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Schizophrenia Research 53 (2002) 123–143

SCHIZOPHRENIA RESEARCH

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Trauma, PTSD, and the course of severe mental illness: an interactive model

Kim T. Mueser[a,*], Stanley D. Rosenberg[a], Lisa A. Goodman[b], Susan L. Trumbetta[c]

[a]*Department of Psychiatry, Dartmouth Medical School, Hanaver, NH, USA*
[b]*School of Education, Boston College, Boston, MA, USA*
[c]*Department of Psychology, Vassar University, Poughkeepsie, NY, USA*

Received 3 July 2000; revised 20 December 2000; accepted 2 January 2001

Abstract

Traumatic life events, as defined by DSM-IV, are common among persons with severe mental illnesses (SMI) such as schizophrenia. Limited evidence suggests concomitantly high rates of posttraumatic stress disorder (PTSD) in this population. However, conceptual models do not exist for understanding the interactions between trauma, PTSD, and SMI. We propose a model, which is an extension of the stress-vulnerability model, in which PTSD is hypothesized to mediate the negative effects of trauma on the course of SMI. Our model posits that PTSD influences psychiatric disorders both directly, through the effects of specific PTSD symptoms including avoidance, overarousal, and re-experiencing the trauma, and indirectly, through the effects of common correlates of PTSD such as retraumatization, substance abuse, and difficulties with interpersonal relationships. We discuss the evidence supporting this model, and consider several intervening variables that are hypothesized to moderate the proposed relationships between PTSD and SMI, including social support, coping and competence, and antisocial personality disorder. Theoretical and clinical implications of the model are considered, as well as several methodological and nosological issues. We conclude with a brief discussion of directions for future research aimed at evaluating components of the model.

Keywords: Trauma; Posttraumatic stress disorder; Severe mental illness

In recent years there has been a growing awareness of the importance of trauma in shaping the course of people's lives. Rates of trauma in the general population are high, and limited evidence suggests that persons with schizophrenia and other types of severe mental illness (SMI) are even more likely to be traumatized throughout their lives. Although exposure to trauma in persons with SMI is related to more severe psychiatric symptoms, substance abuse, and higher use of acute care treatment services, little is understood about how these relationships are mediated, whether trauma influences the course of the mental illness, and how to minimize its toxic effects. More information about the interactions between trauma and SMI is needed in order to develop or refine interventions, and to evaluate their effects in this population.

In this article we present a model in which we posit specific interactions between trauma exposure and the course of SMI. In our model, we propose that posttraumatic stress disorder (PTSD) plays a key role in mediating the negative effects of trauma on SMI.

* Corresponding author. Address. New Hampshire — Dartmouth Psychiatric Research Center, Main Building, 105 Pleasant St., Concord, NH 03301, USA. Tel.: +1-603-271-5747; fax: +1-603-271-5265.

E-mail address: kim.t.mueser@dartmouth.edu (K.T. Mueser).

0920-9964/02/$ - see front matter
PII: S0920-9964(01)00173-6

Based on factors known to influence the course of schizophrenia and other types of SMI, specific symptoms of PTSD, and associated conditions (e.g. substance abuse) are expected to exacerbate the psychiatric disorder, leading to a worse outcome and use of higher cost psychiatric services. This model leads to specific, testable hypotheses with implications for both understanding and modifying the interactions between trauma and SMI.

Our model has been developed with particular reference to schizophrenia, and much of the evidence we draw upon to support it is from research on this disorder. However, as we will review below, rates of trauma are uniformly high across a variety of other SMIs, such as bipolar disorder and treatment refractory major depression. Furthermore, there is abundant research suggesting that the severity and course of schizophrenia, bipolar disorder, and severe major depression are influenced by the same types of biological and environmental factors, including genetic loading, substance abuse, psychotropic medication adherence, and interpersonal and situational stress. The high rates of trauma across these disorders, coupled with similarities in factors affecting their course, suggest that the impact of trauma and its interactions with other factors are common across these disorders. Although it is likely that diagnostic-specific interactions exist between trauma and different types of SMI, we suggest that the importance of their similarities outweighs these differences. This is especially the case, considering that little research has addressed the proposed interactions, and available data do not permit differential predictions based on specific psychiatric disorders. Research testing this model will need to evaluate whether the proposed interactions between trauma and the course of psychiatric illness in fact differ across disorders. For these reasons, we propose that our model is applicable to a broad range of persons with an SMI, while acknowledging the centrality of schizophrenia to the model.

We begin with a discussion of the definition of trauma, followed by a review of the correlates of trauma, including PTSD, among patients with SMI. We next present our model of the interactions between trauma, PTSD, and SMI. In this model, we consider both direct and indirect effects of PTSD on the severity and course of SMI. Following this, we discuss several variables that may moderate the proposed interactions between trauma, PTSD, and outcome, including social support, coping and competence, and antisocial personality disorder. We consider the theoretical and clinical implications of the model, and suggest directions for future research. We conclude with a discussion of methodological and nosological issues relevant to the assessment of PTSD in the SMI population, and clinical implications of the model.

1. Trauma: definitions and prevalence

Psychological *trauma* refers to the experience of an uncontrollable event which is perceived to threaten a person's sense of integrity or survival (Horowitz, 1986; Herman, 1992; Van der Kolk, 1987). In defining a *traumatic event* as a criterion for PTSD, DSM-IV (American Psychiatric Association, 1994) adopts a narrower definition to include events involving direct threat of death, severe bodily harm, or psychological injury, which the person at the time finds intensely distressing or fearful. Common types of trauma include various forms of violent victimization such as rape and assault, combat exposure, natural disasters, the witnessing of or being threatened with bodily harm, and the sudden and unexpected death of a loved one.

By all accounts, rates of lifetime trauma in the general population are high, as exemplified by two large scale epidemiology studies. In the National Comorbidity Survey, 56% of respondents reported exposure to a traumatic event during their lives (Kessler et al., 1995). In a study of trauma in 1007 young persons (ages 21–30) living in southeastern Michigan, 39% reported at least one lifetime trauma; when the full sample was prospectively followed up three years later, 19% had been subsequently exposed to a traumatic event (Breslau et al., 1991, 1995).

In general, men are more likely to have experienced or witnessed physical assault, whereas women are more likely to have been sexually victimized (Breslau et al., 1995; Kessler et al., 1995). Indeed, concern over the high rate of sexual assault in women is amplified by numerous surveys indicating that between 15 and 33% of females are sexually abused as children (Finkelhor et al., 1990; Russell, 1986; Saunders et al., 1992; Wyatt, 1985), and between 14 and 25% of women are raped during adulthood (Burt, 1979;

Kilpatrick et al., 1987; Koss, 1993; National Victims Center, 1992; Russell, 1986; Searles and Berger, 1987; Sorenson et al., 1987; Wyatt, 1985). These high rates of trauma are of special concern considering the bias towards under-reporting traumatic events inherent in retrospective study designs (Kessler et al., 1995). Furthermore, factors such as reluctance to discuss unpleasant memories (Dill et al., 1991), fear of responses of the person to whom the event is disclosed (Symonds, 1982), or desire to protect perpetrators of abuse with whom they may have ongoing relationships (Della Femina et al., 1990) may result in under-reporting traumatic events, at least in the general population.

