Alcohol Abuse in Individuals Exposed to Trauma: A Critical Review

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In this article, the author critically reviews studies on the relationship between exposure to trauma, posttraumatic stress disorder (PTSD), and alcohol abuse. After establishing that strong relationships exist between exposure to traumatic events and alcohol problems, particularly between the diagnoses of PTSD and alcoholism, the author discusses various factors, theories, and possible mechanisms to account for these associations. Moreover, she discusses applications of these findings to the assessment and treatment of people exposed to trauma who abuse alcohol. Finally, the author outlines novel methods for testing theoretical hypotheses and makes suggestions for methodological improvements in future research.

This article critically reviews the evidence for an important functional relationship between posttraumatic stress disorder (PTSD) and alcoholism. Although several reviewers have shown a significant overlap between certain anxiety disorders and alcohol abuse (e.g., B. J. Cox, Norton, Swinson, & Endler, 1990; Kushner, Sher, & Beitman, 1990; Pihl & Stewart, 1991), they did not consider the relationship between PTSD and alcoholism. One review examined the relationship between PTSD and substance abuse (i.e., Keane, Gerardi, Lyons, & Wolfe, 1988). However, their focus was very different from this review, assessing abuse of a wide range of chemical substances by only Vietnam veterans exposed to trauma. The purposes of this article are to (a) examine the empirical evidence for the co-occurrence of trauma exposure and alcohol problems and for the comorbidity between PTSD and alcoholism across various traumatic events; (b) critique methodological issues in the literature; (c) review theoretical mechanisms that may account for the relationship of trauma exposure, PTSD, and alcohol abuse; (d) discuss the implications of this research for the assessment and treatment of individuals comorbid for PTSD and alcoholism; and (e) suggest directions for future research.

It should be made clear at the outset that this review focuses specifically on alcohol abuse in response to psychological trauma (e.g., sexual assault) as opposed to stressful life events (i.e., negative events that are not necessarily traumatic, such as divorce; Holmes & Rahe, 1967) or physical trauma (e.g., head injury). As these latter two issues are not a focus of this article, the interested reader is referred to reviews on the associations between alcohol abuse and stressful life events (Beckman, 1976; Benson & Wilsnack, 1983; Straussner, 1985) and alcohol abuse and physical trauma (Solomon & Malloy, 1992). Although considerable empirical data suggest an overlap between PTSD and the abuse of various illicit substances (e.g., opiates; Cottler, Compton, Mager, Spitznagel, & Janca, 1992), this review focuses only on the abuse of alcohol, given its ready accessibility. Finally, this review examines only the development of alcoholism following trauma, although there is evidence that alcohol abuse increases the likelihood of being a victim or a perpetrator of trauma (Murdoch, Pihl, & Ross, 1990; Pihl, Peterson, & Lau, 1993).

Definitions

Throughout this review, there are frequent references to the diagnoses of PTSD and alcoholism. I provide definitions and base-rate prevalences of these two forms of behavioral pathology to familiarize the reader with these disorders from the outset.

Posttraumatic Stress Disorder

PTSD comprises a characteristic set of symptoms that may develop following the experiencing, witnessing, or confronting of an event involving actual or threatened serious injury or death or a threat to the physical integrity of the self or others (Diagnostic and Statistical Manual of Mental Disorders [DSM–IV] 4th ed.; American Psychiatric Association [APA], 1994). Such events are experienced with fear, helplessness, or horror and are exemplified by combat, sexual and physical assault, natural and man-made disasters, and accidents. The characteristic symptoms include reexperiencing the traumatic event (intrusive thoughts, flashbacks, nightmares, and physiological reactivity to reminders of the trauma), avoidance of reminders of the trauma (efforts to avoid stimuli associated with the trauma and to suppress thoughts or feelings about it), emotional numbing (inability to feel close to others, to experience positive emotions, or to enjoy pleasurable activities), and increased physiological arousal (hypervigilance, exaggerated startle response, concentration difficulties, sleep disturbance, and irritability). A PTSD diagnosis requires that symptoms be present for more than 1 month and that the disturbance causes...
clinically significant distress or impairment in important areas of functioning.

Exposure to life-threatening and catastrophic experiences, whether in the context of war, crime, disasters, or accidents, has lasting consequences for many individuals. A recent epidemiological study estimated the prevalence of PTSD in the adult population to be between 1% and 2% (Helzer, Robins, & McEvoy, 1987). Such estimates potentially yield some 2.4 to 4.8 million cases of PTSD in the United States alone.

Alcoholism

This article takes a broad view of alcohol-related problems, reviewing the literature on both alcohol abuse and alcohol dependence. A DSM-IV diagnosis of alcohol abuse is made when an individual’s pattern of alcohol use is maladaptive, as indicated by at least one of the following symptoms within a 12-month period: recurrent alcohol use that results in not being able to fulfill major role obligations; recurrent use in physically hazardous situations (e.g., drunk driving); recurrent alcohol-related legal problems; or continued use despite persistent social problems, occupational problems, or both caused or exacerbated by alcohol’s effects.

Alcohol dependence involves a cluster of symptoms indicating impaired control over alcohol use. In addition to the characteristic symptoms of alcohol abuse, other symptoms of alcohol dependence include the use of alcohol in larger amounts or over a longer period than the individual intended; prior unsuccessful attempts to control the use of alcohol; a great deal of time spent acquiring alcohol or recovering from its effects; impairment in social, occupational, or recreational activities due to alcohol use; heightened tolerance to alcohol; withdrawal symptoms resulting from discontinuation of alcohol use or alcohol use to relieve or avoid withdrawal symptoms; and continued alcohol use despite knowledge of having a physical or psychological problem caused or exacerbated by alcohol.

Alcohol and drug abuse are the most prevalent of all the psychiatric disorders (Robins et al., 1984). The Epidemiological Catchment Area (ECA) study showed a lifetime prevalence rate of alcohol abuse—dependence in the United States of 12–16% (Robins et al., 1984).

Comorbidity Studies

In the first half of this section, I establish an association between trauma exposure and alcohol problems. In the last half of this section, I review literature on the association between the diagnoses of PTSD and alcoholism. Throughout this section, I attempt to establish whether it is trauma exposure or PTSD that is most highly related to posttraumatic abusive drinking. Following this section, I provide a methodological critique of the comorbidity literature and discuss various factors that might contribute to the different comorbidity rates seen across studies.

Is There a Relationship Between Trauma Exposure and Alcohol Abuse—Dependence?

Because the majority of research on the association between trauma exposure and alcohol abuse—dependence is limited to three specific traumatic events (i.e., disasters, assault, and combat), the literature review is organized around them.

Disasters. The relationship between trauma exposure and alcohol abuse has been investigated retrospectively by examining changes in alcohol consumption and various “social indices” of abusive drinking (e.g., drunk driving charges) following natural disasters. For example, Green, Grace, and Winget (1981) found that alcohol consumption increased by 30% in a 2-year period following the 1972 flood at Buffalo Creek, West Virginia. Similarly, P. R. Adams and Adams (1984) found significant increases in several social indices of alcohol abuse (e.g., drunk driving charges and referrals to community alcohol centers) following the Mount Saint Helens’ volcano eruption in Washington State. These data suggest that exposure to trauma in the form of a natural disaster may, indeed, lead to increases in excessive drinking, at least in terms of social indices of abusive drinking.

Green, Grace, and Gleser (1985) found a relationship between exposure to grotesque death and an alcohol abuse diagnosis in a sample of disaster survivors of the 1977 Beverly Hills Supper Club fire. They also found that the greater the degree of involvement in rescue efforts (i.e., exposure to burned and mutilated bodies), the greater the likelihood of alcohol abuse 2 years after the fire. Thus, studies examining social indices of drinking or alcohol abuse diagnoses suggest a relationship between exposure to disaster and subsequent abusive drinking. Moreover, the data suggest that certain aspects of the trauma (i.e., exposure to grotesque death) may be most highly associated with posttraumatic alcohol abuse.

Assault. Case reports in which alcoholism appears related to a history of sexual assault, physical assault, or both are abundant in the literature. In this section, I review empirical studies that examine the associations among sexual abuse, physical abuse, or both in childhood (both intrafamilial and extrafamilial), sexual assault, physical assault, or both in adulthood (e.g., rape), and levels of alcohol abuse in adolescence or adulthood.

Some researchers have examined rates of alcoholism in psychiatric outpatients (e.g., Mieselman, 1978; Swett, Cohen, Survey, Compaine, & Chavez, 1991). For example, Swett et al. (1991) surveyed 189 women at an adult psychiatric outpatient clinic. Women with a self-reported history of sexual abuse, physical abuse, or both obtained significantly higher scores on the Michigan Alcoholism Screening Test (MAST; Selzer, 1971), a self-report measure of alcohol-related problems, than did women reporting no such history. These results indicate higher levels of alcohol problems in women with abuse histories. Interestingly, when the abuse occurred before the age of 18 with no subsequent abuse in adulthood, the association of abuse history and high MAST scores persisted. Thus, even early abuse may be associated with increased levels of problem drinking in adult women.

Other researchers have examined the relationship between victimization and alcohol abuse within more circumscribed psychiatric samples. For example, Briere and Zaidi (1989) reviewed medical records of 50 women admitted to an urban psychiatric emergency room (85% of whom were unemployed). More of the women reporting a history of sexual abuse met DSM-III-R (3rd ed., rev., APA, 1987) criteria for alcohol...
abuse than those reporting no such abuse histories (37% vs. 20%, respectively). However, both groups had higher rates of alcohol abuse than lifetime prevalence rates seen in the general female population of approximately 4% to 5% (e.g., Robins et al., 1984), a finding that may be due to Briere and Zaidi's sample characteristics (i.e., from a psychiatric emergency room and lower-than-average socioeconomic status [SES]).

The prevalence of abuse histories in individuals (mainly women) seeking or receiving treatment for alcoholism has also been examined. Results consistently reveal exceedingly high rates of self-reported sexual-abuse histories in samples of women with alcoholism (i.e., 24–85%), although the precise rates across studies vary widely (e.g., Cohen & Densen-Gerber, 1982; Kovach, 1986; Wilsnack, 1982, 1984). Despite these differences, even the lowest rate is clinically significant when compared with base rates. For example, the prevalence of incest and sexual abuse, obtained with samples of women with alcoholism, are much higher than the 10–30% rate of incest reported by women in the general population (Finkelhor, 1979; Gagnon, 1965) and the 16% rate of childhood sexual abuse reported by women referred for psychotherapy (Sheldon, 1988).

Covington (1983) included a matched group to control for the effects of extraneous variables such as SES. She compared 35 women with alcoholism with 35 matched female controls without alcoholism. Significantly more women with alcoholism than those without reported a history of incest (i.e., 34% vs. 17%, respectively).

B. A. Miller, Downs, and Testa (1993) examined the relationship between childhood victimization and subsequent alcoholism in adult women, while controlling for the effects of being in treatment. They compared the reports of childhood victimization of a sample of women having alcoholism with those of a sample of women without alcoholism in treatment for other mental health problems. They found a higher rate of childhood victimization in women with alcoholism versus the in-treatment controls, suggesting a specific link between victimization and the later development of alcohol problems in adult women. More important, the significant relationship between alcoholism diagnoses and childhood victimization remained, even after family background and demographic factors (e.g., parental alcohol problems) were statistically controlled. Other studies of samples of both male and female adolescents and adults also support the connection between childhood victimization histories and the development of alcohol abuse problems (e.g., Flanagan, Potrykus, & Marti, 1988; Harrison, Hoffman, & Edwall, 1989; Hernandez, 1992; Hernandez, Ladco, & DiClemente, 1993; Pribor & Dinwiddie, 1992; M. R. Schaefer, Sobieraj, & Hollyfield, 1988; Singer, Petchers, & Hussey, 1989).

Dembo and colleagues conducted extensive interviews with juvenile detainees to determine the causal connections between sexual abuse, physical abuse, or both and alcohol abuse (e.g., Dembo, Dertke, Borders, Washburn, & Schneider, 1988; Dembo, Williams, Wothke, Schneider, & Brown, 1992). Using structural equation modeling, they obtained evidence that both forms of abuse appear to precede and substantially contribute to the development of alcohol abuse. This finding is consistent across gender and over time (Watts & Ellis, 1993).

Other researchers have examined the relationship between sexual assault, physical assault, or both and alcohol abuse in the general population (i.e., non-treatment-seeking). For example, Wilsnack and colleagues (Klassen & Wilsnack, 1986; Wilsnack, 1991) conducted a longitudinal survey of a large representative American sample to investigate the causes and consequences of women's drinking. The sample was divided into "problem" and "nonproblem" drinkers. More than twice as many problem as compared with nonproblem drinkers reported experiencing at least one incident of sexual abuse before the age of 18. A history of sexual abuse significantly predicted the onset of drinking problems over the 5-year follow-up in those classified as nonproblem drinkers at the start of the study (i.e., 51% vs. 19% in abused and nonabused groups, respectively). In another community study, Winfield, George, Swartz, and Blazer (1990) surveyed approximately 1,200 women from the North Carolina site of the ECA study. All women were interviewed with the Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, & Ratcliff, 1981) and were questioned about their histories of sexual assault. DIS diagnoses of alcohol abuse—dependence were significantly higher in the women assaulted versus the women not assaulted.

Some data suggest that the nature of the abuse may differentiate individuals who later develop alcohol problems from those who do not. For example, Covington (1983) found that incest was more violent and more frequent, involved more perpetrators, and was of longer duration in the group with alcoholism as compared with the control group without alcoholism. A similar study by S. Schaefer, Evans, and Sterne (1985) compared 100 women in treatment for alcoholism with 42 matched female controls without alcoholism on a number of childhood sexual abuse measures. When a history of sexual abuse was reported, the first incident occurred significantly earlier, its duration was significantly longer, and informing a parent of the abuse was less frequent in the women with alcoholism relative to the controls without alcoholism. Thus, preliminary data suggest that the severity of sexual abuse may prove important in predicting the development of alcohol abuse. Specifically, more severe levels of abuse appear significantly associated with greater risk for alcohol problems.

**Combat.** An extensive literature exists on the relationship between exposure to combat and the subsequent development of alcohol abuse. For example, a national epidemiological study (Boscarino, 1981) compared a group of Vietnam combat veterans with control groups (noncombat veterans and nonveterans), matched for important demographic characteristics, such as age, education level, and income. Combat veterans exhibited substantially higher levels of binge drinking than the matched controls. Boman (1986) compared a psychiatric sample of 23 male patients who had served in combat in Vietnam with a control psychiatric group of 25 male patients who were currently serving in the military but had never been in combat. Significantly more of the combat-exposed men than the noncombat controls met DSM-III (3rd ed., APA, 1980) criteria for alcohol abuse (78% vs. 36%, respectively). Because preexisting differences between soldiers assigned to combat duty and those not assigned might contribute to the differences in posttrauma problem drinking (Roy, 1983), retrospective diagnoses were made for each participant for the period between leaving high school and entering the military. About 20% of the men in both groups met diagnostic criteria for alcohol abuse.
before military enlistment. The absence of a significant difference between the groups in terms of proportion with alcohol abuse diagnoses before military service suggests that exposure to combat, as opposed to preexisting group differences, is specifically associated with increased rates of alcohol abuse following military service.

