

## The Contribution of Early Traumatic Events to Schizophrenia in Some Patients: A Traumagenic Neurodevelopmental Model

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THE current diathesis-stress model of schizophrenia proposes that a genetic deficit creates a predisposing vulnerability in the form of oversensitivity to stress. This model positions all psychosocial events on the stress side of the diathesis-stress equation. As an example of hypotheses that emerge when consideration is given to repositioning adverse life events as potential contributors to the diathesis, this article examines one possible explanation for the high prevalence of child abuse found in adults diagnosed schizophrenic. A traumagenic neurodevelopmental (TN) model of schizophrenia is presented, documenting the similarities between the effects of traumatic events on the developing brain and the biological abnormalities found in persons diagnosed with schizophrenia, including overreactivity of the hypothalamic-pituitary-adrenal (HPA) axis; dopamine, norepinephrine, and serotonin abnormalities; and structural changes to the brain such as hippocampal damage, cerebral atrophy, ventricular enlargement, and reversed cerebral asymmetry. The TN model offers potential explanations for other findings in schizophrenia research beyond oversensitivity to stress, including cognitive impairment, pathways to positive and negative symptoms, and the relationship between psychotic and dissociative symptomatology. It is recommended that clinicians and researchers explore the presence of early adverse life events in adults with psychotic symptoms in order to ensure comprehensive formulations and appropriate treatment plans, and to further investigate the hypotheses generated by the TN model.

### INTRODUCTION

Schizophrenia is considered to be one of the most biologically based of the mental disorders (Chua and Murray 1996; McGuffin, Asherson, Owen, and Farmer 1994; Walker and DiForio 1997). However, the methodological rigor of the evidence for this proposi-

tion is often described as less than adequate (Bentall 1990; Boyle 1990; Karon 1999; Rose 2001; Ross and Pam 1995). This article explores the possibility that for some adults diagnosed as schizophrenic, adverse life events or significant losses and deprivations cannot only "trigger" schizophrenic symptoms but may also, if they occur early enough or are sufficiently

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severe, actually mold the neurodevelopmental abnormalities that underlie the heightened sensitivity to stressors so consistently found in adults diagnosed schizophrenic. As one example we explore recent research on the prevalence of child abuse in people diagnosed schizophrenic, and emerging similarities between the neurodevelopmental effects of traumatic events and the neurobiological deficits in schizophrenia.

In light of the literature (reviewed later in this article) indicating that child abuse is correlated with psychosis in general and schizophrenia in particular, and the apparent improbability that a diathesis-stress model based on a genetic diathesis will adequately investigate the implications of that literature, we propose a new diathesis-stress model. Our central hypothesis is that for some adults diagnosed schizophrenic the diathesis that leads to the well-documented high responsiveness to stress is the abnormal neurodevelopmental processes originating in traumatic events in childhood. We hope that a Traumagenic Neurodevelopmental (TN) model will facilitate a more integrated approach to diathesis-stress formulations, with the potential to answer questions not answerable, and to ask questions not askable, by the current paradigm.

The specific hypotheses raised by the TN model and examined here are as follows. (1) The neurological and biochemical abnormalities found in adult schizophrenia and cited as evidence of biogenetic etiology are caused, in some schizophrenics, by child abuse via their long-lasting neurobiological effects. (2) This is the case, specifically, for over-reactivity of the hypothalamic-pituitary-adrenal (HPA) axis, abnormalities in neurotransmitter systems, and structural brain changes, including hippocampal damage, cerebral atrophy, ventricular enlargement, and reversed structural cerebral asymmetry. (3) These trauma-induced neurobiological abnormalities may eventually contribute to our understanding of various aspects of schizophrenia, including oversensitivity to stress, cognitive impairments, pathways to negative and positive symptoms, and the relationship between psychotic and dissociative symptomatology.

### *The Biopsychosocial Model*

The diathesis-stress model of schizophrenia, which gained near-consensus status for the last three decades of the 20th century (Norman and Malla 1993a, 1993b; Walker and DiForio 1997), is characterized as a “biopsychosocial” approach, implying an integration of data from various paradigms. However, the assumption that the diathesis is a genetic predisposition seems to have impeded adequate consideration of the relevance of stress, traumatic events (physical or emotional), neglect, and loss by positioning all psychosocial factors exclusively in the stress component of the diathesis-stress equation. “It seemed inarguable at the time that if mental illness was in the brain or in the genes, then stress was merely a precipitant of conditions that were bound to appear sooner or later, or an exacerbator of existing or dormant symptomatology” (Yehuda 1998a, p. xiii).

Proponents of the biopsychosocial model argue that schizophrenics are not exposed to disproportionate amounts of stressors, but merely over-respond to stress. It is this oversensitivity, or “vulnerability,” that is supposedly inherited genetically. While this model allows that hostility from family members can cause relapse by activating an “underlying autonomic hyperarousal” (Tarrrier and Turpin 1992) or “neurocognitive vulnerability” (Rosenfarb, Nuerchterlein, Goldstein, and Subotnik 2000), the causes of the vulnerability are rarely sought in the interpersonal domain. A prominent review on “Stressful Life Events and Schizophrenia” (Norman and Malla 1993a, p. 165) argues that “there is considerably more evidence for variation in stressors being associated with changes in the course of symptoms for schizophrenic patients than for schizophrenics having been exposed to more external life stressors than the general population or patients suffering from other psychiatric disorders.”

However, the preconception that the diathesis in the diathesis-stress process is genetic (and could not be due to psychosocial events) leads to Norman and Malla, like many

other reviewers, including only those studies measuring stressors a few weeks prior to the outbreak of symptoms. Describing this period as “prodromal” allows even these events to be seen as relevant only in so much as they exacerbate premorbid behavioral dysfunction or, at most, hasten the onset of the initial clinical episode (Walker and DiForio 1997). Evaluating a biogenetically based diathesis-stress model without considering any life events that might contribute to a diathesis seems to be an example of a dominant paradigm asking only those questions that confirm its central assumptions (Kuhn 1970). McGuffin et al. (1994) have even argued, using hypothetical nontransmissible changes in gene structure or expression, that environment has no causal role at all, even as exacerbator.

The extent to which we have achieved a balanced integration of the biological, the psychological, and the social is examined in Table 1. While the first three categories (genetics, biochemistry, and neuropsychology) combined have, as a proportion of all schizo-

phrenia research, remained quite stable (7.4% in the 1960s, 8.0% in the 1990s), the proportion of studies investigating stress (of any kind) peaked at 1.2% in the 1980s and declined to 0.8% in the 1990s. Socioeconomic status peaked at 0.6% and declined to 0.2% by the end of the century. Schizophrenia research dealing with child rearing or parent-child relationships attained a 1.6% share in the 1960s and has declined consistently to 0.2% in the 1990s. In the last four decades, for every study on the relationship between child abuse or neglect and schizophrenia there have been 30 on the biochemistry of schizophrenia and 46 on the genetics.

Even research into childhood schizophrenia, including the extensive study by the U.S. National Institute of Mental Health (NIMH) (McKenna, Gordon, and Rapaport 1994), a Schizophrenia Bulletin issue devoted to the topic (e.g., Spencer and Campbell 1994), and reviews of the research (e.g., Volkmar 1996), ignore any stressors beyond birth trauma and viral infection. PsycINFO records

**TABLE 1**  
*Comparison of Numbers of Biogenetic and Psychosocial Research Studies on Schizophrenia Reported Since 1961; and Percentage (in Parentheses) of Total Schizophrenia Studies by Decade*

Research Category	Total <i>n</i> = 33,648	1961–1970 <i>n</i> = 2,014	1971–1980 <i>n</i> = 5,854	1981–1990 <i>n</i> = 10,663	1991–2000 <i>n</i> = 15,117
Genetics	1285 (3.8)	54 (2.7)	197 (3.4)	481 (4.5)	553 (3.7)
Biochemistry	842 (2.5)	95 (4.7)	194 (3.3)	260 (2.4)	293 (1.9)
Neuropsychology	501 (1.5)	0	19 (0.3)	118 (1.1)	364 (2.4)
Socioeconomic Status	110 (0.3)	12 (0.6)	33 (0.6)	36 (0.3)	29 (0.2)
Stress	313 (0.9)	9 (0.4)	50 (0.9)	128 (1.2)	126 (0.8)
Child Rearing Practices or Parent Child Relations	269 (0.8)	33 (1.6)	84 (1.4)	90 (0.8)	62 (0.4)
Child Abuse, Sexual Abuse, Physical Abuse, Child Neglect, Emotional Abuse, or Family Violence	28 (0.1)	0	1 (0.0)	10 (0.1)	17 (0.1)
Ratio of first three categories to last two	8.8	4.5	4.8	8.6	15.3

*Note.* Data gathered from PsycINFO using exact, that is, “matched,” headings.

that none of the 19,099 studies conducted before 2001 on child abuse, sexual abuse, physical abuse, emotional abuse, child neglect, or family violence was related to childhood schizophrenia.

