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Substance abuse and post-traumatic stress disorder comorbidity

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Abstract

This article reviews the extant literature on substance abusers with and without a comorbid diagnosis of post-traumatic stress disorder (PTSD) and reveals the discontinuity between clinical lore and empirical research. Included is an overview of PTSD-substance abuse theoretical models and comorbidity prevalence rates, as well as an evaluation of the comparative data on treatment outcome and psychosocial factors, such as coping skills, for PTSD versus non-PTSD substance abusers. In addition, we discuss the controversy surrounding sequential versus simultaneous treatment approaches for such 'dually-diagnosed' patients. We conclude by identifying gaps in current knowledge about the nature and impact of PTSD on substance abuse treatment outcome and outlining needs for future research.

Key words: Post-traumatic stress disorder; Substance abuse; Comorbidity; Treatment outcome; Treatment interventions

1. Introduction

Research examining adult substance abusers consistently reveals a high degree of psychiatric comorbidity (Powell et al., 1982; Ross et al., 1988) as well as a significant association between such comorbidity and treatment outcome (Helzer and Pryzbeck, 1988; Powell et al., 1992). In the relatively few studies which have assessed post-traumatic stress disorder (PTSD), this disorder has emerged as a common co-occurring diagnosis among substance abusers (Cottler et al., 1991; Hyer et al., 1991). Despite significant comorbidity rates, little is known about the impact of PTSD on outcome following substance abuse treatment. Both clinical theory and programmatic efforts for this 'dually-diagnosed' population have continued to evolve and develop without direction from empirical research. The goal of this article is to review the theoretical and clinical literature regarding PTSD and substance abusers, the influence of such comorbidity on substance abuse treatment outcome, and issues regarding the clinical treatment of patients suffering from both disorders. We conclude by considering the limitations of existing knowledge and research methodologies and identify avenues for future research on substance abuse-PTSD comorbidity.

2. Definition of PTSD and theoretical models of PTSD-substance abuse comorbidity

PTSD is classified as an anxiety disorder (American Psychiatric Association, 1987) and describes the constellation of symptoms associated with exposure to extraordinary traumatic events (e.g., childhood physical and/or sexual abuse, adulthood sexual assault, combat, natural and technical disasters). PTSD is characterized by three classes of symptoms: (a) re-experiencing (e.g., nightmares, 'flashbacks'); (b) avoidance (e.g., efforts to avoid places reminiscent of the trauma) and numbing of responsiveness (e.g., feelings of detachment or estrangement); and (c) hyperarousal (e.g., extreme startle reactions). This disorder is among the most extreme reactions that individuals can have to high magnitude life events and can result in severe and chronic impairment across the major life areas.

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The link between PTSD and substance abuse appears to be particularly complex with cause-and-effect relationships difficult to disentangle and determine. The self-medication hypothesis is commonly invoked in the dual-diagnosis literature (Khantzian, 1985; Khantzian, 1990) and in reference to PTSD as the co-existing disorder, postulates that PTSD develops first and that chemical substances are used as a means of achieving symptom relief. Anecdotal evidence suggests that PTSD patients report using alcohol and/or drugs to overcome the distress of trauma-related events and 'to forget' intrusive, disturbing memories of the trauma (LaCoursiere et al., 1980; Jelinek and Williams, 1984; Schnitt and Nocks, 1984). Although the self-medication hypothesis postulates a relationship between the type of psychiatric diagnosis and the drug of choice, the PTSD-substance abuse literature, as well as the general 'dual diagnosis' literature, shows little evidence of specificity of substance use. However, clinical findings that substance abusing PTSD patients use a wide variety of substances with vastly different pharmacological actions (Brief et al., 1992) may not necessarily argue against the self-medication hypothesis but rather reflect a more complex relationship between the separate symptom clusters of PTSD (i.e., re-experiencing, avoidance/numbing and hyperarousal) and the type of substance used. For example, patients with PTSD characterized primarily by avoidance may use central nervous system stimulants (e.g., amphetamines) to boost sociability, whereas PTSD patients experiencing predominantly sleep difficulties and irritability/agitation may prefer to use sedatives (e.g., alcohol). For patients with comparably severe levels of re-experiencing, avoidance/numbing and hyperarousal symptomatology, a variety of substances may be used to alleviate and manage their diverse ailments. This notion of a complex relationship between specific diagnostic subgroups and self-medication has been supported by schizophrenia research conducted by Dixon and colleagues (1990, 1991) showing that the propensity of schizophrenics to self-medicate is a function of the number of experienced negative symptoms (e.g., unresponsiveness, apathy).