Green, Grace, Lindy, Gieser, and Leonard (1990) examined the relative contribution of prewar (e.g., level of education and peer relationships in school), war (i.e., level of combat exposure), and postwar (e.g., negative life events and social support) factors to the long-term postservice adjustment of a group of Vietnam veterans, most of whom were not seeking treatment. They administered the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) to obtain current diagnostic information. Prewar factors made a significant contribution to the prediction of alcohol abuse diagnoses. However, the addition of war and postwar variables each significantly improved the prediction. In contrast, combat exposure made no additional contribution to the prediction of other psychiatric disorders, including major depression, which were largely explained by prewar factors. Thus, combat exposure was again significantly (and quite specifically) associated with alcohol abuse, even after the consideration of other relevant variables.

Additional data suggest that the level of combat exposure may be specifically associated with an increase in abusive drinking. For example, Laufer, Yager, Frey-Wouters, and Donnellan (1981) found that severity of combat exposure was significantly positively associated with self-reported drinking levels in a clinical sample of combat veterans. Kulka et al. (1990) replicated this finding in a nonclinical sample of combat veterans from the National Vietnam Veterans Readjustment Study (NVVRS). Furthermore, Branchey, Davis, and Lieber (1984) showed in a clinical sample that the association between combat exposure and alcohol abuse persisted over an extended period after service in Vietnam. Yager, Laufer, and Gallops (1984) interviewed a national nonclinical sample of men who were of military age during the Vietnam War. These interviews were conducted 6 to 15 years after veterans in the sample had left military service. When preservice background factors were controlled statistically, combat exposure severity showed a significant positive association with a measure of “daily drinking” (i.e., number of months in the last 2 years the participant had consumed at least a six pack of beer, a bottle of wine, or several drinks of hard liquor almost every day). These findings suggest that the association between combat severity and heavy alcohol consumption persists over an extended period following combat exposure, even in nonclinical participants.

Sutker, Uddo, Brailey, Vasterling, and Errera (1994) extended this work by examining psychiatric diagnoses in military troops performing graves registration duties during the Persian Gulf War. Current and lifetime diagnoses of alcohol dependence tended to be higher in the war-zone deployed group (who had been exposed to the handling of human remains) than in a matched control group consisting of troops stationed stateside. Thus, increased alcohol problems may not only be associated with exposure to combat but also with other war-related traumatic experiences. In summary, research supports the notion that combat exposure, severity of exposure, and other war-related traumatic experiences are associated with increased levels of abusive drinking.

Epidemiological data. Cottler et al. (1992), using data from nonclinical participants in the St. Louis ECA study, examined the relationship between exposure to a wide variety of traumatic events and heavy alcohol use. “Heavy alcohol users” were over three times more likely than “non-heavy alcohol users” and nonusers to report having been exposed to combat-related trauma. However, the three alcohol-use groups did not differ in terms of their reported exposure to other forms of traumatic events, including serious accident, physical attack, or witness to a killing or significant injury. These findings suggest that rates of posttraumatic alcohol abuse may vary as a function of the nature or type of traumatic exposure.

Summary. There is much empirical evidence attesting to an important relationship between exposure to traumatic events and increased abusive drinking. Moreover, the data suggest that objective indices of trauma severity may be positively associated with the degree of abusive alcohol consumption. Thus, exposure to trauma, particularly severe trauma, may lead to the development of alcohol abuse—dependence.

Is There a Relationship Between PTSD and Alcohol Abuse—Dependence?

It is possible that the positive correlations between trauma severity and alcohol problems do in fact reflect an association between PTSD and alcohol abuse because trauma severity is positively associated with the emergence of PTSD symptoms. Thus, some researchers have suggested that an adverse psychological reaction to the trauma (i.e., PTSD symptoms) may better account for the increased abuse of alcohol following exposure to trauma than exposure to trauma or severity of the traumatic event, per se (e.g., LaCoursiere & Coyne, 1984). Recently, researchers have focused more directly on the relationship between the diagnoses of PTSD and alcohol abuse—dependence. In this section, I review studies on the comorbidity of PTSD and alcoholism. Again, this review is organized with reference to the nature of the traumatic event (i.e., disaster, assault, and combat). I also review epidemiological studies spanning a wide variety of traumatic events.

PTSD due to disaster. Green, Lindy, Grace, and Leonard (1992) conducted a 14-year posttrauma follow-up of 193 victims of the 1972 Buffalo Creek flood. Diagnoses were established with the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, & Gibbon, 1987). A current PTSD diagnosis was statistically related to an alcohol abuse diagnosis, suggesting that participants who were experiencing significant flood-related PTSD symptoms at the follow-up were also likely to be abusing alcohol. Although the comorbidity pattern of PTSD with other disorders in individuals exposed to the disaster was similar to a previously tested sample of combat veterans (Green, Lindy, Grace, & Gieser, 1989), a direct comparison revealed that the individuals exposed to the disaster demonstrated lower rates of comorbid PTSD and alcohol abuse than did the combat veterans. These data suggest that some types of trauma may more likely lead to comorbid PTSD and alcohol abuse than others. This result is consistent with Cottler et al.’s (1992) findings that heavy drinking was most highly associated
with prior exposure to combat trauma, as opposed to other types of traumatic events.

**PTSD due to assault.** Kovach (1986) showed that nearly 40% of women attending Alcoholics Anonymous who reported histories of incest met DSM-III criteria for PTSD (about 12% of a sample of 117 women). She postulated that childhood incest may result in delayed onset PTSD, which in turn may contribute to the development of alcohol dependence. Kovach unfortunately did not assess her comparison group to determine if PTSD was more likely to be found among women with alcoholism who were exposed to incest than among women with alcoholism who did not report a history of incest. Thus, Kovach's postulation of a PTSD link between childhood incest and adult alcoholism remains speculative on the basis of her data alone (Hurley, 1991).

Clark and Jacob (1992) examined comorbid psychiatric diagnoses in a sample of adolescents meeting DSM-III-R criteria for alcohol abuse-dependence. Twenty-seven percent of the adolescents with alcoholism were diagnosed with PTSD, the most frequent anxiety disorder diagnosis in the sample. Consistent with Kovach's (1986) postulation of a link with childhood histories of sexual abuse, PTSD, and the later development of alcohol abuse problems, 88% of the adolescents comorbid for PTSD and alcoholism reported prior histories of sexual abuse, physical abuse, or both.

Also relevant to the review of the relationship of alcohol abuse and PTSD due to assault are the emerging data on the effects of exposure to other forms of violent crime. Kilpatrick and Resnick (1993) reviewed data from a large national probability sample of adult women. The women were divided into three groups: those with PTSD who experienced a crime, those without PTSD who experienced a crime, and those not having experienced a crime. All participants were assessed for the presence of alcohol problems. Independent of PTSD status, experiencing a crime increased the risk of developing alcohol problems. PTSD was an additional major risk factor for alcohol problems within the groups who experienced a crime. Those with PTSD were 3.2 times more likely than those without PTSD and 13.7 times more likely than those not having experienced a crime to report serious alcohol problems. These results suggest that criminal victimization may increase the risk for alcohol abuse among adult women. Moreover, those who experience a crime and display extreme emotional distress following the crime (i.e., those with PTSD) may be at highest risk for developing major alcohol problems.

**PTSD due to combat.** Many studies have examined the overlap between combat-related PTSD and alcoholism. In fact, the majority of research on the relationship between PTSD and alcoholism has been conducted with combat veterans. For example, Branchey et al. (1984) reported that alcohol abuse is the most prevalent coexisting psychiatric diagnosis in combat veterans with PTSD. In the studies involving clinical samples of male combat veterans with PTSD, rates of alcohol abuse-dependence are extremely high (41–85%), although the precise rates vary widely across studies (e.g., Davidson, Kudler, Saunders, & Smith, 1990; Davidson, Swartz, Storck, Krishnan, & Hammett, 1985; Escobar et al., 1983; Hendin & Haas, 1984; Keane, Caddell, & Martin, 1983; Keane, Gerardi, et al., 1988; Siersles, Chen, McFarland, & Taylor, 1983; Streimer, Cosstick, & Tennant, 1985). The rates of alcoholism in these studies much exceed the lifetime prevalence rates for alcohol abuse—dependence in the general male population of approximately 19–29% (Robins et al., 1984).

The overlap between combat-related PTSD and alcoholism has also been examined in nonclinical samples of veterans. In the NVVRS (Kulka et al., 1990), conducted with a nonclinical sample of Vietnam veterans, 74% of the male veterans with PTSD and 29% of the female veterans with PTSD also met SCID (Spitzer et al., 1987) lifetime criteria for alcohol abuse. The corresponding figures for current alcohol abuse diagnoses (i.e., within the previous 6 months) were 22% for the men with PTSD and 10% for the women with PTSD. Alcohol abuse was the most common comorbid disorder among men with PTSD, and the third most common comorbid disorder among women with PTSD. Similar figures were obtained in the Center for Disease Control Vietnam Experience Study, also conducted with a nonclinical sample of Vietnam veterans: 70% of Vietnam combat veterans who had ever met the criteria for PTSD also met lifetime criteria for alcohol abuse (cited in Davidson & Fairbank, 1993). Thus, the strong relationship between PTSD and alcohol abuse is evident, even in nonclinical samples.

Streimer et al. (1985) attempted to directly determine whether alcohol abuse is associated with PTSD or merely with exposure to combat. They examined the medical records of a random sample of 126 inpatients who had served in Vietnam. Only 29% of the sample met DSM-III criteria for a primary diagnosis of alcohol abuse—dependence. However, the rate of alcohol abuse—dependence diagnoses rose to 38% in those also diagnosed with combat-related PTSD, suggesting that alcoholism is more closely linked with PTSD than with combat exposure, per se.

Boman (1986) also examined the relationship between combat-related PTSD and alcohol abuse. Vietnam veterans with PTSD, Vietnam veterans without PTSD, and military personnel without PTSD who had not served overseas and who were receiving psychiatric treatment were compared for rates of DSM-III diagnoses of alcohol abuse, established with a standard psychiatric interview. The three groups were comparable on important demographic variables (e.g., age and SES). Diagnoses of alcohol abuse were more prevalent in the Vietnam veterans with PTSD (78%) than in the veterans without PTSD (72%) and more prevalent in the Vietnam veterans without PTSD than in the nonveteran, non-PTSD controls (36%). The groups did not differ with respect to alcohol abuse diagnoses before military enlistment. None of the non-PTSD Vietnam veterans had been exposed to life-threatening combat, whereas all those in the PTSD group had been exposed, introducing a confound between combat exposure and a PTSD diagnosis. Thus, it remains unclear as to whether the somewhat higher rate of alcohol abuse in the Vietnam veterans group with PTSD compared with the Vietnam veterans group without PTSD was due specifically to PTSD symptoms or to the effects of combat exposure itself.

McFall, Mackay, and Donovan (1992) compared MAST (Selzer, 1971) scores in a sample of 108 combat-exposed Vietnam veterans and a sample of 151 Vietnam-era veterans who had not been assigned combat duty. No significant differences in scores on this measure were found between the groups. How
ever, the results were quite different when combat-exposed Vietnam veterans were divided into those who did and did not suffer from PTSD, according to scores on the Mississippi Scale for Combat-Related PTSD (M–PTSD: Keane, Caddell, & Taylor, 1988). Combat veterans with PTSD scored significantly higher overall on the MAST than did combat veterans without PTSD, suggesting greater levels of problem drinking in the group with PTSD than in the combat veterans group without PTSD. These data provide some preliminary evidence that PTSD, rather than combat exposure, is linked to alcohol abuse problems. Correlational results also support this interpretation. In the combat-exposed group, MAST scores were significantly positively correlated with the severity of PTSD symptoms but were not significantly associated with any of the objective indices of severity of combat experience (i.e., scores on a combat scale, months in combat, or age in combat). This study provides further evidence that the relationship between trauma and alcohol problems appears to be mediated by the individual’s psychological reaction to the trauma (i.e., PTSD symptoms) rather than by the severity of the trauma.

Green et al. (1989) examined the degree to which a variety of war-related stressor experiences could predict PTSD and other comorbid diagnoses in veterans recruited from clinical and nonclinical sources. Comorbid PTSD and alcohol abuse were predicted by involvement in or exposure to mutilation, grotesque deaths, or both. This result held even after prewar diagnoses were statistically controlled. Sutker, Uddo, Brailey, Allain, and Errera (1994) also provided evidence that exposure to grotesque death is strongly associated with comorbid PTSD and alcoholism. Using the SCID (Spitzer et al., 1987), Sutker, Uddo, Brailey, Allain, and Errera found that almost half of a sample of troops who performed graves registration duties in the Persian Gulf War met DSM–III–R criteria for PTSD. Many of those diagnosed with PTSD also displayed symptoms of comorbid substance abuse disorder (including high rates of alcohol abuse). These results are consistent with the increased rates of alcohol abuse observed in fire disaster workers exposed to burned and mutilated bodies (Green et al., 1992).

Epidemiological comorbidity studies. All the studies above focus on individuals exposed to specific types of traumatic events, making it difficult to establish the overall comorbidity of PTSD and alcohol abuse—dependence across traumatic events. Several recent epidemiological studies overcome this limitation (see reviews by Davidson & Fairbank, 1993, and Kilpatrick & Resnick, 1993). In the St. Louis ECA (Helzer et al., 1987), it was found that participants with PTSD were 1.6 times more likely than those without PTSD to be diagnosed with a previous or concurrent alcohol abuse disorder. The Mount St. Helens Epidemiological Study similarly found a strong association between PTSD and alcohol abuse diagnoses: 27% of those with PTSD were diagnosed with a concurrent alcohol abuse disorder (cited in Davidson & Fairbank, 1993). Another national probability sample study examined the relationship between PTSD and several indices of alcohol abuse (e.g., car accident, black-out, guzzling for effect, and drinking first thing in the morning). Participants who currently met criteria for PTSD scored higher on all the measured indices of alcohol abuse than controls without PTSD (cited in Kilpatrick & Resnick, 1993). Finally, a study by Breslau and Davis (1992) divided a young adult community sample meeting criteria for PTSD into those with chronic versus nonchronic PTSD: Participants in the chronic group were 2.7 times more likely than those in the nonchronic group to meet diagnostic criteria for alcohol abuse—dependence, although both groups had a higher prevalence of these alcohol disorders than a matched control group without PTSD (Breslau, Davis, Andreski, & Peterson, 1991). Thus, severity of PTSD symptoms (when defined in terms of chronicity) appears positively associated with comorbid alcoholism. On the basis of these epidemiologic data, Davidson and Fairbank and Kilpatrick and Resnick have concluded that there is a high rate of comorbidity between PTSD and alcohol abuse across a variety of traumatic events.

Another study replicates and extends these epidemiological findings. Warshaw et al. (1993) examined the prevalence of trauma exposure, PTSD, and other diagnoses in a sample of patients with anxiety disorder. On the basis of SCID interviews, researchers divided patients into one of three groups: trauma exposed with PTSD, trauma exposed without PTSD, and no history of trauma exposure. The highest rates of alcohol abuse—dependence were seen in the group with PTSD and the lowest rates in the group without a history of trauma exposure, although all groups showed higher rates of alcohol abuse—dependence than rates seen in the general population (Robins et al., 1984). Thus, exposure to trauma in general, and the presence of PTSD in particular, appears to increase the risk for an alcohol disorder, over and above the risk associated with the presence of another anxiety disorder (see Pihl & Stewart, 1991, for a review of the relationship between anxiety disorders, other than PTSD, and alcohol abuse).