Studies that retrospectively examine the childhoods of adults diagnosed schizophrenic tend to search only for evidence of behavioral dysfunction (Neumann, Grimes, Walker, and Baum 1995). Rather than researching what was occurring in the lives of these children, findings are explained in terms of the “constitutional vulnerability” underlying schizophrenia.

A more recent study (Cannon et al. 2001) does investigate quality of relationships with parents in childhood but groups this variable under “symptoms,” with the implied preconception that any disturbance in the relationship is a result of the illness rather than a possible cause. The fact that the children diagnosed schizophrenic as adults were 2.7 times more likely than those who were not mentally ill as adults to have been in an institution or children’s home (a finding absent for those with affective psychosis as adults) did not lead to hypotheses about what might have been going on in these children’s lives to have caused these disruptions.

Researchers for the National Institute of Mental Health Genetics Initiative for Schizophrenia and Bipolar Disorders have demonstrated that after controlling for gender and age, traumatic brain injury (TBI) is significantly related ( $p = .008$ ) to being diagnosed schizophrenic, but not bipolar disorder or major depression (Malaspina et al. 2001). Their study found that 17% of adults diagnosed schizophrenic had suffered TBI. Despite having access to the details of the TBIs, including the patients’ age at the time and the nature of the injury, no mention is made of whether the injuries were accidental or purposefully inflicted. The only explanations considered are whether TBIs “cause a phenocopy of the genetic form of schizophrenia” or “lower the threshold for expressing schizophrenia in those with genetic vulnerability.” One in eight children in the United States between the ages of 10 and 16 have experienced aggravated assault (physical assault involving either use of a

weapon or injury), excluding violence within the family (Boney-McCoy and Finkelhor, 1995). A possible relationship (neurological or psychological) between any TBIs resulting from these assaults and subsequent schizophrenia is covered, however, by the assertion that “early illness features of schizophrenia such as agitation or psychosis might increase exposure to traumatic brain injury. If that is true then the head injury does not cause the schizophrenia” (Malaspina et al. 2001, p. 441).

The biopsychosocial formulation, with its assumption that the diathesis is predominantly or exclusively a genetic predisposition, has thus far not produced a balanced integration of the kind that might readily incorporate the research literature on TBI (accidental or intentional), neglect, loss, deprivation, or sexual abuse. It is beyond the scope of this paper to repeat the many critiques of studies investigating a genetic predisposition to schizophrenia (Boyle 1990; Joseph 2001; Rose 2001; Turkheimer 1998) or other adult disorders (Goldberg 2001). The model proposed here does not imply that early losses, stressors, neglect, and traumatic events are the only determinants of vulnerability to schizophrenic symptoms. In keeping with current multifactorial etiological models of schizophrenia (Tienari and Wynne 1994), our model recommends open-minded consideration and proper research investigation of whether severe adverse events in childhood might contribute, either independently or in interaction with the effects of genetic risk or perinatal factors (Kunugi, Nanko, and Murray 2001), to the production of a neurodevelopmental diathesis for schizophrenia. One example of the results of such open-mindedness is the recent finding that a deficit in performance of smooth pursuit eye-movement tasks, usually assumed to be a biological marker of the genetic predisposition to schizophrenia, is significantly related to physical and emotional abuse in childhood (Irwin, Green, and Marsh 1999).

We are merely proposing a more longitudinal, and therefore more inclusive, approach to the role of stressful life events than the current exclusive focus on perinatal events

and events immediately preceding the first overt psychotic episode. The British Psychological Society (Kinderman, Cooke, and Bentall 2000, p. 28) recently expressed our central hypothesis succinctly by adding one sentence to the traditional genetically based diathesis-stress formulation of schizophrenia: "Sensitivity to particular stresses may, of course, be at least partly a result of events that have happened previously in a person's life."

*Neurodevelopmental Theories  
of Schizophrenia*

Neurodevelopmental theories have gradually replaced neurodegenerative theories. The dysfunction identified in the brains of schizophrenics has now been shown to precede, rather than result from, schizophrenia (Harrison 1995). This could facilitate consideration of the possibility that adverse events in childhood play an etiological role. Even in this area, however, researchers limit investigation of the causes of the neurodevelopmental dysfunction to genetics and perinatal events (McGlashan and Hoffman 2000).

In "Schizophrenia: A Neural Diathesis-Stress Model," Walker and DiForio (1997) reiterate the popular position that stressors can exacerbate symptoms but do not constitute causal factors, and cite the usual evidence that vulnerability to schizophrenia is associated with heightened sensitivity to stressors. They go beyond previous reviews, however, by seeking to elucidate the actual nature of the rather enigmatic but frequently cited "constitutional vulnerability."

Walker and DiForio document that activation of the hypothalamic-pituitary-adrenal (HPA) axis is one of the primary manifestations of the stress response and that the adrenal cortex, stimulated by adrenocortrophic hormone (ACTH) from the pituitary, releases glucocorticoids (including cortisol in primates). The hippocampus contains a high density of glucocorticoid receptors and plays a vital role in the feedback system that modulates the activation of the HPA axis. Studies measuring glucocorticoids show that stressors of sufficient magnitude can produce a sensi-

zation effect instead of the expected habituation effect. "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).

The role of dopamine neurotransmission in the production of behavioral sensitization following exposure to stressors has since been further elaborated, leading to the acknowledgment that "experience-dependent effects may be an important ontogenetic mechanism in the formation, and even stability, of individual differences in DA system reactivity" (Depue and Collins 1999, p. 507).

Walker and DiForio (1997) report findings of higher baseline cortisol levels and a negative response to the dexamethasone suppression test (DST) (demonstrating preexisting HPA axis hyperactivation) in schizophrenics, and go on to demonstrate that schizophrenia appears to be associated with "a unique neural response to HPA activation" (p. 672). Having reviewed studies finding an association between cortisol release and severity of schizophrenic symptoms, they highlight research documenting reduced volume and cellular irregularities in the hippocampus of schizophrenics as further evidence that schizophrenic symptoms are related to dysregulation of the stress response.

Central to their argument for this "unique neural response" is the evidence that there are effects of the HPA axis on the synthesis, reuptake, and receptor sensitivity of dopamine, a neurotransmitter consistently linked to schizophrenia. Their review shows that stress exposure elevates not only the release of cortisol but of dopamine (DA) as well, that the magnitude of cortisol release and DA activity are related, that both DA administration and stress can produce sensitization, that HPA activation augments DA synthesis and receptors, and, synergistically, that DA can enhance HPA activation. In particular, "stress of sufficient magnitude permanently alters the modulation of the HPA axis, such that corticosterone release is augmented and hippocampal glucocorticoid receptors are changed. Thus

long-standing hypersecretion of corticosterone may serve to enhance DA receptors as well as DA release" (Walker and DiForio 1997, p. 676). Walker and DiForio conclude their review thus: "But the dearth of empirical research that addresses the biobehavioral aspects of stress responsivity in schizophrenia has been due, at least in part, to the lack of a theoretical framework that can generate testable hypotheses. We hope that the hypotheses proposed here will stimulate integrative research aimed at elucidating the nature of the diathesis-stress interaction at both the biological and behavioral levels" (p. 679).

This paper is one attempt at such an integrative approach. It responds specifically to their call "to clarify the nature of developmental changes in the HPA system, especially the HPA response to stress and its relation to symptoms" by "identifying the patient characteristics that predict sensitivity to stressors" (p. 679). We propose that one such patient characteristic may be exposure to severely adverse physical or emotional childhood events. As one example of how integration of such events into research and clinical practice regarding schizophrenia can be illuminating, this paper focuses on traumatic events in childhood, particularly sexual and physical abuse.