The validity of people's accounts of traumatic events has been a topic of much controversy, especially reports by adults of childhood sexual abuse (Brandon et al., 1998; Herman, 1992; Loftus and Ketcham, 1994; Pope and Hudson, 1995). Even greater concern pertains to the reports of persons with SMI, whose psychiatric illness may result in psychotic distortions or delusions with themes involving sexual or physical abuse (Coverdale and Grunebaum, 1998). Given the very private nature of most interpersonal traumatic experiences, external verification of trauma reports is not possible for most people, either with or without a psychiatric disorder. While the accuracy of reports of victimization is difficult to ascertain, the reliability (or consistency) of reports over time can be more easily determined. Temporal reliability of trauma reports is a necessary, but not sufficient condition to establish validity. The few studies of the temporal stability of trauma exposure measures in non-SMI individuals report fair to moderate test–retest reliability (Goodman et al., 1998; Green, 1996; Lauterbach and Vrana, 1996; Norris and Perilla, 1996). Less research has addressed the stability of trauma reports in SMI patients, but two recent studies have demonstrated comparable levels of reliability (Goodman et al., 1999; Mueser et al., in press).

While trauma is common in the general population, persons with a SMI appear to be even more likely to be traumatized over the course of their lives. Among persons with a SMI, between 34 and 53% report childhood sexual or physical abuse (Greenfield et al., 1994; Jacobson and Herald, 1990; Mueser et al., 1998; Rose et al., 1991; Ross et al., 1994). Over the lifetime, estimates of exposure to interpersonal violence, either physical or sexual, in persons with SMI range between 43 and 81% (Carmen et al., 1984; Hutchings and Dutton, 1993; Jacobson, 1989; Jacobson and Richardson, 1987; Lipschitz et al., 1996). Furthermore, exposure to interpersonal violence over the past year is high for persons with a SMI living with family members or significant others, as indicated by two studies reporting rates of 79% in the US (Cascardi et al., 1996) and 38% in Sweden (Bergman and Ericsson, 1996). As surveyed by Goodman et al. (1997), studies of the prevalence of interpersonal trauma in women with SMI suggest that these individuals may be especially vulnerable to victimization. For example, one study of episodically homeless women with SMI indicated that 97% had been exposed to interpersonal violence (Goodman et al., 1995), while a second study found that 77% of homeless women with SMI had been sexually or physically abused as children (Davies-Netzley et al., 1996).

The methods used to assess exposure to traumatic events in persons with SMI have varied from study to study, as well as the demographic and diagnostic characteristics of the individuals sampled. Most studies have included convenience samples, rather than probability samples, limiting generalizability of findings to the population of persons with SMI. Evaluating the extent to which persons with SMI are, in fact, more vulnerable to trauma than persons in the general population is further hampered by the fact that the largest and most valid epidemiology studies of trauma exposure contain small numbers of persons with SMI. However, a number of studies of trauma in persons with SMI have employed standard measures of trauma and interpersonal violence, such as the Trauma History Questionnaire (Green, 1996) and the Conflict Tactics Scale (Straus et al., 1996), and have reported rates of violence in excess of those usually found in the general population (Goodman et al., 1997). There is a need for research directly evaluating whether persons with SMI are more likely to have experienced trauma than in the general population. However, regardless of whether trauma rates are in fact elevated, it is important to understand the effects of trauma on patients with SMI, and the interactions between trauma and the course of psychiatric illness.

2. Clinical correlates of trauma in SMI

Aside from the evidence linking childhood abuse, especially sexual abuse, to the later development of adult psychiatric disorders (Bagley and Ramsey, 1986; Browne and Finkelhor, 1986; Bushnell et al., 1992; Duncan et al., 1996; Polusny and Follette, 1995), trauma exposure is also related to the severity of psychiatric symptoms in the SMI population. Specifically, a history of sexual and physical abuse in persons with SMI is related to more severe symptoms such as hallucinations and delusions, depression, suicidality, anxiety, hostility, interpersonal sensitivity, somatization, and dissociation (Beck and van der Kolk, 1987; Briere et al., 1997; Bryer et al., 1987; Craine et al., 1988; Carmen et al., 1984; Davies-Netzley et al., 1996; Figueroa et al., 1997; Greenfield et al., 1994; Muenzenmaier et al., 1993; Ross et al., 1994; Surrey et al., 1990; Swett et al., 1990). Trauma in persons with SMI has also been found to be related to greater utilization of high cost services such as psychiatric hospitalization (Briere et al., 1997; Carmen et al., 1984). The quality of these studies varies widely, as does the nature and type of instruments used to assess trauma and psychiatric symptoms. However, despite differences in methodology, most studies report positive associations between trauma history and severity of psychiatric symptoms. Thus, while questions remain as to the nature and type of relationship between trauma exposure and symptom severity in persons with SMI, the available data indicate a significant association.

Although numerous studies show that trauma in persons with SMI is related to more severe symptoms and higher use of acute care services, less research has examined the relationship between trauma and PTSD in this population. As PTSD is the most rigorously defined and studied psychiatric disorder explicitly linked to trauma in the general population, and evidence indicates that it can be successfully treated, the diagnosis and prevalence of PTSD among persons with SMI is important. Furthermore, as we shall address in the presentation of our model, there are compelling reasons for hypothesizing that PTSD mediates the frequently reported associations between trauma, severity of psychiatric symptoms, and increased use of acute care services in persons with SMI.

3. PTSD and SMI

PTSD is a disorder defined in DSM-IV by three types of symptoms, including *re-experiencing of the trauma*, *overarousal*, and *avoidance of trauma-related stimuli*, which are present at least one month after exposure to a traumatic event (American Psychiatric Association, 1994). Recent estimates of lifetime prevalence of PTSD in the general population range between 7.8 and 12.3% (Breslau et al., 1991; Kessler et al., 1995; Resnick et al., 1993). As discussed below, research on PTSD in patients with SMI indicates even higher prevalence.

3.1. Assessment of PTSD in SMI

Before reviewing research on the prevalence of PTSD in patients with SMI we briefly address two relevant methodological issues: (1) what types of events should be considered as meeting the DSM-IV Criterion A definition of a 'traumatic event', and (2) what is the reliability and validity of PTSD assessments using standard instruments in patients with SMI?

