).

Prevalence of child abuse in severely disturbed clinical populations

We have already seen that interpersonal trauma in adulthood is very common among people who experience psychosis and that child abuse is causally related to a myriad of psychiatric sequelae including overall severity. The question of whether there is also a causal relationship between child-

hood trauma and psychosis is raised, but by no means answered, by the many studies finding very high rates of child abuse among in-patients. In 1997 a review (1) of 15 studies of female in-patients found that 64% had suffered either CSA (50%) or CPA (44%). Table 1 presents 39 studies of female in-patients, and seven studies of female out-patient samples where at least half were diagnosed psychotic. These studies confirm that the majority of female patients (69%) report either CSA (48%) or CPA (48%). Table 2 shows that the majority of male patients (59%) also suffer either CSA (28%) or CPA (50%).

Without an Indian study (85), with the lowest female CSA rate (12%), the average CSA rate for female patients is 50%. The other five studies with samples of over 100 averaged 51% (57, 65, 75, 78, 83). When contacted to ask about possible reasons for the particularly low rate, the primary researcher identified 'underreporting', adding that 'in a culture where sexual matters are seldom discussed openly there is no concept of discourse about sexual experience' (P. Chandra, pers. comm).

Other studies in Tables 1 and 2 may also be underestimates because of under-reporting of child abuse in general (99), especially by men (100); and because, as we have seen, people seem particularly unlikely to report child abuse when they are psychiatric patients (1, 3, 52, 53). It must also be noted that Tables 1 and 2 exclude emotional abuse or neglect (physical or emotional). In a recent study, the child abuse prevalence for men increases from 57% (CSA or CPA) to 85% when these three other factors are included; and from 75% to 100% for women (93).

Not all service users, even in in-patient samples, are psychotic. It would be surprising, however, if in the studies presented in Tables 1 and 2 the relationships of child abuse to specific diagnoses were weaker for the more severe diagnoses like 'schizophrenia' than for other diagnoses. As we have seen, child abuse is related to severity of disturbance however it is measured (3, 10–21).

Child abuse and research measures of psychosis and schizophrenia

A 2004 review (22) identified nine studies showing that abuse survivors score higher than non-abused people on the 'schizophrenia' and 'paranoia' scales of the MMPI and the 'psychosis' scale of the Symptom Checklist 90-Revised (SCL-R). In five of the nine these scales were *more* strongly related to abuse than the other clinical scales. A tenth study, of CSA survivors with PTSD, found that the

MMPI schizophrenia scale was the most elevated of the 10 clinical scales (38).

Child abuse and clinical diagnoses

Studies using clinical diagnoses produce similar findings to the MMPI and SCL-R studies (22). While some find that schizophrenia and psychosis are no more, or less, related to child abuse than other diagnoses (44, 74, 75, 77, 84), many find that the relationship is particularly strong for psychosis and schizophrenia. In a study of over 500 child guidance clinic attenders, 35% of those diagnosed 'schizophrenic' as adults had been removed from home because of neglect; double the rate of any other diagnosis (101). A study of over 1000 people found that those whose interactions with their mothers when three years old were characterized by 'harshness towards the child; no effort to help the child' were, as adults, significantly more likely to be diagnosed with schizophreniform disorder, but not mania, anxiety or depression (102). In a sample of adult out-patients diagnosed 'schizophrenic' 85% had suffered some form of childhood abuse or neglect (CSA 50%) (86). Among female out-patients, 78% of those diagnosed 'schizophrenic' had suffered CSA, compared with panic disorder (26%), anxiety disorders (30%), major depressive disorder (42%) (84). In a study of 426 in-patients diagnosed psychotic (103), the CPA rate for the women was 29%, compared with 5% in the general population using identical methods (104). Among child in-patients, 77% of those who had been sexually abused were diagnosed psychotic, compared with 10% of the other children (105).

Child abuse and symptoms of schizophrenia

Because of the disjunctive and heterogeneous nature of the 'schizophrenia' construct (31, 106, 107) many researchers now focus instead on understanding the origins of specific psychotic symptoms, or as Bentall (106) prefers, 'complaints' (so as to avoid language supporting the kind of assumptions that have led to a minimizing of psychosocial factors in the past). Table 3 summarizes studies of the relationship of childhood abuse to symptoms. A more detailed analysis, and table, of studies of the subtypes of delusions and hallucinations is available elsewhere (22).

In a study of 'chronically mentally ill women' those who had been abused or neglected as children experienced more psychotic symptoms than other patients (68). The same is found in general population studies of psychotic symptoms

Table 3. Relationships between child abuse and symptoms of schizophrenia or related schizotypal traits

	Hallucinations	Delusions	Thought disorder	Negative symptoms
Child abuse (CSA or CPA)				
Goff et al. (14)	0		0	(-)
Famularo et al. (114)	+	0	0	0
Ross et al. (70) ¹	++	++		(-)
Read and Argyle (113)	(+)	(+)	(+)	
Berenbaum (110) ^{1,3,4}	+	+		
Read et al. (49)	++	(+)	(+)	0
Resnick et al. (88) ²	+	+		(-)
Janssen et al. (108) ^{1,2,3}	++	++		
Schenkel et al. (90) ^{3,5}	+	+		
Physical abuse				
Mundy et al. (115) ¹	0	+ ^B		
Goff et al. (116)		(+)		
Honig et al. (117)	(+)			
Berenbaum (112) ^{1,4}	+	(+)		
Read and Argyle (113)	(+)	0	0	
Startup (111) ^{1,4}	(+)	0		
Hammersley et al. (118)	0	0	0	
Morrison and Petersen (119) ^{1,4,6}	++			
Read et al. (49)	++	(+)	0	0
Whitfield et al. (217)	+			
Bowe et al. (93) ⁵	0			
Kilcommons and Morrison (92) ⁶	(+)	0		
Sexual abuse				
Bryer et al. (55)		0	+ ⁹	
Sansonnet-Hayden et al. (19)	++	0		
Mundy et al. (115) ¹	+	0 ^B		
Goff et al. (116)		+		
Ensink (120)	(+)			
Honig et al. (117)	0			
Berenbaum (110) ^{1,4}	+	0		
Read and Argyle (113)	(+)	0	(+)	
Startup (111) ^{1,4}	+	0		
Lysaker et al. (96)				0
Hammersley et al. (118)	++	0	0	
Morrison and Petersen (119) ^{1,4,6}	0			
Offen et al. (87)	(+)			
Read et al. (49)	++	(+)	(+)	0
Bebbington et al. (121) ^{1,2,6}	++	++		
Spataro et al. (2) ^{1,2}	0	0		
Whitfield et al. (217)	+			
Bowe et al. (93) ⁵	++			
Kilcommons and Morrison (92) ⁶	++	0		
Incest				
Beck and van der Kolk (11)		(+)		
Bryer et al. (55) ⁷		0	0 ^B	
Ellenson (122)	(+)			
Heins et al. (123)	(+)			
Mundy et al. (115) ⁷	(-)	0 ^B		
Read and Argyle (113) ⁷	++	0	0	
Read et al. (49) ⁷	(+)	(+)	0	0

+, P < 0.05; ++, P < 0.01; (+) (-), non-significant trend, or high rates with no control group; 0, no difference; blank cells mean relationship not examined in that study.

¹Community/non-patients (all others clinical samples).

²Measure of positive symptoms in general.

³Measure included other forms of childhood trauma and abuse.

⁴Related schizotypal trait/predisposition.

⁵Relationships with severity and/or frequency of abuse.

⁶Includes abuse after childhood.

⁷Comparison of incest to non-familial sexual abuse.

⁸Paranoia.

⁹Psychotic thinking.

(108, 109) and of schizotypal indices such as perceptual aberrations and magical ideation (110, 111). A recent general population study found that childhood neglect was more predictive of schizotypal traits than were CSA or CPA (112).

In one community survey 46% of people with three or more schizophrenic symptoms had suffered CPA or CSA, compared with 8% of those with no symptoms (109). An in-patient study found one or more of the DSM's five characteristic symptoms for schizophrenia in 75% of those who had suffered CPA, 76% of those who had suffered CSA, and 100% of those subjected to incest (113). A study of 200 adult out-patients found that 35% of those abused as children had two or more of the five symptoms, compared with 19% of the non-abused patients (49). Ross and colleagues found, with an in-patient sample of 'schizophrenics', that those who had suffered CSA or CPA had significantly more positive schizophrenia symptoms (but slightly fewer negative symptoms) than those not abused. The symptoms that were significantly related to abuse were, in order of the strength of the relationship: Voices Commenting, Ideas of Reference, Thought Insertion, Paranoid Ideation, Reading Others' Minds and Visual Hallucinations (70).

Hallucinations. Table 3 indicates a particularly strong relationship between childhood abuse and hallucinations. A New Zealand in-patient study found hallucinations in 53% of those subjected to CSA, 58% of those subjected to CPA, and 71% of those who suffered both CSA and CPA (113). Another New Zealand study, of out-patients, found hallucinations in 19% of the non-abused patients but 47% of those subjected to CPA, 55% of those subjected to CSA and 71% of those subjected to both CPA and CSA (49). The figures for 'command hallucinations' to harm self or others were: non-abused 2%, CPA 18%, CSA 15%, CSA + CPA 29%. The figures for voices commenting were: non-abused 5%, CPA 21%, CSA 27%, CSA + CPA 36%. Abused adolescent and child in-patients are also more likely to hallucinate than their non-abused counterparts (19, 114).

The relationship between abuse and hallucinations may exist across diagnostic boundaries. Hammersley et al. (118) found, in adult bipolar affective disorder patients, that although those subjected to CSA were no more likely to have visual hallucinations, they were twice as likely to have auditory hallucinations in general and six times more likely to hear voices commenting.

Among non-patients predisposition to auditory, but not visual, hallucinations was significantly higher in those who reported multiple traumas.

Emotional abuse (strongly) and physical assault were related to predisposition to auditory hallucinations. Bullying was related to predisposition to visual hallucinations (119). Amongst homeless Los Angeles adolescents CSA, but not CPA, was significantly related to auditory hallucinations (115). Experiencing auditory hallucinations as malevolent has been found to be related to paternal overprotection (i.e. being 'controlling and intrusive') and low levels of paternal care (124).

A Dutch study of people diagnosed with schizophrenia or dissociative disorder found that 'in most patients, the onset of auditory hallucinations was preceded by either a traumatic event or an event that activated the memory of earlier trauma' (117, p. 646).

Delusions. One of the largest and best-designed general population studies to date found delusions to be as strongly related, or perhaps even more strongly related, to child abuse as hallucinations (108). High rates of sexual delusions have been found in incest survivors diagnosed psychotic (11). Amongst homeless adolescents in Los Angeles intrafamilial CPA (but neither CSA nor extrafamilial CPA) was significantly related to paranoid psychotic symptoms (115).