#### **THE RELATIONSHIP BETWEEN CHILDHOOD ABUSE AND SCHIZOPHRENIA**

The range of adult disorders in which child abuse or neglect have been shown to have an etiological role includes depression, anxiety disorders, posttraumatic stress disorder, eating disorders, substance abuse, sexual dysfunction, personality disorders, and dissociative disorders (Beitchman et al. 1992; Boney-McCoy and Finkelhor 1995; Kendler et al. 2000). The more severe the abuse, the greater is the probability of psychiatric disorder in adulthood (Fleming, Mullen, Sibthorpe, and Bammer 1999; Mullen, Martin, Anderson, Romans, and Herbison 1993). However, it is widely assumed (Read 1997) that child abuse is less related, or unrelated, to the more severe

psychiatric disorders, such as psychosis in general and schizophrenia in particular. While the effects of traumatic events in childhood are not uniquely linked to schizophrenic symptoms, the literature reviewed next suggests that the relationship between traumatic events in childhood and schizophrenia may be as strong, or stronger, than the relationships between traumatic events in childhood and other less severe adult disorders. Indeed, childhood abuse is related to most measures of severity of disturbance. Compared to other psychiatric patients, those who suffered childhood physical abuse (CPA) or childhood sexual abuse (CSA) are more likely to attempt suicide, have earlier first admissions and longer and more frequent hospitalizations, spend more time in seclusion, receive more medication, and have higher global symptom severity (Beck and van der Kolk 1987; Beitchman et al. 1992; Briere, Woo, McRae, Foltz, and Sitzman 1997; Bryer, Nelson, Miller, and Krol 1987; Goff, Brotman, Kindlon, Waites, and Amico 1991; Pettigrew and Burcham 1997; Read 1998; Read, Agar, Barker-Collo, Davies, and Moskowitz 2001; Sansonnet-Hayden, Haley, Marriage, and Fine 1987).

#### *Child Abuse among Psychiatric Inpatients*

In 13 studies of "seriously mentally ill" women the percentage that experienced either CSA or CPA ranged from 45% to 92% (Goodman, Rosenberg, Mueser, and Drake 1997). Another review, encompassing 15 studies totaling 817 female psychiatric inpatients, calculated that 64% reported either CPA or CSA, with 50% reporting CSA and 44% reporting CPA (Read, 1997). A study of girls in a child and adolescent psychiatric inpatient unit found that 73% had suffered either CSA or CPA (Ito et al. 1993). Read (1997) concluded that women in psychiatric hospitals are at least twice as likely as other women to have been abused as children. This may be a conservative estimate because general population studies, which often involve multiple screenings and extended interviews, tend to produce higher and more accurate rates (Jacobson 1989), and because psychiatric inpatients tend to under-

report abuse (Dill, Chu, Grob, and Eisen 1991; Read 1997).

Four studies of female inpatients, or outpatients with predominantly psychotic diagnoses, found incest prevalences from 22% to 46%, with a total of 112 out of 397 (28%) (Beck and van der Kolk 1987; Cole 1988; Muenzenmaier, Meyer, Struening, and Ferber 1993; Rose, Peabody, and Stratigas 1991).

Male inpatients report rates of CPA similar to their female counterparts (Jacobson and Richardson 1987; Rose et al. 1991). Male inpatient CSA rates range from 22% to 39% (Jacobson and Herald 1990; Rose et al. 1991; Sansonnet-Hayden et al. 1987; Wurr and Partidge 1996), and are at least double the rates of CSA in the general male population in England (Palmer, Bramble, Metcalfe, Oppenheimer, and Smith 1994) and the United States (Jacobson and Herald 1990).

Prevalence rates in a mixed gender sample of child and adolescent inpatients were CSA, 37%; CPA, 44%; emotional abuse, 52%; emotional neglect, 31%; and physical neglect, 61%. The CSA had a mean age of onset of 8 years and a mean duration of 2.1 years. The majority of the CSA was intrafamilial and involved penetration or oral sex. The CPA had a mean age of onset of 4.4 years, lasted an average 6.4 years, and involved physical injury in the majority of cases (Lipschitz et al. 1999).

#### *Child Abuse and Schizophrenia*

Among the "Recent Advances in Understanding Mental Illness and Psychotic Experiences" identified by the British Psychological Society (Kinderman et al. 2000) is the finding that "many people who have psychotic experiences have experienced abuse or trauma at some point in their lives" (p. 28). A growing body of research demonstrates this finding in relation to schizophrenia and child abuse.

*Research Measures.* CSA and CPA are significantly related to research measures of psychosis in general and schizophrenia in particular. The Psychoticism scale of the Symptom Checklist-90-R (SCL-90-R) is often found to be more strongly related to child abuse than any of the other clinical scales (Bryer

et al. 1987; Ellason and Ross 1997; Lundberg-Love, Marmion, Ford, Geffner, and Peacock 1992; Swett, Surrey, and Cohen 1990). The Schizophrenia scale of the Minnesota Multiphasic Personality Inventory (MMPI) has been found to be significantly elevated in adults who suffered CPA (Cairns 1998) and incest (Scott and Stone 1986). Chronically mentally ill women who had been abused score higher than those who were not abused on the Beliefs and Feelings Scale, measuring psychotic symptoms (Muenzenmaier et al. 1993).

CSA is also related, in the general population, to the Unusual Experiences component (including perceptual aberrations) of schizotypy (Startup 1999). Perceptual Aberration Scale scores, which are predictive of clinical psychoses, are 10 times more common in young adults who were maltreated as children than those who were not maltreated (Berenbaum 1999).

One of the rare studies of the genetics of schizophrenia that has evaluated the families adopting the offspring of parents with schizophrenia found that only 4% of those children raised by "healthy" adoptive families were diagnosed as "severe + psychotic," compared to 34% of the children raised by "disturbed" adoptive families (Tienari 1991). Among the family dimensions correlated to the children's mental health, at the  $p < .0001$  level, were "expelling relation to offspring" (i.e., rejection) and "conflict between parents and offspring." Tienari concluded that "in healthy rearing families the adoptees have little serious mental illness, whether or not their biological mothers were schizophrenic" (p. 463). The level of functioning of the adopting families produced an improvement chi-square (measuring the extent to which a variable gives more information, that is, improves the model) of 40.22 ( $p = .000$ ) while the improvement chi-square of the genetic variable (whether or not the biological mother was schizophrenic) was only 5.78 ( $p = .016$ ). Thus the dysfunction of the family, and the maltreatment of the child implied thereby, had 7 times more explanatory power than genetic predisposition.

*Clinical Diagnoses.* Children who are diagnosed schizophrenic as adults are significantly more likely than the general population

to run away from home (Malmberg, Lewis, and Allebeck 1998), to attend child guidance centers (Ambelas 1992), and to be placed in children's homes (Cannon et al. 2001). In a 30-year study of 524 child guidance clinic attenders, 35% of those who became schizophrenic as adults had been removed from their homes because of neglect, twice as many as any other diagnostic group (Robins 1966). Among women inpatients diagnosed schizophrenic, 60% had suffered CSA (Friedman and Harrison 1984). Among chronically hospitalized psychotic women, 46% had suffered incest (Beck and van der Kolk 1987). In a mixed-gender sample of adults diagnosed schizophrenic, 83% had suffered CSA, CPA, or emotional neglect (Honig et al. 1998). Even a chart review (which underestimates abuse, especially for men and schizophrenics; see Clinical Implications; Assessment) found that 48% of women inpatients diagnosed schizophrenic (but only 6% of the men) had suffered definite or probable CSA, and that 52% of the women and 28% of the men had suffered definite "parental violence" (Heads, Taylor and Leese 1997). Of 5,362 children, those whose mothers had poor parenting skills when the children were four were significantly more likely to be schizophrenic as adults (Jones et al. 1994).

Parental hostility precedes, and is predictive of, schizophrenia (Rodnick, Goldstein, Lewis, and Doane 1984). In families where both parents expressed high criticism toward their child, 91% of disturbed but nonpsychotic adolescents were diagnosed (within 5 years) as having schizophrenia or a related disorder, whereas in families in which both parents were rated low on criticism only 10% of similarly disturbed but nonpsychotic adolescents were similarly diagnosed (Norton 1982).