Concerning the first issue, some researchers have construed the experience of developing a psychosis or the process of psychiatric hospitalization as a traumatic event, and have evaluated PTSD secondary to that event. Specifically, several studies have shown that persons with SMI often report PTSD symptoms related to the onset of their psychosis (Shaner and Eth, 1989; McGorry et al., 1991; Williams-Keeler et al., 1994). In addition, one study found that recently hospitalized patients with schizophrenia endorsed high levels of PSTD symptoms related to the process of their hospitalization (Priebe et al., 1998). These studies suggest that the experience of a psychosis and its treatment can, in and of itself, be traumatic. However, the studies also raised the question of whether internally generated events, such as psychotic symptoms, should be included as meeting the DSM-IV Criterion A definition of a traumatic event. Although there is no clear consensus in the field regarding this question, we have adopted a conservative definition of trauma by limiting our review of PTSD in patients with SMI (below) to studies examining more conventional traumatic events, such as physical and sexual abuse, witnessing assault, accidents or disasters.

Table 1
Summary of studies on the prevalence of PTSD in patients with SMI

Study	No. of patients	% Schizophrenia	% Female	PTSD assessment instrument[a]	% With PTSD	% Trauma exposed with PTSD	% With PTSD in chart
Craine et al. (1988)	105	41	100	Standard checklist	34	66	0
Cascardi et al. (1996)	69	29	52	PSS	29	48	0
Mueser et al. (1998)	275	34	56	PCL	43	44	2
Switzer et al. (1999)	181	24	74	CIDI	40	42	3
Mueser et al. (2001)	30	40	53	CAPS	40	50	0
Mueser et al. (submitted for publication)	782	67	41	PCL	35	38	–

[a] CAPS, Clinician Administered PTSD Scale (Blake et al., 1990); CIDI, Composite Diagnostic Interview (Kessler, 1994); PCL, PTSD Symptom Checklist (Blanchard et al., 1995); PSS, PTSD Symptom Scale (Foa et al., 1993).

Second, two recent studies have been conducted regarding the reliability and validity of PTSD assessments in patients with SMI. Goodman et al. (1999) have shown that the internal reliability and the test–retest reliability of SMI patient self-reports of PTSD symptom severity over two weeks is high (r and coefficient $\alpha \geq 0.80$). In addition, a study of structured clinical interviews for the diagnosis of PTSD (the Clinician Administered PTSD Scale; Blake et al., 1990) in patients with SMI demonstrate high internal reliability (coefficient $\alpha = 0.63$–0.85) and inter-rater reliability ($\kappa = 1.0$ for PTSD diagnosis, intraclass correlation coefficients 0.75–0.99 for number of symptoms, frequency, and intensity), moderate test–retest reliability over two weeks ($\kappa = 0.63$), and moderate convergent validity with self-report measures (Mueser et al., in press). Furthermore, when more stringent PTSD severity criteria were employed to define a PTSD case, the test–retest reliability increased to $\kappa = 0.90$ (Mueser et al., in press). These studies provide encouragement that meaningful assessments of PTSD can be conducted in patients with SMI.

3.2. Prevalence of PTSD in SMI

A total of six studies suggest the prevalence of PTSD in patients with SMI is elevated compared to the general population. Table 1 summarizes the results of these studies. As reviewed, the six studies detected a range of 29–43% with PTSD, with fewer than 5% of identified cases as having PTSD documented in their charts.

The high rates of PTSD in patients with SMI are consistent with their increased exposure to trauma, but also suggest an elevated risk for developing PTSD given exposure to a traumatic event compared to the general population. For example, in a sample of persons drawn from a large health maintenance organization, Breslau et al. (1991) reported that the prevalence of PTSD among those exposed to trauma was 24%: 31% for women and 14% for men. These rates of PTSD following trauma exposure are considerably lower than the rates reported in studies of SMI, and suggest that these individuals may be especially vulnerable to developing PTSD following a traumatic event. Unfortunately, no study to date provides adequate information on the phenomenology of PTSD in the SMI population, including type and severity of symptoms, chronicity, and other comorbid conditions, nor the prevalence of PTSD compared to a sample drawn from the general population and assessed using the same procedures. In the next section, we describe a model which posits specific interactions between trauma, PTSD, and SMI.

4. An interactive model of trauma, PTSD, and SMI

Our model is an adaptation and extension of the stress-vulnerability model developed for schizophrenia and other SMIs (Falconer, 1965; Liberman et al., 1986; Zubin and Spring, 1977). The stress-vulnerability model assumes that symptom severity and other characteristic impairments of SMI have genetic and related biological bases (*psychobiological vulnerability*) determined early in life by a combination of genes and early environmental factors, such as

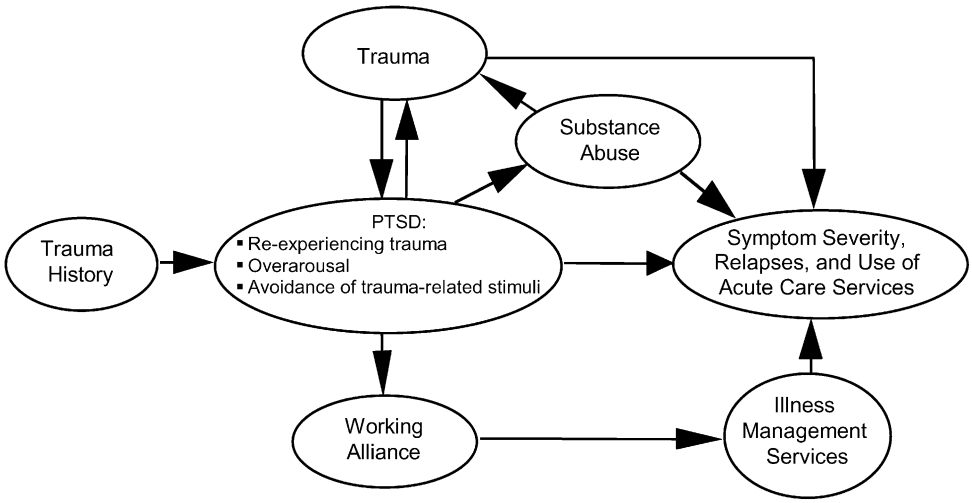

Fig. 1. Interactive model of trauma, PTSD, and severe mental illness. PTSD is hypothesized to worsen the severity and course of serious mental illness through the direct effects of PTSD symptoms (e.g. re-experiencing the trauma, overarousal) and indirectly through the effect of PTSD on substance abuse, retraumatization, and a poor working alliance with clinicians, leading to receipt of fewer preventative illness management services.

the intrauterine hormonal environment and birth complications. This psychobiological vulnerability, and hence symptom severity, can be decreased by medications, and increased by stress and substance abuse. *Stress*, including discrete events such as traumas and exposure to ongoing conditions such as a hostile, overly demanding, or unstructured environment, can impinge on vulnerability, precipitating relapses and contributing to impairments in other domains (e.g. social functioning). Finally, *coping* resources, such as coping skills or the ability to obtain social support, can minimize the effects of stress on relapse and the need for acute care.

