

Child Maltreatment and Psychosis: A Return to a Genuinely Integrated Bio-Psycho-Social Model

*John Read*¹, *Paul Jay Fink*², *Thom Rudegeair*³,
*Vincent Felitti*⁴, *Charles L. Whitfield*⁵

Abstract

For several decades the conceptualization and treatment of mental health problems, including psychosis, have been dominated by a rather narrow focus on genes and brain functions. Psychosocial factors have been relegated to mere triggers or exacerbators of a supposed genetic predisposition. This paper advocates a return to the original stress-vulnerability model proposed by Zubin and Spring in 1977, in which heightened vulnerability to stress is not, as often wrongly assumed, necessarily genetically inherited, but can be acquired via adverse life events. There is now a large body of research demonstrating that child abuse and neglect are significant causal factors for psychosis. Ten out of eleven recent general population studies have found, even after controlling for other factors, including family history of psychosis, that child maltreatment is significantly related to psychosis. Eight of these studies tested for, and found, a dose-response. Interpreting these findings from psychological and biological perspectives generates a genuinely integrated bio-psycho-social approach as originally intended by Zubin and Spring. The routine taking of trauma histories from all users of mental health services is recommended, and a staff training program to facilitate this is described.

Key Words: Psychosocial, Schizophrenia, Psychosis, Mental Health Services, Violence

Understanding the Stress-Vulnerability Model

In 1977 Zubin and Spring published their landmark paper “Vulnerability: A New View of Schizophrenia” (1). Their stress-vulnerability model offered the possibility of a genuine

integration of psychosocial and biological research. Unfortunately, rather than embrace this opportunity, biological enthusiasts decreed that the heightened vulnerability to stress, which everyone agreed lay at the core of psychosis, must be biological in origin, usually genetic but with some attention to perinatal factors. Psychosocial factors were thereby relegated to mere triggers, or exacerbators, of a genetic predisposition without which schizophrenia could supposedly not develop. Zubin and Spring had clearly stated, however, that there is such a thing as “acquired vulnerability” and that this can be “due to the influence of trauma, specific diseases, perinatal complications, family experiences, adolescent peer interactions, and other life events that either enhance or inhibit the development of subsequent disorder” (p. 109).

This gross distortion of what had actually been said produced an illusion of integration. Meanwhile, however, asking

¹ Department of Psychology, University of Auckland, New Zealand

² Temple University School of Medicine, U.S.A.;
past President, American Psychiatric Association

³ Auckland District Health Board, New Zealand

⁴ Kaiser Permanente Medical Care Program, San Diego;
University of California, U.S.A.

⁵ Center for Disease Control, Atlanta, Georgia, U.S.A.

Address for correspondence: John Read, PhD, Associate Professor,
Department of Psychology, University of Auckland, Private Bag 92019,
Auckland 1, New Zealand

Phone: +64 9 373 7999; Fax: +64 9 373 7450;
E-mail: j.read@auckland.ac.nz

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Table 1 Proportions of "Schizophrenia" Research Investigating Child Abuse, Poverty, Biological Etiology and Drug Treatment						
	All	% of All Research	Before 2000	% of All Research	2000–Feb. 2008	% of All Research
Schizophrenia...and...	75,618		51,843		23,775	
Child abuse and neglect*	252	0.3%	142	0.3%	110	0.5%
Poverty [†]	620	0.8%	427	0.8%	193	0.8%
Biological causes [‡]	11,028	14.6%	6,516	12.6%	4,512	19.0%
Drug therapy [§]	14,487	19.1%	8,411	16.2%	6,076	25.6%
Ratio of biological causes to social causes	12.6:1		11.5:1		14.9:1	

Key words entered:
 * "child abuse," "child neglect," "emotional abuse," "sexual abuse," "physical abuse"
 † "poverty," "socioeconomic status"
 ‡ "neurotransmitters," "brain," "genetics"
 § "drug therapy," "antipsychotics"
 || Ratio of biological causes[‡] to (child abuse and neglect* plus poverty[†])

about childhoods and trying to understand the contextual meaning of symptoms continued to be outweighed by an approach that merely counted symptoms, diagnosed and medicated. Research is similarly unbalanced, especially in North America (2). PsycINFO searches, entering "schizophrenia," suggest that the imbalance is worsening since the turn of the century, with research into biological causes increasing its share of research into schizophrenia, outweighing social causes by about fifteen to one (Table 1). Furthermore, of 1,284 publications about childhood schizophrenia only five (0.4%) relate to child abuse and eight (0.7%) to poverty.

Why did so many professionals prefer the distorted version to the real one? Why have so many become uninterested in the childhoods of the people they are trying to help? One explanation is an understandable avoidance of the vicarious traumatization that can result from hearing the awful things that can happen in childhood (3). Another factor has been the influence that the pharmaceutical industry has developed over our training institutions, journals, professional organizations, research, conferences and public opinion (4-8). In 2005, the then president of the American Psychiatric Association warned:

If we are seen as mere pill pushers and employees of the pharmaceutical industry, our credibility as a profession is compromised. ... As we address these Big Pharma issues, we must examine the fact that as a profession, we have allowed the bio-psycho-social model to become the bio-bio-bio model (4, p. 3).

Nevertheless, an announcement for the first Congress of the Schizophrenia International Research Society in 2008,

funded by eight drug companies, listed over two hundred presentations, predominantly about genes, brains and drugs. One mentions stress. One mentions environment. None mention trauma, abuse, neglect, or poverty.

In the spirit of resurrecting the truly integrated approach that Zubin and Spring proposed, with proper emphasis on both the psychosocial and the biogenetic, this paper reviews the research on the relationship of childhood maltreatment to psychosis, and offers both psychological and biological perspectives on that relationship.

Childhood Trauma, Nonpsychotic Disorders and Severity

Child abuse has a causal role in most mental health problems, including depression, anxiety disorders, PTSD, eating disorders, substance abuse, personality disorders, and dissociative disorders (9-12). Psychiatric patients subjected to childhood sexual or physical abuse have earlier first admissions and longer and more frequent hospitalizations, spend longer time in seclusion, receive more medication, are more likely to self-mutilate, and have higher symptom severity (9, 11-15). Patients who were abused as children are far more likely to try to kill themselves (11, 12, 14, 16). Even *within* samples of schizophrenia, those abused as children are more likely to attempt suicide (17, 18).

The Psychosocial Causes of Psychosis

Poverty is even more strongly related to schizophrenia and psychosis than to other disorders (19). British children raised in economic deprivation are four times more likely

to develop nonschizophrenic disorders, but are eight times more likely to grow up to be schizophrenic. Even among those with no family history of psychosis, the deprived children are seven times more likely to develop schizophrenia, demonstrating that you do not need a genetic predisposition to develop schizophrenia (20). Similarly, the relationship between urban living and schizophrenia remains after controlling for family history of psychiatric disorder (21, 22).

Ethnicity is also a powerful predictor of schizophrenia. This has been demonstrated in Australia, Belgium, Denmark, Germany, Greenland, the Netherlands, New Zealand, Israel, Sweden, the United Kingdom and the United States (19). Although racist misdiagnoses are part of the explanation (2, 19), the factors that link ethnicity to schizophrenia are discrimination, financial disadvantage, unemployment and social isolation (19, 23, 24).