*Specific Symptomatology.* A community survey found that 46% of those with three or more Schneiderian symptoms of schizophrenia had experienced CPA or CSA, compared to 8% of those with none (Ross and Joshi 1992). Inpatients who suffered CSA or CPA are significantly more likely than other inpatients to experience voices commenting, para-

noid ideation, thought insertion, ideas of reference, visual hallucinations, or reading others' minds (Ross, Anderson, and Clark 1994). An outpatient study found that hallucinations, across all sensory modalities, are significantly more common in patients who suffered either CSA or CPA than those who did not (Read 2001). In a Dutch study 65% of schizophrenics related the initial onset of hearing voices to traumatic events such as witnessing people being shot in a war, the suicide of a close family member, and CSA and CPA. Furthermore, "the disability incurred by hearing voices is associated with (the reactivation of) previous trauma and abuse" (Honig et al. 1998, p. 646).

Hallucinations have been found to be particularly common among incest survivors (Ensink 1992, pp. 109–138). Ellenson (1985) identified, in 40 women, a "post incest syndrome," including hallucinations, which he reported as "exclusively associated with a history of childhood incest" (p. 526). This was replicated in 10 other incest cases (Heins, Gray, and Tennant 1990). Read and Argyle (1999) found that all female incest survivors in their inpatient study experienced hallucinations and that incest survivors were significantly more likely to do so than those subjected to extrafamilial CSA.

Both parental absence and institutionalization in childhood are related to specific schizophrenic symptoms later in life, with a particularly strong relationship, for boys, to thought disorder, hallucinations, delusions, and hebephrenic traits (Walker, Cudeck, Mednick, and Schulsinger 1981).

Not only do abused psychiatric patients experience schizophrenic symptoms more often than nonabused patients, they do so at a younger age (Goff et al. 1991). Among children admitted to a psychiatric hospital, 77% of those who had been sexually abused were diagnosed psychotic, compared to 10% of those who had not been abused (Livingston 1987). Adolescent inpatients that have experienced CSA are more likely to hallucinate than those who have not (Sansonnnet-Hayden et al. 1987). Famularo, Kinscherff, and Fenton (1992) found that hallucinations were significantly



more likely in a group of severely maltreated 5–10-year-olds than in a control group and, in keeping with an earlier study (Ensink 1992), that “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” (p. 866). Read and Argyle (1999) found that the content of 54% of schizophrenic symptoms in adult inpatients who had been abused were obviously related to child abuse. Examples included command hallucinations to self-harm being the voice of the perpetrator.

*Mediating Variables.* The relationship between child abuse and psychiatric sequelae in adulthood remains after controlling for potentially mediating variables such as socioeconomic status, marital violence, parental substance abuse and psychiatric history, and other childhood traumas (Boney-McCoy and Finkelhor 1995; Downs and Miller 1998; Fleming et al. 1999; Kendler et al. 2000; Pettigrew and Burcham, 1997). After controlling for factors related to disruption and disadvantage in childhood, women whose CSA involved intercourse were 12 times more likely than nonabused females to have had psychiatric admissions (Mullen et al. 1993). Among women at a psychiatric emergency room, 53% of those who had suffered CSA had “nonmanic psychotic disorders (e.g., schizophrenia, psychosis NOS)” compared to 25% of those who were not victims of CSA. The corresponding CPA findings were 49% and 33%. After controlling for “the potential effects of demographic variables, most of which also predict victimization and/or psychiatric outcome,” CSA was related to nonmanic psychotic disorders ( $p = .001$ ) and depression ( $p = .035$ ) but not manic or anxiety disorders (Briere et al. 1997).

#### THE NEURODEVELOPMENTAL EFFECTS OF CHILDHOOD ABUSE AND NEGLECT

Neurodevelopmental research has established that, because of the brain’s extreme malleability and sensitivity to experience in early childhood, traumatic events in the first

few years of life can have long-term impacts on emotional, behavioral, cognitive, social, and physiological functioning (Heim et al. 2000; Ito, Teicher, Glod, Ackerman 1998; Perry, Pollard, Blakely, Baker, and Vigilante 1995). This is particularly likely if the events are severe, unpredictable, or persistent (Perry 1994). Self-regulatory systems, such as the HPA axis, seek to return the brain to prestress levels of sensitivity to stress. However, repeated stressors can sensitize neurobiological process so that the homeostasis returned to is at a higher level of responsivity. This can even occur following single instances of sufficient magnitude or unpredictability because the resultant sensitization process means that stimuli similar to the original traumatic event can elicit the same response as the original trauma; as far as the brain is concerned the stressors are ongoing.

Two interacting patterns of response to traumatic events in childhood have been identified. The first is the hyperarousal (or “fight-or-flight”) response:

This sensitization of the brain stem and mid-brain neurotransmitter systems also means the other critical physiological, cognitive, emotional, and behavioural functions which are mediated by these systems will become sensitized. . . . The child will very easily be moved from being mildly anxious to feeling threatened to being terrorised. In the long run, what is observed in these children is a set of *maladaptive* emotional, behavioral, and cognitive problems, which are rooted in the original *adaptive* response to a traumatic event. (Perry et al. 1995, p. 277)

Perry (1994) found that 29 out of 34 abused children had a resting heart rate of at least 10 beats per minute higher than normal, indicating hyperarousal. This resting tachycardia, and other signs of autonomic nervous system lability, have also been found in both adults (Zahn, Frith, and Steinhauer 1991) and children (Zahn et al. 1997) diagnosed schizophrenic.

The second response to stress, more common in girls and younger children, is the “dissociative” continuum. This is different from the hyperarousal continuum in that it

involves decreasing blood pressure and heart rate, and dissociative “freeze” or “surrender” responses. These responses may be adaptive in the immediate situation but, via a similar process of sensitization, in different brain systems than the hyperarousal pattern, become maladaptive later. Measures of heart rate (baseline and reactivity) show that in traumatized children the adaptive style (hyperarousal or dissociation, or combination thereof) first employed in the face of traumatic events persists for at least 6 months after the trauma (Perry 1994; Perry et al. 1995).

*Evidence That Child Abuse Can Cause Hyper-responsivity of the HPA Axis*

Walker and DiForio’s (1997) neural model of schizophrenia emphasizes that when exposure to stressors persists and heightened glucocorticoid release is chronic, there can be permanent changes in the HPA axis. Child abuse researchers believe the activation of the HPA axis and a concomitant peripheral release of hormones including ACTH, epinephrine (adrenaline), and cortisol are key components in the sensitization of the stress response in traumatized children (Perry and Pate 1994). HPA dysregulation may occur by other means than heightened release of glucocorticoids. Yehuda (1998b) proposes an additional pathway in PTSD, in which low cortisol response to traumatic events is followed by decreased basal cortisol levels that lead to an increase in both the numbers and responsivity of glucocorticoid receptors, resulting in increased negative feedback regulation and, ultimately, a sensitized HPA axis.

DeBellis, Chrousos et al. (1994) found lower basal, net ovine CRH-stimulated, and total adrenocorticotrophic hormones (ACTH) levels in 13 sexually abused girls than in controls. ACTH is the hormone released by the pituitary to stimulate the adrenal cortex to release corticosteroids. DeBellis et al. concluded that sexual abuse, in addition to causing psychiatric morbidity, was associated with clear and sustained changes in the dynamics of the HPA axis. Dysregulation of cortisol secretion by the adrenal cortex component of

the HPA axis has also been found in abused girls in comparison to controls (Putnam, Trickett, Helmers, Dorn, and Everett 1991).

Evidence is now emerging that the HPA changes induced by traumatic events in childhood can persist into adulthood. Women (ages 18–45) who had suffered CSA or CPA exhibit increased pituitary–adrenal and autonomic responses to stress compared to nonabused women: “Our findings suggest that hypothalamic–pituitary–adrenal axis 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” (Heim et al. 2000, p. 592). As is often the case, however, women with a history of psychosis were not included in this study.