However, some studies that found a relationship between child abuse and hallucinations in adolescent in-patients (19), child in-patients (114), and bipolar patients (118) found no relationship with delusions. In a study of 200 out-patients 40% of the CSA patients experienced some form of delusion, compared with 27% of the non-abused; but this was not statistically significant (49). This study did not replicate the relationships found by Ross et al. (70) between child abuse and ideas of reference or mind reading. It did, however, provide a degree of support for their finding of a relationship between child abuse and paranoid ideation; paranoid delusions were present for 40% of the CSA out-patients, compared to 23% of the non-abused patients. This study (49) also found paranoid delusions in 36% of incest survivors but none of the extrafamilial CSA cases.

Thought disorder, catatonia and negative symptoms. While one early study did find a relationship between CSA and 'psychotic thinking' (55) subsequent studies have not found a relationship between CSA and thought disorder and have also found no link to CPA (49, 118). In schizophrenia spectrum patients the cognitive component of the Positive and Negative Syndrome Scale (125) has been found to be significantly related to CSA (96). A high rate of thought disorder has been found in

patients who suffered both CSA and sexual assault later in life (49).

There seems to be no research examining the relationship between trauma and ‘grossly disorganized or catatonic behaviour’. However, in his original 1874 conception of catatonia Kahlbaum (126, p. 4) stated that it was usually precipitated by ‘very severe physical or mental stress... (such as) a very terrifying experience’. Some commentators still see catatonia as an extreme fear response (127).

Studies of adult (49, 88, 96) and child (114) in-patients have found no differences in rates of negative symptoms between abused and non-abused. Two adult in-patient studies found slightly fewer negative symptoms in abused patients (14, 70). A recent study suggests that negative symptoms may be a reaction to the trauma of psychosis and hospitalization (128). Of course, if the psychosis is itself a reaction to childhood trauma in the first place, this would position psychosis and hospitalization as mediating variables in the relationship between child abuse and negative symptoms. Another important research avenue is the overlap, and relationship, between negative symptoms and the clearly trauma-based avoidance and numbing symptoms of PTSD (33, 129).

The content of psychotic symptoms. Several studies have found that the content of hallucinations and delusions experienced by abuse survivors is frequently related to the abuse (11, 49, 122, 123, 130). For example, Ensink found that the content of hallucinations of CSA survivors contain both ‘flash-back elements and more symbolic representations’ of traumatic experiences (120, p. 126). In maltreated 5- to 10-year olds ‘the content of the reported visual and/or auditory hallucinations or illusions tended to be strongly reminiscent of concrete details of episodes of traumatic victimization’ (114, p. 866). In an out-patient study reference to evil or the devil was more common among those who had been sexually abused (49). Another study found that CSA is related to a tendency to regard auditory hallucinations as malevolent (87). Two studies that have attempted to quantify the frequency with which symptom content is obviously related to trauma in psychotic adults who were abused as children both found that this occurs in just over half of cases (93, 113).

Controlling for other variables

Many of the studies discussed thus far are either correlational or uncontrolled group comparisons. Such studies tell us little about whether the relationship between child abuse and psychosis is

a causal one. Other factors might account for the relationship, such as other adverse events or circumstances in childhood, and subsequent re-victimization. However, other mental health problems are related to child abuse after controlling for these potentially mediating variables (6, 9, 131). After controlling for other childhood disadvantages, women whose CSA involved intercourse were 12 times more likely than non-abused females to have had psychiatric admissions, and 26 times more likely to have tried to kill themselves (3). Prior to 2004 only two studies appear to have controlled for these variables in relation to the child abuse–psychosis relationship.

In the first study, of psychiatric emergency room patients, 53% of those who had suffered CSA had ‘non-manic psychotic disorders (e.g. schizophrenia, psychosis not otherwise specified)’ compared with 25% of those who were not victims of CSA (12). After controlling for ‘the potential effects of demographic variables, most of which also predict victimization and/or psychiatric outcome’ CSA was more strongly related to psychotic disorders ($P = 0.001$) than to other diagnoses.

The second study (49) controlled for re-traumatization. In a regression analysis, childhood abuse (CSA or CPA) was a significant predictor of hallucinations even after taking into account (i.e. without) sexual or physical assault in adulthood. This was the case for auditory hallucinations in general and specifically for voices commenting and tactile hallucinations. However for command, visual and olfactory hallucinations, the relationships with child abuse were, in the absence of later re-traumatization, no longer significant. Similarly, while delusions and thought disorder were predicted by childhood and adulthood abuse in combination, childhood abuse was not significantly related to these symptoms in the absence of adulthood abuse.

The three 2004 studies

In 2004 three studies addressed the question under review here with larger samples and more sophisticated methodology than any of the studies discussed above. The first concluded that its findings offered no support for the hypothesis that child abuse has a causal role in psychosis. The other two produced findings supporting the hypothesis.

Spataro et al. The first was an Australian study, from Paul Mullen’s research team (2). This prospective study, of 1612 documented CSA cases, compared subsequent rates of treatment for various diagnoses with rates of treatment in the

general population. A major strength of the design was that all CSA cases were drawn from the records of the Victorian Institute of Forensic Medicine, which assesses suspected CSA for the police and protection agencies. The study thereby circumvented concerns about the accuracy of self-reports of historical abuse.

The abused males were 1.3 times more likely, and the abused females 1.5 times more likely, to have been treated for a 'schizophrenic disorder' than the general population. However, these differences were not statistically significant. The authors concluded: 'The results do not support an association between child sexual abuse and psychosis' (p. 418).

The researchers, however, listed numerous limitations which 'reduce the probability of finding a positive association between CSA and mental disorders'. These included the 'systematic bias' introduced by the presence of people in the general population sample who had suffered CSA. It was acknowledged that this 'major bias' 'will act to reduce or even obscure the differences between cases and controls' (p. 419). Another acknowledged limitation was the inclusion of only the most severe forms of CSA. 'A further systematic bias against detecting higher rates of disorder among those who have suffered child sexual abuse is introduced by comparing child sexual abuse cases established by data matching, with a control population, which is derived from relating all known cases on the register back to the known population base. Data-matching inevitably misses cases' (p. 419). The study was so compromised by these limitations that it also failed to replicate the well-established relationship between CSA and alcohol and drug-related disorders (3, 12).

Beyond all these 'powerful systematic biases against finding differences' in general (p. 419) there was an additional flaw (also acknowledged) that was specific to the failure to find a relationship with schizophrenia. 'The average age of our subjects was in their early 20s, thus many have yet to pass the peak years for developing schizophrenic and related disorders'. In addition, the general population sample was significantly older than the abused sample and therefore had more chance of developing schizophrenia. The researchers therefore acknowledged, in relation to their schizophrenia finding: 'Care must be taken in interpreting this and other negative findings' (p. 419).

One final bias, weakening still further the already diminished probability of finding a relationship between CSA and schizophrenia, was not identified by the researchers (132). This is surprising given how obvious and significant the bias was. The use

of agency samples or official record data has been highlighted by previous researchers as producing biased samples which influence estimates of the consequences of abuse (99). Because all the abuse cases had been identified and verified by the relevant authorities, the abuse was obviously acknowledged by adults, most of the children would have been no longer at risk of ongoing abuse, some would have been removed from their home and some would have received support or therapy. Such situations are very rare. Most child abuse cases are never even reported to the police (133). A previous study, also by Spataro et al. (100), identified that only 20% of Australian girls, and 4% of boys, subjected to CSA are removed from their homes as a precaution against further abuse. Such factors are powerful predictors of long-term outcomes, not only via the reduction of multiple or ongoing abuse but also in terms of attributions about blame for the abuse (134). The CSA sample in this study was therefore grossly unrepresentative of CSA cases. Indeed, one might conclude that the data gathered by Spataro, Mullen and their colleagues, which is so extensively biased as to be unable to contribute meaningfully to the issue of whether child abuse can cause psychosis, actually raises an important research question: Do early acknowledgment of the abuse by adults and the taking of appropriate steps such as removing children from abusive situations, provide some protection from subsequent psychosis?

Bebbington et al. Paul Bebbington and colleagues (121) used interview-based data on 8580 adults from the British National Survey of Psychiatric Morbidity to test the hypothesis that a range of early victimization experiences 'contribute to vulnerability to psychosis' (p. 220). Unlike the Spataro study, this was not a prospective study and relied on retrospective self-report of the victimization experiences. The study's strength, apart from the large sample size (including a total of 60 in the psychosis group, compared with 13 in the schizophrenia group of Spataro et al.), was that it controlled for demographic and mental health variables and for interactions between a range of adverse events.

In the psychosis group, established by a two-stage interview process, 34.5% reported sexual abuse, 46.4% reported bullying, 38.1% 'violence in the home' and 34.5% 'running away from home'. The psychosis group was 15.5 times more likely to have suffered sexual abuse than those without any mental disorder. Contrary to assumptions that psychosis is less related to child abuse than other disorders, the odds ratios for other disorders were:

neurotic disorder 6.9, alcohol dependence 2.4, drug dependence 1.8. A similar pattern, of psychosis having the highest odds ratio, emerged for almost all the victimization experiences.

The individual relationships between each of the victimization experiences and psychosis were then analysed, using logistic regression, to control for the inter-relationships between the various experiences. Sexual abuse was the most powerfully related of all the events to psychosis, being 3.9 times more common than in the 'no disorder' group ($P = 0.001$). Violence in the home was 2.0 times more common ($P = 0.07$) and running away from home 2.9 times more common ($P = 0.004$). Including gender, age and ethnicity in the analysis did not effect the findings.

An acknowledged limitation of this study was that the 'events were not dated, although some were by definition early' (p. 225). Adding that 'it might be expected that sexual abuse in people with psychiatric disorders is a childhood phenomenon' does not remove the possibility that some affirmative responses to this question were probably related to experiences in adulthood. It should be noted however that several events that clearly occurred in childhood, such as 'running away from home' and 'time in a children's institution' were not only significantly related to psychosis, but were more powerfully related to psychosis than to other disorders, and remained significantly related to psychosis after controlling for interactions between all the events.

Despite this lack of precision about the timing of events the study provides strong support for a social causation model, with numerous adverse events, some clearly in childhood, predicting psychosis after controlling for both interactions and demographics. The authors concluded: 'In people with psychosis, there is a marked excess of victimising experiences, many of which will have occurred during childhood. This is suggestive of a social contribution to aetiology' (p. 220).

Janssen et al. The third 2004 study involved a general population sample of 4045 in the Netherlands (108). This was a prospective study, involving interviews with adults who had been free of any expression of psychosis 2 years earlier, both at level of disorder and at level of subclinical experiences. This is a limited form of prospective study which does not involve a cohort of cases identified at the time of the abuse. However, including only new cases of psychosis emerging in adulthood avoids the possible explanation of positive results in terms of pre-existing psychosis somehow having rendered participants more vul-

nerable to maltreatment. An important strength of the study is the comprehensive range of potentially mediating variables controlled for: age, sex, education level, unemployment, urbanicity, ethnicity, discrimination, marital status, presence of any psychiatric diagnosis and life-time drug use. The additional inclusion of positive psychotic symptoms or mental health care in first-degree relatives produced data of relevance to the role of genetic predisposition. Another advantage over the other two 2004 studies was assessment of the severity of abuse – in terms of frequency and number of abuse types (physical, sexual, psychological and emotional). Those abused only 'once' or 'sometimes' were not included in the exposure definition. Furthermore, three measures of psychosis were used. 'Any psychosis' represented any presence of the 'unusual thought content' or 'hallucinations' items of the Brief Psychiatric Rating Scale. 'Pathology level psychosis' represented a score of between 4 and 7 on either of these two 1 to 7 item scales. The most severe level, 'Need-based psychosis', was determined using the Camberwell Assessment of Need in the areas of psychotic symptoms and psychological distress and a consensus agreement among four clinicians about need for mental health care.