Loss of mother during childhood is significantly higher in schizophrenics than for other diagnoses (25). A study of people with a first episode of psychosis found that they were 2.3 times more likely than a control group to have had their mothers die before they were sixteen (26). Both these findings were significant after controlling for parental history of mental illness. In Finland (27) and the United States (28), people whose pregnancies had been unwanted are 2.5 and 1.75 times more likely to be diagnosed with schizophrenia.

Psychosis is also related to war traumas (29). This is the case not only for combat veterans, but also prisoners of war, war rape victims, refugees from the Pol Pot regime and from war in Somalia (30-33), people exposed to bombings and shootings in Northern Ireland (34), and Holocaust survivors (35).

While this review focuses on childhood, it must be acknowledged that most psychiatric patients suffer serious assaults as adults. One study found that in the year prior to hospitalization, 63% suffered violence by partners and 46% of those living at home were assaulted by family members (36). Sexual assaults as adults are experienced by the majority of female patients and about a quarter of male patients (32, 37-41). Assaults also occur in mental health settings (42, 43). A study of 409 female inpatients found that sexual assault was significantly related to schizophrenia, but not to mania, depression, substance abuse or borderline personality disorder (44). In another study of female patients, physical assault was significantly related only to psychosis (45).

Rates of Child Maltreatment in Inpatient and Psychotic Populations

Reliability of Self-Report

Concerns that disclosures of abuse by psychiatric patients may be unreliable are not evidence based. Reports of abuse

by patients, including those diagnosed psychotic, have repeatedly been found to be reliable (38, 46, 47). One study found 93% test-retest reliability for reports of sexual coercion by inpatients (48). Corroborating evidence for childhood sexual abuse disclosures by psychiatric patients was found in 74% (49) and 82% (50) of cases. One study found that: "The problem of incorrect allegations of sexual assaults was no different for schizophrenics than the general population" (51, p. 82).

Fifty-Nine Studies

Tables 2 and 3 report rates of childhood physical and sexual abuse (CPA, CSA) in fifty-two inpatient samples, plus seven outpatient samples in which at least 50% were diagnosed psychotic. See earlier versions of these tables (32, 52, 53), and independent reviews thereof (54-56), for the inclusion criteria and discussion of the methodological reasons for the variability in rates, including a range of instruments and variable cutoff ages for childhood. The weighted averages, accounting for variation in sample size, show that 64.5% of the women, and 55.5% of the men, had been subjected to either CSA or CPA. The combined rate is 60.2%. Earlier reviews have found that most of the CPA is inflicted by family members, and that the CSA is inflicted by family members (incest) for more than half of the sexually abused females and about a quarter of the sexually abused males (32, 53). This is not evidence of a causal relationship. It is evidence that somewhere between a half and two-thirds of inpatients and severely disturbed outpatients were sexually or physically abused as children, often, but not always, by family members.

Estimating the prevalence of childhood maltreatment by using only CSA and CPA leads to gross underestimation. Table 4 presents six studies of neglect and emotional abuse in people diagnosed with schizophrenia. A study of first episode schizophrenia-spectrum inpatients (57), which found CSA and CPA rates of 39% and 78%, respectively, also found the following: childhood emotional abuse 94%, childhood emotional neglect 89%, and childhood physical neglect 89%. A study of eighty-seven inpatients with chronic psychosis found that all had suffered either neglect or emotional abuse as children (58).

Relationship of Child Abuse to Research Measures and Clinical Diagnoses of Psychosis

A 2005 review (32) identified ten studies showing that people abused as children score higher than other people on the schizophrenia and paranoia scales of the Minnesota Multiphase Personality Inventory (MMPI) or the psychosis scale of the Symptom Checklist-90-Revised (SCL-90-R). In

Table 2 Percentages of Reported Child Abuse among Female Psychiatric Inpatients, or Outpatients of Whom at Least Half were Diagnosed Psychotic

Study Author(s), Year (Ref #)	Diagnosis	n	Child Sexual Abuse (CSA) (%)	Child Physical Abuse (CPA) (%)	Either CSA or CPA (%)	Both CSA and CPA (%)
Friedman & Harrison, 1984 (180)	sc	20	60			
Bryer et al., 1987 (181)		66	44	38	59	23
Jacobson & Richardson, 1987 (40)		50	22	44	56	10
Sansonnet-Hayden et al., 1987 (15)	ad	29	38	23		
Craine et al., 1988 (182)		105	51	35	61	26
Goodwin et al., 1988 (183)		40	50			
Hart et al., 1989 (184)	ad	16	75	69	81	62
Chu & Dill, 1990 (185)		98	36	51	63	23
Jacobson & Herald, 1990 (186)		50	54			
Shearer et al., 1990 (187)		40	40	25		
Goff et al., 1991 (188)	ps	21			48	
Lanktree et al., 1991 (189)	ch	18	50			
Margo & McLees, 1991 (190)		38	58	66	76	47
Rose et al., 1991 (191)	op	39	50	38		
Carlin & Ward, 1992 (192)		149	51			
Lobel, 1992 (193)		50	60			
Ito et al., 1993 (194)	ch	51			73	
Muenzenmaier et al., 1993 (195)	op	78	45	51	64	32
Mullen et al., 1993 (11)	ex	27	85			
Greenfield et al., 1994 (196)	ps	19	42	42	53	32
Ross et al., 1994 (80)	sc	25	32	32	48	16
Swett & Halpert, 1994 (197)		88	61	57	76	50
Trojan, 1994 (198)	ps	48	25			
Darves-Bornoz et al., 1995 (51)	ps	89	34			
Goodman et al., 1995 (37)	op	99	65	87	92	60
Cohen et al., 1996 (199)	ad	73	51	52	68	34
Davies-Netzley et al., 1996 (200)	op	120	56	59	77	38
Miller & Finnerty, 1996 (201)	sc	44	36			
Wurr & Partridge, 1996 (202)		63	52			
Briere et al., 1997 (45)	op	93	53	42		
Mueser et al., 1998 (41)	op	153	52	33		
Goodman et al., 1999 (38)	ps	29	78*			
Lipschitz et al., 1999 (17)	ad	38	77	47	90	34
Lipschitz et al., 2000 (203)	ad	57	39	30	65	
Fehon et al., 2001 (204)	ad	71	55	51		
Goodman et al., 2001 (39)	op	321	49	54	67	36
Bowe, 2002 (82)	sc	8	62	75	75	62
Friedman et al., 2002 (25)	sc	9	78			
Chandra et al., 2003 (48)		146	12			
Garon et al., 2003 (205)	sc	54	61	48		
Holowka et al., 2003 (206)	sc	7	57	17	57	17
Offen et al., 2003 (124)	ps	7	71			
Resnick et al., 2003 (207)	sc	30	47			
Compton et al., 2004 (57)	sc	2	100	100	100	100
Shack et al., 2004 (208)		111	51	65	70	46
Axelrod, 2005 (58)	ps	18	72	72	83	61
Kilcommons & Morrison, 2005 (209)	sc	7	14	14		
Schenkel et al., 2005 (210)	sc	15	47	47	53	40
Kim et al., 2006 (211)	sc	100	19	20	21	18
Schafer et al., 2006 (212)	sc	30	37	20	41	13
Klewchuk et al., 2007 (213)	sc	7	43	29	57	14
Lysaker et al., 2007 (214)	sc	16	75			
Rosenberg et al., 2007 (108)	sc	183	43	52	62	33
Ucok et al., 2007 (215)	sc	28	32	21	39	14
Western, 2007 (216)	ps	56	48	46		
Beattie et al., in press (217)	ps	12	50	50	58	42
Davidson et al., in press (218)	ps	3	33	33	33	33
Mullolland et al., 2008 (34)	sc	19	26	11	32	16
Kingdon et al., in prep. (219)	sc	31	58	48	65	42
Weighted Average			46.9%	47.2%	64.5%	34.8%
			1,522	1,137	1,236	618
			3,242	2,407	1,917	1,778