*Evidence That Child Abuse Can Cause Neurotransmitter Abnormalities*

*Dopamine.* Walker and DiForio (1997) argued that adults diagnosed with schizophrenia are so reactive to stressors because stress-induced dysregulation of the HPA axis causes increased dopamine (DA) receptor densities and DA release. These dopaminergic systems are very important in interpretation of stress and threat-related stimuli, and, therefore, the development of persecutory delusions (Spitzer 1995). If childhood traumatic events can cause permanent dysregulation of the HPA axis it follows that it may cause, in some schizophrenics who have been abused as children, the DA abnormalities cited as evidence of a biological etiology of schizophrenia. In animal studies various stress paradigms have demonstrated alterations in dopamine metabolism, dopamine receptor densities, and sensitivity (Perry 1998). Studies have also demonstrated psychostimulant- and stress-induced sensitization of these dopaminergic symptoms and found that they can become increasingly sensitive to a constant stimulus (Perry 1998).

Greater synthesis of DA, norepinephrine (NE), and epinephrine has been found in sexually abused girls than in controls. When all significant biochemical measures were ad-

justed by the covariate effect of height, only homovanillic acid remained significant (DeBellis, Lefter et al. 1994, p. 320). Homovanillic acid is a metabolite of DA, which appears, therefore, to play an orchestrating role in the higher catecholamine functional activity of abused children. It was concluded that elevated DA metabolism may be an adaptive response to environmental stress in these sexually abused girls.

Galvin et al. (1991, 1995) measured dopamine beta hydroxylase (DBH), a neurotransmitter enzyme active in the conversion of DA to NE, in psychiatrically hospitalized boys. Lower levels of DBH were found in those who had suffered CPA, CSA, or neglect early in childhood than in those abused later or never abused. In one study by Galvin et al. (1991) the difference was found only in those 3 years old or younger, and in another the difference emerged at 6 years (Galvin et al. 1995), confirming the importance of the malleability of the brain in the early years. Galvin et al. (1995) concluded that low DBH may be a marker for the effects of prolonged exposure to stress associated with the effects of early maltreatment on the HPA.

*Serotonin.* The fact that serotonin (SN) serves as an inhibitor of DA is the basis of the newer, "atypical," neuroleptics (such as clozapine) since they increase SN activity and decrease DA activity (Kane, Honigfeld, Singer, and Meltzer 1989). Exposure to "adverse-rearing conditions" (including both neglect, in the form of low levels of praise and encouragement, and abuse, as measured by frequent parental anger and physical punishment) is related to lower density SN receptors (Pine et al. 1996) and to dysfunctional SN response to the fenfluramine challenge (Pine et al. 1997).

*Evidence That Child Abuse Can Cause Structural Abnormalities in the Brain*

*Hippocampus.* The hippocampus is crucial for learning and memory. It is very sensitive to stress activation, and threat alters the ability of the hippocampus and connected cortical areas to store certain types of information

(e.g., verbal) while efficiently storing others (e.g., nonverbal) (Perry 1998). The hippocampus is sufficiently sensitive that, under certain stress conditions, its capacity to dampen the reactivity of the HPA axis can be permanently reduced (Walker and DiForio 1997). Hippocampal damage is a common finding in adult schizophrenics (Chua and Murray 1996) and is central to Walker and DiForios' neural model of schizophrenia. Suddath, Christison, Torrey, Casanova, and Weinberger (1990) found reduced hippocampal volume in the affected twin in 14 of 15 cases of monozygotic (MZ) twins discordant for schizophrenia. The reductions were predominantly in the anterior region, which in animals has a greater role than the posterior hippocampus in regulating cortisol levels (Regestein, Jackson, and Peterson 1986).

It is significant, from a neurodevelopmental perspective, that hippocampal damage has recently been demonstrated in first-episode cases of schizophrenia (Velakoulis et al. 1999). Is it possible that the damage to the hippocampus, for those schizophrenics who suffered CPA or CSA, is caused by that abuse? There is now considerable data showing that child abuse can cause dysfunction of the limbic system (hippocampus, amygdala, and septum) (Teicher, Ito, Glod, Schiffer, and Gelbard 1996). Teicher, Glod, Surrey, and Swett (1993) tested 253 adult outpatients on the Limbic System Checklist-33 (LSCL-33), a measure that includes brief hallucinatory events, and visual and dissociative disturbances, and is highly correlated with Psychoticism on the (SCL-90-R). Abuse had a "prominent effect" on LSCL-33 scores ( $p < .0001$ ). CPA was associated with a 52% increase in LSCL-33 scores, CSA with a 66% increase, and combined CSA and CPA with a 147% increase. This research team concludes not only that early deprivation or abuse could result in neurobiological abnormalities responsible for subsequent psychiatric disorders but adds that these disorders include refractory psychosis (Teicher et al. 1997).

Further support comes from research into childhood-onset schizophrenia. As part of the NIMH project mentioned earlier, multislice

proton magnetic resonance spectroscopic imaging of multiple cortical and subcortical regions found neuronal damage or malfunction in the hippocampal and the dorsolateral prefrontal cortex in 14 cases of childhood-onset schizophrenia. Decreased hippocampal volume in childhood-onset schizophrenia has been shown to be progressive (Jacobsen et al. 1998). The researchers concluded that their findings were evidence of a biological continuum between childhood- and adult-onset schizophrenia (Bertolino et al. 1998).

*Cerebral Atrophy and Ventricular Enlargement.* Among the most consistently reported abnormalities in adults diagnosed schizophrenic are ventricular enlargement and cerebral atrophy (Harrison 1995). The NIMH team investigated whether in childhood-onset schizophrenia atrophy occurs in those parts of the brain where it has been found to occur with adult schizophrenics. They found this to be the case for almost all areas studied, including the midsagittal thalamic area (Frazier et al. 1996; Rapoport et al. 1997), the vermis and midsagittal inferior posterior lobe (Jacobsen et al. 1997), and the right temporal lobe, bilateral superior temporal gyrus and posterior superior temporal gyrus, right anterior superior temporal gyrus (as well as the hippocampus) (Jacobsen et al. 1998), and smaller total cerebral volume (Alaghband-Rad, Hamburger, Giedd, Frazier, and Rapoport 1997).

Ventricular enlargement was also found (Frazier et al. 1996; Gordon et al. 1994) and shown to be progressive into adolescence (Rapoport et al. 1997) although Rapoport and colleagues note, "It is unlikely that the degree of change . . . would be sustained, as that would produce improbably large ventricular volume later in life" (p. 901). The hypothesis that ventricular enlargement found in schizophrenics not only has its onset during childhood but has ceased to progress by the time schizophrenia begins is supported by studies spanning 2–8 years which show static ventricular size in adult schizophrenics (Illowski, Juliano, Bigelow, and Weinberger 1988; Vita, Sacchetti, Valvassori, and Cazzullo 1988). It was concluded that these neurobiological as-

sociations support the continuity of early-onset schizophrenia with the later-onset disorder (Alaghband-Rad et al. 1997). Despite acknowledging the existence of a developmental period uniquely sensitive to pathological effects (Rapoport et al. 1997), no mention is made of the possible causes of these effects.

Perhaps this evidence that the brain changes cited in support of a biological etiology of schizophrenia can begin very early in life applies only to that quite small percentage of schizophrenics diagnosed in childhood. There is data to suggest, however, that this might be the case for most or all adult schizophrenics who have enlarged ventricles. Johnstone et al. (1989) found that of eight tests of cognitive functioning, the only one significantly related to ventricular enlargement in adult schizophrenics was a memory test relating to 20–30 years prior to testing. They conclude that the findings suggest that the ventricular anomalies in schizophrenia may arise at a time when the brain is still developing.

*Reversed Cerebral Asymmetry.* Many adult schizophrenics have reversed structural cerebral asymmetry, with the left side of the brain smaller, rather than larger (as is the case for most people), than the right (Chua and Murray 1996). This reversed asymmetry is related to early-onset (Crow, Colter, Frith, Johnstone, and Owens 1989). There is strong evidence that the left hemispheres of many schizophrenics are dysfunctional (Gur and Chin 1999; Petty 1999). Consistent with other studies of hippocampal abnormalities in the left hemisphere (Jeste and Lohr 1989; Velakoulis et al. 1999), the enlarged ventricles found by Suddath et al. (1990) were more likely to be on the left side. The NIMH childhood-onset schizophrenia team have found enlargement of the left ventricular horn area (Gordon et al. 1994).