On these three measures of psychosis people who had been abused before age 16 years were, respectively, 3.6, 13.0 and 11.5 times more likely to become psychotic during the study period than non-abused participants. After controlling for all the variables listed above the odds ratios, 2.5, 9.3 and 7.3 respectively, remained large and statistically significant.

Dose-effect

Studies of non-psychotic disorders provide supporting evidence of a causal relationship to childhood trauma in the form of findings that the more severe the abuse the stronger the relationship (3, 6, 17). Several recent studies show that this 'dose-effect' holds true for the relationship with psychosis (90, 92, 93, 135). As noted earlier New Zealand studies (49, 113) found that being subjected to both CSA and CPA increases the probability of a range of psychotic symptoms beyond the probability related to either CSA or CPA alone. In a study of 100 incest survivors, a cumulative trauma-score (involving multiple types of abuse and multiple abusers) was significantly higher in those who later experienced auditory or visual hallucinations (120). Such findings are consistent with the finding that it was the most highly traumatized prisoners of war that were particularly likely to develop

schizophrenia (39). The dose-effect may also cross diagnostic boundaries. In the Hammersley study, that found high rates of auditory hallucinations in abused bipolar patients, only one of the severely abused patients (defined as before age 6, multiple incidents or intrafamilial) did not experience auditory hallucinations (118).

One of the most compelling examples of this dose-effect comes from the 2004 Janssen study (108). People who had experienced child abuse of mild severity were 2.0 times more likely than non-abused participants to have 'pathology level' psychosis, compared with 10.6 and 48.4 times more likely for those who had suffered moderate and high severity of abuse respectively. The same dose-effect was also found for the other two psychosis measures used in this study.

Discussion

Diagnostic issues

It has been repeatedly found that there is considerable overlap between the diagnostic constructs of schizophrenia, dissociative disorders and PTSD (31, 33, 136). The DSM states that in PTSD the re-experiencing of the trauma includes 'hallucinations and dissociative flashback episodes' (137). Between 46% and 67% of acutely psychotic people also have PTSD (138–140). The overlap between dissociative symptoms and the positive symptoms of schizophrenia is equally striking (31, 70, 109, 120, 141, 142). One study found that in-patients with dissociative disorders have four times as many schizophrenic symptoms as other in-patients (143). Another study found more positive symptoms of schizophrenia in DID patients than in those diagnosed with schizophrenia (144). Similarly, it has recently been shown that hallucinations are not uncommon in people diagnosed with borderline personality disorder (145).

What may be happening is that symptoms which are categorized as psychotic by clinicians (or researchers) before trauma has been identified are recategorized as somehow non-psychotic when the trauma history becomes known. The symptom has not changed, but the observer's perception may have been influenced by a belief that abuse cannot cause psychosis but it can cause PTSD, dissociative disorders and borderline personality disorder. Terms used in these circumstances include 'pseudo-hallucinations', 'dissociative hallucinations', 'psychotic-like hallucinations', etc. (55, 60, 123). Indeed, one recent theory of psychosis suggests that it is the culturally unacceptable nature of appraisals that determines

whether a person is viewed as psychotic or not in relation to trauma (23, 146). It may be that the transparency of the link between the traumatic event and the content and form of psychotic experiences contributes to this process. For example, if someone describes vivid perceptual experiences as being related to past physical or sexual assault, then this is likely to be regarded as consistent with a flashback experience in PTSD, whereas if they report that the experiences are real, current and unrelated to past experience, then they are likely to be regarded as being psychotic.

If we were not constrained by the need for a diagnostic nomenclature we might not need to separate abuse sequelae into seemingly discrete categories such as PTSD, dissociative disorders, schizophrenia, borderline personality disorder, etc. We might be able to understand all these abuse-related symptoms, scattered throughout our diagnostic manuals, as 'related components of a long-term process beginning with adaptive responses to early aversive events and evolving into a range of interacting maladaptive disturbances in multiple personal and interpersonal domains' (22, p. 240).

We therefore turn our attention to trying to understand the processes by which childhood trauma leads to the specific experiences and behaviours currently labelled psychotic or schizophrenic.

How does trauma lead to psychosis?

Not all childhood abuse is traumatic in the narrow, life-threatening sense required for a PTSD diagnosis. It seems that such extreme fear is not necessary to increase the probability of psychosis later in life. Any meaningful theory about how early abuse or trauma leads, years later, to psychotic symptoms must integrate biological and psychological paradigms. A number of theories have been developed (23, 130). At the psychological level of analysis the focus has been on cognitive and attributional processes, dissociation and, to lesser extent, attachment theory. At the biological level the focus has been the recently discovered neurodevelopmental effects of trauma on children's brains, particularly damage to the stress regulation mechanisms in the hypothalamic-pituitary-adrenal (HPA) axis. Our review revealed that promising first attempts to integrate the two levels of analysis are emerging.

Cognitive models. Recent cognitive models of psychosis may help to explain the relationships between the experience of trauma, the development

of psychotic experiences and becoming a patient with a psychotic diagnosis. There may be several ways in which traumatic experiences may confer vulnerability to psychosis via cognitive and behavioural processes (23). As mentioned earlier one theory of psychosis (23, 146) suggests that it is the transparency of the link between the traumatic event and the content and form of psychotic experiences that determines appraisals of whether a person is psychotic or not.

It is also possible that the cognitive and behavioural consequences of trauma may make people vulnerable to psychosis. Negative beliefs about self, world and others (such as 'I am vulnerable' and 'other people are dangerous') have been shown to be associated with psychosis (146–148). A recent study has also shown that such beliefs, specifically formed as a result of trauma, are related to psychotic experiences (92). Positive beliefs about psychotic experiences (such as 'paranoia is a helpful survival strategy') have been shown to be associated with the development of psychosis, and may also be related to trauma (149).

It is likely that psychotic experiences are essentially normal phenomena that occur on a continuum in the general population (150, 151). It would seem that the occurrence of trauma in the life history of a person experiencing such phenomena may represent the difference between patients and non-patients (117). It seems that catastrophic or negative appraisals of early psychotic experiences result in the associated distress (152, 153), and that such appraisals are more likely if people have a trauma history. This hypothesis is supported by the finding that adults who were abused as children are 10 times more likely to experience distress when first having psychotic experiences than those who were not abused (154). It is also consistent with the Traumagenic Neurodevelopmental model (reviewed below) which postulates that a central effect of trauma on children's brains is a heightened sensitivity to stress.

Hallucinations: decontextualized trauma flashbacks? We have seen that hallucinations are strongly related to child abuse. Some psychotic hallucinations appear to be nothing more or less than memories of traumatic events identical to the split-off flashbacks usually considered indicative of PTSD rather than schizophrenia (30, 33, 114, 120). As discussed earlier, hallucinations can be recategorized when trauma is identified simply because of the assumption that trauma cannot cause psychosis but can cause PTSD. A recent study of patients with a diagnosis of schizophrenia who hear voices found that the severity of childhood abuse was

associated with the content and severity of auditory hallucinations, and that the content of auditory hallucinations was often directly linked to traumas and other major life events (93).

Other 'psychotic hallucinations' are also trauma based but involve confusion between inner and outer experience. Some intrusive, flashback memories of child abuse occur with awareness that the experience is indeed an internal event relating to the past, i.e. a memory of the trauma. Others, however, occur without this awareness and are experienced as external events in the present. This misattribution of an internal event to an external source (faulty 'source monitoring') has become a central tenet of many of the recent advances made by British cognitive psychologists in understanding psychotic phenomena (106, 148, 155–157).

To experience the memory of the smell of semen, or of the perpetrator's voice calling you a 'slut' as external events in the present can serve as a defence against reliving what actually happened as a child. Although the misattribution may lead to considerable distress and to delusional explanations of the experience (see below) at least one does not remember or relive the actual trauma. From a less psychodynamic perspective Bentall (106, p. 483) offers the following additional explanation:

Source-monitoring failures tend to occur when we experience intrusive or automatic thoughts.... It follows that a person who has poor source-monitoring skills will be most vulnerable to hallucinations when experiencing a flood of intrusive thoughts and images. Trauma (we know from the research literature on PTSD) often has exactly this effect.

The findings of a 2005 study confirm that source monitoring difficulties are a 'prominent feature of schizophrenia' and 'suggest that they form a more enduring characteristic of this disorder than has previously been assumed' (157, p. 57). Faulty source monitoring (or projection, from a psychodynamic perspective) might seem to apply more readily to visual, tactile and olfactory hallucinations than to voices. Flashbacks of the sounds, sights, touch sensations and smells involved in the abuse can be experienced, piecemeal as it were, as hallucinations occurring in the external present. Sensory and perceptual components of traumatic events can be re-experienced without any awareness of their relatedness to past events (158). Nadel and Jacobs (159) offer an elegant integration of psychology and biology in their discussion of trauma-induced inactivation of the hippocampus, which is responsible for the contextualization of memories. Another approach suggests that the strength of contextual integration, which occurs during encoding,

influences the frequency and nature of subsequent intrusive experiences; consequently, individual differences in schizotypal personality traits, which are known to be associated with levels of contextual integration, are also assumed to be related to the phenomenology of trauma-related intrusions (160). In support of this, a recent study found that individuals scoring high in positive schizotypy were more vulnerable to experiencing trauma-related intrusions than low scoring schizotypes, following the viewing of a stressful film (161).

Dissociation. Conceptualizing hallucinations as dissociative events offers a valuable additional perspective (162). Schneiderian and other positive psychotic symptoms follow logically from the existence of a structurally dissociated psyche. In the dissociated psyche, intrusions of thought, feeling, memory, will and behaviour into the executive self can give rise to auditory hallucinations, passivity experiences, delusions of being possessed, thought insertion and automatisms. Inversely, it is conceivable that dissociated part selves can remove psychic elements from the executive self, potentially resulting in thought withdrawal, catatonia, amotivational syndromes and other negative symptoms (31).

The failure to integrate traumatic events at the time of their occurrence can result in the persistence of disaggregated stimuli which, compartmentalized and independent of context, emerge later in life (162). Voices commenting, a common hallucination in schizophrenia, is also very common in patients with DID (163). The most common voices in both schizophrenia and DID are punitive and hostile (117). Such powerfully negative internal self-statements are precisely the kind of inner speech that we might be better off, in the short-term at least, experiencing as located outside ourselves and as unrelated to anything real in our past.