*midpoint of two measures
 sc=all diagnosed schizophrenic or schizophrenia spectrum; ps=all diagnosed psychotic; op=outpatients with at least 50% diagnosed psychotic; ad=adolescent inpatients; ch=child inpatients; ex=inpatients

Table 3 Percentages of Reported Child Abuse among Male Psychiatric Inpatients, or Outpatients of Whom at Least Half were Diagnosed Psychotic

Study Author(s), Year (Ref #)	Diagnosis	n	Child Sexual Abuse (CSA) (%)	Child Physical Abuse (CPA) (%)	Either CSA or CPA (%)	Both CSA and CPA (%)	Study Author(s), Year (Ref #)	Diagnosis	n	Child Sexual Abuse (CSA) (%)	Child Physical Abuse (CPA) (%)	Either CSA or CPA (%)	Both CSA and CPA (%)
Jacobson & Richardson, 1987 (40)		50	16	54	58	12	Offen et al., 2003 (124)	ps	19	26			
Sansonnet-Hayden et al., 1987 (15)	ad	25	24	52			Resnick et al., 2003 (207)	sc	17	18			
Metcalfe et al., 1990 (220)	op	100	34*				Compton et al., 2004 (57)	sc	16	31	75	75	31
Jacobson & Herald, 1990 (186)		50	26				Lysaker et al., 2004 (222)	sc	37	38	57	63	32
Goff et al., 1991 (188)	ps	40			42		Shack et al., 2004 (208)		160	25	33	41	17
Lanktree et al., 1991 (189)	ch	17	12				Axelrod, 2005 (58)	ps	69	57	74	80	51
Rose et al., 1991 (191)	op	50	22	38			Kilcommons & Morrison, 2005 (209)	sc	25	12			
Ito et al., 1993 (194)	ad	53			34		Schenkel et al., 2005 (210)	sc	25	16	24	40	0
Greenfield et al., 1994 (196)	ps	19	16	47	53	11	Lysaker et al., 2005 (223)	sc	65	28	32		
Ross et al., 1994 (80)	sc	56	30	23	43	11	Calhoun et al., 2007 (30)	sc	165	31	61		
Trojan, 1994 (198)	ps	48	27				Klewchuk et al., 2007 (213)	sc	44	27	25	41	9
Cohen et al., 1996 (199)	ad	32	34	47	62	19	Lysaker et al., 2007 (214)	sc	112	38			
Wurr & Partridge, 1996 (201)		57	39				Rosenberg et al., 2007 (103)	sc	386	29	59	65	23
Mueser et al., 1998 (41)	op	122	36	38			Ucok et al., 2007 (215)	sc	29	28	7	28	7
Goodman et al., 1999 (38)	op	21	45*				Western, 2007 (216)	ps	44	27	54		
Lipschitz et al., 1999 (17)	ad	33	33	45	66	12	Beattie et al., in press (217)	ps	35	12	26	29	9
Lipschitz et al., 2000 (203)	ad	38	16	55	71		Davidson et al., in press (218)	ps	28	21	25	36	11
Fehon et al., 2001 (204)	ad	59	12	68			Mulholland et al., 2008 (34)	sc	63	16	11	21	6
Goodman et al., 2001 (39)	op	461	29	58	65	22	Kingdon et al., in prep. (219)	sc	40	10	30	32	7
Lysaker et al., 2001 (221)	sc	52	35				Weighted Average			28.7%	48.7%	55.5%	19.7%
Bowe, 2002 (82)	sc	14	36	57	57	36				777	1,045	960	315
Friedman et al., 2002 (25)	sc	13	0							2,710	2,144	1,731	1,600
Holowka et al., 2003 (206)	sc	19	47	21	53	16							

*midpoint of two measures

sc=all diagnosed schizophrenic or schizophrenia spectrum; ps=all diagnosed psychotic; op=outpatients with at least 50% diagnosed psychotic; ad=adolescent inpatients;

ch=child inpatients; ex=ex-inpatients

six of the ten—or seven of eleven including a later study (59)—these scales were more strongly related to abuse than the other clinical scales.

Studies using clinical diagnoses produce similar findings. The 2005 review identified five studies finding that schizophrenia or psychosis are no less, or more, related to child abuse than other diagnoses, and six studies finding that schizophrenia or psychosis are *more* strongly related than other diagnoses. For example, in a study of female outpatients, 78% of those diagnosed schizophrenic had suffered CSA, compared to 30% of those with anxiety disorders and 42% of those with depressive disorder (25). Among child inpatients, 77% of those who had been sexually abused were diagnosed psychotic, compared to 10% of the other children (60).

Neglect and Emotional Abuse

More subtle, ongoing childhood adversities seem to be just as related to psychosis as overt acts of abuse (see Table 4). Research on Expressed Emotion (EE), a euphemism for parental hostility, criticism and intrusiveness, while avoiding naming these behaviors as abusive so as not to be accused of family blaming, is relevant here. Most EE researchers adopted the distorted version of the stress-vulnerability model and, therefore, focused only on the effects of EE on relapse rates, avoiding its causal role. One U.S. study, however, followed adolescents for fifteen years and found that 36% of those whose parents had both scored high on EE were now diagnosed with schizophrenia, compared to 0% if only one, or neither, parent was high EE (61).

The Parental Bonding Instrument (PBI) retrospectively measures perceptions of parent-child relationships up to age sixteen. The Caring scale measures a dimension from empathy and affection to neglect and emotional coldness. The Over-protection scale ranges from intrusion, control, and infantilization, to autonomy and allowance of independence (62). High protection and low care is characterized as affectionless control. The opposite is optimal parenting (63). Studies of psychosis and schizophrenia consistently find high rates of affectionless control parenting (64, 65).

In an early community survey, women emotionally abused as children were five times more likely to have had a psychiatric admission (66). People whose mother-child interactions at age three were characterized by harshness toward the child and no effort to help the child are, at age twenty-six, more likely to be diagnosed with schizophreniform disorder, but not mania, anxiety or depression (67). Some studies find that neglect and emotional abuse are even more strongly related to psychosis than CPA and CSA (58, 68, 69).