Summary: Evidence suggests that exposure to a wide variety of trauma frequently leads to the development of alcohol abuse. Moreover, some data support the notion that trauma severity is positively associated with level of alcohol abuse. Finally, it appears that psychological response to trauma (i.e., PTSD) may be more highly associated with alcohol abuse than exposure to trauma or the severity of trauma exposure, per se.

Methodological Critique of Comorbidity Studies

In this section, methodological issues in the comorbidity studies are critiqued. This critique considers methodological issues related to operational definitions of alcoholism, trauma, and PTSD and the methods for assessing these clinical conditions, sample selection concerns, inclusion of control groups, statistical control of third variables, and specificity of overlap of alcoholism, trauma, and PTSD. All of these issues must be considered when planning future studies examining the comorbidity of trauma, PTSD, and alcoholism. This section concludes with a consideration of factors that may contribute to the significant variability in comorbidity rates across studies. Specifically, I review evidence suggesting that the nature of the traumatic event, certain individual difference variables, or both might be related to the development of alcohol abuse or PTSD plus alcohol abuse following exposure to a traumatic event.

Operational Definitions and Assessment Issues

Although research suggests that exposure to traumatic events frequently overlaps with alcohol problems, and that the diagno-
ses of PTSD and alcohol abuse frequently co-occur, the rates of overlap vary markedly across studies. Factors that may contribute to this variability include differences in the definitions of alcoholism, trauma, and PTSD and differences in the way they were assessed across studies.

Alcoholism. A major methodological limitation in early prevalence studies was the frequent omission of the operational criteria used to assess alcoholism (Yeary, 1982). When such criteria were reported, they varied considerably across studies. Criteria for defining alcohol abuse-dependence have included attendance at Alcoholics Anonymous meetings (e.g., Kovach, 1986), changes in overall rates of alcohol consumption in regions exposed to disaster (e.g., Gleser et al., 1981), a variety of social indices of alcohol abuse (e.g., R. Adams & Adams, 1984), scores on self-report measures of alcohol-related problems (e.g., McFall et al., 1992), and the use of DSM-III or DSM-III-R criteria for alcohol abuse-dependence. Rates of comorbid alcoholism and PTSD obtained using DSM-III criteria are difficult to compare with those obtained using DSM-III-R criteria because the latter place less emphasis on physiological tolerance and withdrawal than did the former (Nace, 1988). A return to an emphasis on symptoms of tolerance and withdrawal in DSM-IV may present even further difficulties in comparing comorbidity rates across studies in the future. In addition, a wide variety of methods for establishing diagnoses have been used, again making comparisons across studies difficult. These include a number of structured interviews, such as the SADS (Endicott & Spitzer, 1978; e.g., Davidson et al., 1985) and SCID (Spitzer et al., 1987; e.g., Green et al., 1992), and self-report instruments, such as the MAST (Selzer, 1971; e.g., McFall et al., 1992). The use of more consistent methodology in terms of establishing alcoholic status is essential.

Moreover, problems are evident in the retrospective case report methodology used in several previous studies. Indeed, not all patient charts are compiled in the same way, and not all clinicians record the same type of information in charts. Thus, the results of studies using such methods (e.g., Streimer et al., 1985) may represent conservative estimates of the rates of alcoholism in traumatized samples (Brinson & Treatan, 1989).

Trauma exposure. Similarly, the definitions of traumatic events, and the methods by which exposure to traumatic events has been established, have varied markedly across studies. This issue is particularly evident within the area of abuse. The definitions of abuse used across studies have ranged from childhood incest (e.g., Kovach, 1986) to broader definitions of abuse including childhood neglect (e.g., Cohen & Densen-Gerber, 1982). Until researchers reach a consensus on the operational definition of abuse, studies on the relationship between abuse and alcohol problems must be evaluated in terms of the specific definition of abuse used within each study (Briere, 1992).

In addition to the definitional confusion, the methods by which abuse histories have been established have also varied widely across studies. There is a need for the development of validated psychometric instruments to assess prior exposure to abuse. The Trauma Symptom Checklist-40 (TSC-40; Elliott & Briere, 1992) is an instrument that appears promising in this regard. In a national survey of approximately 3,000 professional women, the TSC-40 was found to be a reliable and valid measure of childhood sexual victimization (Elliott & Briere, 1992).

There are two potential biases that may operate when participants provide self-reports of their prior exposure to certain traumatic events. First is the possibility of overreporting. Asking participants to describe their traumatic experience through the use of leading questions or "probes" may increase the likelihood that they report experiencing a trauma that they did not experience, leading to an overestimate of the prevalence of traumatic experiences. In fact, the use of probes has been shown to increase the frequency of reporting of sexual abuse (Fullilove et al., 1993). This issue appears particularly important given emerging concerns about the possibility of a "false memory syndrome." This syndrome involves the notion that some individuals, when searching for an explanation for their current psychological problems, may come to recall a traumatic childhood abuse history that may not have actually occurred. These false memories might represent either intentional falsifications or unintentional memory distortions (see Loftus, 1993; Loftus, Garry, & Feldman, 1994; and L. M. Williams, 1994a, 1994b, for reviews of the relevant controversy).

Second is the possibility that participants may underreport their actual levels of exposure to certain traumatic events because of feelings of shame, fear, or discomfort with the subject matter, lack of trust in the interviewer, reticence to share personal information with a stranger (Fullilove et al., 1993), or all of the above. Researchers have argued that this reporting bias is characteristic of individuals exposed to abuse (e.g., Briere & Zaidi, 1989), but it might also be characteristic of individuals exposed to other forms of trauma. For example, political refugees might be reluctant to report experiences of torture due to their vulnerable political immigrant status (APA, 1994).

Briere (1992) has reviewed three methods that researchers use to increase the validity of self-reports on abuse histories: (a) obtain independent corroborate of abuse reports from other sources (e.g., family and friends; see also Frankel, 1993); (b) use only cases validated by the child protection system or justice system; and (c) attempt to decrease rewards for falsifying abuse histories (e.g., avoiding the use of differential payment when recruiting research participants exposed to abuse). Although useful in allaying concerns about false memory syndrome, the first two methods have some associated practical problems. Individuals exposed to abuse may be unwilling or unable to provide corroborating evidence of their abuse histories. Limiting study to only those cases identified by the child protection or justice systems may introduce sampling biases (e.g., selecting for the most severe cases). Given these problems, Briere has suggested that, instead, researchers should include in their studies other measures relevant to reporting bias (e.g., social desirability, tendency toward repression, and attitudes about disclosure) and that they should more fully investigate the test-retest reliability of abuse reports. In addition, he noted that this research area could benefit from an increased focus on the potential role of interview variables in determining the frequency and extent of participants' abuse reports. For example, attention should be given to the context of the interview (part of a clinical vs. research evaluation), the nature and length of the interview relationship (long-term therapy vs. one-session evaluation), and the wording and specificity of the questioning.
PTSD. Caution should also be noted about the definitions of PTSD and the methods by which PTSD diagnoses are established across studies. For example, a variety of instruments have been used in studies using structured interviews. Previous research has shown, for example, that the SCID (Spitzer et al., 1981) and DIS (Robins et al., 1981) elicit markedly different PTSD prevalence rates. In fact, the sensitivity of the DIS has been questioned (e.g., Davidson & Fairbank, 1993). The NVVRS showed that only one in five cases of PTSD diagnosed by SCID criteria was accurately identified by the DIS (Kulka et al., 1988). Therefore, in studies that have used DIS criteria to establish comorbidity (e.g., Winfield et al., 1990), the overlap between alcohol abuse and PTSD may have been underestimated. The use of both current and lifetime diagnostic assessments is preferable, given the possibility that current PTSD symptoms might be masked if the individual is abusing alcohol at the time of assessment (e.g., Brett & Ostroff, 1985), resulting in underestimates of the actual overlap between PTSD and alcoholism.

Some studies (e.g., McFall et al., 1992) have relied on self-report scales alone (e.g., M-PTSD scale; Keane, Caddell, et al., 1988) to assess PTSD. Although such instruments may be useful indicators of the severity of PTSD symptoms, they should not be used as the sole method of diagnosing PTSD. Lyons, Gerardi, Wolfe, and Keane (1988), for example, advocate the importance of multimodal assessment techniques for establishing the diagnosis of PTSD.

Sample Selection Concerns

Studies using psychiatric outpatients as participants may underestimate levels of alcohol abuse. First, such patients may be reluctant to admit to abusive drinking because of concerns about potential ineligibility for anxiety-focused or trauma-focused treatment. Second, individuals with significant alcohol problems may seek treatment at an addictions facility, rather than at a psychiatric outpatient clinic (B. J. Cox et al., 1990; Otto, Pollack, Sachs, O’Neil, & Rosenbaum, 1992). Alternatively, studies conducted with patients in treatment may be subject to “Berkson’s” selection bias (i.e., individuals with more than one disorder may be more likely to seek treatment than individuals with only one disorder). This bias could inflate comorbidity rates obtained with clinical samples above rates present in the general population. Studies using nonclinical samples have overcome these potential selection biases. In fact, the strong relationship of trauma exposure, PTSD, and alcohol abuse appears to be evident in both clinical and nonclinical samples. However, researchers should be aware of these selection bias issues when designing future studies.

The large majority of comorbidity studies have been conducted with circumscribed samples of individuals exposed to particular forms of traumatic events, rendering it difficult to make general statements about the relationship of trauma exposure, PTSD, and alcoholism across traumatic events. The relations between some forms of traumatic events (e.g., man-made disasters) and alcohol abuse remain relatively understudied. However, the literature from studies conducted within a variety of specific traumatic events, and the emergence of epidemiological studies in the general population that examine exposure to a wide variety of traumatic experiences, provides preliminary data that the relationship between PTSD and alcohol abuse can be found across several traumatic experiences (for reviews, see Davidson & Fairbank, 1993, and Kilpatrick & Resnick, 1993). I consider the potential impact of different types of traumatic events on subsequent abusive drinking in a later section, Nature of the traumatic event.

Control groups. Another limitation in early comorbidity studies was the general absence of matched control groups. In these studies, it was unclear whether traumatic histories, PTSD diagnoses, or both were more common in alcoholic samples than in nonalcoholic samples matched for important demographic variables, such as age and SES. However, more recently researchers (e.g., Covington, 1983) have made headway in establishing a relationship between childhood abuse trauma and later alcoholism through the inclusion of appropriately matched control groups.

Statistical control of third variables. Even with the use of control groups, the question remains whether trauma exposure is causally antecedent to alcohol abuse or whether other variables (such as the impact of other events before or following the trauma) are responsible for the overlap between trauma exposure and alcohol abuse. For example, family dysfunction (broadly defined) and familial alcoholism have been suggested as potential third variables which may be responsible for the high degree of overlap between childhood sexual abuse, physical abuse, or both and alcohol problems in adulthood (American Medical Association, 1993). Some researchers have used statistical methods to control for the possible mediating effects of third variables on the relationship between trauma exposure and subsequent alcohol abuse. For example, B. A. Miller et al. (1993) showed that the relationship between childhood victimization and alcohol abuse persisted after the effects of family background (e.g., parental alcohol problems) were statistically controlled.

However, Briere and Elliott (1993) have questioned the validity of such statistical control procedures, cautioning that such analyses are often misleading in quasi-experimental research (i.e., research involving comparisons between nonrandomly assigned participants—traumatized vs. nontraumatized). They suggested alternatives to statistical control procedures for controlling for third variables, including multivariate tests such as canonical correlation analysis or causal modeling. Some researchers have begun to use such techniques in the study of the relationship between trauma and alcohol abuse. The study by Green et al. (1989) is a fine example of the use of multivariate techniques to assess the relative contributions of trauma exposure, pretraumatic factors, and posttraumatic factors to the prediction of alcohol abuse in combat veterans. Similarly, Dembo and colleagues (e.g., Dembo et al., 1988, 1992) have effectively used structural equation modeling techniques to study the potential causal relationship of abuse histories, other family background characteristics, and the development of alcohol problems in adolescents. Such research has demonstrated...
that trauma exposure contributes unique variance to the prediction of alcohol abuse, over and above the effects of other relevant variables.

Specificity of overlap. Although the prevalence of traumatic histories among people with alcoholism appears significantly higher than rates in the general population (Hurley, 1991), if anything is to be made etiologically of these statistics, it must be demonstrated that traumatic histories are specifically associated with alcoholism diagnoses, as opposed to being more generally associated with a wide variety of behavioral pathologies. This type of discrimination is seldom made and is seldom clear. Some researchers have attempted to address this issue. For example, Briere (1988) has shown that not only do individuals exposed to childhood abuse show higher rates of alcoholism than do other patients, but they also display significantly more insomnia, sexual dysfunction, dissociation, anger, suicidality, self-mutilation, and other forms of drug addiction (see also a review by Herman, 1992). These results suggest that alcohol abuse may be merely one type of maladaptive response to dealing with traumatic experiences. However, other researchers have addressed the question of specificity by examining the relative overlap of PTSD and other diagnoses including alcohol abuse—dependence. For example, Branchey et al. (1984) found that alcohol abuse and PTSD are the most prevalent coexisting psychiatric diagnoses in combat veterans. Similarly, in NVVRS (Kulka et al., 1988), conducted with a nonclinical sample of Vietnam veterans, alcohol abuse was the most common comorbid disorder among men with PTSD, and the third most common comorbid diagnosis among women with PTSD. Thus, alcohol abuse does appear to show some specificity in terms of its overlap with PTSD, at least in veteran samples. This issue is worthy of further study, particularly with respect to other forms of traumatic events.

Nature of the traumatic event. Some studies suggest that the rates of PTSD in individuals exposed to trauma may vary as a function of the nature of the traumatic event. For example, PTSD rates are higher in individuals who have been exposed to heavy combat (Kulka et al., 1988) and sexual assault (Rothbaum & Foa, 1993) than in individuals who have been involved in serious accidents (Helzer et al., 1987). Researchers have only just begun to examine whether there might be similar differences in rates of alcohol abuse as a function of the nature of the traumatic event. For example, the community study by Cottler et al. (1992) showed heavy alcohol use to be significantly associated with prior exposure to combat but not to other forms of traumatic events. Similarly, Green et al. (1992) made a post hoc comparison of data from two comorbidity studies and showed that comorbid alcoholism and PTSD diagnoses were higher in a sample of combat veterans than in a sample of individuals exposed to a natural disaster. However, methodological limitations preclude interpreting these results as firmly supporting the notion that combat is more likely to lead to alcohol abuse plus PTSD than is a natural disaster. Differences in the methodologies used in the two studies, and differences in demographic characteristics of the samples, might explain the observed variability in comorbidity rates (Green et al., 1992). First, the study with combat veterans (Green et al., 1989) used the SADS (Endicott & Spitzer, 1978) to establish diagnoses, whereas the study with individuals exposed to a flood (Green et al., 1992) used the SCID (Spitzer et al., 1987), thus making direct comparisons across the two studies difficult to interpret given the slightly different diagnostic criteria used on the SADS and SCID (Green et al., 1992). Second, the combat veterans sample was younger than the disaster sample, and the former was composed only of men, whereas the latter was a mixed gender group. Because younger ages and the male gender have been associated with increased rates of alcohol abuse—dependence in the general population (e.g., Robins et al., 1984), such differences are important to consider before any definitive conclusions about the relative degree of overlap of PTSD and alcohol abuse across various trauma can be made. Age and gender are considered further in the following section on the potential impact of various individual difference factors.