The self-medication model can be considered a modification of the more general tension-reduction hypothesis, which originated from animal experiments (Masserman et al., 1945; Masserman and Yum, 1946; Conger, 1951). The early version of this theory was based on conditioning principles and held that alcohol directly reduced an underlying drive state of anxiety. Although this original version of the tension-reduction hypothesis received inconsistent empirical support in studies of both human and infrahuman species (Cappell and Greeley, 1987), as noted by Wilson (1987), 'conflicting data... are surprising only if it is assumed that there is an automatic, invariant relation between alcohol consumption and stress reduction'(p. 325). In the past decade, social learning and cognitive theories of the effects of alcohol have rejuvenated the tension reduction hypothesis by proposing alternative conceptualizations that are more empirically supported and sophisticated. This line of research has shown that the effect of alcohol/drugs on tension and other emotional states is mediated by a plethora of variables, such as learned expectations about the effects of alcohol, prior experience with alcohol, and the temporal pattern of alcohol use (Young et al., 1990; Orcutt and Harvey, 1991). In view of all these influences, Wilson (1988) summarizes that 'it is simply no longer useful to ask the question: Does alcohol reduce tension? The more meaningful question to pose is: Under what conditions, at which doses, in whom... and how does alcohol affect specific forms of tension?' (p. 259).

Our understanding of the use of alcohol and drugs following trauma exposure and the development of PTSD may ultimately be amplified by neurobiologic research on PTSD. Although few studies have been conducted to date (Charney et al., 1993), this line of research may be promising in elucidating the pathophysiological changes that occur following psychological trauma and how these changes may predispose, exacerbate, or interact with personal and sociodemographic variables to develop PTSD and perhaps substance abuse problems.

Although the self-medication and tension-reduction models described above suggest that substance abuse is secondary to PTSD, the converse is an equally likely possibility. An investigation of PTSD among substance users from the general population shows that the onset of substance use precedes the onset of PTSD symptoms (Cottler et al., 1992). It may be that early substance abuse occurs in the context of other 'high risk' behaviors (e.g., prostitution), which increase the likelihood of exposure to potentially traumatizing events (e.g., being seriously beaten or injured) and, hence, the likelihood of developing PTSD.

Individuals who began abusing substances at an early age may also be more susceptible to developing PTSD following traumatic exposure because they have historically relied on alcohol and drugs as a way to combat stress and have failed to develop more effective stress inoculation strategies. At the present time, it is an open issue whether PTSD following primary substance abuse is the result of associated risk factors, pre-existing vulnerabilities, persistent changes in brain physiology/neurochemical systems caused directly by the abused substance, or a complex interplay between all of the above.

Research identifying a history of conduct disorder/antisocial personality as a predisposing variable for the development of both substance abuse disorders and PTSD (Helzer et al., 1987; Cottler et al., 1992) suggests that PTSD and substance abuse may not be directly re-

lated per se but rather share a common etiological pathway. It may be similarly argued that psychosocial factors such as homelessness are both risk factors as well as maintenance factors for chronic substance abuse and PTSD.

In concluding this section on theories which might account for the connection between substance abuse and PTSD, it is clear that a host of mechanisms and factors are implicated. Both PTSD and substance abuse are multifactorial disorders, and the understanding of their comorbidity will require a rigorous integration of biological, psychological and environmental factors.