In contrast to the almost exclusively biogenetic framework of the NIMH study, another research team (Teicher et al. 1996) has proposed

a model through which we can perhaps more completely understand the sequelae of child abuse, by considering the possible effects of abuse on brain development. . . .

This framework provides an important bridge between psychological and biological theories of psychopathology and . . . hope that this hypothesis helps foster the development of a more comprehensive understanding of the mechanisms through which early severe stress may produce pervasive psychiatric difficulties. (p. 59)

Nonspecific EEG abnormalities have been found in abused children, including victims of CPA without head trauma (Green, Voeller, and Gaines 1981), incest survivors (Davies 1979), and psychologically abused children (Teicher et al. 1997). Teicher and colleagues highlight the relationship of EEG abnormalities to limbic system dysfunction and hemispheric asymmetry. While EEG abnormalities were found in 27% of nonabused child and adolescent inpatients, they were found in 54% of abused inpatients and 72% of those severely abused. Left-sided abnormalities were found to be "2.5-fold more prevalent" than right-sided abnormalities in the abused group, even more so for the psychologically abused group (Ito et al. 1993, p. 405). They also found reversed asymmetry in 15 severely abused children. These children also had higher levels of left hemisphere coherence (assumed to stem from a deficit in left cortical differentiation) than controls or their own right hemispheres. They conclude: "EEG coherence measures appear well suited for detecting the relatively subtle structural brain abnormalities that presumably occur in schizophrenia" (Ito et al. 1998).

Adult survivors of CSA and CPA have been shown, by magnetic resonance imaging, to have reduced left-sided hippocampal volume compared to nonabused adults, after controlling for age, sex, race, handedness, education, SES, body size and alcohol abuse (Bremner et al. 1997).

Teicher et al. (1997) mention that early stress activates NE, SN and DA, which are asymmetrically distributed and, in keeping with the early malleability of the brain, point out that dendritic growth in the left hemisphere surpasses that of the right hemisphere at about 6 months, and that, therefore, abuse from 6 months until 3 to 6 years of age may

have the greatest differential effect on the left hemisphere (Ito et al. 1998; Teicher et al. 1997)

None of the 10 papers in a recent edition of *Schizophrenia Bulletin* (titled "Is Schizophrenia a Lateralized Brain Disorder?") consider any childhood events (other than neonatal stressors) as possible contributing factors to the lateralization (Gur 1999).

Finally, it seems relevant to note a recent study of two sets of "indices of developmental abnormality which are consistently reported to be more frequent in patients with schizophrenia than in healthy controls" (Lawrie et al. 2001, p. 524). None of 26 tests for neurological abnormality was related to either of two measures of genetic liability for schizophrenia. The other set of indices, minor physical anomalies, were more frequent in those with least genetic liability.

#### *Summary*

In each of the areas discussed the biochemical and neuroanatomical abnormalities particularly associated with schizophrenia, including the key components of Walker and DiForio's neural model, have been shown to be consistent with stress-induced damage or dysfunction and have been specifically linked to child abuse or neglect. Additional support for our traumagenic, neurologically mediated pathway to hypersensitivity to stressors and to schizophrenic symptoms is provided by the fact that much of the damage or dysfunction was not only found in abused children but also in children diagnosed schizophrenic. Furthermore, a recent study, described as the "first direct examination of the longitudinal relationship between psychotic symptoms in childhood and adulthood," found that psychotic symptoms in 11-year-old children were highly predictive of schizophrenic symptoms (but not of mania or depression) in adulthood (Poulton et al. 2000). A review of research into childhood-onset schizophrenia "confirms the hypothesis that the disorder is the same one that affects adults" (Merry and Werry 2000).

Investigation of our hypothesis that some children diagnosed as schizophrenic, and showing the same brain damage or dysfunction as

adult schizophrenics, have been maltreated would be facilitated if researchers of childhood-onset schizophrenia would inquire into the life circumstances of the children. While we agree with Murray (1994) that focusing on the neurodevelopmental aspects of schizophrenia may lead us to the “true” dementia praecox, we suggest, on the basis of the evidence reviewed here, that such a discovery is more likely if we ask what adverse life events or circumstances might be related to the “psychotic deviation in brain development” (Rapport et al. 1997, p. 901).

*Evidence That Child Abuse Can Cause Deficits in Cognitive Functioning*

Another body of research that appears to lend some support to a TN approach concerns the deficits in cognitive function that result from the trauma to the developing brain discussed earlier in this paper. It must be acknowledged that the studies presented in the following section have been selected to demonstrate that there appears to be sufficient preliminary evidence, albeit sometimes only correlational, to warrant more rigorous investigation.

*Deficits in Verbal Functions.* Because damage to the left hemisphere is common in both adult schizophrenics and victims of child abuse, we should also expect to see reduced performance, in both groups, in functions dependent on that hemisphere, such as verbal learning and memory. Hippocampal damage or dysfunction, also common to both groups, predicts difficulties encoding cognitive information.

A recent review of the literature on cognitive impairment in schizophrenia (Heinrichs and Zakzanis 1998) found global verbal memory impairment to be the most consistent finding of the 22 domains reviewed. The reduced verbal intelligence of adult schizophrenics, compared to their own nonverbal (or “performance”) intelligence and that of control groups, has been repeatedly demonstrated (Rains, Sauer, and Kant 1995) and is, within that population, correlated with dysfunction in the left inferior frontal cortex (Stevens,

Goldman-Rakic, Gore, Fulbright, and Wexler 1998) and with reversed asymmetry (Luchins, Weinberger, and Wyatt 1982). A review of the relevant literature concludes that the structural abnormalities seen in adult schizophrenics occur in childhood (Chua and Murray 1996):

Cerebral ventricular enlargement is the best replicated finding and this tends to be associated with impairment of neuropsychological performance. The idea that these abnormalities have a neurodevelopmental origin gains indirect support from the, admittedly less consistent, evidence of abnormalities of cerebral asymmetry and of neural migration in adult schizophrenics, as well as from the better established behavioural, psychomotor, and cognitive impairments reported in pre-schizophrenic children. (p. 547)

The question for the TN model is “Do abused children, like children who become schizophrenic in adulthood, perform poorly on verbal, compared to nonverbal tasks?” Children subjected to CSA or CPA have been shown to be 6 times more likely, and those psychologically abused 8 times more likely, to have less verbal than visuospatial ability than to have less visuospatial than verbal ability (Ito et al. 1993). Perry (1999) reports that 108 children raised in chronically traumatic environments performed significantly worse on the Verbal subscale of the Wechsler Intelligence Scale for Children than they do on the Performance subscale.

*Intellectual Decline in Childhood.* People diagnosed as schizophrenic, but not those diagnosed with bipolar or unipolar depression, show a progressive decline in intelligence and educational performance from their premorbid level to a lower but stabilized level (Goldberg et al. 1993). Until recently it was believed that this decline is somehow a consequence of the “illness” or at least was not evident until after its onset (Elliott and Sahakian 1995). It has, however, been found that educational deficits in adult schizophrenics, compared to controls, can be identified by age eight (Jones, Rodgers, Murray, and Marmont 1994). Far from being a consequence of the schizophrenic illness, the deficit exists well before onset

and does not deteriorate after onset (Russell, Munro, Jones, Hemsley, and Murray 1997).

Furthermore, there is evidence that these children are not born with the deficit but that intellectual functioning in children who become schizophrenics as adults declines during childhood. A study with 547 participants found that the 10% with substantial IQ declines from age 4 to 7 had a rate of psychotic symptoms, at age 23, nearly 7 times as high as the rate for other persons, and that they were not more likely to manifest symptoms of mania, depression, anxiety disorders, antisocial personality disorder, or alcohol or drug abuse (Kremen et al. 1998). "Parallels between the present study and studies of schizophrenia further suggest that our findings are relevant to schizophrenia. . . . If childhood IQ decline is specific for schizophrenia and not just psychotic symptoms, this explanation would also be consistent with the increasingly accepted notion of schizophrenia as a neurodevelopmental disorder" (p. 676). Other reviewers (Doody, Goetz, Johnstone, Frith, and Owens 1998) posit "a form of schizophrenia which manifests in childhood with cognitive impairment prior to the onset of psychotic symptoms. Such a hypothesis is consistent with current neuro-developmental theories of schizophrenia and lends support to a specific cognitive impairment of a non-progressive nature being associated with the disease" (p. 403).