Paranoid delusions: faulty attempts to explain trauma-based hallucinations? Some people, when faced with negative, emotionally loaded, or unusual or anomalous experiences quickly jump to the suspicion of external threat, i.e. they become paranoid. Hearing voices when there is nobody there is often (but not always) a negative experience, and is often experienced as unusual or anomalous. Paranoid delusions are sometimes, therefore, understandable attempts to make sense of hallucinations (in various sense modalities) (106, 146–148, 155–157).

Paranoid delusions can, of course, develop in the absence of hallucinations. Is there a difference between the hypervigilance to threat acknowledged in PTSD patients to be the outcome of

trauma and the belief that people are out to get you which is labelled ‘delusional’ in traumatized people diagnosed psychotic? (30, 33). Having been severely or repeatedly abused as a child is likely to render other people a serious potential threat, a threat that can easily be generalized to anyone or anything that is reminiscent of the perpetrator or the circumstances surrounding the abuse. The processes by which hypervigilance develops into fixed paranoid delusions would appear to be a fruitful research avenue. Again, Nadel and Jacob’s (159) work on the impact of trauma on the brain is salient. Whether we label this PTSD, DID or schizophrenia, the resulting fear, distortions and impoverishment of lives remain.

Heightened sensitivity to stressors: the Traumagenic Neurodevelopmental (TN) model

Many of the theories attempting to explain trauma’s relationships with hallucinations and delusions, such as high levels of distress in the face of anomalous experiences and hypervigilance to threat, are consistent with a heightened sensitivity to stress in general. A study of 271 severely ill in-patients found that the two subscales of the Brief Symptom Inventory most strongly related to sexual and physical abuse were ‘psychoticism’ and ‘interpersonal sensitivity’ (164).

Heightened reactivity to stressors is a cardinal feature of ‘schizophrenia’ (165) and is considered the core of the ‘constitutional vulnerability’ that forms the diathesis in the stress-diathesis model. Activation of the HPA axis is one of the primary manifestations of the stress response. In 1997, Walker and Diforio (165, p. 672) identified ‘a unique neural response to HPA activation’ in schizophrenia. The adrenal cortex, stimulated by adrenocorticotrophic hormone from the pituitary, releases glucocorticoids (including cortisol in humans). The hippocampus contains a high density of glucocorticoid receptors and plays a vital role in the feedback system modulating activation of the HPA axis. ‘When exposure to stressors persists and heightened glucocorticoid release is chronic, there can be permanent changes in the HPA axis. Most notably, the negative feedback system that serves to dampen HPA activation is impaired’ (p. 670).

Studies of the role of dopamine in producing increased sensitivity following prolonged or severe exposure to stress have led to the realization that ‘experience-dependent effects may be an important ontogenetic mechanism in the formation, and even stability, of individual differences in dopamine

system reactivity' (166, p. 507). Thus stress exposure elevates not only the release of cortisol but of dopamine as well, a neurotransmitter consistently linked to 'schizophrenia'.

Walker and Diforio (165, p. 679) stressed the importance, for understanding the causes of 'schizophrenia', of 'identifying the patient characteristics that predict sensitivity to stressors'. One response was a paper entitled 'The contribution of early traumatic events to schizophrenia in some patients: a Traumagenic Neurodevelopmental model' (167). This paper documents the research showing that over-reactivity, and dysregulation, of the HPA axis is found in abused children. It also presents evidence that the dopamine irregularities so frequently cited as evidence that schizophrenia is a predominantly or purely biogenetic phenomenon are found in traumatized children. Of crucial importance were findings that these brain 'abnormalities' caused by childhood trauma can persist into adulthood. Heim et al. (168, p. 592) had already suggested that: 'HPA and autonomic nervous system hyperreactivity, presumably due to cortisol releasing factor hypersecretion, is a persistent consequence of childhood abuse that may contribute to the diathesis for adulthood psychopathological conditions'.

The TN model is an example of a more genuine integration of the reciprocal, complex interactions between social, psychological and biological factors than the 'bio-psycho-social'/'stress-diathesis' model which has for decades underemphasized the fact that our brains are affected by our environment throughout life (11, 22, 167). This is especially true – due to high plasticity – during childhood (169, 170).

The TN model also documented that the neurological abnormalities that have also been cited as evidence that schizophrenia is a brain disease are found in the brains of traumatized children, including: hippocampal damage, cerebral atrophy, ventricular enlargement, and reversed cerebral asymmetry. The cognitive deficits that occur in association with these structural brain abnormalities can also be the result of child abuse (167).

Since that 2001 paper (167) the evidence that child abuse causes long-lasting adverse changes in the HPA axis, hippocampus and dopamine system has continued to accumulate (169, 171–173). These long-term changes can even be caused by prenatal stress on the mother (174), suggesting that even brain abnormalities that exist from birth need not be genetically based. It is relevant to note here that one of the best designed studies of the genetics of schizophrenia found that among children at biological risk for schizophrenia those adopted into dysfunctional families had higher than normal

rates of schizophrenia, while those adopted into healthy families had rates similar to the general population (175).

The question 'Does stress damage the brain?' (173) is finally and firmly on the international research agenda. The focus, however, continues to be on PTSD, depression or anxiety. The implications for psychosis are, however, beginning to be recognized (174, 176, 177). A recent paper, discussing the role of *N*-methyl-D-aspartate receptors in the lack of coordination between specialized brain functions in 'schizophrenia', argued:

Physical and sexual abuse in childhood has been linked to an increased risk of schizophrenia... this may be as fruitful an area to explore as that of behaviour genetics of cognitive coordination in schizophrenia. (178, p. 117)

The TN model proposes that trauma, if sufficiently prolonged, severe or early, can actually create the vulnerability in the vulnerability-stress equation. It can contribute to the oversensitivity to later stress, with or without a genetic predisposition. Recent studies have confirmed that adverse life events can render individuals more vulnerable to the onset of psychotic experiences via increasing their emotional reactivity to subsequent stressors (179), and that the consequent emotional reactivity to daily stress can contribute to the underlying vulnerability for psychotic disorders (180).

These biological sequelae to childhood trauma are not irreversible. Nemeroff's (169, p. 25) review highlights 'intriguing data showing that cross-fostering of rodent pups and environmental enrichment may reverse some of the neurobiological consequences of early-life stress'. Recent neuroimaging studies have also demonstrated that psychotherapy significantly changes functions and structures of the brain (181).

Psychosis as a state of trauma-induced, dopamine-mediated, aberrant salience?

Another example of the theoretical progress that occurs when the goal is a genuine integration of psychological and biological paradigms (31, 33, 106, 167, 182–184) is a 2003 *American Journal of Psychiatry* paper entitled 'Psychosis as a state of aberrant salience: a framework linking biology, phenomenology and pharmacology in Schizophrenia' (28). Shitij Kapur examines the role of dopamine in 'mediating the conversion of the representation of an external stimulus from a neutral and cold bit of information into an attractive or aversive entity' (p. 14).

Kapur does not discuss the origins of the dysregulated dopamine transmission underlying

his theory. His paper is certainly consistent with, and indeed goes some way to integrating, much of what we have covered thus far in our attempts to understand how trauma might lead to psychosis, particularly the TN model and the cognitive theories of hallucinations and delusions. In Kapur's model, hallucinations 'reflect a direct experience of the aberrant salience of internal representations', and arise from 'the abnormal salience of the internal representations of percepts and memories'. 'Delusions in this framework are a "top-down" cognitive explanation that the individual imposes on these experiences of aberrant salience in an effort to make sense of them. Since delusions are constructed by the individual they are imbued with the psychodynamic themes relevant to the individual and are embedded in the cultural context of the individual' (pp. 13–16).

Kapur acknowledges that although 'aberrant dopamine/aberrant salience has a privileged role in expressing psychosis-in-schizophrenia, this role is played out with other actors – neurodevelopmental, cognitive and interpersonal deficits – that take to the stage before the presumed hyperdopaminergic abnormality' (p. 18). The TN model has documented that childhood trauma can cause long-standing neurodevelopmental, cognitive and interpersonal deficits of the kind found in people diagnosed as schizophrenic. More importantly it also demonstrates that childhood trauma can cause the long-term dysregulation of the dopaminergic system that is so common in people diagnosed as schizophrenic and which forms the biological basis for Kapur's aberrant salience theory of hallucinations and delusions.

Can trauma increase risks for psychosis without a genetic predisposition?

Because the diagnosis of 'schizophrenia' is rare and the heritability in liability high at around 80%, it is often assumed that the underlying genetic liability is similarly rare and is always involved in the few individuals who develop the disorder. Following this line of reasoning, it could be argued that individuals exposed to trauma who develop a condition that fulfils the diagnostic criteria for schizophrenia would have become psychotic anyway on account of their genetic risk, and only became psychotic a little earlier. However, although the diagnosis of schizophrenia has been defined in such a way that it appears to be a discrete disorder, it is widely accepted that the underlying genetic vulnerability is a function of multiple genes that contribute to an underlying continuous dimension with a distribution in pop-

ulations (185). The genetic risk for psychosis therefore is ubiquitous and variable so that the more important question becomes what type of relationship genes and environment may have in shaping the risk for psychosis. Some environmental factors may show *synergism* with genetic risk, i.e. genes and environment reinforce each other, so that for example their separate weaker effects become a joint strong effect; other environmental factors may have a strong effect in isolation that however in combination with genetic risk is reduced (*antagonism*), and yet other environmental effects may 'compete' with genetic risk factors to cause psychosis (*parallelism*) (186, 187). Recent work suggests that some environmental factors with an effect early in life act synergistically with genetic risk (188) although a degree of parallelism also appears to be present (189). The suggestion that synergism is the dominant class of gene–environment relationships in relation to psychosis may explain why the prevalence of psychosis, although arguably much more common than the diagnosis of schizophrenia, is not approximating 100%, given the fact that genetic risk is distributed in the population and the population rate of exposure to environmental risk factors is also high.

Clinical implications

Treatment. The clinical implications of a relationship between childhood trauma and psychosis seem obvious. A range of effective, evidence-based psychosocial treatments for psychosis are available (155, 156, 190–199). They should be made available to everyone diagnosed psychotic, including, perhaps especially, those who have been traumatized as children (141). It is possible, as recently found in a large, multi-centre study of chronic depression (200), that psychological approaches are more effective than medication for psychotic people who suffered childhood trauma.

Some researchers are beginning to tease out what interventions may be particularly helpful for people who experience psychosis and have suffered child abuse (33, 201–203), including, for some, a group approach (123, 204, 205). Not everyone will need or want psychotherapy. For some, simply making a connection between their life history and their previously incomprehensible symptoms may have a significant therapeutic effect (206).

Assessment. First, however, we must move beyond ideologically based presuppositions and learn to ask people with psychotic complaints about trauma. The majority of sexual or physical abuse cases remain unidentified by mental health staff, in

both child and adult psychiatric services (18, 51, 53, 56, 57, 63, 77, 85, 133, 207–209). The identification of neglect and psychological abuse appears to be no better (210). CSA in particular is frequently kept secret, from everyone, for years (99). A New Zealand study found that the average time it took women to tell another human being about having been sexually abused as a child was 16 years (133). Patients are unlikely to spontaneously disclose abuse, particularly sexual abuse, to mental health staff (1, 51–53). We therefore have an obligation to ask.