Individual Differences

Traditionally, researchers have examined a number of individual difference factors to determine whether such variables influence the development of PTSD or alcoholism (e.g., W. M. Cox, 1987; McNally & Shin, 1995). However, few researchers have focused on individual difference factors that might contribute to the development of alcohol problems, or comorbid PTSD and alcoholism, following exposure to a traumatic event. Several such possible individual difference factors are reviewed here.

Age. Age must be considered as a potentially important individual difference variable, both in terms of the age of the participant at the time of study and the age at which the participant was exposed to the traumatic event. There are well-known problems inherent in retrospective reporting. Studies with adults suffer the limitation that participants are often asked to report on traumatic events that may have occurred a long time in the past (e.g., childhood abuse). Work with adolescents (e.g., Clark & Jacob, 1992; Dembo et al., 1988, 1992) may, to some degree, overcome this limitation by examining the participants’ reports of the traumatic events closer in time to the occurrence.

Recent research suggests that a substantial proportion of adult individuals exposed to childhood sexual abuse (particularly those with histories of more severe forms of abuse) report some prior period of amnesia for the abuse (i.e., Bernet, Deutscher, Ingram, & Litrownik, 1993; Briere & Conte, 1993; Herman & Schatzow, 1987) and that this amnesia is likely to have occurred in adolescence (Briere & Conte, 1993). These findings, however, are directly contradicted by the work of Terr (1981, 1983) with the children involved in the Chowchilla school bus kidnapping. In contrast to Briere and Conte’s findings of significant amnesia for traumatic events in adolescence, Terr found that the participants’ memories for the traumatic event remained intact and detailed both immediately following the kidnapping (when they were children) and at a 4-year follow-up (when they were adolescents). These two types of traumatic events (kidnapping vs. sexual abuse) may differ in important respects, however. Terr (1991) distinguished between the effects of a single traumatic event (e.g., kidnapping) and those of prolonged, repeated trauma (e.g., sexual abuse). According to Terr (1991), the latter type of trauma is more likely than the former to be associated with denial, numbing, and dissociative types of reactions in children (see also Herman, 1992). Until
this issue is further clarified, researchers need to carefully consider the possibility of amnesia for certain types of traumatic events when examining the relationship between self-reported traumatic histories and alcohol problems in adolescent samples.

With respect to the age at which the traumatic event occurred, it might be speculated that the earlier the traumatic exposure, the less likely drinking problems will develop because adults might be more likely to use alcohol as a coping strategy than children. However, Swett et al. (1991) showed that even early exposure to trauma is associated with high rates of alcohol abuse in adulthood. Kovach (1986) has postulated a delayed-onset PTSD model to explain these findings. She suggested that the link between childhood abuse and adult alcohol problems might be mediated by a delayed onset of PTSD symptoms in adulthood with which the individuals attempt to cope through alcohol abuse.

**Gender.** Epidemiological evidence indicates that men are approximately five to seven times more likely to receive a lifetime diagnosis of alcohol abuse—dependence than are women (Robins et al., 1984). Thus, in future studies designed to compare the incidence of alcohol abuse in patients with PTSD across specific trauma, gender differences in alcoholism diagnoses in the general population should be considered. For example, should it be found that PTSD due to combat is more highly associated with subsequent alcohol abuse than PTSD following sexual assault, it should be kept in mind that these two types of trauma tend also to occur disproportionately across gender, with men more likely to be exposed to combat and women more likely to experience sexual assault (Norris, 1992). Thus, comparison of rates of alcohol abuse in patients with PTSD must be made with base rates by gender before any definitive statements can be made about the relative prevalences of comorbid PTSD and alcohol abuse in individuals who have been exposed to various types of trauma (Green et al., 1992).

**Cultural and racial factors.** Rates of alcohol abuse—dependence vary across cultures. These prevalence differences appear related to each culture's acceptance of alcohol use (APA, 1994). However, few researchers have focused on how these cultural norms might relate to the likelihood of using alcohol to cope with exposure to traumatic events. Cross-cultural studies have shown that the basic symptom patterns of PTSD appear constant across cultures (e.g., Carlson & Rosser-Hogan, 1994). Similar cross-cultural studies on posttraumatic changes in drinking would be useful to determine the influence of cultural variables on the likelihood of developing drinking problems following trauma exposure.

Previous data suggest that racial factors may be related to the likelihood of developing PTSD following exposure to trauma. For example, in the NVVRS (Kulka et al., 1988), rates of PTSD in Vietnam veterans were highest in Hispanic veterans and lowest in Caucasian veterans. Whether these effects represent true racial differences, or rather differences in severity of combat exposure, remains to be determined (Kulka et al., 1988). Some data similarly suggest that overlapping rates of PTSD plus alcoholism may vary as a function of racial factors. For example, Escobar et al. (1983) showed much higher levels of comorbid PTSD and alcohol abuse—dependence diagnoses (i.e., 80%) by SCID criteria in a Hispanic sample of Vietnam combat veterans than those previously established in non-Hispanic or mixed racial samples of veterans (e.g., Sierles et al., 1983). However, because the Escobar et al. study did not include other racial groups matched with the Hispanic sample for important demographic variables, such as SES, the conclusion that Hispanic veterans are at increased risk for comorbid PTSD—alcoholism remains speculative.

**Intelligence and cognitive factors.** Recent data suggest an association between intelligence levels and the development of PTSD in a sample of Vietnam veterans (McNally & Shin, 1995). Specifically, researchers found that veterans with lower IQs were more likely than those with higher IQs to develop PTSD. Although these data are correlational in nature, one of the potential explanations for the overlap is that cognitive deficits may be a risk factor for the development of PTSD following trauma exposure. If cognitive disadvantage leads to impaired coping abilities, then lower levels of intelligence may complicate recovery. Alcohol abuse might be viewed as a maladaptive coping strategy for dealing with extremely stressful experiences. Thus, lower intelligence levels might be one individual difference factor that predisposes to alcohol abuse following trauma exposure.

**Personality factors.** Previous research has attempted to determine whether certain personality factors might be related to the development of alcohol abuse (for a review, see W. M. Cox, 1987). One often-researched trait is trait anxiety, the tendency to react with fear across a wide variety of situations. Although it makes intuitive sense that individuals who experience more trauma might be related to whether a given individual would turn to alcohol following exposure to a traumatic event, research shows only weak and inconsistent relations between trait anxiety and alcohol abuse (Sher, 1987).

In contrast, work on "anxiety sensitivity" might potentially contribute to understanding the relationship between PTSD and alcohol abuse. Anxiety sensitivity is a cognitive individual difference variable that is both conceptually and empirically distinct from the traditional concept of trait anxiety (McNally, 1996). Anxiety sensitivity is characterized by fear of anxiety symptoms, based on beliefs that these symptoms have harmful consequences (Reiss, 1987; Reiss & McNally, 1985). An individual with high-anxiety sensitivity, for example, may misinterpret rapid heart rate as a sign of an impending heart attack, whereas an individual with low-anxiety sensitivity may regard these sensations as merely unpleasant.

Several recent studies focus on the potentially important relationship between anxiety sensitivity levels and the use or abuse of alcohol (McNally, 1996; Peterson & Reiss, 1992; Reiss, 1991; Stewart, 1995a). For example, higher than normative levels of anxiety sensitivity are characteristic of individuals meeting DSM-III-R criteria for alcohol dependence (Karp, 1993). Anxiety sensitivity levels are positively correlated with self-reported, weekly alcohol use both in clinical (B. J. Cox, Swinson, Shulman, Kuch, & Reichman, 1993) and in nonclinical (Stewart, Peterson, & Pihl, 1995) samples. Levels of anxiety sensitivity are significantly positively related to the use of alcohol for coping-related motives (i.e., to reduce or avoid negative emotional states; e.g., Stewart, Karp, Pihl, & Peterson, 1995; Stewart & Zeitlin, 1995). Karp has also shown that levels of anxiety sensitivity are positively associated with the belief that
alcohol consumption leads to relaxation and tension reduction. Moreover, anxiety sensitivity levels are strongly positively correlated with the self-reported frequency of drinking in situations involving unpleasant emotions and conflict with others (i.e., potentially negatively reinforcing drinking situations; Stewart, 1995b). Finally, a series of studies by Stewart and colleagues has shown that anxiety sensitive participants displayed responses to alcohol administration in the laboratory that could potentially explain their increased weekly use of alcohol: High-anxiety sensitive participants are particularly sensitive to the subjective-emotional, physiological, and cognitive tension-reducing effects of alcohol when compared with low-anxiety sensitive controls (Stewart & Pihl, 1994, 1995).

Anxiety sensitivity may be one common factor contributing to the high degree of overlap between PTSD and alcohol abuse. There exists some initial, albeit indirect, evidence to support this contention. First, anxiety sensitivity, PTSD, and some forms of alcohol abuse (i.e., secondary or “neurotic” forms; W. M. Cox, 1987) are all characterized by avoidance behavior (e.g., Reiss, Peterson, Gursky, & McNally, 1986). Second, not all anxiety disorders are associated with elevated anxiety sensitivity. Significant fear of anxiety appears to figure prominently in individuals with PTSD and panic disorder, whereas individuals with simple phobias, generalized anxiety disorder, and obsessive-compulsive disorder display more normative levels of anxiety sensitivity (McNally, 1992; Stewart, Knize, & Pihl, 1992; S. Taylor, Koch, & McNally, 1992). An increased risk for alcoholism appears associated with the anxiety disorders characterized by high levels of anxiety sensitivity, such as panic disorder (see a review by B. J. Cox et al., 1990) and PTSD. In contrast, anxiety disorders, such as a simple phobia, generalized anxiety disorder, and obsessive-compulsive disorder that are characterized by lower levels of anxiety sensitivity, do not appear to be associated with an increased risk for alcoholism (Kushner et al., 1990; Riemann, McNally, & Cox, 1992). The potential role of anxiety sensitivity in mediating the relationship between PTSD and alcohol abuse requires further exploration.

Summary

Future comorbidity studies should carefully consider various methodological issues, including the definitions and choice of instruments for assessing trauma exposure, PTSD, and alcohol abuse—dependence, and inclusion of appropriate control groups, to increase the quality of research in this area and thus allow researchers to draw more firm conclusions. Researchers should also pay more attention to factors that may potentially affect comorbidity rates across studies, such as trauma-related and individual difference variables. Moreover, researchers may wish to go beyond traditional comorbidity research methods and begin to probe the functional relationship of trauma exposure, PTSD, and alcoholism.

Functional Associations

This section begins with a review of several possible causal relationships that may explain the overlap between PTSD and alcoholism. I consider ways in which each disorder may serve as an etiological factor, maintenance factor, or both for the other. Then I review studies that have extended traditional epidemiological comorbidity methods to examine the functional associations between these two disorders.

Possible Causal Relationships

Several causal pathways may explain the co-occurrence of PTSD and alcoholism (Pihl & Stewart, 1991). These possibilities are not necessarily mutually exclusive. First, alcohol abuse might heighten the susceptibility to the development, maintenance, or both of PTSD. Alcohol abuse might increase anxiety and arousal levels through psychological processes, such as stressful life events occurring due to heavy drinking or guilt associated with heavy drinking (B. J. Cox, Norton, Dorward, & Ferguson, 1989; Kushner et al., 1990). Alcohol abuse might also increase anxiety and arousal levels through physiological processes, such as prolonged drinking effects, acute ingestion of very high doses of alcohol, or alcohol withdrawal effects (e.g., Lacroisiere, Godfrey, & Ruby, 1980). These effects could serve to induce a hyperaroused state in which the individual may be more vulnerable to develop PTSD following a traumatic event. Alcohol abuse might also serve to maintain PTSD by interfering with important psychological mechanisms of processing or “working through” the traumatic experience or by preventing desensitization to traumatic events (Herman, 1992). Second, PTSD might be involved in the development, maintenance, or both of alcohol abuse. Some patients with PTSD might begin or continue abusing alcohol in an attempt to reduce or control (i.e., self-medicate for) their PTSD symptoms. This self-medication notion of drug abuse proposes that individuals who are susceptible to certain aversive states, such as anxiety, are at a high risk of abusing drugs capable of reducing these aversive states (Khantzian, 1985). Third, some other variable (e.g., anxiety sensitivity; McNally, 1996) might be related to the onset, maintenance, or both of PTSD and alcohol abuse. Thus, similar etiological mechanisms involved in the two disorders might explain their high degree of overlap, rather than one disorder causing the other.

Studies of Functional Connectedness

The data on the comorbidity between PTSD and alcohol abuse presented in earlier sections of this article are correlational and do not necessarily provide evidence for a functional interplay between the two disorders. To overcome this limitation, several studies have examined the relative order of onset of the comorbid diagnoses of PTSD and alcoholism and patients’ perceptions of the relationship between their problems, in attempts to assess the direction of causality.

Order of onset. Although not specifically examining the diagnosis of PTSD, Winfield et al. (1990) provided evidence that sexual assault tends to precede alcohol abuse in women. In a large community sample of women who were both sexually assaulted and diagnosed with alcohol abuse—dependence, drinking problems developed following the assault. None of the women had a drinking problem before the assault.

In a sample of adolescents with alcoholism, Clark and Jacob (1992) found that anxiety disorders appeared before alcohol
abuse in 85% of the comorbid cases. Unfortunately, order of onset data were not reported separately for each anxiety disorder. Based on the statistics provided, however, it can be estimated that in the large majority (i.e., 75-100%) of the PTSD cases, PTSD developed before the alcohol disorder. However, the relatively small sample size in this study precludes firm conclusions.

Davidson et al. (1985) studied a sample of Vietnam veterans with PTSD. They found that in the cases comorbid for PTSD and alcohol or drug abuse, PTSD developed 3½ years before the onset of the substance abuse disorder, on average. Similarly, in a study of World War II (WW2) and Vietnam veterans with PTSD (Davidson et al., 1990) in cases comorbid for alcohol abuse and PTSD, the alcohol abuse did not develop until approximately 7 years after the emergence of PTSD in the WW2 veterans group and tended to emerge around the same time as PTSD in the Vietnam veterans group. However, the studies above (i.e., Clark & Jacob, 1992; Davidson et al., 1985, 1990) examined the relative order of onset of diagnoses, not the onset of subthreshold symptoms. It is possible that some veterans in the Vietnam group (Davidson et al., 1990) began abusing alcohol to dampen subthreshold PTSD symptoms before the onset of full-blown PTSD. Davidson et al. (1990) noted that the earlier onset of alcoholism in the Vietnam as opposed to the WW2 veterans group may reflect the greater availability of alcohol at the time.