3. Review of comorbidity data

Investigations examining comorbidity among patients seeking substance abuse treatment reveal that PTSD is a common comorbid diagnosis. For example, an evaluation of 120 male veterans admitted to an in patient substance abuse program showed that approximately 35% of the sample met DSM-III-R diagnostic criteria for PTSD (Brief et al., 1992). Another investigation of 489 male veterans presenting for substance abuse treatment found that 46% evidenced clinically significant PTSD symptoms (McFall et al., 1991), as measured by the Mississippi Scale for Combat-Related PTSD (Keane et al., 1988). A study of 33 females receiving substance abuse treatment reports that nearly 40% were diagnosed as suffering from PTSD (Kovach, 1986). Despite methodological differences in sampling and assessment, these investigations similarly show the high prevalence of concurrent PTSD among clinical samples of substance abusing male veterans and civilian women. Although no data are currently available on the rates of PTSD for male civilians or female veterans seeking substance abuse treatment, findings from a large-scale general population study indicate that substance abuse-PTSD comorbidity cuts across gender and military status (Cottler et al., 1992).

4. Treatment outcome in substance abusers with and without concurrent PTSD

Although clinical lore suggests that substance abusers with PTSD have poorer outcomes compared to their non-PTSD counterparts (Root, 1989; Bollerud, 1990; Hyer et al., 1991), only three studies provide empirical data on this issue. A retrospective study of 60 000 hospital discharge records indicates that a comorbid diagnosis of PTSD is not a risk factor for irregular discharge from VA alcoholism treatment; the rate of irregular discharges for patients with co-diagnoses of alcohol dependence and PTSD was similar to that of substance abusers without PTSD (Booth et al., 1992). The significance of this finding, however, is unclear given the use of chart reviews and the attendant reliability issues. Furthermore, the 2% rate of PTSD for the total sample, when compared to the substantially higher rates reported by other veteran studies (McFall et al., 1991) suggests that the prevalence of PTSD was grossly underestimated in this study. Another investigation indicates that male veterans with both PTSD and alcohol problems have higher rates of recidivism than alcoholics without PTSD (Druley and Pashko, 1988). In tracking patient records following discharge from in patient substance abuse treatment, these researchers report that 70% of the PTSD group's files, compared to 30% of the non-PTSD group, were on another unit. These findings are interpreted by the authors as showing that non-PTSD patients seek substance abuse treatment and then exit the VA health care delivery system, whereas PTSD patients almost immediately re-enter the treatment system, presumably because of a relapse episode. However, since the nature of the services received by patients post-discharge from the substance abuse unit was not specified, competing hypotheses cannot be ruled out. For example, PTSD patients' continued use of VA services may be explained by their greater number of chronic physical problems (Litz et al., 1992; Wolfe et al., in press).

Kuhne et al. (1986) report a study of trauma-oriented therapy as an adjunct to residential alcoholism treatment for veterans with high combat exposure. Comparisons of abstinence rates at one-year follow-up revealed no significant differences between the heavy combat exposure, light combat exposure, and non-theater groups. Although the authors interpret these findings as indicating that trauma-oriented therapy ameliorated the negative effects of severe combat trauma, their results must be considered inconclusive given the absence of a heavy combat exposure control group. Also, since patients were not differentiated by PTSD diagnosis, no definitive statements about the impact of PTSD on substance abuse treatment outcome can be made.

Anecdotal evidence also indicates that PTSD substance abusers have an earlier onset of relapse than non-PTSD substance abusers. During the early stages of recovery, PTSD patients frequently report increased PTSD symptomatology and may be particularly vulnerable to relapsing (Kovach, 1986; Young, 1990). These clinical reports are supported by physiological studies showing that withdrawal from alcohol, opiates, and benzodiazepines is associated with central noradrenergic arousal, which mimics the biological effects of PTSD. Even after detoxification, conditioned withdrawal symptoms may continue to exacerbate PTSD symptomatology (Kosten and Krystal, 1988). Thus, it appears that PTSD substance abusers may be at higher risk for early relapse since their clinical picture may be exacerbated by the additive affects of drug withdrawal states and PTSD symptomatology.