The few studies that have investigated the response of mental health services to disclosures of abuse suggest that our response is frequently inadequate, specifically in terms of offering information, support or treatment, or considering reporting to legal or protection agencies (53, 72, 211, 212). People diagnosed psychotic or schizophrenic are particularly unlikely to receive a therapeutic response (53, 211, 212) and are particularly unlikely to be asked about abuse in the first place, especially by psychiatrists or staff with strong biogenetic aetiological beliefs (53, 207, 209).

All mental health services should establish policy guidelines for how and when to ask about trauma in general, including child abuse. In the absence of staff training, however, such policies seem to be relatively ineffective (51, 53, 209, 213). One such training programme has been running for several years in New Zealand (53). A recent evaluation has demonstrated its effectiveness, except in one domain: its ability to attract psychiatrists to the training sessions (213).

Prevention

Finally, if we look beyond our research and our clinical practice there are some important broader implications. How much money would be saved by providing timely, appropriate help for child abuse survivors (psychotic or otherwise) in the mental health system? (214) Recent reviewers have argued that schizophrenia, being as socially based as other mental health problems, is just as preventable (215). A recent study has shown that an environmental enrichment programme at age 3–5 years reduced ‘schizotypal personality’ scores in early adulthood (216).

Postscript

Just prior to going to press the largest study to date was published (217). This retrospective survey of 17337 Californians found, for both men and women, that CPA, CSA, childhood emotional

abuse, and several other adverse childhood events, all significantly increased the risk of hallucinations. The study also confirmed the dose–response findings reported above. After controlling for substance abuse, gender, race and education, those who had experienced the greatest number of types of adverse events in childhood were 4.7 times more likely to have experienced hallucinations. We concur with the researchers’ conclusions that ‘our data and those of others suggest that a history of child maltreatment should be obtained by health care providers with patients who have a current or past history of hallucinations. This is important because the effects of childhood and adulthood trauma are treatable and preventable. ...Finding such a trauma-symptom or trauma-illness association may be an important factor in making a diagnosis, treatment plan, and referral and may help patients by lessening their fear, guilt or shame about their possibly having a mental illness’ (p. 806).

References

1. READ J. Child abuse and psychosis: a literature review and implications for professional practice. *Prof Psychol Res Pr* 1997;**28**:448–456.
2. SPATARO J, MULLEN P, BURGESS P, WELLS D, MOSS A. Impact of child sexual abuse on mental health: prospective study in males and females. *Br J Psychiatry* 2004;**184**:416–421.
3. MULLEN P, MARTIN J, ANDERSON J, ROMANS S, HERBISON G. Childhood sexual abuse and mental health in adult life. *Br J Psychiatry* 1993;**163**:721–732.
4. BUSHNELL J, WELLS J, OAKLEY-BROWNE M. Long-term effects of intrafamilial sexual abuse in childhood. *Acta Psychiatr Scand* 1992;**85**:136–142.
5. CHRISTOFFERSEN M, POULSEN H, NIELSEN A. Attempted suicide among young people: risk factors in a prospective register based study of Danish people born in 1966. *Acta Psychiatr Scand* 2003;**108**:350–358.
6. FERGUSSON D, HORWOOD L, LYNKEY M. Childhood sexual abuse, and psychiatric disorder in young adulthood. *J Am Acad Child Adolesc Psychiatry* 1996;**35**:1365–1374.
7. GRILO C, MASHEB R. Childhood maltreatment and personality disorders in adult inpatients with binge eating disorder. *Acta Psychiatr Scand* 2002;**106**:183–188.
8. HYUN M, FRIEDMAN S, DUNNER D. Relationship of childhood physical and sexual abuse to adult bipolar disorder. *Bipolar Dis* 2000;**2**:131–135.
9. KENDLER K, BULIK S, SILBERG J, HETTEMER J, MYERS J, PRESCOTT C. Childhood sexual abuse and adult psychiatric and substance use disorders in women. *Arch Gen Psychiatry* 2000;**57**:953–959.
10. LANGE A, KOOIMAN K, HUBERTS L, VAN OOSTENDORP E. Childhood unwanted sexual events and degree of psychopathology of psychiatric patients. *Acta Psychiatr Scand* 1995;**92**:441–446.
11. BECK J, VAN DER KOLK B. Reports of childhood incest and current behavior of chronically hospitalized psychotic women. *Am J Psychiatry* 1987;**144**:1474–1476.
12. BRIERE J, WOO R, MCRAE B, FOLTZ J, SITZMAN R. Lifetime victimization history, demographics, and clinical status in

- female psychiatric emergency room patients. *J Nerv Ment Dis* 1997;**85**:95–101.
13. GARNO J, GOLDBERG J, RAMIREZ P et al. Impact of childhood abuse on the clinical course of bipolar disorder. *Br J Psychiatry* 2005;**186**:121–125.
 14. GOFF D, BROTMAN A, KINDLON D, WAITES M, AMICO E. Self-reports of child abuse in chronically psychotic patients. *Psychiatry Res* 1991;**37**:73–80.
 15. LIPSCHITZ D, KAPLAN M, SORKENN J, FAEDDA G, CHORNEY P, ASNIS G. Prevalence and characteristics of physical and sexual abuse among psychiatric outpatients. *Psychiatr Serv* 1996;**47**:189–191.
 16. READ J. Child abuse and severity of disturbance among adult psychiatric inpatients. *Child Abuse Negl* 1998;**22**:359–368.
 17. READ J, AGAR K, BARKER-COLLO S, DAVIES E, MOSKOWITZ A. Assessing suicidality in adults: integrating childhood trauma as a major risk factor. *Prof Psychol Res Pr* 2001;**32**:367–372.
 18. ROSE S, PEABODY C, STRATIGEAS B. Undetected abuse among intensive case management clients. *Hosp Community Psychiatry* 1991;**42**:499–503.
 19. SANSONNET-HAYDEN H, HALEY G, MARRIAGE K, FINE S. Sexual abuse and psychopathology in hospitalized adolescents. *J Am Acad Child Adolesc Psychiatry* 1987;**26**:753–757.
 20. LEVERICH G, McELROY S, SUPPES T et al. Early physical and sexual abuse associated with an adverse course of bipolar illness. *Soc Biol Psychiatry* 2002;**51**:288–297.
 21. NOLEN W, LUCKENBAUGH D, ALTSHULER L et al. Correlates of 1-year prospective outcome in bipolar disorder. *Am J Psychiatry* 2004;**161**:1447–1454.
 22. READ J, GOODMAN L, MORRISON A, ROSS C, ADERHOLD V. Childhood trauma, loss and stress. In: READ J, MOSHER L, BENTALL R, eds. *Models of madness: psychological, social and biological approaches to schizophrenia*. Hove, UK: Brunner-Routledge, 2004:223–252.
 23. MORRISON A, FRAME L, LARKIN W. Relationships between trauma and psychosis: a review and integration. *Br J Clin Psychol* 2003;**42**:331–353.
 24. WINDLE M, SCHEIDT R, DOUGLAS M, MILLER D, GREGORY B. Physical and sexual abuse and associated mental disorders among alcoholic inpatients. *Am J Psychiatry* 1995;**152**:1322–1328.
 25. BROWN G, ANDERSON B. Psychiatric morbidity in adult inpatients with childhood histories of sexual and physical abuse. *Am J Psychiatry* 1991;**148**:55–61.
 26. KIRBY J, CHU J, DILL D. Correlates of dissociative symptomatology in patients with physical and sexual abuse histories. *Compr Psychiatry* 1993;**34**:258–263.
 27. ELLASON J, ROSS C, FUCHS D. Lifetime Axis I and II comorbidity and childhood trauma history in dissociative identity disorder. *Psychiatry* 1996;**59**:255–266.
 28. KAPUR S. Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *Am J Psychiatry* 2003;**160**:13–23.
 29. BUTLER R, MUESER K, SPROCK J, BRAFF D. Positive symptoms of psychosis in posttraumatic stress disorder. *Biol Psychiatry* 1996;**39**:839–844.
 30. SAUTTER F, BRAILEY K, UDDO M et al. PTSD and comorbid psychotic disorder. *J Trauma Stress* 1999;**12**:73–88.
 31. ROSS C. *Schizophrenia: innovations in diagnosis and treatment*. New York: Haworth Press, 2005.
 32. VAN PUTTEN T, EMORY W. Traumatic neuroses in Vietnam returnees: forgotten diagnosis? *Arch Gen Psychiatry* 1973;**29**:695–698.
 33. MUEZENMAIER K, CASTILLE D, SHELLEY A. Comorbid PTSD and schizophrenia. *Psychiatr Ann* 2005;**35**:51–56.
 34. HAMNER M, FRUEH B, ULMER H et al. Psychotic features in chronic posttraumatic stress disorder and schizophrenia: comparative severity. *J Nerv Ment Dis* 2000;**188**:217–219.
 35. ELHAI J, FRUEH C, GOLD P, HAMNER M, GOLD S. Posttraumatic stress, depression and dissociation as predictors of MMPI Scale 8 scores in combat veterans with PTSD. *J Trauma Dissociation* 2003;**4**:51–64.
 36. PIVAC N, KOZARIC-KOVACIC D, MUCK-SELER D. Olanzapine versus fluphenazine in an open trial in patients with psychotic combat-related posttraumatic stress disorder. *Psychopharmacology* 2004;**175**:451–456.
 37. ORUC L, BELL P. Multiple rape trauma followed by delusional parasitosis. *Schizophr Res* 1995;**16**:173–174.
 38. ELHAI J, FRUEH C, GOLD P, GOLD S, HAMNER M. Clinical presentations of posttraumatic stress disorder across trauma populations. *J Nerv Ment Dis* 2000;**188**:708–713.
 39. BEEBE G. Follow-up studies of World War II and Korean war prisoners. *Am J Epidemiol* 1975;**101**:400–402.
 40. KINZIE J, BOEHNLEIN J. Posttraumatic stress among Cambodian refugees. *J Trauma Stress* 1989;**2**:185–198.
 41. BHUI K, ABDISALAMA A, ABDI M et al. Traumatic events, migration characteristics and psychiatric symptoms among Somali refugees. *Soc Psychiatry Psychiatr Epidemiol* 2003;**38**:35–43.
 42. CASCARDI M, MUESER K, DEGIRALOMO J, MURRIN M. Physical aggression against psychiatric inpatients by family members and partners. *Psychiatr Serv* 1996;**47**:531–533.
 43. DAVIDSON J, MCNAMARA L. Systems that silence. In: BRECKENRIDGE J, LAING L, eds. *Challenging silence: Innovative responses to sexual and domestic violence*. Sydney: Allen and Unwin, 1999:35–48.
 44. RITSHER J, COURSEY R, FARRELL E. A survey on issues in the lives of women with severe mental illness. *Psychiatr Serv* 1997;**48**:1273–1282.
 45. CLOITRE M, TARDIFF K, MARZUK P, LEON A, PORTERA L. Childhood abuse and subsequent sexual assault among female inpatients. *J Trauma Stress* 1996;**9**:473–482.
 46. MEYER I, MUEZENMAIER K, CANCIENNE J, STRUENING E. Reliability and validity of a measure of sexual and physical abuse histories among women with serious mental illness. *Child Abuse Negl* 1996;**2**:213–219.
 47. GOODMAN L, THOMPSON K, WEINFURT K. Reliability of violent victimization and PTSD among men and women with serious mental illness. *J Trauma Stress* 1999;**12**:587–599.
 48. HERMAN J, SCHATZOW E. Recovery and verification of childhood sexual trauma. *Psychoanal Psychol* 1987;**4**:1–14.
 49. READ J, AGAR K, ARGYLE N, ADERHOLD V. Sexual and physical assault during childhood and adulthood as predictors of hallucinations, delusions and thought disorder. *Psychol Psychother Theory Res Pract* 2003;**76**:1–22.
 50. DARVES-BORNOZ J-M, LEMPERIERE T, DEGIOVANNI A, GAILLARD P. Sexual victimization in women with schizophrenia and bipolar disorder. *Soc Psychiatry Psychiatr Epidemiol* 1995;**30**:78–84.
 51. BRIERE J, ZAIDI L. Sexual abuse histories and sequelae in female psychiatric emergency room patients. *Am J Psychiatry* 1989;**146**:1602–1606.
 52. DILL D, CHU J, GROB M, EISEN S. The reliability of abuse history reports. *Compr Psychiatry* 1991;**32**:166–169.
 53. READ J. Breaking the silence: learning how to ask about trauma. In: LARKIN W, MORRISON A, eds. *Understanding trauma and psychosis*. Hove, UK: Brunner-Routledge, 2005 (in press).
 54. FRIEDMAN S, HARRISON G. Sexual histories, attitudes and behavior of schizophrenic and normal women. *Arch Sex Behav* 1984;**13**:555–567.