In contrast to these results, Cottler et al. (1992) found that alcohol abuse symptoms generally predated the onset of PTSD symptoms in nonclinical participants from the St. Louis ECA study. However, this study used very liberal criteria for defining alcohol problems, including individuals who drank heavily once a week for at least 2 months, with no objective definition of heavy drinking provided. Because alcohol problems were not adequately diagnosed, differences in results across studies that examine the relative order of onset may be attributable to differences in diagnostic criteria. Moreover, Cottler et al. examined the relative onset of subthreshold symptoms, whereas the previously cited studies examined the point at which participants met diagnostic criteria for PTSD and alcoholism. Thus, these two findings are not necessarily mutually exclusive. For example, an individual could be a heavy drinker on weekends and experience only subthreshold symptoms of alcohol abuse or dependence. If this individual were to experience a traumatic event and develop PTSD symptoms, he or she might be predisposed to use alcohol to cope with or dampen PTSD symptoms and over time develop an alcohol disorder.

With the exception of Cottler et al.'s (1992) study, the majority of data support the notion that PTSD symptoms tend to precede the development of alcohol abuse problems. However, the mere fact that one disorder tends to precede the other does not establish a causal link between the two problems. Rachman (1991) has recommended several strategies for overcoming problems inherent in traditional comorbidity research (i.e., observed high-comorbidity rates may be merely statistical relationships rather than functional associations). He suggested that, in addition to the simple determination of the rates of co-occurrence, attention should be given to the psychological connectedness of the two disorders. This can be assessed in a variety of ways, including having participants estimate the degree to which they consider their two problems to be associated.

**Patients' perceptions of associations.** Some researchers have applied Rachman's (1991) suggestion that patients be queried as to their perceptions regarding the functional interplay between their PTSD symptoms and their drinking problems. For example, Clark and Jacob (1992) found that in the majority of cases comorbid for anxiety and alcohol abuse disorders, participants perceived that the anxiety disorder was causally linked to the development of the alcohol abuse problem. Although the data were not presented as a function of the type of anxiety disorder, PTSD was by far the most common comorbid anxiety disorder in this sample. Thus, the Clark and Jacob data provided evidence that patients do perceive a causal connection between PTSD and the later development of their drinking problems. Therefore, like the data on chronological patterns, initial results on perceived relationships provide preliminary evidence that anxiety disorders, particularly PTSD, may contribute to the development of alcohol disorders.

More research on participants' perceptions of the causal links between their PTSD and their alcohol abuse symptoms is needed to examine the functional relationship between the two problems. However, as Rachman (1991) cautioned, such self-estimates may bear little relationship to the actual functional connectedness of the two problems because individuals are not always aware of the motives for their behaviors (McClelland, 1985; Nisbett & Wilson, 1977). Rachman recommended that, in addition to having participants make general estimates of connectedness, in the future researchers should use questions probing participants' predictions of what would happen to one problem if the other problem were to increase or to decrease. Such research has yet to be conducted with respect to comorbid PTSD and alcoholism.

**Drinking patterns.** Some researchers have examined the patterns of drinking behavior in patients with PTSD to better understand the functional relationship between these two problems. Jellinek and Williams (1984) reported that combat veterans exhibit unusually high levels of alcohol consumption and engage in continuous, sustained drinking patterns, as well as frequent binge drinking. In contrast, Boccarino (1981) showed that Vietnam combat veterans exhibited only substantially higher rates of binge drinking than did Vietnam-era veterans after statisitically controlling for important demographic differences between the two groups. This is consistent with the belief among many clinicians that combat veterans abuse alcohol in an episodic, rather than habitual, manner (Hyer, Leach, Boudeywys, & Davis, 1991).

Hyer et al. (1991) specifically examined the typical drinking patterns of Vietnam veterans with alcoholism as a function of the presence or absence of concomitant PTSD. All veterans were on a chemical dependency treatment unit, and all had a current DSM-III-R diagnosis of alcohol abuse. The PTSD group was identified according to cutoff scores on the MMPI-PTSD scale (Keane, Malloy, & Fairbank, 1984). All veterans were given the Alcohol Use Inventory (AUI; Horn, Wanberg, & Foster, 1987), a measure of alcohol use patterns. The group with PTSD achieved significantly higher scores on the AUI than the group without PTSD, reflecting an overall profile of deteriorated binge drinking patterns in veterans with PTSD.
Thus, preliminary data suggest that war veterans comorbid for PTSD and alcoholism tend to be binge drinkers. Hyer et al. (1991) have speculated that the episodic heavy drinking of combat veterans with PTSD may coincide with periods of intrusive PTSD symptoms. Although McFall et al. (1992) demonstrated that levels of problem drinking (i.e., MAST scores) were significantly positively correlated with the severity of intrusive PTSD symptoms, no researchers have yet examined whether heavy drinking episodes actually overlap in time with the occurrence of intrusive PTSD symptoms.

LaCoursiere et al. (1980) provided a series of case studies containing some preliminary observations regarding the functional relationship between PTSD symptoms and abusive drinking. They presented three case examples of war veterans with comorbid diagnoses of PTSD and alcohol abuse. They noted that these patients tended to consume large amounts of alcohol just before bed, reportedly to prevent sleep difficulties and the experience of trauma-related nightmares. More research in this area is warranted and should be extended to examine individuals with PTSD due to other trauma, such as sexual or physical abuse. Both retrospective and prospective self-report methodologies (e.g., daily diaries of PTSD symptoms and drinking behaviors) could be used in future research to further evaluate the typical drinking profiles of patients with and without PTSD, the situations in which heavy drinking occurs, patients' perceptions of their motivations for abusive drinking, and the behavioral consequences of heavy drinking. Examination of the relation between anniversary dates (around which patients' reexperiencing symptoms are often exacerbated; APA, 1994) and alcohol use might provide important information about the functional relationships between PTSD symptoms and abusive binge drinking.

Summary

Studies of both order of onset and patients' perceptions of the causal connection between their PTSD symptoms and alcohol abuse suggest an important functional relationship. PTSD symptoms tend to precede the onset of problem drinking, and patients' perceive that their anxiety symptoms led to the development of their alcohol abuse. Moreover, it appears that comorbid patients with PTSD and alcoholism tend to drink in an episodic (binge drinking) fashion. More research is required to determine whether the binges are actually triggered by PTSD symptoms, external reminders of the traumatic event, or both.

Mechanisms Proposed to Account for Comorbid PTSD–Alcohol Abuse

Most explanations offered to date to account for the high degree of overlap between PTSD and alcohol abuse have centered on the notion that some individuals with PTSD abuse alcohol to reduce or control their PTSD symptoms. For the purposes of this review, the postulated self-medication effects of alcohol are divided into four categories that may or may not be mutually exclusive: physiological, behavioral, affective, and cognitive. Although the effects of alcohol on each of these conceptually distinct categories of PTSD symptoms are examined separately, it should be recognized that if alcohol proves to exert its effects on more than one of these symptom clusters simultaneously, the resultant interactive effect of alcohol on PTSD symptoms may prove to be greater than the sum of its effects on individual PTSD symptom components.

In this section, I review the relationship between PTSD and alcohol abuse from within the framework of the many existing stress-related drinking theories. I discuss the advantages and disadvantages of each theory in the most relevant section. For example, given that the tension-reduction theory (TRT; Conger, 1951) of alcohol use–abuse historically developed out of the behavioral tradition, it is reviewed in the Behavioral Effects section.

Physiological Effects of Alcohol in PTSD

One of the characteristics of PTSD is persistent symptoms of increased arousal, as indicated by such symptoms as difficulty falling or staying asleep, exaggerated startle response, and physiological reactivity to exposure to events that symbolize or resemble an aspect of the trauma (APA, 1994). Alcohol abuse in PTSD may function to dampen this increased physiological arousal. For example, LaCoursiere et al. (1980) speculated that alcohol abuse in patients with PTSD may represent an attempt to prevent sleep disturbances. Keane, Gerardi, et al. (1988) have also suggested that substance abuse in combat veterans with PTSD may promote sleep or help them to alleviate physiological arousal states.

These hypothesized mechanisms can be viewed from within the framework of Sher's (1987) stress response dampening (SRD) theory of alcohol abuse. According to this theory, individuals drink to dampen their physiological reactivity to stressful events. It could be speculated that patients with PTSD may be even more susceptible to alcohol's SRD effects than individuals not exposed to trauma and thus may be highly susceptible to negative reinforcement from drinking.

No researchers have specifically examined the effects of alcohol consumption on physiological reactivity within PTSD samples. However, much research suggests that alcohol reduces physiological reactivity to stressful stimuli or events (for a review, see Sher, 1987). Researchers have examined the effects of alcohol on a variety of measures of physiological responses to stress in humans. Levenson, Sher, Grossman, Newman, and Newlin (1980), for example, examined the effects of alcohol on physiological arousal before and during exposure to two stressors (i.e., signalled shock and a self-disclosing speech) in male college students. The consumption of alcohol, but not of a placebo, markedly attenuated the magnitude of physiological responses to the stressors. A consistent finding across several studies was a reduction in the magnitude of heart rate and skin conductance responses to stress (e.g., Finn & Pihl, 1987, 1988; Finn, Zeitouni, & Pihl, 1990; Lindman, Alexanderson, & Kvamnstrom, 1979; Sher & Levenson, 1982, 1983; Stewart, Finn, & Pihl, 1992; Stewart & Pihl, 1994; Wilson, Abrams, & Lipscomb, 1980).

Some studies have not shown SRD effects of alcohol on measures of autonomic reactivity to stress (e.g., D. B. Abrams & Wilson, 1979; Dengerink & Fagan, 1978; Keane & Lisman, 1980; Wilson & Abrams, 1977). These studies have used relatively low doses of alcohol. In fact, the clear dose effects ob-
tained by Stewart, Finn, et al. (1992) support the conclusion that relatively high doses of alcohol (i.e., those producing blood alcohol levels [BALS] of greater than 0.08%) are needed to achieve significant SRD effects (see also Stewart, Finn, & Pihl, 1995). In summarizing the results of various studies in this area, Cappell and Greeley (1987) and Sher (1987) have concurred that the majority of evidence suggests that alcohol attenuates autonomic responses to stress at least when moderately high doses of alcohol are used.

Gray (1982) has described an anxiety system, termed the behavioral inhibition system (BIS), that responds to threat cues with inhibition of ongoing behavior and increased physiological arousal. Alcohol is known to increase activity of the neurotransmitter gamma-aminobutyric acid in septal–hippocampal brain regions (i.e., BIS, Gray, 1982; Pihl & Peterson, 1995). In other words, one of alcohol’s principal physiological effects is to enhance the actions of an inhibitory neurotransmitter (i.e., one that ultimately leads to decreased neural activity) in the regions of the brain thought to be associated with anxiety production. This neurochemical mechanism may underlie alcohol’s physiological SRD effects. Gray (1985) suggested that anxious individuals are high in BIS activity and may be particularly sensitive to alcohol-induced SRD effects. These physiological effects of alcohol may reinforce drinking in patients with anxiety disorders such as PTSD.

More research is required to determine the degree to which these previous findings on alcohol’s physiological SRD effects are actually related to alcohol abuse in individuals with PTSD. One study provides some preliminary data on this issue. Studying Vietnam combat veterans, McFall et al. (1992) found that problem drinking levels (i.e., MAST scores) were significantly positively correlated with self-report measures of the severity of physiological arousal symptoms. This finding could suggest that alcohol abuse represents an attempt to dampen physiological reactivity in PTSD. However, the correlational nature of these data again begs the question of causation. Moreover, the effects of alcohol on other arousal-related PTSD symptoms (e.g., sleep disturbance and exaggerated startle response) remain to be studied.

The alcohol challenge paradigm (Newlin & Thomson, 1990) would prove useful in this endeavor. In this paradigm, patients with PTSD and controls could be administered a moderately intoxicating dose of alcohol, and the effects of alcohol on physiological reactivity could be monitored to determine whether patients with PTSD exhibit unique responses to alcohol (e.g., increased sensitivity to alcohol’s SRD effects), which might begin to explain their apparently increased risk for alcohol abuse. Although the SRD theory appears to account well for some of the data (e.g., McFall et al., 1992), any theory that purports to explain the increased abuse of alcohol in patients with PTSD by way of alcohol’s effects on physiological reactivity must account for the fact that alcohol consumption has repeatedly been shown to lead to a significant increase in “tonic” levels of arousal (i.e., baseline levels of physiological arousal taken in the absence of situational stressors; e.g., Jones, Parsons, & Rundell, 1976; Niatoh, 1972; Stewart, Finn, et al., 1992).

Even if physiological reactivity symptoms in patients with PTSD are reduced following alcohol consumption, one needs to demonstrate that such alcohol-induced SRD effects actually reinforce alcohol consumption in patients with PTSD. The stress-induced drinking methodology, first used by Higgins and Marlatt (1973, 1975), might prove helpful. In this paradigm, participants are exposed to a stress-induction manipulation, and then the experimenters record participants’ levels of alcohol consumption in an unobtrusive taste-rating test. Although the true variable of interest is the amount of alcohol consumed, participants are led to believe that the experimenters are interested in their ratings of a variety of beverages including alcohol on a number of dimensions of taste (e.g., Higgins & Marlatt, 1973; Samoluk & Stewart, 1996a; Sweet, Stewart, Samoluk, & MacDonald, 1995). Previous research has shown that anticipatory stress, measured by increased autonomic arousal, is associated with increased alcohol consumption (Strickler, Tomaszewski, Maxwell, & Suib, 1979). It can be hypothesized that patients with PTSD, but not controls without PTSD, would be particularly susceptible to drinking in response to manipulations of physiological arousal levels.

A final physiological stress-related drinking theory that provides a useful framework to view the PTSD–alcoholism comorbidity data is Volpicelli’s (1987) endorphin-compensation theory. This theory rests on evidence that stressful events lead to release of endogenous endorphins. However, with chronic stressors (or chronic responses to the initial stressor, such as in PTSD), stores of endorphins become depleted. Ingestion of alcohol is said to be reinforced following a chronic stressor because the alcohol compensates for the depleted endorphin stores (Volpicelli, 1987). In fact, Pitman, van der Kolk, and Orr (1990) have suggested alterations in the regulation of endogenous opioids as a biological correlate of PTSD in combat veterans. Future research is needed to establish the validity of endorphin compensation as an explanation for abusive drinking in patients with PTSD.

Behavioral Effects of Alcohol in PTSD

Another of the characteristic symptoms of PTSD is the persistent avoidance of stimuli associated with the trauma, which is behaviorally evidenced by efforts to avoid activities or situations that arouse recollections of the trauma (APA, 1994). Thus, the self-medication behavior of patients with PTSD may involve attempts to reduce behavioral avoidance. For example, Skorina and Kovach (1986) have postulated that, in sexually abused women with alcoholism, alcohol may be used to reduce avoidance of sexual activity.

The behavioral effects of alcohol can be viewed from within the framework of Conger’s (1951) TRT of alcohol abuse. This behavioral theory consists of two tenets. First, alcohol leads to tension reduction; second, tension reduction reinforces alcohol consumption (i.e., through negative reinforcement). Although it is recognized that the TRT is not a global explanation for abusive drinking, researchers have suggested that certain individuals might be at a heightened risk for learning to drink alcohol for its tension-reducing properties (Cappell & Greely, 1987). Individuals exposed to trauma might prove to be one such group. In fact, Skorina and Kovach (1986) reported that women with alcoholism and sexual abuse histories report sexual dysfunction (e.g., avoidance of sexual activity) before abusive drinking more often than women with alcoholism and no...
histories of sexual abuse, suggesting that alcohol may be used by those exposed to trauma to reduce behavioral avoidance of trauma reminders.