Children later diagnosed schizophrenic are more likely to have had: less satisfactory relationships with their parents

Table 4 Rates of Emotional Abuse (EA), Emotional Neglect (EN) and Physical Neglect (PN) in Samples of People Diagnosed with Schizophrenia or Schizophrenia Spectrum Disorders

Study Author(s), Year (Ref #)	n	Female %	EA %	EN %	PN %
Bowe, 2003 (80)	22	36	77	91	63
Holowka et al., 2003 (201)	26	27	35	73	42
Compton et al., 2004 (57)	18	11	94	89	89
Schafer et al., 2006 (206)	30	100	40	43	37
Ucok et al., 2007 (209)	57	49	32	35	19
Davidson et al., in press (212)	31	10	42	19	
Weighted Average			46.7%	51.1%	41.2%
			<u>86</u>	<u>94</u>	<u>63</u>
			184	184	153

(using a measure including parents’ reports) (70), stressful family characteristics (parental attitudes and poverty combined) (71), poor family relationships (72), families with high levels of conflict in general (73) and, consistent with the EE and PBI research, over-involved or hostile parenting (74).

A study of Israeli adolescents with schizophrenia (75), reporting high levels of life events associated with impaired family function, reminds us that positive (protective) life events can be as important as negative ones (76). Negative life events differentiated the schizophrenia and control groups, but did not differentiate between suicidal and non-suicidal subsets of the schizophrenia sample. However, the percentage of negative events (out of total events) did so: suicidal–74%; nonsuicidal–57%.

Specific Symptoms

Many researchers are abandoning research into the heterogeneous and disjunctive category “schizophrenia,” largely because of its poor reliability and predictive validity (77-79), and are focusing instead on the causes of more discrete phenomena such as hallucinations or delusions. There is a pattern emerging in which the strongest relationships with abuse and neglect appear to be for hallucinations, particularly voices commenting, and paranoid delusions (32, 53, 69, 80). One study found that although hallucinations, delusions, and thought disorder were all predicted by child abuse, regression analysis revealed that only hallucinations

were predicted by child abuse in the absence of abuse in adulthood (50). The relationship between child abuse and hallucinations exists across diagnostic boundaries. A study of bipolar affective disorder found that those subjected to CSA were twice as likely to have auditory hallucinations in general and six times as likely to hear voices commenting (81).

Content

Two studies found that the content of just over half of the schizophrenic symptoms of adults abused as children is obviously related to the abuse (82, 83). A third study found that 12.5% had hallucinations with similar themes and content to their traumas, and 45% had hallucinations in which the themes were the same, but not the specific content (84).

A study of severely maltreated children found that the content of their hallucinations or illusions was strongly reminiscent of episodes of traumatic victimization (85). The same has been found to be true for many adolescents (86) and adults (87, 88) who suffered CSA. The content of hallucinations of adult CSA survivors contains both flashback elements and more symbolic representations of traumatic experiences (89).

Examples from studies of incest survivors include: one believed that her body was covered with ejaculate and another that she had had sexual relations with public figures (90, p. 1475); a man who had been raped several times by an uncle at age seven heard voices telling him he was “sleazy” and should kill himself; a woman who had been sexually assaulted by her father from a very young age, and raped as a teenager, had the delusion that people were watching her as they thought she was “a sexual pervert” and had auditory hallucinations accusing her of doing “dirty sexy things” (86). Examples from another study (50, p. 12) include:

One person, whose chart included a forensic report stating “was abused over many years through anal penetration with the use of violence,” hears the perpetrator’s voice telling the patient to touch children ... Another’s chart read “Sexual abuse from an early age. ... Raped several times by strangers and violent partners.” This person believes that he is being tortured by people getting into body, for example, the Devil and the Beast and “had bleeding secondary to inserting a bathroom hose into self, stating ‘wanting to wash self as people are trying to put aliens into my body.’”

Eleven General Population Studies: 2004-2008

When investigating causality there is a hierarchy of types of studies, generating a hierarchy of evidence. The relatively

small, correlational and uncontrolled group comparisons described thus far lie toward the lower end of this hierarchy, while large-scale population studies controlling for multiple factors are positioned toward the top—with prospective studies sitting above cross-sectional ones. In 2004, three general population studies were published (Table 5).

In the U.K. people who had suffered sexual abuse (not all childhood) were 15.5 times more likely to be psychotic (91). The Odds Ratios (ORs) for sexual abuse, and for the four other victimization events (see Table 5), were all higher than the ORs for the three other diagnostic groupings studied.

In a Netherlands study people subjected to any form of child abuse were between 3.6 and 13 times more likely to be psychotic, depending on psychosis severity (92). In both of these studies the relationships remained significant after controlling for other variables. The Netherlands study, which was prospective rather than cross-sectional, and had a clear age cutoff point for the child abuse, found that even after controlling for twelve variables, people abused as children were 7.3 times more likely than nonabused participants to experience the most severe level of psychosis measured.

The third study, also prospective, studied CSA and treatment for schizophrenic disorders in Australia (93). The ORs of 1.3 for females and 1.5 for males were not statistically significant. The authors acknowledged, however, “powerful systematic biases against finding differences between cases and controls.” These included the fact that the comparison group, the general population, included people who had suffered CSA, “which will act to reduce or even obscure the differences between cases and controls.” These biases were so powerful that they even obscured the well-established relationship between CSA and substance abuse. Another crucial bias led the authors to caution, in relation to their schizophrenia finding, that “care must be taken in interpreting this;” the abuse sample was younger than the general population sample and, therefore, less likely to have developed schizophrenia. An even more important bias was not acknowledged. The abuse sample had been through the court system. It was, therefore, a very atypical sample of abuse victims, because the majority of victims never tells anyone for years about the abuse, let alone goes to court. This unrepresentative group had told someone and been believed. Many would have been separated from the abuser (thereby preventing ongoing abuse) and received some help. One interpretation of this study, therefore, is that early intervention following child abuse is a preventive factor for schizophrenia (94).

In 2005 a U.S. study found that CSA and CPA significantly predicted hallucinations in adulthood after controlling for substance abuse (69). It also found that childhood emotional abuse was slightly more predictive of hallucinations than CPA or CSA.

These first four population studies, together with the myriad of other studies reported above, led a 2005 review to conclude that child abuse is a causal factor for psychosis and schizophrenia (32). The research presented was described in the media as “tectonic plate shifting evidence” and led to the prediction that the “psychiatric establishment is about to experience an earthquake that will shake its intellectual foundations” (95). The Institute of Psychiatry in the U.K. invited the first author to debate the motion: “This house believes that child abuse is a cause of schizophrenia.” Following a rebuttal from leading geneticist Professor Peter McGuffin, and lengthy discussion among the assembled psychiatrists and others present, the motion was carried by 114 to 52 votes (96). Meanwhile a British High Court judge ruled that:

The likelihood as it seems to be is that the terrible abuse to which A was subjected led to both his suffering PTSD and that disorder of the mind which is symptomatic of schizophrenia. ... What is important is that his adult psychiatric problems, however they are classified, were caused by his childhood sexual abuse (97).

Nevertheless, scholarly critiques of the 2005 review argued that more research was needed (54-56). We summarize next, therefore, the seven population-based studies that have been published since the 2005 review.