In summary, the limited empirical literature provides equivocal support for the assumption that PTSD has a deleterious impact on substance abuse treatment outcome. To date, no investigation has compared substance abusers with and without a comorbid PTSD diagnosis on their alcohol and drug use following substance abuse treatment. In addition, the hypothesis that PTSD patients relapse earlier than non-PTSD substance abusers has not been empirically tested.

5. Additional psychopathology

Existing comparative studies of substance abusers with and without concurrent PTSD suggest that the clinically observed between-group differences in treatment outcome may be attributable to significant differences in psychopathology. In a study of Vietnam veterans receiving in patient substance abuse treatment, Hyer et al. (1991) found that those veterans with combat-related PTSD scored the highest on almost all MMPI validity scales and clinical scales. Although substance abuse was comparable among both PTSD and non-PTSD veterans (as assessed by the MAC content scale), the PTSD group was more decompensated on a wide range of psychopathological dimensions. Similarly, a study by McFall et al. (1991) compared substance abusers with and without a comorbid PTSD diagnosis and found that the PTSD patients reported significantly higher lifetime and current rates of psychiatric difficulties, such as trouble controlling violent tendencies/behavior, trouble with thoughts of suicide, and suicide attempts.

Although the above studies indicate that PTSD substance abusers have a greater degree of psychiatric symptomatology, comorbid diagnoses were not formally assessed. To date, no published study has examined whether substance abusers with concurrent PTSD have a greater number or different types of additional DSM-III-R (American Psychiatric Association, 1987) Axis I and Axis II disorders compared to substance abusers without a diagnosis of PTSD. Such research would shed light on whether group differences result from PTSD per se or from the variety of comorbid disorders that accompany both PTSD and substance abuse disorders. The high rate of concurrent psychiatric disorders among individuals with PTSD (Keane and Wolfe, 1990) has led some researchers to question the validity of PTSD as a unique diagnostic entity (Goodwin and Guze, 1984). While considerable support for PTSD as a distinct diagnostic category has now emerged (Keane et al., 1987; Solomon et al., 1991), no published study to date has specifically examined whether PTSD is more likely than other disorders to appear in conjunction with other syndromes. However, drawing on separate findings from the Epidemiological Catchment Area Survey, we can conclude that comorbidity in PTSD (Helzer et al., 1987) and comorbidity in alcohol abuse/dependence (Helzer and Pryzbeck, 1988) occur at comparable rates. Individuals with PTSD are twice as likely to have some other disorder compared to persons without PTSD and, similarly, individuals with alcohol abuse/dependence are 2.8 times more likely to suffer from a co-existing disorder than are individuals without a substance abuse/dependence diagnosis. Further comparative analysis of the specific types of comorbidity (e.g., the likelihood of major depression occurring with PTSD versus substance abuse) will allow us to more accurately gauge the special significance of PTSD-substance abuse comorbidity.

6. Coping skills

Although little is known about factors which potentially moderate or mediate the relationship between psychiatric comorbidity and substance abuse treatment outcome, theoretically guided studies of substance abusers consistently show that coping skills play an important role in the recovery process (Marlatt and Gordon, 1985). Empirical research documents that the more frequent use of cognitive coping predicts abstinence at six-months post-treatment (Ito and Donovan, 1990) and that the use of coping strategies is significantly associated with the prevention of relapse (Sjoberg and Samsonowitz, 1985; Wells et al., 1989). Similarly, other investigations have shown that increased drinking following treatment is associated with both skills deficits (Chaney et al., 1978; Marlatt and Gordon, 1980) and the failure to use alternative coping responses (Cronkite and Moos, 1980).