The time course of drinking in response to PTSD symptoms is clearly specified in TRT (Conger, 1951): Drinking occurs before entry into feared situations (i.e., those situations that symbolize or resemble the traumatic events) to reduce anticipatory anxiety, and thereby reduce behavioral avoidance. This stands in marked contrast to predictions emerging from Volpicelli's (1987) endorphin-compensation theory, which implies that drinking should follow exposure to the traumatic event or reminders of the event (see also Fidler & LoLordo, 1996). More evidence on the time course of drinking in patients with PTSD is needed to further evaluate the usefulness of each of these theories in explaining alcohol abuse in patients with PTSD.

Although no researchers specifically have examined alcohol's effects on behavioral avoidance in samples of participants with PTSD, previous research suggests that alcohol may be an effective agent in reducing behavioral avoidance in anxiety-provoking situations, in general (for a review, see Cappell & Greeley, 1987). Early tests of the TRT focused on the effects of alcohol on avoidance behavior in animals (e.g., Conger, 1951; Masserman & Yum, 1946). Alcohol has repeatedly been shown to reduce fear- or conflict-related avoidance behavior in animals (for reviews, see Cappell & Greeley, 1987; Cappell & Herman, 1972; and Hodgson, Stockwell, & Rankin, 1979). Similar results have been found with human participants. Measures of approach toward a feared object provide a clear behavioral index of the avoidance-reducing effects of alcohol. For example, Alexander and Lindman (1980) examined the effect of alcohol on a naturally occurring fear. Female participants were asked to transfer a mouse from one jar to another. The behavioral measure of avoidance was the amount of time taken to complete this transfer. A moderately high dose of alcohol, but not a placebo beverage, significantly reduced behavioral avoidance (i.e., significantly shorter transfer latencies following alcohol consumption). Thyer and Curtis (1984) examined the effects of alcohol on performance in a behavioral avoidance test (BAT; Lang, 1968) in participants diagnosed with a simple phobia. Degree of approach toward the phobic object was assessed both before and after participants consumed either a moderately intoxicating dose of alcohol or a placebo. The Thyer and Curtis results are clearly consistent with reduced avoidance behavior following beverage consumption in the group administered alcohol but not the placebo group (Sher, 1987). A similar study by Rimm, Bridell, Zimmerman, and Caddy (1981) of undergraduates with a snake phobia did not replicate findings of reduced behavioral avoidance following alcohol consumption, but they did use a significantly lower dose of alcohol than the doses used in other studies. Taken together, these results suggest that alcohol, at least at moderately intoxicating doses, does reduce behavioral avoidance in anxiety-producing situations.

More research is required to determine the degree to which alcohol's reduction of avoidance behavior is related to the motives for alcohol abuse in individuals with PTSD. One study provides some preliminary data on this issue. McFall et al. (1992) found that MAST scores were positively correlated with scores on the Numbing/Avoidance subscale of the M–PTSD in a large sample of Vietnam veterans. The degree of correlation between MAST scores and degree of numbing/avoidance was not as strong as the correlation between MAST scores and degree of physiological arousal (McFall et al., 1992). This finding suggests that alcohol abuse in patients with PTSD may be motivated by a desire to control behavioral avoidance symptoms but that this factor may contribute less to posttraumatic alcohol abuse than the desire to control physiological arousal symptoms. However, the inclusion of both emotional numbing and avoidance symptoms in this M–PTSD subscale, and the subscale's focus on both behavioral and cognitive avoidance symptoms, precludes drawing firm conclusions on this issue. Moreover, the McFall et al. data are correlational. Experimental studies on alcohol's effects on behavioral avoidance in PTSD with the alcohol challenge paradigm (Newlin & Thomson, 1990) and the BAT (Lang, 1968) are required before researchers can make any definitive statements on causality. Retrospective and prospective self-report studies examining the patterns of alcohol use in patients with PTSD could help determine whether alcohol use in the daily lives of patients with PTSD occurs in response to anticipation of entering feared situations, as would be predicted by the TRT (Conger, 1951; Fidler & LoLordo, 1996).

**Affective Effects of Alcohol in PTSD**

Another characteristic symptom of PTSD is a numbing of general emotional responsiveness, as indicated by efforts to avoid feelings associated with the trauma, restricted range of affect, feelings of detachment or estrangement from others, and diminished interest in significant activities (APA, 1994). Other affective symptoms characteristic of PTSD include emotional lability (i.e., irritability or outbursts of anger) and associated complications of feelings of depression, and guilt (APA, 1994). As has been previously suggested with respect to other anxiety disorders (e.g., B. J. Cox et al., 1990; Kushner et al., 1990; Otto, Pollack, Sachs, et al., 1992), alcohol abuse in patients with PTSD may represent an attempt to reduce the negative affective symptoms of PTSD. More specifically, LaCoursiere et al. (1980), Kosten and Krystal (1988), and Schnitt and Nocks (1984) suggested that alcohol may be used in PTSD to relieve subjective feelings of anxiety or to self-medicate for the depression-like dysphoria and guilt associated with PTSD symptoms. Alternatively, Krystal (1984) hypothesized that combat veterans with PTSD develop a dread of the more expressive elements of emotion and strive to block these through alcohol abuse. Similarly, Evans (1988) hypothesized that individuals exposed to sexual abuse, physical abuse, or both may use alcohol to dampen the experience of strong feelings.

Given these hypotheses, research on alexithymia might contribute to understanding the motives for abusive drinking in patients with PTSD. The term *alexithymia*, which literally means no words for mood, refers to a disturbance in affective and cognitive functions, characterized by difficulties in recognizing and verbalizing feeling states, a relative lack of fantasy life, descriptions of somatic symptoms as opposed to affective states, and speech and thought patterns that are concrete and closely tied to external events (Nemiah & Sifneos, 1970; Sifneos, 1972, 1973; for a review on alexithymia, see G. J. Taylor, 1994).

Researchers have found that people exposed to trauma, such
as individuals held in concentration camp (Krystal, 1982), and combat veterans (Hyer, Woods, Summers, Boudewyns, & Harrison, 1990; Shipko, Alvarez, & Noviello, 1983) are often highly alexithymic. Krystal proposed that alexithymia may develop in response to extreme trauma to reduce the experience of painful affect. McNally, Litz, Prassas, Shin, and Weathers (1994) provided experimental data to support this argument. They showed that Vietnam veterans with PTSD exhibit “overgeneral” memories (i.e., memories lacking in specific detail) in response to emotional cue words, which may be indicative of attempts to avoid affect (J. M. G. Williams, 1995). Shipko et al. have suggested that constriction of emotional expression may be adaptive in some traumatic situations (e.g., combat).

In addition to being characteristic of people exposed to trauma, high levels of alexithymia have also been observed in people with currently symptomatomatic alcoholism (Krystal, 1979), newly abstinent alcoholism (Haviland, MacMurray, & Cummings, 1988), and those with high familial-genetic risk for alcohol abuse (Finn, Martin, & Pihl, 1987)—the last study suggesting that alexithymia in people with alcoholism is more likely a premorbid vulnerability factor than a consequence of chronic abusive drinking. Like alexithymia, alcohol abuse might develop as a means of avoiding strong affect in patients with PTSD. In fact, alcohol consumption, motivated primarily by desires to cope with (i.e., avoid or reduce) negative emotional states, is a strong predictor of alcohol-related problems. Frequency of drinking for coping-related motives predicts drinking problems over and above drinking quantity and frequency information alone (Cooper, Russell, Skinner, & Windle, 1992). Additionally, McFall et al. (1992) found that level of Numbing/Avoidance symptoms on the M-PTSD was positively correlated with MAST (Selzer, 1971) scores, providing initial empirical support for Krystal’s (1984) emotional-numbing hypothesis of alcohol abuse in combat veterans. However, the correlational nature of these data and the fact that numbing and avoidance symptoms are included in the same subscale make interpretation of McFall et al.’s findings problematic.

Although no researchers have directly examined the effects of alcohol on affect in either people exposed to trauma in general, or patients with PTSD in particular, a good deal of empirical research exists that supports the notion that alcohol can, at times, be effective in reducing negative mood states (Pihl & Smith, 1983; Russell & Mehrabian, 1975). In general, alcohol leads to improvements in self-reported mood (i.e., increases in positive mood and decreases in negative mood; Cappell & Greenley, 1987). The finding that alcohol consumption reduces negative mood (i.e., subjective anxiety or tension) provides support for the TRT (Conger, 1951). However, results in this area have not been as consistent or robust as the findings of alcohol-induced SRD effects on physiological measures (Sher, 1987). Moreover, there is significant variability in the methodologies used across studies, including differences in the doses of alcohol used and the point at which self-rating measures are completed relative to the phase of the blood alcohol curve (Pihl & Smith, 1983). With regard to dose, the effects of alcohol on subjective mood appear to be dose dependent, with larger acute doses of alcohol being associated, in general, with greater reductions in self-reported negative mood states (e.g., Kalin, McClelland, & Kahn, 1965; Persson, Sjoberg, & Svensson, 1980; Warren & Raynes, 1972). Self-reported mood also appears related to the direction of the change in blood alcohol concentration: Participants tend to report mood enhancement when their BALs are ascending and mood deterioration during the descending limb of the blood alcohol curve (e.g., Goldberg, 1966; Jones, 1973).

Paradoxically, alcohol at times leads to increases in negative affect. For example, in the laboratory, prolonged drinking has been associated with increases in self-reports of anxiety and other negative affect in participants with alcoholism (e.g., McNamee, Mello, & Mendelson, 1968; Mendelson, LaDou, & Solomon, 1964). Moreover, the ingestion of very high doses of alcohol has been shown to lead to increased anxiety (Freed, 1978), as has withdrawal from continued drinking (Peyser, 1982). Any theory that purports to explain the overlap between PTSD and alcohol abuse in terms of attempts to reduce negative emotional states must consider these paradoxical findings. For example, it could be that patients with PTSD may be less sensitive to these aversive effects of alcohol consumption than controls without PTSD. Alternatively, it could be that patients with PTSD are initially motivated to drink to decrease or avoid negative emotional states but eventually drink to alleviate aversive alcohol withdrawal symptoms (Mello, 1981).

Moreover, any hypothesis purporting to explain the overlap between PTSD and alcohol abuse on the basis that alcohol is used to avoid strong affect (e.g., Krystal, 1984) must account for findings that alcohol not only reduces negative emotions but also increases positive emotions. For example, it may be that the motive for drinking in patients with PTSD is not the avoidance of strong affect, in general (Krystal, 1984), but rather the avoidance of only painful and negative affects. Alternatively, it might be that patients with PTSD experience a dampening of painful affect following alcohol consumption but, unlike controls, experience no change in levels of positive affect. Finally, if patients with PTSD only dread strong emotions (Krystal, 1984) in the sober state but are able to find strong positive emotions pleasurable and reinforcing when intoxicated, then it could be that they experience both decreased negative affect (negative reinforcement) and increased positive affect (positive reinforcement) following alcohol consumption. These various possibilities are deserving of further study. Methods to assess the relationship between various aspects of affect and alcohol consumption in patients with PTSD include retrospective and prospective self-reports, the alcohol challenge paradigm (Newlin & Thomson, 1990), and the stress-induced drinking methodology (e.g., Higgins & Marlatt, 1973).

**Cognitive Effects of Alcohol in PTSD**

The hallmark symptom of PTSD is the persistent reexperiencing of the traumatic event (APA, 1987). Such intrusive cognitive symptoms can involve recurrent and distressing recollections of the event, recurrent distressing dreams of the event, sudden acting or feeling as if the traumatic event were recurring, and intense psychological distress at exposure to events that symbolize or resemble an aspect of the trauma (APA, 1994). Other characteristic cognitive symptoms of PTSD include difficulty concentrating, hypervigilance, and an effort to avoid thoughts associated with the trauma (i.e., thought suppression; APA, 1994). Thus, abusive drinking to self-medicate in patients...
with PTSD may involve attempts to control one or more of these cognitive symptoms. For example, both LaCoursiere et al. (1980) and Krystal (1984) postulated that alcohol abuse in patients with PTSD may serve to eliminate terrifying nightmares. More specifically, LaCoursiere et al. suggested that patients with PTSD may be using alcohol to suppress rapid eye movement (REM) sleep and its associated nightmares. They also postulated that the abuse of alcohol in such patients would escalate as tolerance to the REM-suppressant effects of alcohol develops over time. However, traumatic nightmares are not confined to REM sleep (Kramer, Schoen, & Kinney, 1984). Therefore, alcohol may suppress REM sleep, eliminating some, but not all, nightmares. Not being able to eliminate their dream disturbances completely may reinforce heavier drinking in patients with PTSD.

LaCoursiere et al. (1980) further postulated that in patients with PTSD, heavy drinking might block intrusive memories associated with the traumatic event during the waking state. Similarly, Keane, Gerardi, et al. (1988) and Lindy, Grace, and Green (1984) suggested that substance abuse in combat veterans with PTSD exists to quell intrusive cognitive experiences. Alcohol abuse in patients with PTSD might also focus attentional resources (i.e., reduce the concentration difficulties characteristic of PTSD; Steele & Josephs, 1988), reduce hypervigilance, or both.

In Wilson's (1988) review of the relation between alcohol abuse and various anxiety disorders, he noted that there may be important pathophysiological differences among the various anxiety disorders that interact differentially with alcohol. Several current theories interpret alcohol's anxiolytic properties in terms of direct or indirect effects on cognitive processes (e.g., threat appraisal, Levenson et al., 1980; attention allocation, Steele & Josephs, 1988). Wilson has speculated that alcohol's self-medicating effects may be cognitively mediated only in patients with anxiety disorders with a large cognitive component. With intrusive cognitive symptoms often hailed as the disorder's "hallmark" symptom, PTSD could certainly be viewed as one disorder meeting criteria for inclusion in this category. However, are the cognitive symptoms of PTSD influenced by alcohol consumption, and does this hypothesized reduction in intrusive cognitive symptoms reinforce heavy drinking in patients with PTSD?

Preliminary results support the notion that alcohol abuse in patients with PTSD may represent an attempt to dampen intrusive cognitive symptoms. For example, in the study by McFall et al. (1992) of participants with combat-related PTSD, degree of cognitive reexperiencing of the trauma was significantly positively correlated with MAST (Selzer, 1971) scores. However, these data are correlational and thus do not establish a causal connection between cognitive reexperiencing and alcohol abuse. Future studies that monitor daily symptoms in relation to alcohol use would be useful in this regard, as would experimental alcohol challenge (Newlin & Thomson, 1990) studies.