Stress-vulnerability models, including the ameliorative effects of social support and coping efforts, have also been proposed to account for the development and course of primary PTSD (Allen, 1995; McFarlane, 1996). Genetic vulnerability to generalized anxiety disorder increases vulnerability to PTSD. Specifically, PTSD was significantly more prevalent among twins with anxiety disorders than among twins with other non-psychotic DSM-III-R disorders, and more prevalent in monozygotic (MZ) than in dizygotic co-twins (Skre et al., 1993). Quantitative genetic analysis of the Vietnam Twin Registry showed that after adjusting for differences in combat exposure, genetic factors accounted for up to one-third of the variance in liability to symptoms of re-experiencing, avoidance, and arousal (True et al., 1993). When genetic factors are held constant, there is a clear effect of the environment on PTSD prevalence: MZ twins who experienced high levels of combat in southeast Asia showed a nine-fold increase in prevalence of PTSD over that of their co-twins whose military service was not in southeast Asia (Goldberg et al., 1990).

In our model, shown in Fig. 1, we hypothesize that PTSD is a comorbid disorder which mediates the relationships between trauma, increased symptom severity, and higher use of acute care services in persons with a SMI. PTSD is given a central role in this model because the symptoms which define it, as well its common clinical correlates, can be theoretically linked to a worse prognosis of SMI. We hypothesize that PTSD can both directly and indirectly increase symptom severity, risk of relapse, and use of acute care services in patients with a SMI. PTSD symptoms can directly affect SMI through the avoidance of trauma-related stimuli, distress related to re-experiencing the trauma, and overarousal. Common correlates of PTSD can also indirectly influence SMI, including substance abuse, retraumatization, and a poor working alliance with treatment providers. Before explicating our model and

providing support from the SMI and PTSD literatures for the hypothesized links, we digress briefly to address the relevance of our model for the etiology of SMI in patients with trauma history and PTSD.

4.1. Etiologic considerations

Our model is silent on the etiology of non-PTSD Axis I SMI, although we assume these disorders have genetic and related biological bases. The causal relationships between PTSD and SMI are probably complex and interacting. There is evidence that early trauma predicts the later development of psychiatric illness (e.g. Bagley and Ramsey, 1986; Duncan et al., 1996), and that persons with mental disorders are more vulnerable to developing PTSD after exposure to a traumatic event than non-mentally ill persons (e.g. Blanchard et al., 1995; Breslau et al., 1995). Furthermore, as previously reviewed, patients with a SMI are more likely to be interpersonally victimized, thereby increasing their vulnerability to develop PTSD.

The prominent overlap between PTSD and other psychiatric disorders is illustrated by the National Comorbidity Survey, which found that 88% of males and 79% of females with PTSD had another DSM-III-R disorder (Kessler et al., 1995). Determining the etiological role of trauma in SMI is an exceedingly complex (if not impossible) task. Rather than attempting to explain etiology, we propose that PTSD provides a useful framework for understanding how history of trauma may contribute to a worse course of SMI. Our model posits that PTSD in patients with SMI leads to worse symptoms and increased use of acute care services, regardless of the etiology of the SMI. One study has reported that PTSD in patients with SMI is indeed correlated with higher utilization of acute care, and more costly psychiatric services (Switzer et al., 1999). Our model suggests mechanisms which may lead to this association.

In the next section we elaborate on the direct effects of PTSD on SMI posited by our model, followed by the indirect effects.

4.2. Direct effects of PTSD on SMI

We hypothesize that each of the three symptom clusters that define PTSD according to DSM-IV, avoidance of stimuli related to the trauma, distress related to re-experiencing the trauma, and overarousal, affect SMI. Based on factors known or believed to influence SMI, each of these PTSD symptom clusters may be expected to directly impact on the comorbid psychiatric disorder.

Because most violence in the lives of persons with SMI is interpersonal in nature (Mueser et al., 1998), *avoidance of trauma-related stimuli* often extends to close relationships, leading to reduced social contacts and social isolation (Allen, 1995; American Psychiatric Association, 1994; Jordan et al., 1992). Multiple studies have shown that lack of social contacts is a strong predictor of symptom relapses and rehospitalizations in persons with SMI (Avison and Speechley, 1987; Harrison et al., 1996; Rajkumar and Thara, 1989; Strauss and Carpenter, 1977). It has been hypothesized that social isolation may increase vulnerability to psychotic symptoms because of the lack of opportunities for reality testing with others, the absence of meaningful stimulation such as work, or the failure to experience the buffering effects of a supportive social network (Bell et al., 1996; Wing and Brown, 1970; Cresswell et al., 1992). Thus, severe avoidance and social isolation due to PTSD is expected to worsen symptoms and related impairments in other areas of functioning in persons with SMI.

Based on our model, *distress due to re-experiencing the trauma* can be conceptualized as an intermittent or chronic stressor. Abundant evidence shows that both discrete stressors (e.g. life events) and exposure to chronic stress (e.g. tense and critical family relationships) can worsen SMI, resulting in symptom relapses and rehospitalizations (Bebbington and Kuipers, 1992, 1994; Butzlaff and Hooley, 1998; Goodwin and Jamison, 1990). Persons with SMI and PTSD who re-experience traumatic events in the form of intrusive memories, nightmares, or flashbacks may be at increased vulnerability to relapses due to the stressful nature of these symptoms. Furthermore, extreme re-experiencing symptoms may take on delusional intensity in persons prone to psychotic symptoms (Hamner et al., 1997; Sautter et al., 1999) in a way that defies reality testing.

Overarousal, the third symptom cluster of PTSD, is also linked to a worse prognosis of SMI. Numerous studies have shown that increased physiological

arousal, especially chronic autonomic arousal, is associated with a poor prognosis in persons with SMI (Dawson and Nuechterlein, 1984; Straube and Öhman, 1990; Zahn, 1986). Primary PTSD is associated with chronic overarousal reflected across a wide range of different measures, such as heart rate, skin conductance, and catecholamine excretion, especially in response to stimuli reminiscent of the traumatic event (Orr and Kaloupek, 1997; Southwick et al., 1997). Therefore, comorbid PTSD may worsen the course of SMI by further increasing arousal in persons who are already physiologically compromised and who often, even in the absence of trauma, evince high levels of activation. In addition, overgeneralization of vigilance may be to the detriment of the person's ability to assess actual probabilities of threat, increasing vulnerability to retraumatization (see below).

Little research has evaluated the relationship between specific PTSD symptoms or PTSD diagnosis and symptom severity or course of SMI. However, as previously reviewed, the suggested associations between symptoms and trauma exposure in persons with SMI, coupled with the evidence of high rates of trauma and PTSD in this population, are consistent with the hypothesis that PTSD symptoms directly effect other psychiatric symptoms.

4.3. Indirect effects of PTSD on SMI

In addition to the direct effects of PTSD on SMI, our model posits that common clinical or behavioral correlates of PTSD indirectly affect psychiatric illness. Three common correlates of PTSD are hypothesized to worsen SMI, including *substance abuse*, *retraumatization*, and interpersonal difficulties (e.g. establishing trust) leading to a *poor working alliance*. We discuss each of these indirect effects in turn below.