Several studies (Nezu and Carnevale, 1987; Green et al., 1988) show that PTSD is associated with more avoidant, emotion-focused coping strategies (i.e., efforts to regulate emotions that are associated with or result from stressor exposure) than with problem-focused strategies (i.e., efforts to eliminate the impact of a stressor). Research also shows that patients with rape-related PTSD (Foa et al., 1989) or combat-related PTSD (Fairbank et al., 1991) have more avoidant coping styles than non-PTSD comparison groups. In a study comparing the coping behaviors of male substance abusing veterans with and without concurrent PTSD, PTSD patients were found to evidence significantly more avoidant coping styles (Penk et al., 1988).

Although available research suggests that differences exist in the coping styles of PTSD and non-PTSD substance abusers, no study to date has examined diagnostic subgroup differences in skills for coping with high risk relapse situations. Research by Fairbank et al. (1991), however, suggests that PTSD patients' avoidance coping style may be generalized to their coping behaviors in the specific context of a high risk relapse situation. In their examination of the coping efforts of PTSD patients, Fairbank and his colleagues

found that avoidance coping was used regardless of the type of stressor. These authors speculate that PTSD patients with a long history of coping with their trauma symptomatology may have developed a preferred or characteristic coping response to stressors. Although this hypothesis contrasts sharply with the prevailing view of coping as a fluid, stressor-specific phenomenon, those who have been exposed to extreme events and subsequently develop PTSD may be an exception. Thus, PTSD may differentially influence relapse through deficits in skills for managing high risk relapse situations.

7. Treatment issues: sequential versus concurrent approaches

The treatment of PTSD patients with comorbid substance abuse has been an extremely controversial topic, with the core dilemma being whether to treat these two disorders sequentially or simultaneously. In the sequential approach, the patient's substance abuse is targeted first with the goal of achieving a substantial period of sobriety before beginning treatment for the PTSD (Roy, 1984; Moyer, 1988; Nace, 1988; Brinson and Treanor, 1989). The underlying assumption is that substance abuse is primary and its successful treatment will enhance the prognosis for successful treatment of PTSD. In contrast, the simultaneous approach involves the treatment of both PTSD and substance abuse problems at the beginning of the recovery process. Advocates of the concurrent approach (Bollerud, 1990; Boudewyns et al., 1991; Friedman, 1991) argue that postponing trauma-related treatment increases the patient's risk of relapsing. The assumption is that regardless of whether PTSD preceded substance abuse or vice versa, the two disorders are interwined and that PTSD patients will quickly return to using alcohol and/or drugs to manage their PTSD symptomatology unless they begin to address their trauma issues and learn alternative coping or symptom-management skills. As noted above, anecdotal evidence suggests that without treatment focused on their trauma, PTSD substance abusers experience more difficulty in the early stages of sobriety and may be more susceptible to relapse (Kovach, 1986; Young, 1990). Additional support for the concurrent treatment of substance abuse and PTSD comes from single subject design case studies in the literature. These studies suggest that marked reductions in alcohol abuse can be obtained by successful treatment of PTSD anxiety symptoms (LaCoursiere et al., 1980; Fairbank and Keane, 1982; Keane and Kaloupek, 1982; Fairbank et al., 1983).

To date, no empirical data are available on whether substance abuse treatment outcome is affected by the introduction and/or timing of PTSD treatment. Until such research is conducted, the efficacy of specific treatment interventions for PTSD substance abusers will remain indeterminate.

8. Methodological issues in interpreting studies on substance abuse and PTSD

This literature review of studies on substance abuse and PTSD reveals a number of limitations in current research. The purpose of this section is to highlight four substantive methodological issues which need to be addressed in future studies of this 'dually-diagnosed' population. These problems involve the assessment of substance abuse, the assessment of PTSD, the nature of the stressor examined, and treatment outcome measures.