Researchers have begun to use methods derived from experimental cognitive psychology to investigate information-processing biases in participants with high levels of anxiety (e.g., Samoluk & Stewart, 1996b; Stewart & Gignac, 1996). Previous studies whose researchers used such methods have shown patients with PTSD to be characterized by information-processing biases that may be related to intrusive cognitive symptoms. First, an attentional bias favoring the processing of trauma-related information has been repeatedly demonstrated in patients with PTSD but not in controls (e.g., Bryant & Harvey, 1995; Foa, Feske, Murdock, Kozak, & McCarthy, 1991; Kaspi, McNally, & Amir, 1995; McNally, English, & Lipke, 1993; McNally et al., 1987; Thrasher, Dalgleish, & Yule, 1994). For example, Bryant and Harvey (1995) compared the performance of individuals involved in motor vehicle accidents with PTSD, simple phobia of driving, or low anxiety on an emotional Stroop color-naming paradigm. This paradigm is commonly used to assess degree of attentional bias for fear-related information in the emotional disorders (see reviews by Logan & Goetsch, 1993; Mathews & MacLeod, 1994; Segal, 1996; and J. M. G. Williams, Watts, MacLeod, & Mathews, 1988). Participants were asked to name the color in which a variety of words were printed, as quickly as possible, while ignoring the meaning of the words. Words included strong threat words (e.g., smash), mild threat words (e.g., traffic), positive words (e.g., smile), and neutral words (e.g., blanket). In contrast to individuals with a simple phobia of driving and low-anxious controls, those with PTSD were significantly slower to color name strong threat words than positive and neutral words. Such findings suggest that trauma-related information captures and holds the attention of patients with PTSD, thereby interfering with task performance (i.e., color naming). These findings support the notion that patients with PTSD are hypervigilant toward trauma-related information (see reviews by Chemtob, Roitblatt, Himada, Carlson, & Twentonman, 1989; and Litz & Keane, 1989). These results also support the hypothesis that information about trauma is primed in memory and parallel phenomenological reports of the ease with which traumatic memories involuntarily "come to mind" for patients with PTSD.

This latter hypothesis has been evaluated in patients with PTSD using memory paradigms derived from experimental cognitive psychology. Initial research in this area used cued recall and word-stem completion tests to assess relative explicit and implicit memory for trauma-relevant versus trauma-irrelevant material in patients with PTSD and controls (see a review by McNally, 1994). Explicit memory refers to conscious recollection of previous experiences, whereas implicit memory is revealed when performance on a task is facilitated by prior experience without the necessity of conscious intentional recollection of the previous experience (Schacter, 1987). Work with the cued recall (explicit memory) and word-stem completion (implicit memory) tests in patients with PTSD and controls demonstrated enhanced memory for trauma-related words in patients with PTSD (see a review by McNally, 1994). However, recent research demonstrates that the word-stem completion test is sensitive to manipulations of perceptual (Rodiger & Blaxton, 1987) but not semantic (Gignac & Stewart, 1995) attributes of stimuli. This task may therefore have little relevance for the study of implicit memory for emotional meanings in anxiety disorders (Gignac & Stewart, 1995; McNally, 1994). Researchers have thus begun to use alternative implicit memory paradigms adapted from cognitive psychology to study implicit memory biases for trauma-related material in PTSD. For example, Amir, McNally, and Wiegartz (1995) used
a noise judgement task developed by Jacoby, Allan, Collins, and Larwill (1988) to investigate implicit memory bias for combat material in Vietnam veterans with and without PTSD. Veterans were first exposed to combat relevant (e.g., “the chopper landed in a hot LZ” [landing zone receiving small arms fire]) and neutral (e.g., “the shiny apple sat on the table”) sentences. Veterans then rated the volume of white noise accompanying the presentation of these primed sentences as well as novel unprimed sentences. Implicit memory for the meaning of previously heard sentences is revealed when veterans rate the white noise accompanying unprimed sentences as louder than the noise accompanying the primed sentences (Jacoby et al., 1988). Veterans with PTSD, but not controls, were found to rate the noise accompanying primed combat sentences as louder than noise accompanying primed combat sentences, suggesting implicit memory for the meaning of combat-related material in patients with PTSD. Such implicit memory biases for trauma-related material may underlie the intrusive cognitive symptoms (e.g., flashbacks) characteristic of PTSD.

Researchers have begun to investigate whether alcohol and related drugs, such as the benzodiazepines (Stewart, Pihl, & Padjen, 1992), might affect the tendency of anxious participants to selectively process fear-related information (e.g., Golombok, Stavrou, & Bonn, 1991). Although no researchers have specifically examined the effects of alcohol consumption on intrusive cognitive symptoms in patients with PTSD, some preliminary research suggests that alcohol may be an effective agent in reducing selective attention to threat in participants with high anxiety. For example, a set of two experiments reviewed by Stewart and Pihl (1995) examined the effects of alcohol on the processing of threatening information in an analogue sample of students with high-anxiety sensitivity and controls with low-anxiety sensitivity. In the first study (Stewart, Achille, Dubois-Nguyen, & Pihl, 1992), the processing bias favoring threatening words observed in sober students with high-anxiety sensitivity was eliminated following the administration of a moderately intoxicating dose of alcohol. These results were replicated and extended in a second study (Stewart, Achille, & Pihl, 1993): The degree of attentional bias for threatening information in a group of students with high-anxiety sensitivity who had received a moderately intoxicating dose of alcohol was significantly attenuated relative to the degree of bias for threat in a control group of students with high-anxiety sensitivity who had received a placebo beverage. Thus, some preliminary evidence suggests that alcohol may be effective in reducing the tendency of participants with anxiety sensitivity to selectively attend to threatening material. The degree to which such results are applicable to explaining the association between alcohol abuse and PTSD remains to be determined.

No researchers have specifically assessed the effects of alcohol on memory biases for trauma-related material in patients with PTSD. However, much basic research attests to the fact that acute doses of alcohol and the structurally and functionally similar benzodiazepines (Stewart, Pihl, et al., 1992) have significant effects on human memory abilities. Initial research suggested that alcohol (Hastroudi, Parker, DeLisi, Wyatt, & Mutter, 1984) and the benzodiazepines (Danion, Zimmerman, Willard-Schroeder, Grange, & Singer, 1989; Fang, Hinrichs, & Ghoneim, 1987; also see a review by Curtan, 1991) significantly impaired performance on traditional tests of explicit memory, while leaving performance on implicit memory tasks intact. However, recent evidence suggests that certain benzodiazepines may not only reduce explicit memory performance but also impair implicit memory processes when memory is tested at peak drug concentrations (e.g., Legrand et al., 1995; Stewart, Rioux, et al., 1995), additional basic research on the effects of alcohol on implicit memory is required to test this latter speculation. In addition, studies addressing the effects of acute doses of alcohol on memory biases for trauma-related material (e.g., Amir et al., 1995) are clearly needed. Finally, if future research determines that alcohol administration is indeed effective in reducing and controlling intrusive cognitive symptoms in patients with PTSD, it must also be determined that these cognitive effects of alcohol actually reinforce alcohol consumption. Alcohol consumption in response to cognitive manipulations (e.g., exposure to primes resembling some aspect of the traumatic event; McNally et al., 1994) could be explored using the unobtrusive taste-rating task (e.g., Higgins & Marlatt, 1973; MacDonald, Stewart, Samuluk, & Sweet, 1995).

Several cognitive theories have been proposed to explain the high degree of comorbidity between the anxiety and alcohol disorders. Each of these theories appears potentially applicable in explaining the increased abuse of alcohol in patients with PTSD. For example, Levenson et al. (1980) suggested that alcohol might disrupt the cognitive processes involved in evaluating a situation as stressful or threatening. Thus, alcohol consumption by patients with PTSD might influence the appraisal of various situations as stressful; Alcohol consumption might reduce the distressing nature of intrusive recollections of the traumatic event or reduce the distress associated with exposure to events that symbolize or resemble an aspect of the traumatic event through its influence on the threat-appraisal process.

Another cognitive theory is the attention allocation model proposed by Steele and Josephs (1988) to account for alcohol’s effects on anxiety (see also Josephs & Steele, 1990). Alcohol is said to narrow the focus of attention to the most immediate internal and external cues. When intoxication is accompanied by a moderately demanding cognitive activity, attention is directed toward that activity, leaving less attentional resources available for the processing of threatening information, leading to anxiety reduction. Alcohol abuse in patients with PTSD might represent an attempt to focus attentional resources to-
ward the task at hand and away from trauma-related cues. Thus, alcohol might serve to reduce hypervigilance toward threat and difficulty concentrating in the patient with PTSD. More research is required on this attention allocation model, particularly with respect to its potential to explain the overlap between PTSD and alcohol abuse problems.

Marlatt and Rohsenow (1980) have also proposed a cognitive theory of alcohol abuse. Their expectancy hypothesis places emphasis on the beliefs about the expected effects of alcohol consumption. Such “alcohol expectancies” produce significant effects on both drinking and postdrinking behaviors, which are often as strong or even stronger than the actual pharmacological effects of the drug (Hull & Bond, 1986). In particular, individuals with alcoholism are more likely to attribute stress-reducing properties to alcohol than are controls (Calahan, 1970). Alcohol expectancies in patients with PTSD versus controls without PTSD remain to be assessed. It would be interesting to determine whether patients with PTSD are more likely than controls without PTSD to attribute stress-reducing properties to alcohol. It also remains to be determined whether those patients with PTSD who believe alcohol to be capable of reducing or controlling PTSD symptoms (e.g., intrusive distressing thoughts) are more likely to abuse alcohol than those patients with PTSD who report fewer of these specific alcohol expectancies.

Once alcohol abuse is established, its maintenance in patients with PTSD might also be viewed through the framework of cognitive urge models of addiction (e.g., Baker, Morse, & Sherman, 1987). These models propose that urges are organized at a cognitive level within a propositional network that encodes information on eliciting cues, drug-related responses (e.g., verbal reports of craving and drug-procurement behaviors), and the interpreted meanings of stimuli and responses. Baker et al. drew contrasts between positive- and negative-affect urges. Patients with PTSD might be hypothesized to be a group at high risk for alcohol use following negative-affect urges. Stimulus information encoded within this type of urge network for patients with PTSD might include internal symptoms (e.g., intrusive cognitive symptoms, physiological arousal or reactivity cues, and negative affect cues), external stimuli (e.g., events that serve as reminders of the initial traumatic event), or both. According to Baker et al.’s urge model, the responses produced by the activation of such a network should promote alcohol-seeking behavior and self-reports of urges or cravings to drink in alcohol abusing patients with PTSD.

Given data that self-reported urges do not always predate relapse in addicts (for a review, see Tiffany, 1990), Tiffany proposed an alternative cognitive theory of addictions that contends that drug use in the addict is controlled by automatized action schemata. Given a relatively accessible drug like alcohol and a history of repeated practice of alcohol use in response to certain cues, alcohol procurement and consumption behaviors may become highly automatized in the alcohol abusing patient with PTSD. Tiffany also proposed that the procedures for alcohol administration are stored in memory in the form of automatized action schemata, which are activated by certain internal and external triggers. A modification of Tiffany’s theory to explain the maintenance of alcohol abuse in patients with PTSD might assert that PTSD symptoms, reminders of the traumatic event, negative emotions, or all three could serve as eliciting cues for this automatized action schemata of alcohol administration behavior. Unlike the traditional urge models (e.g., Baker et al., 1987), Tiffany’s theory asserts that alcohol administration can occur in the absence of self-reported urges or cravings for alcohol.

Researchers have begun to test predictions made by Baker et al.’s (1987) and Tiffany’s (1990) models with respect to several addictive drugs including alcohol (e.g., Rohsenow et al., 1992). For example, if alcohol cues are stored in the memory of the individual who abuses alcohol along with eliciting cues, as is suggested by both Baker et al. and Tiffany, then exposure to eliciting cues should lead to enhanced processing of alcohol-related information. Samoluk and Stewart (1996b) used the Stroop paradigm to test this prediction in an analogue sample at risk for alcohol abuse. Similar cognitive methodologies could be used to test the hypothesis that alcohol-related information is stored in memory networks along with eliciting cues (e.g., PTSD symptoms, information related to the trauma, or both) in patients with PTSD who abuse alcohol. Such findings would be predicted by both traditional urge models (e.g., Baker et al., 1987) and Tiffany’s theory. However, the two models appear to make different predictions regarding the level of awareness of alcohol urges or cravings in the individual who is alcohol dependent, with Baker et al.’s theory stating that urges are necessary for alcohol administration to occur in response to triggering stimuli and Tiffany’s theory positing that urges are unnecessary. These predictions could be evaluated by using cognitive paradigms designed to examine automatic versus nonautomatic information-processing biases (e.g., Mogg, Bradley, Williams, & Mathews, 1993) and by having participants report on their level of alcohol urges or cravings (see Rankin, Hodgson, & Stockwell, 1979) in response to hypothesized triggering stimuli.

Alcohol-Induced PTSD Symptom Enhancement

Alternatively or in addition to the self-medicating role of alcohol abuse in individuals with PTSD are notions that alcohol might increase susceptibility to PTSD or worsen PTSD symptoms following exposure to a traumatic event. For example, Kolb (1985) has hypothesized that alcohol, like the structurally and functionally similar barbiturates, might serve as a “narcosynthetic” agent in the expression of PTSD symptoms (i.e., certain PTSD symptoms, such as flashbacks, are more likely to be triggered following heavy drinking). Kolb used the sodium amytal interview (i.e., barbiturate administration) as a clinical tool for identifying catastrophic stressors that were too terrifying for discussion in the control state of consciousness. As defined by Kolb (1985), narcosynthesis is a drug-induced recall of repressed material through an abreactive experience. Through this procedure, repressed material becomes consciously available for later integration and synthesis by the personality (for reviews, see Friedman, 1990, and Kolb, 1993).

Similar to Kolb’s (1985) notion of alcohol-induced PTSD symptom enhancement, Greenstein, Kitchner, and Olsen (1986) suggested that alcohol-induced disinhibition may contribute to flashbacks and nightmares in patients with PTSD. Even LaCoursiere et al. (1980), some of the earliest and strongest supporters of the self-medication hypothesis for alcohol...
abuse in patients with PTSD, noted that long-term heavy use of alcohol may significantly exacerbate the symptoms of PTSD. For example, the peripheral nerve damage engendered by chronic heavy drinking may make some patients with PTSD even more susceptible to startle reactions. Furthermore, heavy drinking eventually disturbs the natural sleep process, and thus continued alcohol abuse may exacerbate the sleep disturbances typically seen in patients with PTSD.

The high degree of overlap between panic disorder and alcoholism has been explained on the basis of the effects of repeated alcohol withdrawal. More specifically, neurochemical changes accompanying alcohol withdrawal, such as noradrenergic (NA) system activation have been proposed to kindle panic attacks (George, Nutt, Dwyer, & Linnoila, 1990). Southwick et al. (1993) have proposed that altered sensitivity of the NA system may underlie PTSD symptoms and thus serve as a biological marker for the disorder. However, this notion has been challenged by Wiseman (1994) on the basis that Southwick et al. did not consider the effects of repeated alcohol withdrawal on the NA system of patients with PTSD. Thus, rather than the NA system disturbances being a biological marker for PTSD, the NA changes noted by Southwick et al. may be secondary to repeated alcohol withdrawal in many patients with PTSD. LaCoursiere et al. (1980) also commented on the role of the well-known alcohol withdrawal effects that emerge following prolonged drinking. They noted that after chronic use of alcohol, attempts to discontinue this self-medication often lead to an exacerbation of the initial PTSD symptoms. Given the similarities between several alcohol withdrawal and PTSD symptoms (e.g., autonomic overarousal and sleeping difficulties; APA, 1994), it is possible that patients misinterpret alcohol withdrawal symptoms as signs of anxiety or that these symptoms serve as reminders of the trauma, thereby further increasing anxiety and arousal levels and motivating continued alcohol consumption.