Finally, 23% of childhood-onset schizophrenia cases (but only 9% of childhood bipolar disorder cases) have been found to have IQs of less than 80 (Werry, McLellan, and Chard 1991), with none of the first-degree relatives or grandparents of any of the schizophrenia cases having been diagnosed schizophrenic.

A review of 204 studies (Heinrichs and Zakzanis 1998) concluded that the literature on neurocognition in schizophrenia is limited and often inadequate. Even basic participant attributes, such as age, education and gender, were not reported by some studies. As was the case for the biochemical and neurological literatures, insufficient attention is paid, even in this more psychological domain, to psycho-

social events that might have an etiological role in the cognitive impairments discussed in this paper.

An additional area worthy of further theoretical and empirical study is the possibility of a relationship between child abuse and the development of "theory of mind" deficits and social cognition abnormalities, especially in relation to the development of persecutory delusions (Blackwood, Howard, Bentall, and Murray 2001; Frith and Corcoran 1996; Kinderman and Bentall 2000).

### OTHER RESEARCH IMPLICATIONS

The TN Model may possibly be of assistance in understanding the heterogeneity of schizophrenia by linking phenomenological subsets to their neuropsychological concomitants (Mortimer 1992). As an illustration we wish to open discussion, and encourage research, in relation to positive and negative symptoms.

#### *Pathways to Positive and Negative Schizophrenic Symptoms*

Having found that positive (e.g., hallucinations, delusions) and negative (e.g., social withdrawal, anhedonia) symptoms of schizophrenia are negatively correlated with each other, Andreasen and Olsen (1982) argued that these symptoms form the basis of clearly defined schizophrenic subtypes. Ross et al. (1994) found that abused inpatients are significantly more likely than nonabused inpatients to experience positive symptoms of schizophrenia, and suggested that "there may be at least two pathways to positive symptoms of schizophrenia. One may be primarily endogenously driven and accompanied by predominant negative symptoms. The other may be primarily driven by childhood social trauma and accompanied by fewer negative symptoms" (p. 491). It is also possible, however, that both positive and negative subtypes are related to child abuse, with two different pathways to negative or positive symptoms in adulthood

beginning with the two patterns of stress response (hyperarousal in males, and dissociation in females).

Positive symptoms are related to “an underlying pathologic process that is predominantly neurochemical,” and are more responsive to dopamine-targeted neuroleptics and less related to cerebral atrophy than negative symptoms (Andreasen and Olsen 1982, p. 790). Dopamine has “an increased relative importance” in dissociative responses to child abuse compared to hyperarousal responses (Perry, Pollard, Blakely, Baker, and Vigilante 1995).

Just as the pathway to adulthood hallucinations, delusions, and dissociative symptoms may begin with a predominantly dissociative response to traumatic events in childhood and be mediated predominantly by biochemical processes (especially dopaminergic), it may be possible to map a second pathway, mediated primarily by atrophy of the brain, from the hyperarousal response to child abuse through to the schizophrenia subtype with predominantly negative symptoms.

Anxiety and oversensitivity in preschizophrenic children correlates with passivity symptoms in adulthood (Cannon, Kargin, Jones, Hollis, and Murray 1995). With repeated traumatic events children can switch from high to low autonomic responsivity in adolescence (Perry et al. 1995). This shift might somehow be related to neuronal loss (“pruning”) in adolescence (Andersen and Teicher 2000; McGlashan and Hoffman 2000).

#### *Specificity and Severity*

An apparent weakness of the TN model of schizophrenia is that the majority of adults who were abused as children never display schizophrenic symptoms, and not all adults diagnosed schizophrenic suffered traumatic events or neglect as children. Indeed, child abuse is related to many other diagnoses besides schizophrenia (Beitchman et al. 1992; Mullen et al. 1993).

As noted earlier, the severity of child abuse is related to the probability of developing nonpsychotic adult disorders. Similarly, the severity of the abuse (e.g., age at onset,

degree of violence, duration, and intrafamilial abuse) may partly determine who does and does not develop schizophrenic symptoms. In an unreplicated study of 100 incest survivors, a cumulative trauma-score was significantly higher in those who later experienced auditory or visual hallucinations (Ensink, 1992, pp. 109–138). In keeping with the evidence provided earlier that severe abuse in the first 6 years of life is particularly likely to cause changes in the HPA axis, this study also found that CSA before age seven involving physical aggression and abuse from multiple family members was the most powerful predictor of auditory hallucinations.

Our TN model would hypothesize that for a small proportion of traumatized children, especially those who suffer severe, ongoing abuse which commences in the first 6 years of life, dissociative coping mechanisms will not be sufficient to prevent overtly psychotic symptoms in childhood (childhood-onset schizophrenia). A far greater number will struggle through childhood and early adolescence with high levels of dissociative and hyperarousal responses to stressors reminiscent of the original traumatic events, and multiple other symptoms, but no overt psychosis, until they hit the multiple stressors of late adolescence. Among the best childhood predictors of schizophrenia (but not of affective psychosis) are “abnormal suspiciousness or sensitivity,” “social withdrawal” and “disturbance of relationship with peers” (Cannon et al. 2001). In cases where these predictors are the result of traumatic events such a frightened and lonely existence offers little protection against the familial conflicts (Norton 1982; Rodnick et al. 1984) and extrafamilial social and sexual demands of mid-to-late adolescence that can reactivate the trauma response and thereby trigger first episodes of schizophrenia. Indeed, the best predictors of whether 18-year-old males will later develop schizophrenia, besides having run away from home as children, are being “more sensitive than others,” and having “fewer than two friends” and “no steady girlfriend” (Malmberg et al. 1998). As dissociation is, in the face of new stressors, joined by inaccurate interpretations of others’ behavior (Blackwood et al. 2001), there is no avenue



for checking reality with, or receiving support from, trusted peers.

Furthermore, it has just been demonstrated that within an adult schizophrenia sample CSA is associated with poorer psychosocial functioning (Lysaker, Meyer, Evans, Clements, and Marks 2001).

#### *A "Posttraumatic Dissociative Psychosis"?*

Many researchers have challenged the reliability, validity, and clinical utility of the "schizophrenia" construct (Bentall 1990; Boyle 1990; McGorry et al. 1995; Read 1997, 2000). Its heterogeneity alone dictates that a single primary cause, psychosocial or biogenetic, will never be discovered. It may be more constructive to focus on what people have in common etiologically rather than in terms of symptoms. Instead of separating the sequelae of abuse into putatively discrete diagnoses (PTSD, dissociative disorders, schizophrenia, etc.), it might be more productive to view them as interacting components of a long-term process beginning with adaptive responses to early aversive events and evolving into a range of maladaptive disturbances in multiple personal and interpersonal domains (Ensink, 1992). In other words: "Many investigators suggest that this diversity is more apparent than real and that a set of basic developmental disruptions link ostensible differences" (Putnam and Trickett 1997, p. 152).

There is a remarkable similarity, for instance, between Bleuler's description of the defining characteristic of schizophrenia as the "splitting" of psychic functions and modern definitions of dissociation, such as that in the DSM-IV, "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (American Psychiatric Association 1994, p. 766). In children, dissociation is highly correlated with the MMPI Schizophrenia scale (Friedrich, Jaworski, Huksahl, and Bengston 1997). Studies have demonstrated extensive overlap between dissociative symptoms, and the positive, or Schneiderian, symptoms of schizophrenia (Ellason, Ross, and Fuchs 1996). Significantly more positive symptoms occurred in 108 dissociative identity disorder cases than in 240 cases of

schizophrenia ( $p < .00001$ ) (Ellason and Ross 1995). "Many clinicians cannot differentiate dissociative symptoms from psychotic ones. It is an open research question whether reliable qualitative differentiations between dissociative and psychotic Schneiderian symptoms are possible" (Ross and Joshi 1992, p. 272).

Certainly the strong link between traumatic events and pathological dissociation (Putnam and Carlson 1997) and the huge overlap between dissociative and positive schizophrenic symptoms suggest that our current categorizations hinder rather than help our understanding of the sequelae of abuse. Nurcombe et al. (1996) have addressed this issue by postulating "dissociative hallucinosis" in adolescents as a severe variant of PTSD but distinct from schizophrenia. However, many of the effects of traumatic events on the developing brain which are so similar to the dysfunctions found in the brains of adult schizophrenics are also found in PTSD (Lipschitz, Rasmusson, and Southwick 1998; Sapolsky 2000).