Substance abuse. Persons with PTSD often abuse alcohol and drugs in order to avoid or minimize unpleasant, intrusive memories of traumas, to decrease arousal, or to improve sleep (Briere, 1992; Stewart, 1996), and there is evidence that PTSD tends to precede the development of substance use disorders in the general population (Chilcoat and Breslau, 1998a,b; McFarlane, 1998; Stewart et al., 1998). Consequently, substance use disorders are very common in PTSD (Deering et al., 1996; Duncan et al., 1996; Keane and Wolfe, 1990; Kessler et al., 1997; Rundell et al., 1989; Triffleman et al., 1995). There is also a high prevalence of substance use disorders in the SMI population (Cuffel, 1996; Mueser et al., 1990, 1992, 2001; Regier et al., 1990), and such substance abuse has been linked with higher lifetime exposure to trauma, especially in childhood (Briere et al., 1997; Carmen et al., 1984; Craine et al., 1988; Goodman and Fallot, 1998; Rose et al., 1991; Rosenberg et al., in press).

Prospective research has shown that substance abuse in patients with SMI contributes to a wide range of negative outcomes, including worse symptoms and relapses (Drake et al., 1989, 1996b; Kozaric-Kovacic et al., 1995; Linszen et al., 1994), as well as more hospitalizations and higher use of other acute care services (Bartels et al., 1993; Dickey and Azeni, 1996; Haywood et al., 1995; Swartz et al., 1998). PTSD, therefore, may indirectly worsen the course of SMI via its effects on increased substance abuse.

In addition to the direct effects of substance abuse on course of SMI, our model hypothesizes that substance abuse also increases vulnerability to trauma (or retraumatization), which (as discussed below) can also lead to a worse course of illness. Several studies indicate that within the SMI population substance use disorders are associated with violent victimization (Hiday et al., 1999; Lam and Rosenheck, 1998). Although the mechanism of increased risk is unclear, it may be speculated that substance abuse leads to trauma via use of substances in unsafe situations, impaired judgment, or disinhibitory effects of substances. Research has not been conducted to examine the direction of effects between substance abuse and trauma in the SMI population, but some evidence from the general population indicates that substance abuse increases risk of subsequent traumatization (Kilpatrick et al., 1997).

Retraumatization. Research on trauma has shown that earlier victimization, especially childhood sexual abuse, increases risk of later victimization and PTSD over the lifetime (Burnam et al., 1988; Nishith et al., 2000; Polusny and Follette, 1995), and the high number of traumas experienced by persons with SMI has been noted by many (Goodman et al., 1995; Lipschitz et al., 1996; Mueser et al., 1998;

Muenzenmaier et al., 1993). Furthermore, the number of traumas experienced is a strong predictor of PTSD in both the general population (Astin et al., 1995; King et al., 1996; Resnick and Kilpatrick, 1994) and among persons with SMI (Mueser et al., 1998). Analog research of ambiguous situations involving potential interpersonal threat show that women with a history of sexual victimization have poorer recognition of risk and indicate they would leave a threatening situation later than women with no history of sexual victimization (Wilson et al., 1999). Such problems with social perception may be magnified in persons with SMI, whose impairments in social cognition can further hamper accurate detection of potential interpersonal threat, and their ability to avoid interpersonal victimization (Gearon and Bellack, 1999; Penn et al., 1997).

Multiple traumatization may both contribute to the development of PTSD and be a byproduct as well. For example, a foreshortened sense of future, a symptom of PTSD, may lead to retraumatization due to the inability or unwillingness to anticipate and prevent negative events. Intense hypervigilance to potential immediate threats associated with a previous trauma may limit the ability of persons with PTSD to attend to the current environment, or to anticipate longer-term threats to their well-being. Alternatively, in their desperate attempts to avoid memories of traumatic events, individuals may fall prey to Santayana's (1905–1906) observation that those who forget the past are condemned to repeat it. Similarly, retraumatization may interact with other effects of trauma and PTSD, such as substance abuse. For example, trauma and PTSD may lead to substance use disorders (reviewed above), which may subsequently increase exposure to retraumatization, leading to a vicious cycle, as reported for rape victims by Kilpatrick et al. (1997).

In addition, early trauma and onset of PTSD may interfere with the acquisition of social skills necessary to avert exposure to interpersonal violence (Harris, 1996; Mueser and Taylor, 1997). Skills for averting exposure to traumatic events may be further hampered by the general deficits in social skills prominent in persons with SMI (Bellack et al., 1990, 1992, 1994). Regardless of the precise pathways, PTSD is related to revictimization and this association may be even stronger among persons with SMI.

The experience of recent life events, including traumas, has been repeatedly linked to an increased risk of relapse and rehospitalization in persons with SMI (e.g. Bebbington and Kuipers, 1992; Briere et al., 1997; Carmen et al., 1984). Revictimization therefore, either in the form of exposure to recent traumas or ongoing interpersonal victimization in patients with SMI and PTSD, can have a deleterious effect on the psychiatric disorder, similar to other types of life stress. To the extent that PTSD contributes to such revictimization, it will indirectly worsen the course of SMI.

Working alliance. Because of the interpersonal nature of most traumas, PTSD and social dysfunction, including pervasive feelings of mistrust, are closely linked (American Psychiatric Association, 1994; Carmen et al., 1984; Figley, 1985). Problems related to hypervigilance, recurrent disturbing memories, efforts to avoid trauma-related stimuli, anger, and mistrust can have an impact on the ability to form and maintain close relationships (Browne and Finkelhor, 1986; Roesler and McKenzie, 1994). Efforts to keep secrets or avoid topics related to traumatic events can further interfere with close relationships, and ultimately exacerbate anxiety about the experiences themselves (Kelly and McKillop, 1996).

Based on our model, we hypothesize that interpersonal problems related to PTSD may interfere with the ability of patients with SMI to establish a working alliance (or therapeutic alliance) with clinicians. The concept of working alliance has been defined by Bordin (1976) as including: (1) the perceived relevance of the *tasks* involved in treatment, (2) agreement as to the *goals* of the intervention, and (3) the strength of the interpersonal *bonds* between the clinician and individual (e.g. mutual trust and acceptance). To the extent that a therapeutic relationship includes these three components, a good working alliance will exist that can serve as a vehicle for change towards desired outcomes.

For patients with a SMI, poor relationships with clinicians can result in their receiving fewer illness management services than necessary for the optimal management of their disorder (e.g. medication, case management), thereby increasing their risk of relapses and rehospitalizations. Evidence supporting this is found in several studies indicating that the quality of the therapeutic alliance with the case manager is related to symptom severity and hospitalizations in

patients with SMI (Gehrs and Goering, 1994; Neale and Rosenheck, 1995; Priebe and Gruyters, 1993; Solomon et al., 1995). Furthermore, one study demonstrated that severe sexual abuse is related to the quality of the working alliance among homeless women working with case managers (Johnson, 1998). The relationship between therapeutic alliance and PTSD has not been the topic of much research, although many clinicians have written anecdotally about its importance in working with trauma survivors (e.g. Harris, 1996; Herman, 1992). Thus, PTSD may worsen the course of SMI by interfering with patients' ability to form a good working relationship with their treatment providers.