55. BRYER J, NELSON B, MILLER J, KROL P. Childhood sexual and physical abuse as factors in psychiatric illness. *Am J Psychiatry* 1987;**44**:1426–1430.
56. JACOBSON A, RICHARDSON B. Assault experiences of 100 psychiatric inpatients. *Am J Psychiatry* 1987;**144**:908–913.
57. CRAINE L, HENSON C, COLLIVER J, MACLEAN D. Prevalence of a history of sexual abuse among female psychiatric patients in a state hospital system. *Hosp Community Psychiatry* 1988;**39**:300–304.
58. GOODWIN J, ATTIAS R, MCCARTY T, CHANDLER S, ROMANIK R. Reporting by adult psychiatric patients of childhood sexual abuse. *Am J Psychiatry* 1988;**145**:1183–1184.
59. HART L, MADER L, GRIFFITH K, DEMENDONCA M. Effects of sexual and physical abuse. *Child Psychiatry Hum Dev* 1989;**20**:49–57.
60. CHU J, DILL D. Dissociative symptoms in relation to childhood physical and sexual abuse. *Am J Psychiatry* 1990;**147**:887–892.
61. JACOBSON A, HERALD C. The relevance of childhood sexual abuse to adult psychiatric inpatient care. *Hosp Community Psychiatry* 1990;**41**:154–158.
62. SHEARER S, PETERS C, QUAYTMAN M, OGDEN R. Frequency and correlates of childhood sexual and physical abuse histories in adult female borderline inpatients. *Am J Psychiatry* 1990;**147**:214–216.
63. LANKTREE C, BRIERE J, ZAIDI L. Incidence and impact of sexual abuse in a child outpatient sample: the role of direct inquiry. *Child Abuse Negl* 1991;**15**:447–453.
64. MARGO G, MCLEES E. Further evidence of the significance of a childhood abuse history in psychiatric inpatients. *Compr Psychiatry* 1991;**32**:62–66.
65. CARLIN A, WARD N. Subtypes of psychiatric inpatient women who have been sexually assaulted. *J Nerv Ment Dis* 1992;**180**:392–397.
66. LOBEL C. Relationship between childhood sexual abuse and borderline personality disorder in women psychiatric inpatients. *J Child Sex Abuse* 1992;**1**:63–80.
67. ITO Y, TEACHER M, GLOD C, HARPER D, MAGNUS E, GELBARD H. Increased prevalence of electrophysiological abnormalities in children with psychological, physical, and sexual abuse. *J Neuropsychiatry* 1993;**5**:401–408.
68. MUENZENMAIER K, MEYER I, STRUENING E, FERBER J. Childhood abuse and neglect among women outpatients with chronic mental illness. *Hosp Community Psychiatry* 1993;**44**:666–670.
69. GREENFIELD S, STRAKOWSKI S, TOHEN M et al. Childhood abuse in first-episode psychosis. *Br J Psychiatry* 1994;**164**:831–834.
70. ROSS C, ANDERSON G, CLARK P. Childhood abuse and positive symptoms of schizophrenia. *Hosp Community Psychiatry* 1994;**45**:489–491.
71. SWETT C, HALPERT M. High rates of alcohol problems and history of physical and sexual abuse among women inpatients. *Am J Drug Alcohol Abuse* 1994;**20**:263–273.
72. TROJAN O. Sexual experiences of psychotic patients. *Int J Adolesc Med Health* 1994;**7**:209–217.
73. GOODMAN L, DUTTON M, HARRIS M. Physical and sexual assault prevalence among episodically homeless women with serious mental illness. *Am J Orthopsychiatry* 1995;**65**:468–478.
74. COHEN Y, SPIRITO A, STERLING C et al. Physical and sexual abuse and their relation to psychiatric disorder and suicidal behavior among adolescents who are psychiatrically hospitalized. *J Child Psychol Psychiatry* 1996;**37**:989–993.
75. DAVIES-NETZLEY S, HURLBURT M, HOUGH R. Childhood abuse as a precursor to homelessness for women with severe mental illness. *Violence Victims* 1996;**11**:129–142.
76. MILLER L, FINNERTY M. Sexuality, pregnancy, and child-rearing among women with schizophrenia-spectrum disorders. *Psychiatr Serv* 1996;**47**:502–506.
77. WURR C, PARTRIDGE I. The prevalence of a history of childhood sexual abuse in an acute adult inpatient population. *Child Abuse Negl* 1996;**20**:867–872.
78. MUESER K, TRUMBETTA S, ROSENBERG S et al. Trauma and PTSD in severe mental illness. *J Consult Clin Psychol* 1998;**66**:493–499.
79. LIPSCHITZ D, WINEGAR R, NICOLAU A, HARTNICK E, WOLFSON M, SOUTHWICK S. Perceived abuse and neglect as risk factors for suicidal behavior in adolescent inpatients. *J Nerv Ment Dis* 1999;**187**:32–39.
80. LIPSCHITZ D, WINEGAR R, HARTNICK E, FOOTE B, SOUTHWICK S. PTSD in hospitalized adolescents: diagnostic comorbidity and clinical correlates. *J Am Acad Child Adolesc Psychiatry* 1999;**38**:385–392.
81. LIPSCHITZ D, GRILO C, FEHON D, MCGLASHAN T, SOUTHWICK S. Gender differences in the associations between posttraumatic stress symptoms and problematic substance use in psychiatric inpatient adolescents. *J Nerv Ment Dis* 2000;**188**:349–356.
82. FEHON D, GRILO C, LIPSCHITZ D. Gender differences in violence exposure and violence risk among adolescent inpatients. *J Nerv Ment Dis* 2001;**189**:532–540.
83. GOODMAN L, SALYERS M, MUESER K et al. Recent victimization in women and men with severe mental illness. *J Trauma Stress* 2001;**14**:615–632.
84. FRIEDMAN S, SMITH L, FOGEL D et al. The incidence and influence of early traumatic life events in patients with panic disorder: a comparison with other psychiatric outpatients. *Anxiety Dis* 2002;**16**:259–272.
85. CHANDRA P, DEEPTHIVARMA S, CAREY M, CAREY K, SHALINI-NANT M. A cry from the wilderness: women with severe mental illness in India reveal their experiences with sexual coercion. *Psychiatry* 2003;**66**:323–334.
86. HOLOWKA D, KING S, SAHEB D, PUKALL M, BRUNET A. Childhood abuse and dissociative symptoms in adult schizophrenia. *Schizophr Res* 2003;**60**:87–90.
87. OFFEN L, WALLER G, THOMAS G. Is reported childhood sexual abuse associated with the psychopathological characteristics of patients who experience auditory hallucinations? *Child Abuse Negl* 2003;**27**:919–927.
88. RESNICK S, BOND G, MUESER K. Trauma and PTSD in people with schizophrenia. *J Abnorm Psychol* 2003;**112**:415–423.
89. COMPTON M, FURMAN A, KASLOW N. Preliminary evidence of an association between childhood abuse and cannabis dependence among African American first-episode schizophrenia-spectrum disorder patients. *Drug Alcohol Depend* 2004;**76**:311–316.
90. SCHENKEL L, SPAULDING W, DILILLO D, SILVERSTEIN S. Histories of childhood maltreatment in schizophrenia: relationships with premorbid functioning, symptomatology, and cognitive deficits. *Schizophr Res* 2005;**76**:273–286.
91. SCHÄFER I, HARFST T, ADERHOLD V. et al. Childhood trauma and dissociation in female patients with schizophrenia spectrum disorders. *J Nerv Ment Dis* (in press).
92. KILCOMMONS A, MORRISON A. Relationships between trauma and psychosis: an exploration of cognitive factors. *Acta Psychiatr Scand* 2005;**112**:351–359.
93. BOWE S, MORRISON A, MORLEY S. The relationship between past childhood trauma and auditory hallucinations in individuals with psychosis. *Br J Clin Psychol* (in press).
94. METCALFE M, OPPENHEIMER R, DIGNON A, PALMER R. Childhood sexual experiences reported by male psychiatric patients. *Psychol Med* 1990;**20**:925–929.