8.1. Assessment of substance abuse problems

In the studies reviewed above, a range of different definitions of substance abuse problems are employed. For the most part, the criteria used to diagnose substance abuse have not been specified. For those studies utilizing chart diagnoses (Booth et al., 1991), it is impossible to determine the definitions/criteria employed and to assess the degree of diagnostic reliability.

Another methodological shortcoming is that the majority of studies focus on alcohol and fail to assess for concurrent drug use (Druley and Pashko, 1988). With respect to the assessment of drug abuse and dependence, Cottler et al. (1992) found that rates of exposure to traumatic events and PTSD vary across type of substance used. These findings suggest that future studies should include the specific category of drug abused rather than merely the presence or absence of any form of a drug problem.

8.2. Assessment of PTSD

Our review of the literature reveals that there is great variability in the methods used to assess PTSD. Although the majority of studies have used some form of a structured clinical interview, others have employed only symptom checklists (Druley and Pashko, 1988). Most studies may also be criticized for attempting to measure a very complex disorder by only one means. The most advocated assessment approach is a comprehensive, multiaxial one, which incorporates information from a broad range of independent sources (e.g., psychometric instruments, clinical interview), thereby providing a way to obtain convergence of findings and enhance diagnostic reliability and validity (Keane et al., 1987; Wolfe et al., 1987; Lyons et al., 1988; Kulka et al., 1990; Wolfe and Keane, 1992). Only one study reviewed in this paper (Brief et al., 1992) has used such a multimethod approach to assess PTSD.

Two psychological instruments in particular — the Mississippi Scale for Combat-Related PTSD and its civilian version (Keane et al., 1988) as well as the MMPI PTSD Subscale (Keane et al., 1984) — have received

empirical support for their diagnostic utility. The Mississippi Scale for Combat-Related PTSD consists of 35 items selected from an initial pool of 200 items reflecting DSM-III criteria and additional items assessing associated features, such as depression and substance abuse. This scale has excellent internal consistency and test-retest reliability and has been shown to reliably and accurately distinguish veterans with and without PTSD, using a cut-off score of 107 (Keane et al., 1988). A 39-item version of the Mississippi Scale for assessing civilian trauma is now available from Keane and his colleagues. Preliminary research on this instrument shows that it possesses acceptable psychometric properties (Vreven et al., 1993), although the cut-off score may need to be adjusted depending on the clinical setting (e.g., in patient psychiatric unit versus community mental health center).

The 49-item MMPI PTSD (PK) scale (Keane et al., 1984) can be calculated from either the original MMPI (49 items) or the revised MMPI-2 (46 items). The MMPI-2 PK scale has been shown to possess excellent internal consistency and test-retest reliability (Graham, 1990) and performs well in differentiating cases of PTSD among both veteran (Keane et al., 1984; Watson et al., 1986; Cannon et al., 1987) and civilian groups (Koretzky and Peck, 1990).

In conjunction with the aforementioned self-report measures, experts in the field of PTSD advocate the use of a structured clinical interview (Litz et al., 1992; Wolfe and Keane, 1992). Currently, the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990) represents the state-of-the-art structured interview for assessing PTSD. The CAPS measures DSM-III-R (American Psychiatric Association, 1987) symptoms of PTSD, associated symptoms of PTSD (e.g., survivor guilt), overall symptom severity, impairments in social and occupational functioning, and validity of responses. Studies indicate that the CAPS has sound psychometric properties (Blake et al., 1990) and excellent diagnostic utility against the SCID PTSD diagnosis (Weathers et al., 1992).