4.4. Other potential intervening factors

In explicating our interactive model, we have emphasized the role of factors related to PTSD that can directly or indirectly influence the course of SMI. However, several other factors may play a critical role in mediating the course of both SMI and PTSD, or in moderating the direct and indirect relationships between the disorders suggested by the model. The measurement of these additional variables may be important in testing the hypothesized associations between PTSD and SMI. We consider three intervening factors below, including social support, coping and competence, and antisocial personality disorder. The hypothesized directions of the relationships between these intervening variables and other factors in the interactive model are described below.

Social support. Social support has been the focus of extensive research over the past 30 years as a determinant of well-being and mental health, and as a buffer of the noxious effects of stress (Wethington and Kessler, 1986; Veiel, 1985). Social support generally refers to an individual's perception that he or she is cared for, is esteemed and valued, and is a member of a network of others (Cobb, 1976). Such support is thought to both influence the chances that a vulnerable individual will develop a psychiatric illness, given exposure to sufficiently high levels of stress, and to interact with a host of factors that determine the course and outcome of SMI, including well-being and relapses (Brown and Harris, 1978; Pearlin et al., 1981).

Social support has been found to be related to the course of SMI, as well as the development of adverse psychological consequences following trauma, including PTSD. For example, the extent and quality of social relationships among persons with a first episode of schizophrenia or major affective disorder was found by Erickson et al. (1989) to predict outcome at 18 months. This finding (and similar associations reported by others) is consistent with previous research alluded to here showing that the degree of social contacts is predictive of relapse in persons with SMI, suggesting that social support and social contacts are closely related.

With respect to social support and the consequences of trauma, Romans et al. (1995) reported that the quality of family relationships and support in adolescence of women who were sexually abused as children predicted psychological outcome in adulthood. Similarly, Runtz and Schallow (1997) found that perceived social support for individuals who were maltreated as children was a strong predictor of adult adjustment, while Norris and Kaniasty (1996) reported that social support decreased psychological distress secondary to natural disasters. As might be expected, other studies show that higher levels of perceived social support and family stability are related to lower rates of PTSD or lower severity of PTSD symptoms (e.g. Fontana et al., 1997; King et al., 1996).

How might social support interact with the other factors in mediating the effects of PTSD on SMI? Aside from higher levels of social support decreasing the overall probability of a traumatic event leading to the development of PTSD, or decreasing the severity of PTSD symptoms, we suggest that such support may reduce some of the direct and indirect effects of PTSD on SMI, especially effects that are social in nature. Three pathways between PTSD and severity of SMI are most likely to be affected by social support: avoidance of trauma-related stimuli, retraumatization, and the quality of the working alliance with the clinician.

First, because avoidance of trauma-related stimuli frequently generalizes to close relationships and results in social isolation, greater social support might minimize such avoidance, thereby lessening the negative effects of isolation on SMI. Second, exposure to retraumatizing events, which are often interpersonal like the initial precipitating events, may be decreased to the extent that the individual enjoys

good, supportive relationships that are not abusive or otherwise traumatizing. In addition to these relationships not being the source of retraumatizing experiences to the person, they may help individuals avoid trauma from other sources, by decreasing the need to seek affiliation from other, less familiar persons, involving potentially traumatic situations. Third, better social support, through the experience of positive relationships with others, may improve the capacity of persons to establish and maintain a good working alliance with treatment providers. Pervasive problems with trust, so common in persons with a trauma history (e.g. Herman, 1992), may be lessened to the extent that persons perceive and receive emotional support from others.

Coping and competence. Closely tied to the construct of social support are coping and competence, individual characteristics that serve to minimize the negative effects of stress, and to allow or facilitate the attainment of goals. Zubin and Spring (1977) define and distinguish coping and competence as follows:

> Coping efforts are exemplified by the persistent application of energy toward problem solving and abstract thinking in situational dilemmas. Competence is developed by exerting coping efforts and consists of social skills, intellectual strategies, and other acquired capacities that equip the individual to deal with life exigencies. Coping efforts should not be confused with competence. The former refers to the attitudinal, motivational stance of an individual faced with a task; the latter refers to his abilities, skills, and accumulated know-how in solving life problems (p. 111).

Coping and competence reflect a broad constellation of efforts and abilities the person can deploy to bear on managing a difficult situation or achieving a goal. There is a substantial literature demonstrating that coping, and in particular competence, are generally decreased in the SMI population (Bellack et al., 1990; Zigler and Glick, 1986). However, we hypothesize that depending on the availability of role models during the formative years, and the severity of the SMI, coping and competence will be variable among persons with SMI and PTSD. Traumas that occur at a relatively early age and within the family leading to PTSD may pose a greater barrier to the acquisition of coping skills and development of social competence than when the PTSD is due to other traumas. While we expect that coping has a direct effect on decreasing the severity of PTSD (and other psychiatric) symptoms, similar to other research on each of these disorders, we also propose that more effective coping efforts and social competence interact with other intervening variables to influence the course of SMI.

Poor coping may render persons with PTSD more susceptible to maladaptive strategies for coping with their PTSD symptoms, such as substance abuse. For example, among women who are victims of rape and who develop PTSD, motives for using alcohol to cope with negative effects are predicted by the severity of sleep disturbance and lower levels of education, suggesting that alcohol is used as a coping strategy among these women (Nishith et al., in press). A modicum of coping effort and social competence is necessary to marshal social support that does not otherwise spontaneously occur from family or friends. In this way, higher levels of coping and competence, in concert with social support, may buffer individuals from the negative effects of exogenous stressors, such as retraumatization.

Antisocial personality disorder. Antisocial personality disorder (ASPD), or the closely related construct of psychopathy, may be critical because of its relationship to several other factors in our model. First, ASPD and its precursor, conduct disorder, are more common among persons with SMI than in the general population (Bland et al., 1987; Hodgins et al., 1996; Jackson et al., 1991; Robins, 1966). Thus, these behavioral predispositions are common complicating factors that may interact with trauma exposure and the course of SMI. Second, ASPD increases vulnerability to substance use disorders, both in the general population (Grande et al., 1984; Regier et al., 1990) and among patients with SMI (Caton et al., 1994, 1995; Mueser et al., 1999). To the extent that ASPD contributes to substance abuse independent of PTSD (but see third point below), its assessment is important for determining the impact of PTSD on substance abuse in patients with SMI.