95. PALMER R, BRAMBLE D, METCALFE M, OPPENHEIMER R, SMITH J. Childhood sexual experiences with adults: adult male psychiatric patients and general practice attenders. *Br J Psychiatry* 1994;**165**:675–679.
96. LYSAKER P, MEYER P, EVANS J, CLEMENTS C, MARKS K. Childhood sexual trauma and psychosocial functioning in adults with schizophrenia. *Psych Serv* 2001;**52**:1485–1488.
97. LYSAKER P, WICKETT A, LANCASTER R, DAVIS L. Neurocognitive deficits and history of child abuse in schizophrenia spectrum disorders. *Schizophr Res* 2004;**68**:87–94.
98. LYSAKER P, HUNTER N, STRASBURGER A, DAVIS L. Reported history of child sexual abuse in schizophrenia: associations with heightened symptom levels and poorer participation over four months in vocational rehabilitation. *J Nerv Ment Dis* (in press).
99. FERGUSSON D, HORWOOD L, WOODWARD L. The stability of child abuse reports: a longitudinal study of the reporting behaviour of young adults. *Psychol Med* 2000;**30**:529–544.
100. SPATARO J, MOSS S, WELLS D. Child sexual abuse: a reality for both sexes. *Aust Psychol* 2001;**36**:177–183.
101. ROBINS L. *Deviant children growing up*. London: Williams Wilkins, 1996.
102. CANNON M, CASPI A, MOFFITT T et al. Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder. *Arch Gen Psychiatry* 2002;**59**:449–456.
103. NERIA Y, BROMET E, SIEVERS S, LAVELLE J, FOCHTMAN L. Trauma exposure and PTSD in psychosis. *J Consult Clin Psychol* 2002;**70**:246–251.
104. KESSLER R, SONNEGA A, BROMET E, HUGHES M, NELSON C. PTSD in the National Comorbidity Survey. *Arch Gen Psychiatry* 1995;**52**:1048–1060.
105. LIVINGSTON R. Sexually and physically abused children. *J Am Acad Child Adolesc Psychiatry* 1987;**26**:413–415.
106. BENTALL R. *Madness explained: psychosis and human nature*. London: Penguin, 2003.
107. READ J. Does ‘schizophrenia’ exist? Reliability and validity. In: READ J, MOSHER L, BENTALL R, eds. *Models of madness: psychological, social and biological approaches to schizophrenia*. Hove, UK: Brunner-Routledge, 2004: 43–56.
108. JANSSEN I, KRABBENDAM L, BAK M et al. Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatr Scand* 2004;**109**:38–45.
109. ROSS C, JOSHI S. Schneiderian symptoms and childhood trauma in the general population. *Compr Psychiatry* 1992;**33**:269–273.
110. BERENBAUM H. Peculiarity and reported child maltreatment. *Psychiatry* 1999;**62**:21–35.
111. STARTUP M. Schizotypy, dissociative experiences and childhood abuse. *Br J Clin Psychol* 1999;**38**:333–344.
112. BERENBAUM H, VALERA E, KERNS J. Psychological trauma and schizotypal symptoms. *Schizophr Bull* 2003;**29**:143–152.
113. READ J, ARGYLE N. Hallucinations, delusions and thought disorders among adult psychiatric inpatients with a history of child abuse. *Psychiatr Serv* 1999;**50**:1467–1472.
114. FAMULARO R, KINSCHERFF R, FENTON T. Psychiatric diagnoses of maltreated children: preliminary findings. *J Am Acad Child Adolesc Psychiatry* 1992;**31**:863–867.
115. MUNDY P, ROBERTSON M, ROBERTSON J, GREENBLATT M. The prevalence of psychotic symptoms in homeless adolescents. *J Am Acad Child Adolesc Psychiatry* 1990;**29**:724–731.
116. GOFF D, BROTMAN A, KINDLON D, WAITES M, AMICO E. The delusion of possession in chronically psychotic patients. *J Nerv Ment Dis* 1991;**179**:567–571.
117. HONIG A, ROMME M, ENSINK B, ESCHER S, PENNING S, DEVRIES M. Auditory hallucinations: a comparison between patients and nonpatients. *J Nerv Ment Dis* 1998;**186**:646–651.
118. HAMMERSLEY P, DIAS A, TODD G, BOWEN-JONES K, REILLY B, BENTALL R. Childhood traumas and hallucinations in bipolar affective disorder. *Br J Psychiatry* 2003;**182**:543–547.
119. MORRISON A, PETERSEN T. Trauma and metacognition as predictors of predisposition to hallucinations. *Behav Cogn Psychother* 2003;**31**:235–246.
120. ENSINK B. *Confusing realities*. Amsterdam: Vu University, 1992.
121. BEBBINGTON P, BHUGRA D, BRUGHA T et al. Psychosis, victimization and childhood disadvantage: evidence from the second British National Survey of Psychiatric Morbidity. *Br J Psychiatry* 2004;**185**:220–226.
122. ELLENSON G. Detecting a history of incest: a predictive syndrome. *J Contemp Soc Work* 1985;**66**:525–532.
123. HEINS T, GRAY A, TENNANT M. Persisting hallucinations following childhood sexual abuse. *Aust N Z J Psychiatry* 1990;**24**:561–565.
124. OFFEN L, THOMAS G, WALLER G. Dissociation as a mediator in the relationship between recalled parenting and the clinical correlates of auditory hallucination. *Br J Clin Psychol* 2003;**42**:231–241.
125. BELL M, LYSAKER P, GOULET J, MILSTEIN R, LINDENMAYER J. Five component model of schizophrenia: factorial invariance of the Positive and Negative Syndrome scale. *Psychiatry Res* 1994;**52**:295–303.
126. KAHLBAUM K. *Catatonia*. Baltimore, MD: Johns Hopkins University [1874], 1973.
127. MOSKOWITZ A. ‘Scared stiff’: catatonia as an evolutionary-based fear response. *Psychol Rev* 2004;**111**:984–1002.
128. HARRISON C, FOWLER D. Negative symptoms, trauma, and autobiographical memory. *J Nerv Ment Dis* 2004;**192**:745–753.
129. GENRON J, BELLACK A, TENHULA W et al. Preliminary reliability and validity of the Clinician-Administered PTSD Scale for Schizophrenia. *J Consult Clin Psychol* 2004;**72**:121–125.
130. ROMME M, ESCHER S. Trauma and hearing voices. In: LARKIN W, MORRISON A, eds. *Understanding trauma and psychosis*. Hove, UK: Brunner-Routledge (in press).
131. PETTIGREW J, BURCHAM J. Effects of childhood sexual abuse in adult female psychiatric patients. *Aust N Z J Psychiatry* 1997;**31**:208–213.
132. READ J, HAMMERSLEY P. Child sexual abuse and schizophrenia. Correspondence. *Br J Psychiatry* 2005;**186**:76.
133. READ J, MCGREGOR K, COGGAN C, THOMAS D. Mental health services and sexual abuse: the need for staff training. *J Trauma Dissociation* (in press).
134. BARKER-COLLO S, READ J. Models of response to childhood sexual abuse: their implications for treatment. *Trauma Violence Abuse* 2003;**4**:95–111.
135. SPAUWEN J, KRABBENDAM L, LIEB R, WITTCHEN H, VAN OS J. Impact of psychological trauma on the trajectory of psychosis liability. *Br J Psychiatry* (in press).
136. SEEDAT S, STEIN M, OOSTHUIZEN P, EMSLEY R, STEIN D. Linking PTSD and psychosis: a look at epidemiology, phenomenology, and treatment. *J Nerv Ment Dis* 2003;**191**:675–681.
137. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders, 4th edn, Text revision*. Washington, DC: APA, 2000.
138. FRAME L, MORRISON A. Causes of PTSD in psychosis. *Arch Gen Psychiatry* 2001;**58**:305–306.

139. MCGORRY P, CHANEN A, MCCARTHY E, VAN RIEL R, MCKENZIE D, SINGH B. PTSD following recent-onset psychosis: an unrecognized postpsychotic syndrome. *J Nerv Ment Dis* 1991;**179**:253–258.
140. SHAW K, MCFARLANE A, BOOKLESS C, AIR T. The aetiology of postpsychotic PTSD following a psychotic episode. *J Trauma Stress* 2002;**15**:39–47.
141. READ J, ROSS C. Psychological trauma and psychosis: another reason why people diagnosed schizophrenic must be offered psychological therapies. *J Am Acad Psychoanal Dynamic Psychiatry* 2003;**31**:247–267.
142. NURCOMBE B, MITCHELL W, BEGRUP R et al. Dissociative hallucinosis and allied conditions. In: VOLKMAR F, ed. *Psychoses and pervasive developmental disorders in childhood and adolescence*. Washington, DC: American Psychiatric Press, 1996:107–128.
143. TUTKUN H, SAR V, YARGIC I, OZPULAT T, YANIK M, KIZILTAN E. Frequency of dissociative disorders among psychiatric inpatients in a Turkish university clinic. *Am J Psychiatry* 1998;**155**:800–805.
144. ELLASON J, ROSS C. Positive and negative symptoms in dissociative disorder and schizophrenia. *J Ment Nerv Dis* 1995;**83**:236–241.
145. YEE L, KORNER A, MCSWIGGAN S, MEARES R, STEVENSON J. Persistent hallucinations in borderline personality disorder. *Compr Psychiatry* 2005;**46**:147–154.
146. MORRISON A. The interpretation of intrusions in psychosis: an integrative cognitive approach to hallucinations and delusions. *Behav Cogn Psychother* 2001;**29**:257–276.
147. BENTALL R, CORCORAN R, HOWARD R, BLACKWOOD R, KINDERMAN P. Persecutory delusions: a review and theoretical integration. *Clin Psychol Rev* 2001;**22**:1–50.
148. GARETY P, KUIPERS E, FOWLER D, FREEMAN D, BEBBINGTON P. A cognitive model of the positive symptoms of psychosis. *Psychol Med* 2001;**31**:189–195.
149. MORRISON A, GUMLEY A, SCHWANNAUER M et al. The beliefs about paranoia scale: preliminary validation of a meta-cognitive approach to conceptualising paranoia. *Behav Cogn Psychother* 2005;**33**:153–164.
150. JOHNS L, VAN OS J. The continuity of psychotic experiences in the general population. *Clin Psychol Rev* 2001;**21**:1125–1141.
151. MILLHAM A, EASTON S. Prevalence of auditory hallucinations in nurses in mental health. *J Psychiatr Ment Health Nurs* 1998;**5**:95–99.
152. CHADWICK P, BIRCHWOOD M. The omnipotence of voices: a cognitive approach to auditory hallucinations. *Br J Psychiatry* 1994;**164**:190–201.
153. MORRISON A, NOTHARD S, BOWE S, WELLS A. Interpretations of voices in patients with hallucinations and non-patient controls. *Behav Res Ther* 2004;**42**:1315–1323.
154. BAK M, KRABBENDAM L, JANSSEN I, DE GRAAF R, VOLLEBERGH W, VAN OS J. Does early trauma increase the risk for psychotic disorders by impacting on emotional response and perception of control associated with psychotic experience? *Acta Psychiatr Scand* 2005;**112**:360–366.
155. MORRISON A. Cognitive therapy for people with psychosis. In: READ J, MOSHER L, BENTALL R, eds. *Models of madness: psychological, social and biological approaches to schizophrenia*. Hove, UK: Brunner-Routledge, 2004:291–306.
156. MORRISON A. *Cognitive therapy for psychosis: a formulation-based approach*. Hove, UK: Brunner-Routledge, 2004.
157. HENQUET C, KRABBENDAM L, DAUTZENBERG J, JOLLES J, MERCKELBACK H. Confusing thoughts and speech: source monitoring and psychosis. *Psychiatry Res* 2005;**133**:57–63.
158. VAN DER KOLK B, FISLER R. Dissociation and the fragmentary nature of traumatic memories. *J Trauma Stress* 1995;**8**:505–525.
159. NADEL L, JACOBS W. The role of the hippocampus in PTSD, panic and phobia. In: KATO N, ed. *The hippocampus: functions and clinical relevance*. Amsterdam: Elsevier, 1996:455–463.
160. STEEL C, FOWLER D, HOLMES E. Trauma related intrusions and psychosis: an information processing account. *Behav Cogn Psychother* 2005;**33**:139–152.
161. HOLMES E, STEEL C. Schizotypy: a vulnerability factor for traumatic intrusions. *J Nerv Ment Dis* 2003;**192**:28–34.
162. MOSKOWITZ A, READ J, FARRELLY S, RUDEGEAIR T, WILLIAMS O. Are psychotic symptoms traumatic in origin and dissociative in kind? In: DELL P, O'NEIL J, eds. *Dissociation and the dissociative disorders: DSM-IV and beyond*. New York: International Society for the Study of Dissociation (in press).
163. ROSS C, MILLER S, REAGOR P, BJORNSEN L. Schneiderian symptoms in multiple personality disorder and schizophrenia. *Compr Psychiatry* 1990;**31**:111–118.
164. SHACK A, AVERILL P, KOPECKY C, KRAJEWSKI K, GUMMATTIRA P. Prior history of physical and sexual abuse among the psychiatric inpatient population: a comparison of males and females. *Psychiatr Q* 2004;**75**:343–359.
165. WALKER E, DIFORIO D. Schizophrenia: a neural diathesis-stress model. *Psychol Rev* 1997;**104**:667–685.
166. DEPUE R, COLLINS P. Neurobiology of the structure of personality: dopamine, facilitation of incentive motivation, and extraversion. *Behav Brain Sci*, 1999;**22**:491–569.
167. READ J, PERRY B, MOSKOWITZ A, CONNOLLY J. The contribution of early traumatic events to schizophrenia in some patients: a Traumagenic Neurodevelopmental model. *Psychiatry* 2001;**64**:319–345.
168. HEIM C, NEWPORT D, GRAHAM Y et al. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *J Am Med Assoc* 2000;**284**:592–597.
169. NEMEROFF C. Neurobiological consequences of childhood trauma. *J Clin Psychiatry* 2004;**65**(suppl. 1):18–28.
170. PERRY B. Memories of fear. In: GOODWIN J, ATTIAS R, eds. *Splintered reflections*. Washington, DC: Basic Books, 1999:9–38.
171. CICHETTI D, WALKER E. Stress and development: biological and psychological consequences. *Dev Psychopathol* 2001;**13**:413–418.
172. AGEMAN I, ANDERSEN H, JORGENSEN M. Post-traumatic stress disorder: a review of psychobiology and pharmacotherapy. *Acta Psychiatr Scand* 2001;**104**:411–422.
173. BREMNER J. *Does stress damage the brain?* London: Norton, 2002.
174. HUIZNICK A, MULDER E, BUTTELAAR J. Prenatal stress and risk for psychopathology. *Psychol Bull* 2004;**130**:115–142.
175. TIENARI P, WYNNE L, SORRI A et al. Genotype-environment interaction in schizophrenia-spectrum disorder: long term follow-up study of Finnish adoptees. *Br J Psychiatry* 2004;**184**:216–222.
176. SODHI M, SANDERS-BUSH E. Serotonin and brain development. *Int Rev Neurobiol* 2004;**59**:111–126.
177. TEICHER M, ANDERSEN S, POLCARI A, ANDERSON C, NAVALTA C, KIM D. The neurobiological consequences of early stress and childhood maltreatment. *Neurosci Biobehav Rev* 2003;**27**:33–44.
178. PHILLIPS W, SILVERSTEIN S. Convergence of biological and psychological perspectives on cognitive coordination in schizophrenia. *Behav Brain Sci* 2003;**26**:65–138.