8.3. Type of stressors examined

In reviewing the clinical literature on PTSD among substance abusers, it is apparent that most studies typically restrict their exposure assessment to only one type of traumatic event (e.g., combat). More comprehensive assessments of PTSD across the life span are needed to elucidate the relationships between the type and number of traumatic stressors experienced and substance abuse. Epidemiological studies have shown that individuals are at risk of being exposed to a wide range of potentially traumatizing events, including robbery, physical assault, injury or serious accident, etc., and that different types of exposure occur at different rates and are associated with different rates of PTSD (Breslau et al., 1991; Norris, 1992). It may similarly be that the development of a substance abuse problem is a function of the type of stressor experienced. This notion receives some support from a study by Green et al., (1989) showing that veterans who were exposed to grotesque death were more likely to have PTSD accompanied by alcohol abuse and depression compared to veterans exposed to terrifying, high-risk situations who were more likely to have PTSD accompanied by a phobic disorder.

Several studies have indicated that individuals who have been exposed to a traumatic event are at increased risk for additional exposure (Russell, 1986; Koss and Harvey, 1991). There is also some evidence that certain types of stressors may co-occur frequently, for example, war-zone exposure and sexual assault/rape during military service (Wolfe et al., 1992). To date, no study has examined the contribution of multiple exposure to the probability of developing substance abuse problems. Although data from Breslau et al. (1991) suggest that the lifetime prevalence of PTSD might increase geometrically with increasing exposure to traumatic events, this hypothesis remains untested with respect to presence and severity of substance abuse problems.

Collectively, these findings suggest the need for comprehensive assessments of exposure to traumatic events across the life span. In particular, a history of childhood physical/sexual abuse and adulthood sexual assault/rape should be routinely assessed, given that these types of trauma occur at alarming rates (Russell, 1986; Wyatt, 1986; Finkelhor et al., 1989; National Crime Center, 1992). In addition to clinical interviews, screening instruments (Norris, 1990; Krinsley and Young, 1992; Wolfe and Levin, 1992) may be helpful in eliciting information about specific traumatic experiences.

8.4. Treatment outcome measures

In all but one of the treatment outcome studies reviewed above (Kuhne et al., 1986), results were limited by the absence or brevity of the follow-up period. In addition, all of these studies may be criticized for using single outcome measures and single sources of data. Emrick and Hansen (1983) emphasize the importance of employing a broad array of outcome variables in follow-up assessment so as to determine whether achievement of abstinence or reduced use is associated with other improvements in functioning. In addition to assessing substance use behaviors and PTSD symptomatology, the following indices should be assessed: (a) treatment completion; (b) treatment use (e.g., number of out patient sessions attended, number of hospitalizations);(c) family/social functioning; (d) vocational and legal status; (e) emotional functioning; (f) physical health; (g) mortality; and (h) life stressors. Emrick and Hansen (1983) also recommend using a variety of data sources, such as patient self-report, reports by significant others and collaterals, biological markers, and examination of hospital, police, and employment records, to increase validity.

9. Conclusion

The review of the literature on substance abusers with and without PTSD reveals a discontinuity between theory, clinical practice, and empirical research. Despite an abundance of anecdotal evidence supporting claims of the 'high risk' status of PTSD substance abusers, relatively little is known about the treatment prognosis for this 'dually diagnosed' population. Several basic questions still need to be addressed: (a) Are substance abusers with a comorbid diagnosis of PTSD at a greater risk for substance abuse relapse than their non-PTSD counterparts? (b) Do PTSD substance abusers have an earlier onset of relapse than non-PTSD substance abusers? (c) Are rates of relapse significantly affected (and in what way) by the inclusion of trauma/PTSD treatment? (d) Which treatment approach (sequential versus simultaneous) is more effective? (e) How do PTSD substance abusers and non-PTSD substance abusers compare on factors which have been associated with substance abuse treatment outcome (e.g., coping skills, additional psychopathology)? (f) Are the predictors of treatment outcome similar or different for PTSD and non-PTSD substance abusers?

Future progress in successfully treating PTSD substance abusers is contingent upon our ability to answer these questions. Ultimately, longitudinal and prospective studies are needed to shed light on the impact of PTSD comorbidity on substance abuse treatment outcome.

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