The team that had published the 2004 Netherlands study turned its attention to nonclinical delusional ideation and hallucinatory experiences in adolescents (98). These experiences are highly predictive of psychosis in adulthood (99). The OR for the adolescents who had experienced sexual trauma was 5.1, while the OR for those bullied once a week or more in the preceding year was 3.1. This latter finding is consistent with the U.K. study, which found an OR for bullying of 4.2 (91). Meanwhile, another study found, in a German sample of young people, an OR for CSA of 3.4 for having three or more symptoms of psychosis (100). In both studies, the findings remained significant after controlling for other variables.

In 2007 four more population-based studies were published. Irish researchers published another study of hallucinations, with a U.K. sample. Being molested or raped before age sixteen was, after controlling for eight potential mediating factors, significantly predictive of auditory, visual and tactile hallucinations. Childhood neglect was only predictive of visual hallucinations and, interestingly, CPA predicted only tactile hallucinations (101). An Australian study found that those who had been sexually molested were seven times more likely to experience delusions (102), but, as in the 2004 U.K. study (91), it was not clear whether all the molestation had occurred in childhood.

Two more studies from the Irish team used actual di-

agnoses of psychosis. The first used the same U.K. sample as its hallucinations study. It reported ORs, after adjusting for ten potentially confounding variables, of 5.7 for sexual abuse and 2.1 for violence in the home (103). The same paper also reported a study on a U.S. sample which, after controlling for the same ten factors, produced ORs of 2.5 for sexually molested and 4.2 for CPA.

The final, much smaller, study found that among Irish adolescents, those with one or more psychotic symptoms were four times more likely to have suffered CSA, six times more likely to have suffered CPA and ten times more likely to have witnessed domestic violence (104). These findings were not confounded by comorbid psychiatric illness. The researchers concluded that: “Our findings add to the evidence that childhood trauma may increase the risk of psychotic experiences.”

Thus there are now eleven population-based studies, by seven independent research teams, using nine different samples drawn from six countries. All found higher levels of child maltreatment or neglect in their psychosis groups. Ten of the eleven found that this was statistically significant, even after controlling for a range of other factors.

Artificial Minimization Produced by Controlling for Other Factors

The process of controlling for other variables is important in establishing whether factor X has a statistically independent effect on factor Y. It does not, however, reflect the real world. Some studies in Table 5, for instance, controlled for depression or substance abuse, or for the effects of the other adverse events in the study. This renders the significant findings that survived this procedure particularly impressive. However, many abused children become depressed. Many use drugs and alcohol, sometimes to self-medicate the effects of the abuse (57, 105). Indeed, within a schizophrenia sample, childhood trauma predicts who has abused substances (106). The majority of children subjected to one particular form of maltreatment will also have experienced other forms of abuse, loss or neglect (57, 58, 82, 107). Controlling for such factors artificially lowers the probability of finding a relationship between a specific type of adverse event and psychosis. For example, in the 2004 U.K. study, adults who had been in a children’s institution were 11.9 times more likely to be diagnosed psychotic, with ORs of 9.0 for violence in the home and 4.2 for being bullied (91). After controlling for depression and interactions with other adverse events, the relationship between each of these specific types of events and psychosis was no longer significant. It would be silly, however, to conclude that these three factors do not contribute to psychosis. In reality the effects of multiple types of trauma are cumulative, as illustrated by our next topic.

Dose-Response

Although it is impossible to be absolutely certain that events at age twenty-five were caused by events twenty years earlier, the probability increases if we find that X not only predicts Y, but that more of X, in this case greater severity or frequency of abuse, or both, is more related to Y, i.e., that there is a dose-response. Table 5 shows that eight of the eleven studies investigated this dose-response hypothesis. All eight confirmed the hypothesis.

We have already seen that the content of hallucinations and delusions is often based, directly or symbolically, on memories of childhood trauma.

In the 2007 U.S. study, for example, the ORs for psychosis were 1.6 for one type of trauma only, 3.7 for three types of trauma and 30.2 for five types (103). The 2007 U.K. study showed a similar pattern, with those who had experienced five or more types of trauma being 198 times more likely to be diagnosed psychotic than nonabused people (103).

The dose-response hypothesis can be investigated in the other direction, too; i.e., does child abuse lead to more psychosis? Two studies tested this hypothesis. The 2006 Netherlands study found, in relation to CSA, ORs for one, two, and three psychotic symptoms of 1.2, 1.9, and 3.4 respectively (101). In the study of German adolescents, those who had suffered sexual trauma were 4.7 times more likely to have one type of psychotic experience, but 6.4 times more likely to experience two types (100).

A dose-response has also been found within a sample of 569 people with schizophrenia. Cumulative exposure to adverse childhood events was related to suicidality, psychiatric hospitalizations, HIV infection, homelessness, and criminal justice involvement (108).

The Genetic Predisposition Hypothesis

The 2004 Netherlands study controlled for both family mental healthcare and history of hallucinations or delusions in first-degree relatives and found that people who have been abused as children are nine times more likely than nonabused people to experience pathology-level psychosis (92). This is consistent with studies cited earlier showing that psychosis is related to poverty, urban living and ethnicity in the absence of family history of psychosis (20-22, 25, 26).

It is indisputably true that all human behavior involves gene-environment interactions. It seems, however, that the hypothesis that there is a specific genetic predisposition for schizophrenia, overemphasized for thirty years, partly because of the misunderstanding of the stress-vulnerability model, is turning out to be one of the costliest blind alleys in

the history of medical research. Reviews of the methodologies and concepts deployed suggest there is no robust evidence at all in support of a genetic predisposition (109).

A recent *American Journal of Psychiatry* paper (110), described by the editor as: "The most comprehensive genetic association study of genes previously reported to contribute to the susceptibility to schizophrenia" (111, p. 420), found that "none of the polymorphisms were associated with the schizophrenia phenotype at a reasonable threshold for statistical significance" and "of the 69 SNPs (single nucleotide polymorphisms) ... only four showed even nominal association. ... The distribution of test statistics suggests nothing outside of what would be expected by chance" (111, p. 421).

Psychological Mechanisms Explaining the Causal Relationship

Many researchers are now tackling the fascinating question of *how* childhood abuse and neglect lead to psychosis (33, 112). Some try to explain the relationship within the existing *Diagnostic and Statistical Manual of Mental Disorders (DSM)* categorical framework. U.S. researchers, having demonstrated that psychosis is exacerbated by the trauma-based hyperarousal, intrusive memories and avoidance of PTSD, continue to perceive PTSD and psychosis as separate disorders, thereby avoiding the possibility that trauma itself is causal for psychosis (30, 41).

The bulk of this body of research, however, demonstrates the utility of focusing on specific symptoms of psychosis, thereby circumventing *DSM* categories which mask the reality that schizophrenia, PTSD, dissociative disorders, borderline personality disorder, etc., portrayed in the *DSM* as separate entities with separate causes, all include symptoms that are trauma based. The theories and research emanating from this symptom-focused approach, summarized below, are described in more depth in the first book dedicated to trauma and psychosis (112).