179. MYIN-GERMEYS L, KRABBENDAM P, DELESPAUL P, VAN OS J. Do life events have their effect on psychosis by influencing the emotional reactivity to daily life stress? *Psychol Med* 2003;**33**:327–333.
180. MYIN-GERMEYS L, VAN OS J, SCHWARTZ J, STONE A, DELESPAUL P. Emotional reactivity to daily life stress in psychosis. *Arch Gen Psychiatry* 2001;**58**:1137–1144.
181. FUCHS T. Neurobiology and psychotherapy: an emerging dialogue. *Curr Opin Psychiatry* 2004;**17**:479–485.
182. RIECHLER-ROSSLER A, HAFNER H. Gender aspects in schizophrenia: bridging the border between social and biological psychiatry. *Acta Psychiatr Scand* 2000;**102**(suppl. 407):58–62.
183. ROSE S. The 21st century brain: exploring, mending and manipulating the mind. London: Jonathan Cape, 2005.
184. STRAUSS J. The interactive developmental model revisited. *Acta Psychiatr Scand* 2000;**102**(suppl. 407):19–25.
185. SHAM P. Statistics in human genetics. London: Arnold, 1998.
186. VAN OS J, SHAM P. Gene-environment interactions. In: MURRAY R, JONES P, SUSSER E, VAN OS J, CANNON M, eds. The epidemiology of schizophrenia. Cambridge, UK: Cambridge University Press, 2003:235–253.
187. VAN OS J, MARCELIS M. The ecogenetics of schizophrenia: a review. *Schizophr Res* 1998;**322**:127–135.
188. VAN OS J, PEDERSEN C, MORTENSEN P. Confirmation of synergy between urbanicity and familial liability in the causation of psychosis. *Am J Psychiatry* 2004;**161**:2312–2314.
189. HENQUET C, KRABBENDAM L, SPAUWEN J et al. Prospective cohort study of cannabis use, predisposition for psychosis, and psychotic symptoms in young people. *BMJ* 2005;**330**(7481):11.
190. ADERHOLD V, GOTTWALZ E. Family therapy and schizophrenia: replacing ideology with openness. In: READ J, MOSHER L, BENTALL R, eds. Models of madness: psychological, social and biological approaches to schizophrenia. Hove, UK: Brunner-Routledge, 2004:335–348.
191. BRENNER H. Psychological therapy in schizophrenia: what is the evidence? *Acta Psychiatr Scand* 2000;**102**(suppl. 407):74–77.
192. GOTTDIENER W. Psychodynamic therapy for schizophrenia: empirical support. In: READ J, MOSHER L, BENTALL R, eds. Models of madness: psychological, social and biological approaches to schizophrenia. Hove, UK: Brunner-Routledge, 2004:307–318.
193. JOHANNESSEN J-O. The development of early intervention services. In: READ J, MOSHER L, BENTALL R, eds. Models of madness: psychological, social and biological approaches to schizophrenia. Hove, UK: Brunner-Routledge, 2004:319–334.
194. KINGDON D, TURKINGTON D. Cognitive-behavioural therapy of schizophrenia. London: Guilford Press, 2004.
195. LEFF J. Family work for schizophrenia: practical application. *Acta Psychiatr Scand* 2000;**102**(suppl. 407):78–82.
196. MARTINDALE B, BATEMAN A, CROWE M, MARGISON F, eds. Psychosis: psychological approaches and their effectiveness. London: Gaskell, 2000.
197. MOSHER L, HENDRIX V, FORT D. Soteria: through madness to deliverance. Philadelphia, PA: Xlibris, 2005.
198. SILVER A, KOEHLER B, KARON B. Psychodynamic psychotherapy of schizophrenia: its history and development. In: READ J, MOSHER L, BENTALL R, eds. Models of madness: psychological, social and biological approaches to schizophrenia. Hove, UK: Brunner-Routledge, 2004:209–222.
199. TURKINGTON D. Effectiveness of brief cognitive behavioural therapy intervention in the treatment of schizophrenia. *Br J Psychiatry* 2002;**180**:523–531.
200. NEMEROFF C, HEIM C, THASE M et al. Differential responses to psychotherapy versus pharmacotherapy in patients with chronic forms of major depression and childhood trauma. *Proc Natl Acad Sci U S A* 2003;**100**:14293–14296.
201. GOODMAN L, ROSENBERG S, MUESER K, DRAKE R. Physical and sexual assault history in women with serious mental illness. *Schizophr Bull* 1997;**23**:685–686.
202. HARRIS M, LANDIS C. Sexual abuse in the lives of women diagnosed with serious mental illness. London: Harwood, 1997.
203. ROSENBERG S, MUESER K, FRIEDMAN M et al. Developing effective treatments for posttraumatic disorders among people with severe mental illness. *Psychiatr Serv* 2001;**52**:1453–1461.
204. SHELLEY A, BATTAGLIA J, LACEY J, ELLIS A, OPLER L. Symptom-specific group therapy for inpatients with schizophrenia. *Einstein Q* 2001;**18**:21–28.
205. HERDER D, REDNER L. The treatment of childhood sexual abuse in chronically mentally ill adults. *Health Soc Work* 1991;**16**:50–57.
206. FOWLER D. Psychological formulation of early episodes of psychosis. In: BIRCHWOOD M, FOWLER D, JACKSON C, eds. Early interventions in psychosis. London: Wiley, 2000:112–137.
207. YOUNG M, READ J, BARKER-COLLO S, HARRISON R. Evaluating and overcoming barriers to taking abuse histories. *Prof Psychol Res Pr* 2001;**32**:407–414.
208. LOTHIAN J, READ J. Asking about abuse during mental health assessments: clients' views and experiences. *N Z J Psychol* 2002;**31**:98–103.
209. READ J, FRASER A. Abuse histories of psychiatric inpatients: to ask or not to ask? *Psychiatr Serv* 1998;**49**:355–359.
210. THOMPSON A, KAPLAN C. Emotionally abused children presenting to child psychiatry clinics. *Child Abuse Negl* 1999;**23**:191–196.
211. AGAR K, READ J. What happens when people disclose sexual or physical abuse to staff at a community mental health centre? *Int J Ment Health Nurs* 2002;**11**:70–79.
212. READ J, FRASER A. Staff response to abuse histories of psychiatric inpatients. *Aust N Z J Psychiatry* 1998;**32**:206–213.
213. CAVANAGH M-R, READ J, NEW B. Childhood abuse inquiry and response: a New Zealand training programme. *N Z J Psychol* 2004;**33**:137–144.
214. KNAPP M. Schizophrenia costs and treatment cost-effectiveness. *Acta Psychiatr Scand* 2000;**102**(suppl. 407):102–116.
215. DAVIES E, BURDETT J. Preventing 'schizophrenia': creating the conditions for saner societies. In: READ J, MOSHER L, BENTALL R, eds. Models of madness: psychological, social and biological approaches to schizophrenia. Hove, UK: Brunner-Routledge, 2004:271–282.
216. RAINE A, MELLINGEN K, LIU J, VENABLES P, MEDNICK S. Effects of environmental enrichment at ages 3–5 years on schizotypal personality and antisocial behavior at ages 17 and 23 years. *Am J Psychiatry* 2003;**160**:1627–1635.
217. WHITFIELD C, DUBE S, FELITTI V, ANDA R. Adverse childhood experiences and hallucinations. *Child Abuse Negl* 2005;**29**:797–810.
218. READ J, MOSHER L, BENTALL R. Models of madness: psychological, social and biological approaches to schizophrenia. Hove, UK: Brunner-Routledge, 2004.

Copyright of Acta Psychiatrica Scandinavica is the property of Blackwell Publishing Limited. The copyright in an individual article may be maintained by the author in certain cases. Content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.