Ellason and Ross (1997) emphasize that trauma-driven psychotic symptoms may occur in conjunction with other symptom clusters, including dissociative, mood, anxiety, somatic, borderline, and substance abuse symptoms. In accordance with our own TN model, Ross and Joshi (1992) add: "It will be of interest in future studies to determine whether the traumatized subgroup of various psychiatric disorders, including schizophrenia, exhibits a distinct phenomenology, family history, psychobiology, course, response to psychotherapy and medication, and prognosis" (p. 272). Others include our neurodevelopmental perspective in raising the same possibility (Ito et al. 1993): "Early childhood abuse may alter the course of limbic system maturation, producing neurobiological alterations, and these alterations may provide the biological substrate for a panoply of psychiatric consequences, including affective instability, inability to modulate anger, poor impulse control, limited stress tolerance, episodic aggression, dissociative disturbances, memory impairment, and hallucinatory phenomena" (p. 401).

Heins et al. (1990), however, suggest that the hallucinations they consistently found

in incest survivors were “pseudo-hallucinations” because they were “emanating from the mind” rather than “perceived as out there in the world” and were “vague, fleeting and ill-defined” (p. 563). They added that truly psychotic hallucinations in incest survivors are only found in those who are substance abusers. A recent inpatient study, however, found that none of the hallucinations of childhood abuse survivors matched either the exclusion criteria of Heins et al. for true hallucinations or the exclusion criteria of DSM-IV for “hallucinations characteristic of Schizophrenia” (American Psychiatric Association 1994, p. 275), and only 27% of the hallucinating abuse survivors were substance abusers (Read and Argyle 1999).

### CLINICAL IMPLICATIONS

#### *Assessment*

Clinicians identify an alarmingly small proportion of the abuse that is identified when researchers survey samples of psychiatric patients: 30% (Wurr and Partridge 1996); 28% (Lipschitz et al. 1996); 20% (Goodwin, Attias, McCarty, Chandler, and Romanick 1988); 12% (Jacobson, Koehler, and Jones-Brown 1987) and 0% (Rose et al. 1991). Emotional abuse may be similarly unrecognized by clinicians (Thompson and Kaplan 1999). Even when an inpatient admission form included a specific section for abuse history, only 32% of patients were asked the abuse questions (Read and Fraser 1998a). Adults diagnosed with schizophrenia are especially unlikely to be asked (Read and Fraser 1998a), particularly by biogenetically oriented clinicians (Young, Read, Barker-Collo, and Harrison, 2001).

The response of mental health staff once abuse has been identified has received little research attention. The inpatient study discussed above found that support during hospitalization (e.g., counseling, opportunity to discuss abuse-related issues, or information about abuse) was considered for only 12% of CSA cases and 8% of CPA cases. This was significantly less likely for diagnoses indicative of psychosis such as schizophrenia. Only 12%

were actually referred for postdischarge abuse counseling (Read and Fraser 1998a). Referral was not even considered for any of the schizophrenic patients.

In a survey designed to explore the reasons for this low level of inquiry and response, psychiatrists and psychologists identified “Too many more immediate needs and concerns” as the most common reason for not asking about abuse. The item “Client may be experiencing psychotic symptoms and imagine abuse that did not occur” was rated 2.8 on a scale from 1 (not at all relevant to not asking) to 6 (extremely relevant). The item “My inquiring could be suggestive, and therefore possibly induce false memories,” although rated only at 1.9, was significantly correlated to actual likelihood of asking. Thus, clinicians who believe more strongly that false memories are relatively common are less likely to ask at all. The available research, however, indicates that psychiatric patients under-report rather than over-report abuse (Dill et al. 1991; Read, 1997) and that their reports have high test-retest reliability (Goodman et al. 1999). Another study found that the problem of incorrect allegations of sexual assaults was no different for schizophrenia than for the general population (Darves-Bornoz et al. 1995).

The reasons why the relationship between child abuse and schizophrenia is minimized or ignored, and why, therefore, people with this diagnosis are even less likely than other patients to be asked about abuse, have been discussed earlier in this paper, and previously (Read 1997). However, in addition to the various ways that over-reliance on a simplistic biological paradigm leads to minimization, there is another, perhaps related, factor at work. Fear of being accused of “family-blaming” is particularly powerful in relation to schizophrenia. The pendulum has swung from a period around the 1960s, when the study of child-parent relations was at its meager height, to the current prevailing attitude that researching ways in which families may have contributed to severe mental disturbance seems almost taboo.

Nevertheless, many researchers and clinicians, with varying views about the causal

relationship of abuse to psychosis, have called for routine abuse inquiry in all mental health settings (Briere et al. 1997; Bryer et al. 1987; Dill et al. 1991; Goodman et al. 1997; Jacobson and Richardson 1987; Lipschitz et al. 1996; Rose et al. 1991; Sansonnet-Hayden et al. 1987; Swett et al. 1990). It has been suggested (Read and Fraser 1998b) that every mental health unit should develop its own policy and training packages about when and how to ask, and how to respond, informed by local circumstances and resources, and delineating the roles of various professions (Read, 2000). The training will not only need to be skills-based but will benefit from enhancing knowledge of the sequelae to child abuse (Briere 1999; Young et al. 2001), including the schizophrenic symptoms that seem to mediate against being asked about abuse and against receiving an adequate clinical response when abuse is disclosed.

#### *Treatment*

Regardless of one's beliefs about etiology there can be little doubt that, for both humane and economic reasons (Franey, Geffner, and Falconer 2001), effective treatments for survivors of child abuse who have been diagnosed schizophrenic are urgently needed. The most productive therapeutic approach may be an integration of the trauma models for abuse survivors in general (Briere in press; Courtois 1991; Herman 1992; McGregor 2001), with psychological approaches demonstrated to be effective with schizophrenic symptoms (Gottdiener 2000; Martindale, Bateman, Crowe, and Margison 2000; Nestoras 1997), including cognitive therapy (Birchwood, Todd, and Jackson 1998; Garety, Fowler, and Kuipers 2000; Kingdon and Turkington 1994), psychodynamic approaches (Karon and VandenBos 1981; Siani and Siciliani 2000; Sullivan 1962), family therapy (Wynne 1994), and psychosocial-residential treatment (Mosher, Vallone, and Menn 1995), with particular emphasis given to recent developments in early intervention (McGorry 2000; Johannessen, Larsen, McGlashan, and Vaglum 2000).

A rare discussion of the treatment of "seriously mentally ill" (SMI) women who have been abused (Goodman et al. 1997) notes that because researchers have excluded psychotic women from abuse treatment studies there is "currently a paucity of well-articulated and validated treatments for trauma effects in SMI women" (p. 690). Even approaches that address the family, but do so from the biopsychosocial model, may pay no attention to abuse (Allen and Read 1997). Recently, however, models of how some of these psychological and psychosocial approaches can be applied to abused women with serious mental illness are emerging (Harris, 1996; Harris and Landis 1997; Rosenberg et al. 2001).

Group therapy for women who suffered CSA may reduce paranoid ideation (Talbot et al. 1999). However, in terms of treatment specifically targeted at adults who experience psychotic symptoms and who were abused as children, all we have is tentative evidence that group therapy with "chronically mentally ill" females who were sexually abused as children has produced promising outcomes (Herder and Redner 1991) and that, in the case of female incest survivors with hallucinations, "sharing and clarifying traumatic events over several meetings appears to have assisted all of the cases reported. Hallucinations have become less preoccupying and much less frequent" (Heins et al. 1990, p. 565).

#### **CONCLUSION**

At this stage the hypotheses generated by the Traumagenic Neurodevelopmental model of schizophrenia are best described as tentative and in need of further exploration. The purpose of this paper was to identify some issues not yet sufficiently integrated into a diathesis-stress paradigm in the hope that they will be more adequately addressed by researchers and clinicians in future. The questions raised could quite economically be addressed if schizophrenia research programs would include abuse histories in their designs, and if abuse researchers would include schizophrenia when studying the long-term effects.

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