Cognitive researchers have demonstrated that hallucinations and delusions, and their links to early trauma, can be understood in terms of problematic thinking styles originating in abuse and other childhood disadvantages. Hallucinations have been shown to originate from a source monitoring difficulty in accurately attributing experiences, especially inner thoughts, to internal or external sources (33, 77, 112, 113). We have already seen that the content of hallucinations and delusions is often based, directly or symbolically, on memories of childhood trauma. The source monitoring difficulty may serve the defensive function of protecting the person from the distress of realizing the true source, an inner memory of childhood trauma in the past, by experiencing it as an external event in the present (53). This represents an intriguing convergence of a cognitive focus on thought processes and a psychodynamic understanding of projec-

Table 5 Large-Scale, General Population, Cross-Sectional and Prospective Studies						
Study Author(s), Year, Country, Design*, n, (Ref #)	Psychosis Measure	Adverse Childhood Event	Odds Ratio (OR)	Adjusted OR	Factors Controlled for in Adj. OR	Dose-Response: OR for Different Levels of Abuse/Trauma
Bebbington et al., 2004, U.K., C., 8580, (91)	diagnosis of psychotic disorder	sexual abuse (not all childhood) bullied violence in home running from home children's institution	15.5 4.2 9.0 11.5 11.9	2.9 [†] 1.4 1.4 2.8 [†] 1.5	1, 2	not tested (see Shevlin et al., 2007)
Janssen et al., 2004, Netherlands, P, 4045, (92)	psychosis severity: a. any psychosis b. pathology level c. need for care	physical, sexual, emotional or psychological abuse	a. 3.6 b. 13.0 c. 11.5	a. 2.5 [†] b. 9.3 [†] c. 7.3 [†]	3-14	abuse: mild severe a. 1.4 9.8 [§] b. 2.0 48.4 [§] c. 1.0 31.7 [§]
Spataro et al., 2004, Australia, P, 1612, (93)	d. hallucinations e. delusions treatment for schizophrenic disorder	sexual abuse	d. 4.0 e. 3.9 male: 1.5 ^{RR} female: 1.3	d. 2.5 e. 2.8 [†]		not tested
Whitfield et al., 2005, U.S., C., 17337, (69)	hallucinations	sexual abuse physical abuse emotional abuse battered mother	1.8 1.7 2.5 1.6	1.7 [†] 1.7 [†] 2.3 [†] 1.5 [†]	3-5, 7	1 event: 1.1 4 events: 1.8 ≥7 events: 6.7 [§]
Lataster et al., 2006, Netherlands, C, 1290 (adolescents), (98)	nonclinical psychotic experiences	sexual trauma bullied	5.1 3.1	4.8 [†] 2.9 [†]	1, 3, 4, 15	bullied once/twice: 1.9 bullied once/twice per month: 3.5 [§]
Spauwen et al., 2006, Germany, P, 2524 (aged 14-24), (100)	three or more positive psychotic symptoms	sexual abuse any trauma before 13 years	3.4 2.6	1.55 2.2 [†]	4, 8, 10, 14-16	1 severe event: 1.8 2 severe events: 3.1 [†]
Shevlin et al., 2007, U.K., C, 5877, (101)	visual hallucinations	"sexually molested" "rape" "physical abuse" "serious neglect"	1.7 2.4 1.5 2.1	1.6 [§] 2.4 [§] 1.0 1.6 [†]	1-4, 8, 14, 15, 17	1 trauma: 2.7 4 traumas: 7.8 [†]
(subsample of Shevlin et al., 2007 U.K. sample—see next study)	auditory hallucinations	"sexually molested" "rape" "physical abuse" "serious neglect"	1.8 1.8 1.5 1.7	1.9 [§] 1.7 [†] 1.2 1.3		1 trauma: 1.9 4 traumas: 4.5 [†]
	tactile hallucinations	"sexually molested" "rape" "physical abuse" "serious neglect"	2.1 1.9 2.4 1.5	1.8 [§] 1.7 [†] 1.9 [§] 0.8		1 trauma: 2.7 4 traumas: 8.7 [†]
Shevlin et al., 2007, U.K., C, 8580 (same as Bebbington), (103)	diagnosis of psychotic disorder	sexual abuse violence in home	15.5 9.0	5.7 [†] 2.2 [†]	1-5, 7, 8, 14, 15, 18	1 trauma: 1.7 1 trauma: 1.7 3 traumas: 18.0 [†] 5 traumas: 193.0 [†]
Shevlin et al., 2007, U.S., C, 5782, (103)	diagnosis of nonaffective psychotic disorder	sexually molested physical abuse		2.5 [†] 4.2 [†]	1-5, 7, 8, 14, 15, 18	1 trauma: 1.6 3 traumas: 7.4 [†] 5 traumas: 30.2 [†]

- Continued

Table 5 Large-Scale, General Population, Cross-Sectional and Prospective Studies - continued

Study Author(s), Year, Country, Design*, n, (Ref #)	Psychosis Measure	Adverse Childhood Event	Odds Ratio (OR)	Adjusted OR	Factors Controlled for in Adj. OR	Dose-Response: OR for Different Levels of Abuse/Trauma
Scott et al., 2007, Australia, C, 10641, (102)	delusions	"sexually molested" (not all childhood) any trauma without PTSD any trauma with PTSD	7.1 2.7 9.2	2.3 ^{§RR} 2.0 [§] 6.4 [§]	19 3, 4, 14, 20	1 or 2 traumas: 2.5 3 or 4 traumas: 2.9 ≥5 traumas: 9.5 ^{§RR}
Kelleher et al., in press, Ireland, C, 222 (adolescents), (104)	one or more psychotic symptoms	sexual abuse physical abuse bullied exposure to domestic violence	4.2 6.0 1.2 10.1	5.4 6.2 [†] 1.5 7.8 [†]	10	not tested

n=number; RR=relative risk
*Design: P=prospective; C=cross-sectional
† p <.05; § p <.01; § p <.001

Factors controlled for in adjusted OR: 1=interrelationship between adverse events; 2=depression; 3=age; 4=gender; 5=education level; 6=discrimination; 7=ethnicity; 8=urbanicity; 9=marital status; 10=nonpsychotic diagnosis; 11=family history of psychiatric care; 12=family history of psychosis; 13=unemployment; 14=substance use; 15=socioeconomic status; 16='psychosis proneness' on SCL-R-90 at start of study; 17=family history of depression; 18=living arrangements; 19=PTSD; 2.3=without PTSD; 2.3=with PTSD; 20=past diagnosis of schizophrenia

tion of distressing emotions and memories onto the external world (114).

Paranoid delusions have also been shown to originate in faulty cognitive processes learned in childhood. These include negative beliefs about self and others, a rigid tendency to attribute negative experiences to external, personal events, and the tendency to jump to conclusions on the basis of limited information, especially when confronted with ambiguous or anomalous experience (53, 77, 112, 113). The latter explanation suggests that delusions are often an attempt to explain hallucinations, which, as we have seen, often involve traumatic material (53).

Cognitive theorists have also studied the role of affect (115). The amount of distress when first having a psychotic experience is determined by one's attributions about the experience, and these attributions are predicted by the presence of earlier trauma (116, 117). There is good evidence to support a "catastrophic interaction hypothesis" whereby the cognitive difficulties of traumatized people exacerbate their distress when they later encounter normal stressful events, and that this exaggerated distress reciprocally exacerbates the cognitive problems (118).

Attachment theory may be particularly useful (64, 65, 119). A recent review which found high rates of the affectionless control parenting style in the parents of people diagnosed psychotic and schizophrenic (64), also reported numerous studies showing high rates of insecure and disorganized attachment. Attachment theory may help us integrate the other psychological theories, and, as we shall see later, biological processes.