Third, persons with ASPD are more likely to be exposed to traumatic events, both in childhood and

adulthood, than their non-ASPD counterparts (Burnam et al., 1988; Breslau et al., 1991, 1995; King et al., 1996). This increased exposure appears to reflect both early exposure to pathological environments that may contribute to the development of ASPD (Reiss et al., 1995; Rutter, 1997; Snyder et al., 1997) and self-exposure to more dangerous situations in adulthood due to personality characteristics such as impulsivity, monotony avoidance, and sensation seeking (Schalling, 1978). Because of the higher exposure of persons with ASPD to traumatic events, some studies have also found higher rates of PTSD among these individuals (Breslau et al., 1991; Helzer et al., 1987).

Traditional characterizations of ASPD and (primary) psychopathy have emphasized that individuals with this disorder (or these personality predispositions) are less prone to experiencing anticipatory anxiety, worry, and fear, and consequently are less able to learn from unpleasant experiences (Cleckley, 1976; Gough, 1948; Lykken, 1957). This would suggest that ASPD serves as a protective factor from the development of PTSD, given exposure to a traumatic event. However, this 'protection' may be diminished or compensated for by the increased tendency to be exposed to traumatic events, resulting in comparable or higher rates of PTSD in persons with ASPD.

As is evident from the three points above, the assessment of ASPD may be useful to evaluating our model. Independent of PTSD, ASPD may increase exposure of persons to multiple traumatic events, as well as substance abuse. If PTSD contributes to substance abuse and retraumatization in persons with SMI, as we suggest, the identification of persons at risk to these behaviors due to other (non-PTSD) predisposing factors may be important.

5. Theoretical and clinical implications

We have proposed that PTSD mediates the negative effects of trauma on the severity and course of SMI through both direct and indirect mechanisms. To facilitate the evaluation of the model, we have taken care to operationalize our model in terms of specific, measurable constructs. This model has both theoretical and clinical implications for understanding factors which influence the course of SMI, and developing interventions designed to lessen the hypothesized effect of trauma on these disorders. We consider several of the implications here.

In our model we suggest that PTSD is the most important factor that mediates the negative effects of trauma on the severity and course of SMI. While the model does not assume that PTSD is the only significant sequelae of trauma, or that other hypothesized consequences do not also influence SMI, it does posit a central role for PTSD. Trauma, including childhood sexual abuse, does not invariably lead to negative psychological outcomes in adulthood (Binder et al., 1996), nor, therefore, would it be expected to necessarily interact with SMI. If PTSD is indeed the crucial variable that mediates the effects of trauma on SMI, stronger associations between PTSD (and specific dimensions of PTSD, as hypothesized) and SMI would be expected than between trauma and SMI, as PTSD has the more proximal impact. Support for such a hypothesis would suggest that not all persons with a trauma history and SMI should be provided with, or would benefit from intervention aimed at addressing trauma experiences and their possible consequences.

The hypothesized effect of PTSD on SMI may explain, in part, the high rate of substance abuse in this population. As previously reviewed, there is a high prevalence of substance use disorders (Regier et al., 1990) and trauma exposure (Goodman et al., 1997) in the population of persons with SMI, and limited research also suggests a high rate of PTSD in this population. In contrast to research on PTSD and substance abuse in the general population (e.g. Chilcoat and Breslau, 1998a,b), little research has examined whether PTSD contributes to substance abuse in persons with SMI, as suggested by our model. While it is unlikely that PTSD alone is responsible for the high comorbidity between SMI and substance abuse, it may account for some of the increased risk.

Similar to the importance of PTSD in increasing risk of substance abuse in patients with SMI, our model also suggests that the high rates of victimization may be related to the interactions between trauma and PSTD. Does trauma exposure itself predispose individuals to subsequent trauma, and if so how? Are elevated rates of retraumatization attributable to a common predisposing variable such as ASPD? We

propose that PTSD is an important intervening variable that increases subsequent risk of exposure to trauma via both direct pathways (i.e. retraumatization related to foreshortened sense of future) and indirect pathways (i.e. substance abuse). PTSD symptoms may serve as a protective mechanism that has evolved to help prevent retraumatization, avoidance of harm, etc. but that extremes of these symptoms (as seen in the syndrome) have paradoxical effects of increasing traumatization. The relationship of retraumatization to PTSD severity may be parabolic, with non-anxious and over-anxious types being repeatedly retraumatized and with the 'optimally anxious' being at lowest risk for retraumatization. Evidence supporting the effects of PTSD on revictimization would provide valuable insights into the high rates of trauma that could be considered 'normative' in the SMI population (Goodman and Dutton, 1996; Mueser et al., 1998).

Aside from the theoretical implications of our model for understanding the effects of trauma on the course of SMI, it has several clinical implications as well. Despite the high rates of trauma for persons with SMI, there is a dearth of research examining PTSD in this population (Mueser et al., 1998), with most studies of PTSD limiting the scope of assessment to either narrow time-frames or certain types of trauma (e.g. Cascardi et al., 1996; Craine et al., 1988). Furthermore, in the few studies that have evaluated PTSD in this population, virtually none of the persons with confirmed diagnoses had PTSD noted in their medical charts. The practical implications are that PTSD appears to be a frequently neglected comorbid diagnosis among patients with SMI, despite the growing recognition of the common problem of trauma in these persons.

If some of the hypothesized pathways between PTSD and poor outcome of SMI are supported, it would suggest a need to increase the awareness of clinicians of PTSD as a common comorbid disorder in this population and to provide training in appropriate assessment methods. Eilenberg et al. (1996) pointed out that despite state mandated inquiry into trauma history for all psychiatric outpatients, PTSD was rarely diagnosed, and few clinicians incorporated trauma history into their treatment plans. A parallel may be drawn between the lack of awareness in the past of substance abuse in the SMI and the current underdetection of PTSD in these patients. A decade ago substance use disorders were rarely diagnosed in patients with SMI, whereas in recent years there has been a growth in both assessment of these disorders and recognition of their negative effects on the course of SMI (Drake et al., 1996a,b). Understanding of the role of trauma and PTSD in influencing the course of SMI may lead to similar changes in practice, as the assessment of these difficulties becomes more routine and accepted as a necessary standard of practice.

One possible obstacle to the routine assessment of trauma in men and women with SMI has been the absence of clear treatment guidelines for these individuals. Clinicians may not address trauma history in their patients simply because they do not know what to do. A second practical implication of the proposed model is that it may provide clinicians with a clear direction to pursue in treating comorbid PTSD in their patients. In contrast to the paucity of rigorous research on the generic consequences of trauma, controlled studies have demonstrated that time-limited cognitive-behavioral treatment is effective in reducing or eliminating symptoms in primary PTSD populations secondary to combat, rape, and other traumas (Boudewyns and Hyer, 1990; Cooper and Clum, 1989; Foa et al., 1991, 1999; Keane et al., 1989; Marks et al., 1998; Resick and Schnicke, 1992; Tarrier et al., 1999). If PTSD is found to contribute to a worse course and outcome of SMI, as we hypothesize, a next step would be to adapt and evaluate PTSD interventions for this population. Such an approach would hold the most immediate promise for improving the outcome of persons with SMI and PTSD, while potentially decreasing use of high cost services required to respond to symptom relapses.