It may be that early adverse experiences contribute to the development of core predictors associated with the development of psychosis and the emergence of a negative trajectory of psychosis. This is characterised by problematic emotional and interpersonal adaptation, heightened sensitivity to interpersonal stress (e.g., criticism and emotional over-involvement), poor pro-social coping and help-seeking, social withdrawal and avoidant and/or conflicted coping styles, and impoverished reflective function and affect regulation (120, p. 46).

One outcome of insecure or disorganized childhood attachment is increased levels of dissociation (107). Dissociation is another process that can facilitate the integration of different mechanisms, including biological processes. High levels of dissociative symptoms have been found in people diagnosed psychotic, and vice versa (31, 121-124). The developmental integration of the various separate affective states of the human infant requires a safe and consistent social environment in which a primary caregiver attends to the child's needs and minimizes emotional and physical

trauma. Ongoing social stressors inhibit the integrative process, in part by impairing the capacity of the hippocampus to provide the contextual component of memory, resulting in the persistence of separate affective and cognitive states, i.e., of dissociation. The absence of an integrating frontal executive function and the impoverishment of the capacity for contextualization leave the emerging adult vulnerable to the intrusions of these persistent sequestered intrapsychic states into each other. In addition to the characteristic symptoms of dissociation, including memory lapses, identity confusion, and affective instability, symptoms of psychosis are also logical consequences of this uninhibited internal dialogue. For example, decontextualized voices, signals from one compartmentalized state to another, are easily misunderstood as originating externally, which might account for the source monitoring problem discussed earlier. Likewise, decontextualized feelings of fear are easily ascribed to people and events occurring in the present social environment, generating paranoia. Abrupt shifts between states, especially at times of stress, can easily generate disorganization of thought (121, 125-128).

A Genuine Integration: The Traumagenic Neurodevelopmental Model

None of these psychological researchers argue that biological processes are irrelevant. Neither, however, do they distort the original stress-vulnerability model by relegating social causes to mere triggers of a supposed genetic predisposition. Naturally there are biological processes underlying the mechanisms by which trauma leads to psychosis. All mental processes, all human experience, have neurological and biochemical correlates. Some researchers, however, believe that because we can sometimes identify these correlates, we have, thereby, discovered a cause. This is akin to assuming that because the brain operates differently when we are grieving, it is the brain that caused our sadness. Brain researchers studying schizophrenia often operate as if the brain exists in a social vacuum, ignoring the fact that a primary function of the brain is to react to the environment.

It is, nevertheless, brain research that points the way to a genuine integration of the biological, the social, and the psychological in understanding how childhood trauma can lead to psychosis. The Traumagenic Neurodevelopmental (TN) model of psychosis (129) is based on research demonstrating that the biological differences traditionally cited as evidence that schizophrenia is a brain disease are also found in the brains of abused children. These include: overactivity of the hypothalamic-pituitary-adrenal (HPA) axis; dopamine, norepinephrine and serotonin abnormalities; hippocampal damage; cerebral atrophy; ventricular enlargement; and reversed cerebral asymmetry. Thus, the heightened sensitivity to stress evidenced by dysregulation of the brain's stress

regulation mechanisms is not necessarily inherited. It can be caused by childhood trauma.

The authors also reviewed research linking child abuse and neglect to the psychological deficits in childhood that are typically interpreted as early signs of the illness rather than as the result of adverse events in childhood. Even reviewers skeptical about the causal relationship between trauma and psychosis acknowledge:

There are studies that have found HPA dysregulation in abused girls and in women who were physically or sexually abused in childhood. Heightened sensitivity to stress has long been considered a central feature of schizophrenia, and recent research has found patients with a first episode of psychosis to have enlarged pituitary glands compared with normal controls, independent of antipsychotic treatment (55, p. 8)

An explicit test of the TN model found that within a sample of schizophrenia patients those abused as children, especially those emotionally abused, have greater HPA axis dysregulation, measured by cortisol levels, than their non-abused counterparts (130). Similarly, problems in childhood attachment can alter the structures, neurochemicals, and connectivity of the brain. Like CPA and CSA, severe neglect of children affects the ability of the HPA axis to regulate the body and brain's response to stress (131, 132).

There is a considerable literature which genuinely integrates psychological, social and biological models, some of which predates the TN model of psychosis (125, 127, 133-136). One example of progress stimulated by real paradigm integration is a 2003 paper entitled "Psychosis as State of Aberrant Salience: A Framework Linking Biology, Phenomenology and Pharmacology in Schizophrenia" (136). 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). "It is proposed that in psychosis there is a dysregulated dopamine transmission that leads to stimulus-independent release of dopamine" (p. 15). Kapur's theory is consistent with, and helps integrate, the various attempts to understand how trauma might lead to psychosis, particularly the TN model and the cognitive theories of hallucinations and delusions.

Recent reviewers of schizophrenia research agree that "the traumagenic neurodevelopmental model is an example of a genuine integration between social, psychological and biological factors" (137, p. 279). This approach is not, of course, only applicable to psychosis. A recent study found a relationship between adverse childhood experiences and eighteen different outcomes, which "parallels the cumulative exposure of the developing brain to the stress response with

resulting impairment in multiple brain structures and functions” (9, p. 174).

Research Implications

A 2004 review of the trauma-psychosis research literature identified thirty-seven research questions generated by an integrated approach to psychosis (53). One of these was: “Can research on the effects of trauma on the developing brains of children shed some light on why men experience more negative symptoms and have earlier onset of schizophrenia than women” (p. 242).

Gender issues in schizophrenia are under researched from a psychosocial perspective (19, 134). An exception is a recent British study which replicated the findings of the general population studies reported above in relation to both CSA and CPA, but only in the women, not in the men (138). This is consistent with an earlier study, which found that women who had suffered CSA were ten times more likely than nonabused women to have a schizophrenic disorder, but found no such relationship for men (139). The hypothesis that these findings are an artifact of more abused boys than abused girls ending up in the justice system, or killing themselves, needs to be addressed.

A particularly depressing outcome of the distorted stress-vulnerability model is that, instead of lobbying governments to fund primary prevention programs, the psychiatric community gives governments a perfect excuse to do nothing.

More research into the biological and psychological mechanisms involved is needed. More exploration of which type of abuse or neglect leads to which sort of psychotic symptom might also be important. In a sophisticated elaboration of focusing on symptoms rather than diagnoses, two recent general population studies used latent class analysis to identify homogeneous subtypes of psychosis. Both studies found strong relationships between the subtypes and child maltreatment, with, again, a dose-response (140, 141). Since trauma-based symptoms, psychotic and nonpsychotic, are found in multiple *DSM* categories, there are major implications for the validity of these diagnoses as separate entities (32, 33, 53, 77, 121-125, 142).

Structural equation modeling (143) could facilitate analysis of the complex interrelationships of multiple internal and external factors in the development of psychotic symptoms subsequent to child abuse.

Prevention Implications

An obvious, but until recently taboo, implication is that psychosis can be prevented (32, 76). In the field of psychosis, prevention usually refers not to primary prevention, in which social causes are identified and efforts made to minimize those causes, e.g., wealth redistribution where poverty is a causal factor, but to either secondary prevention, targeting at risk groups, or tertiary prevention, treatment to prevent exacerbation of existing conditions. Yet if child abuse is a causal factor for psychosis, to the same or greater extent that it is for other psychological problems, then the same primary prevention programs targeted at keeping children safe, and supporting families, that work for other problems will work for psychosis (76). For example, an environmental enrichment program at age three to five has reduced schizotypal personality scores in early adulthood (144). A particularly depressing outcome of the distorted stress-vulnerability model is that, instead of lobbying governments to fund primary prevention programs, the psychiatric community gives governments a perfect excuse to do nothing.

Treatment Implications

Given the limited efficacy and serious adverse effects of antipsychotic medication, and the high proportion of patients who decline to take these drugs (7, 77, 145, 146), it seems important to further develop and fund psychological therapies and to ascertain who is likely to benefit from medication, who needs psychotherapy, and who needs both (146). It may be relevant that a study of depression found that psychotherapy was more effective than medication for participants with a history of childhood trauma (147).

The fact that early trauma affects the brain does not imply that those brain changes are irreversible. Neuroimaging studies show that psychotherapy can change brain functions and structure (148). Space permits just a few references to the literature on the effectiveness of psychological and integrated treatments for psychosis in general (77, 149-154) and trauma-related psychosis in particular (112, 114, 120, 121, 155, 156). For instance, psychological treatment of youth at risk for psychosis has been shown to be effective, without medication (157). Families should be offered the opportunity to talk about their understandings of what is going on in their family and be offered genuine family therapy, rather than receive an ideologically driven education program based on a distortion of the stress-vulnerability model (158, 159).

The International Society for the Psychological Treatment of Schizophrenia (www.isps.org) disseminates information on psychosocial approaches in its book series. In 2009, ISPS will launch its journal, *Psychosis: Psychological, Social and Integrative Approaches* (www.informaworld.com/psychosis), without drug company funding.

Asking About Abuse

Until recently researchers have failed to ask about abuse. The same is true of many mental health professionals, resulting in the majority of abuse cases being unidentified by mental health services (3, 160-163). Professionals also frequently fail to respond appropriately when they are told about child abuse (3, 161-166). Many of the studies cited in this paper recommend that clinicians take trauma histories with all their patients. This is especially important because people abused as children rarely disclose abuse spontaneously (160, 162, 167). Whether or not one is convinced about the causal relationship discussed in this paper, the high numbers reported in Tables 2, 3 and 4, and the undisputed relationship with suicide, render it imperative that all patients are asked. Even reviewers unconvinced of the causal relationship make this recommendation (56).

*I just wish they would have said,
“What happened to you? What happened?”
But they didn’t.*

There is another body of literature that supports these recommendations. In sixteen countries, the public believes that mental health problems, including schizophrenia, are caused by adverse life events rather than faulty genes or brains (168-172). Research shows that this psychosocial perspective is also held by patients, often dismissed as lack of insight, and family members, which raises the question of how representative are drug-company sponsored groups, like NAMI in the United States and SANE in the United Kingdom. The public holds these beliefs despite vast amounts of money, often donated by drug companies, being spent to educate us to adopt a biomedical disease model. These public campaigns ignore all the research showing that the disease model increases, rather than decreases, fear and prejudice (168-172). The important point here, however, is that most people using mental health services have a psychosocial formulation of their problems and, therefore, expect to be asked about what has gone on in their lives (173).

A one-day training program about when and how to ask about abuse, and how to respond, has been developed and evaluated in Auckland, New Zealand (3, 174). In 2007, it was presented in the professional journal of the Royal College of Psychiatrists in the U.K. (175). Table 6 summarizes the factors associated with not asking. These were used in the design of the training program, the principles of which are outlined in Table 7.

The U.K. Department of Health has just published a briefing paper on Implementing National Policy on Violence and Abuse (176). Consistent with the policy on which the Auckland training program was based, the document states (p. 1):

Table 6 Barriers to Inquiry and Appropriate Response

Other more immediate needs and concerns (3)
Concerns about offending or distressing clients (3, 224)
Fear of vicarious traumatization (3, 225)
Unnecessary fear of inducing “false memories” (3, 45, 174)
The client being male (160, 164, 165, 174, 226, 227)
Client being older (164), i.e., over 60 (174)
Client having a diagnosis indicative of psychosis (160, 164, 165, 174), particularly when the clinician has strong bio-genetic causal beliefs (3)
Clinician being a psychiatrist (165, 227), especially with strong bio-genetic causal beliefs (3)
Strong bio-genetic causal beliefs in general—psychiatrists and psychologists (3)
Clinician being male (165, 228) or opposite gender to client (174)
Lack of training in how to ask and how to respond (174, 175, 229)

Table 7 Principles of Taking, and Responding to, a Trauma History

Principles of Asking
Ask all clients/patients
At initial assessment (or if in crisis, as soon as person is settled)
In context of a general psychosocial history
Preface with brief normalizing statement
Use specific questions, with clear examples of what you are asking about (e.g., “As a child, did an adult ever hurt or punish you in a way that left bruises, cuts or scratches?” and “As a child, did anyone ever do something sexual that made you feel uncomfortable?”)
Principles of Responding
Affirm that it was a good thing to tell
Do not try to gather all the details
Ask if the person has told anyone before—and how did that go?
Offer support (make sure you know what is available)
Ask whether the client relates the abuse to their current difficulties
Check current safety—from ongoing abuse
Check emotional state at end of session
Offer follow-up/check in

Department of Health policy is that adult mental health services should acknowledge and address the links between violence and abuse and mental health by ensuring staff, once satisfactorily trained, raise issues of violence and abuse routinely and consistently in assessments (both at first contact and at assessment reviews).

It should be noted (p. 2) that the British Department of Health understands that:

The effects of child sexual abuse can be a significant contributory factor to all diagnoses and presentations from depression, anxiety, eating and obsessive compulsive disorders and perinatal mental health through to bipolar disorder, psychosis and schizophrenia, dual diagnosis and personality disorder diagnoses.

When experts debate the causes of psychosis, we rarely include the voices of those who actually experience psychosis (177, 178). They have much to contribute. For example, Wilma Boevink, from the Netherlands, suggests another mechanism by which childhood trauma can lead to psychosis:

I don't think that abuse itself is a strong cause for psychosis. It hurts, but it is too simple. I think that the threat and the betrayal that come with it feed psychosis. The betrayal of the family that says you must have asked for it, instead of standing up for you. That excuses the offender and accuses the victim. And forces the child to say that the air is green, while she sees clearly that it is not green but blue. That is a distortion of reality that is very hard to deal with when you're a child. You are forced to betray yourself. That is what causes the twilight zone. What makes you vulnerable for psychosis (179, p. 19).

We leave the last words to a group of service users who, during the planning of the Auckland training program, were asked what they thought about asking all patients about child abuse (173).

There were so many doctors and registrars and nurses and social workers in your life asking you about the same thing, mental, mental, mental, but not asking you why.

I think there was an assumption that I had a mental illness and you know because I wasn't saying anything about the abuse I'd suffered no one knew.

I just wish they would have said, "What happened to you? What happened?" But they didn't.

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