6. Methodological and nosological issues

Our model raises a number of methodological and nosological issues relevant to the assessment and hypothesized interactions between PTSD and SMI. The most important of these issues include PTSD as a continuum disorder and PTSD as a SMI. As these considerations are beyond the central scope of our theory, and are more speculative in nature, we discuss them only briefly below.

6.1. PTSD: discrete or continuum disorder?

Up to this point we have accepted the prevailing wisdom of treating PTSD as a discrete disorder, and discussing its interactions with SMI. However, our model does not require that PTSD be conceptualized as a discrete disorder, instead of on a continuum or series of three continua (corresponding to the symptom dimensions used to define PTSD). There are several reasons why the severity of PTSD symptoms should be considered as an alternative to PTSD diagnoses in evaluating the hypothesized interactions between trauma, PTSD, and SMI.

First, many studies of PTSD in the general population model PTSD symptom severity and not only PTSD diagnoses (e.g. King et al., 1996). Among persons with PTSD, symptom severity is related to different factors such as the extent of trauma exposure, receipt of injuries, etc., and the same may be true for persons with subsyndromal PTSD. Second, among persons with SMI, subsyndromal levels of PTSD may nevertheless predict worse outcomes due to their increased sensitivity to stress, as suggested by traditional formulations of the stress-vulnerability model (Nuechterlein and Dawson, 1984).

Third, the course of PTSD can be episodic (Friedman and Rosenheck, 1996; Ronis et al., 1996). The frequently reported association between number of traumas and PTSD suggests that recent traumas may exacerbate PTSD symptoms in some individuals with lifetime histories of PTSD that are currently in remission. The assessment of subsyndromal PTSD in such individuals may identify persons who are vulnerable to the later development of PTSD (or relapses of PTSD) as well as the interactions between PTSD and SMI. Last, our model posits some specific interactions between PTSD symptoms and the course of SMI. For example, we propose that the avoidance of trauma-related stimuli leads to increased social isolation and problems in establishing a therapeutic alliance. These hypothesized pathways can be evaluated in individuals, regardless of whether they meet full diagnostic criteria for PTSD.

6.2. PTSD as a SMI

Our discussion of the interactions between PTSD and SMI may lead to the impression that PTSD is not (or is never) a SMI. Yet data on the severity of impairment and course of PTSD suggest otherwise (Friedman and Rosenheck, 1996). Although Winje (1996) found considerable resolution of both intrusive and avoidance symptoms three and five years posttrauma, Yehuda et al. (1995) reported increased PTSD symptoms in holocaust survivors who suffered more recent stressors. PTSD runs a chronic course in about one-third of cases, with greatest recovery after one year, and a 50% chance of recovery after two years (Kessler et al., 1995). However, past trauma exposure remains a risk factor for the development of PTSD following exposure to a new traumatic event (Resnick et al., 1993).

Clearly, PTSD is often chronic and, in a small proportion of cases, results in severe debilitation across a range of different areas similar to other SMIs such as schizophrenia. These cases of severe PTSD are further complicated by multiple comorbid diagnoses, with intensity and duration of trauma predictive of higher severity of psychiatric morbidity (Deering et al., 1996). The distinction between PTSD and other SMIs can also be difficult to establish when the clinical presentation of PTSD includes psychotic symptoms, such as hallucinations and delusions, that are not clearly due to another Axis I disorder (Butler et al., 1996; Hamner, 1997; Hamner et al., 1999; Mueser and Butler, 1987; Sautter et al., 1999). Such individuals may be present in populations of persons with SMI who are not properly diagnosed with PTSD. The etiologies of PTSD and other Axis I disorders may be inextricably linked for some individuals, separable (in theory) but interacting for others, and wholly independent for still other persons. It is possible that the interactions between PTSD and the course of SMI differs as a function of the etiological relationships between the disorders, yet we have few clues at this time to permit the disentangling of these relationships, and suspect that the task is not possible for many cases. We accept that certain forms of PTSD are complex, severe, and have a course similar to other SMIs. Nevertheless, the basic predictions that derive from our model apply to such cases of complex 'primary' PTSD. In addition, the almost total neglect of PTSD as a comorbid disorder in samples of patients with SMI suggests that the assessment and treatment of PTSD may improve the course and outcome of these disorders.

7. General conclusions and future directions

Our model posits that PTSD is a primary mechanism that is responsible for the frequently reported associations between trauma history and the severity and course of SMI. PTSD is but one of many concepts advanced to understand the psychological consequences of trauma (e.g. Finkelhor and Browne, 1985; Briere, 1984). Other writers have, for example, emphasized the chronicity and heterogeniety of symptoms observed in survivors of chronic trauma exposure, including dissociation, somatization, and difficulties in effect regulation (Herman, 1992; Newman et al., 1995). Indeed, dissociative symptoms are central to two other DSM-IV disorders, dissociative identity disorder (DID) and acute stress disorder, thought to be related to traumatic experiences (Ross et al., 1989, 1991; Putnam et al., 1986). As discussed above, partial or sub-syndromal PTSD symptomatology may also have important clinical implications for patients with SMI who suffer traumatic events. However, we have chosen to focus on PTSD partly because it is the most widely and thoroughly studied disorder related to trauma, and thus there is a large scientific literature devoted to it. Furthermore, both the criteria for the diagnosis of PTSD as well as common clinical correlates lead to the formulation of specific hypotheses concerning direct and indirect effects of PTSD on SMI, based on factors known to influence SMI. Finally, controlled research indicating that PTSD can be effectively treated in the general population suggests that its interactions with SMI, and its suitability as a potential focus of treatment, require further evaluation.

Despite the importance PTSD to SMI in our proposed model, and the limited data suggesting high rates of PTSD in this population, it is virtually never diagnosed in patients with SMI. Consequently, little is known about the stability of PTSD in this population, its demographic and clinical correlates, and the interactions between PTSD, clinical status, and service utilization. Longitudinal research on the natural history of PTSD in patients with SMI would be useful to evaluate the proposed model. Alternatively, components of the model could be examined in the context of controlled treatment trials designed to reduce PTSD in this population.

Traumatic life events, PTSD, and subsequent retraumatization are common but neglected problems in the lives of patients with SMI. While there has been some increased awareness of trauma in this population, there is a need for a theory that guides exploration into how trauma influences SMI. The conceptual model presented here is intended to serve as a heuristic in guiding research, and potentially clinical practice, in addressing the effects of trauma on the course of SMI.

Acknowledgements

Preparation of this manuscript was supported by NIMH grant no. R24 MH56147. Portions of this paper were presented at the Fourteenth Annual Public Sector Psychiatry Conference on Treatment and Mistreatment of Women with Chronic Mental Illness, Worcester, MA in May, 1998, and the Fourteenth Annual Meeting of the International Society for Traumatic Stress Studies, Washington, DC in November, 1998. We thank Robert E. Drake, Julian Ford, and Mathew J. Friedman for their helpful discussions concerning this paper.

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