In the future, researchers may wish to focus on the degree to which alcohol abusing patients with PTSD are able to distinguish between alcohol withdrawal and PTSD symptoms. If withdrawal and anxiety symptoms are shown to be easily confused by patients with PTSD (as they are by patients with comorbid panic disorder–alcoholism; George, Zerby, Noble, & Nutt, 1988), researchers might increase the understanding of the particular difficulties patients with alcoholism and PTSD face during alcohol detoxification. Such knowledge in turn could lead toward improving interventions for effectively withdrawing such patients from alcohol. For example, a cognitive-behavioral treatment approach to benzodiazepine discontinuation has been developed for patients with panic disorder, based on the notion that these patients tend to misinterpret withdrawal symptoms as signs of reemerging anxiety symptoms (i.e., Otto, Pollack, Meltzer-Brody, & Rosenbaum, 1992). This novel treatment approach has been shown to be more successful in assisting patients to withdraw from the drug than a traditional slow taper discontinuation procedure (Otto, Pollack, Meltzer-Brody, et al., 1992). Similar treatments might be beneficial in helping patients with alcoholism and PTSD through the withdrawal phase.

Summary

In conclusion, it appears that a single unidirectional pathway to explain the overlap between PTSD and alcohol abuse is unlikely to be found. Instead, it seems possible that both self-medication and alcohol intoxication or withdrawal-induced intensification of PTSD symptoms contribute to the high degree of comorbidity between alcohol abuse and PTSD diagnoses. Although preliminary evidence suggests that the initial motivation for abusive drinking in patients with PTSD is the relief of PTSD symptoms, alcohol intoxication, withdrawal effects, or both could serve to heighten anxiety in the long term, requiring further heavy drinking to dampen emerging PTSD symptoms. Thus, a vicious cycle may be at play between PTSD and alcohol abuse, where one disorder sustains the other (Davidson et al., 1990; LaCoursiere et al., 1980). This cyclical interplay may render comorbid PTSD and alcohol abuse disorders more difficult to treat than either disorder alone (see Davidson et al., 1990).

Clinical Implications

The results of the studies reviewed have several important implications for the assessment and treatment of people exposed to trauma who abuse alcohol.

Assessment

The consistent finding of an association between trauma exposure and alcohol abuse suggests that clinicians working with individuals who present alcohol abuse symptoms should carefully assess for a history of exposure to trauma and PTSD symptoms. Information regarding the nature and severity of the trauma should also be obtained. This includes, for example, determining the type of trauma to which the individual was exposed (e.g., disaster, assault, or combat), the age at which the event occurred, the duration of exposure (e.g., acute vs. chronic), and the individual's sense of control during the trauma (see Foà, Sticeete, & Rothbaum, 1989; and Foà, Zinbarg, & Rothbaum, 1992).

In the assessment of PTSD symptoms, clinicians should, whenever possible, use a multimodal assessment (Lyons et al., 1988), focusing on physiological, behavioral, affective, and cognitive components of posttraumatic stress symptoms. Some clinical researchers have also advocated the use of cognitive psychology paradigms, such as the emotional Stroop task, as additional clinical assessment tools (e.g., McNally et al., 1993). There are several interviews available for diagnosing PTSD (for a review, see Blake et al., 1995). There are also self-report assessment instruments available for assessing PTSD symptoms in general, such as the Impact of Events Scale (Horowitz, Wilner, & Alvarcz, 1979), and for assessing PTSD symptoms associated with specific trauma, such as the TSC–40 (Elliott & Briere, 1992) and the PTSD Symptom Scale (PSS; Foà, Rigg, Dancu, & Rothbaum, 1993) for sexual abuse or sexual assault and the MMPI–PTSD (Keane, Gerardi, et al., 1988) and M–PTSD (Keane, Gerardi, et al., 1988) scales for combat trauma.

When inquiring about a history of trauma exposure, clinicians should consider the social stigma (e.g., feelings of shame and guilt) that are associated with some traumatic experiences (e.g., sexual assault) and the potential for underreporting such events due to discomfort with the subject matter. This issue highlights the importance of developing rapport with a patient
before inquiring about highly personal experiences. However, concerns about underreporting must be balanced with concerns about overreporting (see Briere & Zaidi, 1989, and Loftus, 1993, respectively). The effects of the use of leading questions and probes on the accuracy of patients' reports of traumatic history remain to be determined.

Clinicians working with individuals exposed to trauma should carefully assess the individual's alcohol use patterns (Nace, 1988). Information regarding the individual's use of alcohol before and after the trauma should be obtained. The frequency of drinking occasions and quantity of alcohol used per occasion (e.g., Stewart, Peterson, et al., 1995; Vogel-Sprott, 1983), frequency of drinking to intoxication (e.g., Stewart, Peterson, et al., 1995), situational triggers for drinking (e.g., Annis, Graham, & Davis, 1987; Carrigan & Stewart, 1995; Stewart, 1995b), and self-perceived drinking motives (e.g., Cooper et al., 1992; Stewart & Zeitlin, 1995; Stewart, Zeitlin, & Samoluk, 1996) should be assessed, as should any areas of functioning (i.e., social, occupational, and physical) that have been adversely affected through heavy alcohol use (Nace, 1988). There are several interviews available for diagnosing alcohol abuse—dependence (for a review, see Jacobson, 1989). There are also several self-report assessment instruments available for assessing problem drinking symptoms, such as the MAST' (Selzer, 1971).

Assessment can serve as a useful bridge to treatment or as a brief intervention in its own right. Most researchers and clinicians today espouse the view that alcoholism is a complex set of behaviors associated with a wide variety of causes (e.g., Penk et al., 1981). Clinicians should recognize that trauma exposure and PTSD symptoms may be one possible pathway to the development of alcoholism. It is important to recognize that the motivations for alcohol abuse in this group may be distinct from other subgroups of people who are alcohol abusers. Focusing on the individual's motives for alcohol use during the interview can serve as a first step in the intervention process (W. R. Miller, 1989). Providing information on the relationship between PTSD symptoms and alcohol abuse, how the two disorders may be sustaining each other, and advice on breaking this cycle can be a useful step in increasing clients' motivation for change (W. R. Miller, 1989).

Druley and Pashko (1988) highlighted the importance of accurate and early detection and triage of individuals who have been exposed to a traumatic event, including implementation of measures to prevent the development of alcohol-related problems. They described a group of WW2 and Korean War combat veterans, all over the age of 49, with chronic PTSD and alcoholism diagnoses. PTSD in most of these veterans had been undiagnosed for decades. These veterans had chronically abused alcohol to manage their PTSD symptoms, resulting in many hospital admissions for alcohol abuse and related medical problems. Obviously, early detection of such patients, along with the institution of effective interventions, could help to relieve the long-term maladjustment of many individuals exposed to trauma and would relieve the burden on the health care system associated with the chronic care of such patients.

Treatment

In patients with comorbid diagnoses of PTSD and alcohol abuse, treatment needs to address both disorders. Unfortunately, many clinicians who specialize in the treatment of people exposed to trauma view alcohol abuse as secondary to PTSD and thus assume that the drinking will normalize following the alleviation of PTSD symptoms (Brinson & Trenor, 1989; Gottheil, 1988; Hurley, 1991). There are several problems inherent in this viewpoint. Independent of etiological factors, once the abusive drinking has begun, the alcohol disorder may take on a life of its own (Brinson & Trenor, 1989; Hurley, 1991; Nace, 1988; Pihl & Stewart, 1991). Thus, discontinuing abusive drinking, even following PTSD symptom management, is difficult for these individuals. Similarly, many substance abuse clinicians focus on the alcohol problem and overlook the PTSD symptoms (Brinson & Trenor, 1989; Hurley, 1991). However, if the PTSD symptoms are not treated, the patient may experience a recurrence or intensification of the distressing PTSD symptoms following sobriety (or during detoxification) and may again turn to alcohol for temporary symptom relief.

Some researchers speculate that successful treatment of one disorder may be hindered by the presence of a coexisting disorder (e.g., Davidson et al., 1990; Roy, 1984). Kuhne, Nohner, and Baraga (1986) empirically evaluated this possibility by assessing the efficacy of chemical dependency treatment as a function of combat exposure in Vietnam veterans. In addition to the chemical dependency treatment, all veterans were offered concurrent trauma-oriented group therapy. Combat veterans with varying degrees of combat exposure completed chemical dependency treatment at equal rates and showed equal rates of abstinence at a 1-year follow-up posttreatment, suggesting no effects of trauma severity on chemical dependency treatment efficacy (at least when patients are offered concurrent trauma-oriented therapy). However, this study was limited by a small sample size and did not use rigorous measures of combat exposure (see critique by Hyer et al., 1991). Moreover, given data that PTSD symptoms, rather than trauma severity, appear more highly related to posttraumatic alcohol abuse (e.g., McFall et al., 1992), the Kuhne et al. study is particularly lacking in its assessment of the effects of PTSD symptoms on the efficacy of chemical dependency treatment.

Although most experts agree that both problems need to be a focus of treatment when dealing with patients comorbid for PTSD and alcohol abuse, there are divergent opinions about which problem should be dealt with first or if both problems should be treated simultaneously. Some experts recommend treating the alcohol disorder first (e.g., Bartucci & Stewart, 1986; Brinson & Trenor, 1989; Nace, 1988; Schnitt & Nocks, 1984; Skorina & Kovach, 1986). Barlow (1988), for example, suggested that the efficacy of psychological treatments for anxiety may be diminished when patients are concurrently taking arousal-dampening drugs, such as alcohol. The efficacy of treatment for anxiety in general, and for PTSD in particular, in individuals who continue to abuse alcohol awaits empirical study. Other experts suggested an integrated simultaneous treatment approach, where both alcoholism and PTSD are a focus of treatment from the outset (e.g., Hyer et al., 1991; Kuhne et al., 1986; Turner & Colao, 1985). Intervention outcome studies in which these various approaches to the treatment of dual diagnoses are empirically compared are required before statements
Clinicians should also be aware of issues related to the use of benzodiazepines in patients comorbid for PTSD and alcoholism. Although benzodiazepines are effective in eliminating some symptoms for some patients with PTSD, Friedman (1981) recommended that, if used, they should be prescribed with caution for patients with PTSD and concurrent alcoholism. In addition to the growing concerns about addiction and withdrawal problems associated with benzodiazepine use (Wardle, 1990), the benzodiazepines have dangerous effects when combined with alcohol (Stewart, Pihl, et al., 1992). Moreover, cross-tolerance effects between the two drugs may mean that an alcohol-dependent patient with PTSD may require higher than normal levels of benzodiazepines to achieve symptom management. Concerns have also been expressed that the benzodiazepines may actually interfere with the efficacy of psychological treatments for anxiety disorders (e.g., Wardle, 1990). Finally, Friedman (1988) noted that pharmacotherapy is rarely sufficient to provide complete remission of PTSD symptoms.

Hyer et al. (1991) recommended that the binge drinking pattern of patients with alcoholism and PTSD be a specific focus of treatment. They recommended application of Marlatt and Gordon’s (1985) relapse prevention model. More specifically, knowledge that these patients tend to drink heavily in an episodic fashion allows the clinician to assist them in identifying high-risk situations that trigger their drinking and to educate them in the use of alternative coping strategies to deal with these situations when they occur. Similarly, Schnitt and Nocks (1984) recommended making such patients aware of the link between their abusive drinking and their memories of the traumatic event. They also recommended training comorbid patients in coping strategies to deal with urges to abuse alcohol in these high-risk situations. If future researchers establish that intrusive PTSD symptoms, negative affect, reminders of the trauma, or all three serve as cues for eliciting binge drinking behavior, then it may be useful to teach comorbid patients with PTSD and alcoholism to attend to their reactions to these cues, to conduct coping skills training in the presence of these cues, and to combine exposure to these cues with response (i.e., drinking-) prevention to reduce these cue reactivity effects (Rohsenow et al., 1992).

Finally, some researchers commented on difficulties arising in the detoxification phase in patients comorbid for alcoholism and PTSD (e.g., Bartucci & Stewart, 1986; LaCoursiere et al., 1980; Schnitt & Nocks, 1984). For example, Schnitt and Nocks noted that patients with traumatic histories often experience a sense of uncontrollable anxiety or insomnia during detoxification. In addition, Bartucci and Stewart cited data showing an excess of REM sleep in people with chronic alcoholism undergoing detoxification (H. L. Williams & Rundell, 1981). They argued that because patients with PTSD may be using alcohol to suppress REM sleep and its associated nightmares, this period of REM rebound may be particularly difficult for them in attempting to discontinue alcohol abuse. A gradual tapering of alcohol intake is recommended, in combination with preparing patients for these effects using psychoeducational and cognitive-behavioral techniques (e.g., Otto, Pollack, Meltzer-Brody, et al., 1992).

**Summary**

We require a good deal more knowledge about the nature of the relationship between PTSD and alcohol abuse to better structure therapeutic efforts to the special needs of these comorbid patients. Clinicians working with such populations should be aware of the data currently available, indicating a high degree of overlap between the two disorders and the characteristic binge drinking profile of these patients. As is apparent in the review above of current therapeutic strategies, clinicians are responsive to the emerging data on PTSD and alcoholism comorbidity and appear to be adapting their therapeutic strategies appropriately.

**Directions for Further Research**

Throughout this review, a number of suggestions for future research have been made. Important remaining issues are highlighted in the following section, including some comments on suggested methodologies for examining these issues.

First, future comorbidity studies should attempt to use the multimodal assessment method (Lyons et al., 1988) for establishing PTSD whenever possible. As part of this assessment, researchers should use validated measures, such as structured interviews, for establishing PTSD and alcohol abuse dependence diagnoses. In addition, more research should be conducted on comorbid patients’ perceptions of the nature of the relationship between their two problems. Rachman’s (1991) suggestions regarding suitable methodologies for studies on functional connectedness may prove useful.

Second, given the primary nature of intrusive cognitive symptoms in PTSD, more emphasis should be placed on understanding the potential relationship between these symptoms and the abuse of alcohol. Paradigms derived from experimental cognitive psychology (such as the emotional Stroop task and explicit and implicit memory tasks) are useful for studying the nature of intrusive cognitive symptoms in patients with PTSD; such paradigms might also prove useful in shedding light on the nature of the relationship between intrusive cognitive symptoms and alcohol abuse in patients comorbid for PTSD and alcoholism. For example, patients with PTSD may be highly susceptible to abusive drinking because of the reduction in hypervigilance toward trauma-related cues that alcohol affords. Moreover, given data on alcohol-induced reductions in cognitive biases for threatening information in participants with anxiety sensitivity (Stewart & Pihl, 1995), alcohol might similarly reduce the cognitive biases for trauma-relevant information in patients with PTSD, thereby controlling the cognitive recexperiencing symptoms of PTSD.

Third, this area could benefit greatly from studies using the alcohol challenge paradigm (Newlin & Thomson, 1990) to determine whether patients with PTSD show responses to alcohol that might begin to explain their propensity to develop post-traumatic drinking problems. Given that individuals are not always aware of the motivations for their behaviors (McClelland, 1985; NiBett & Wilson, 1977), alcohol chal-
lenges could provide objective data to supplement data obtained through self-reports.

Fourth, even if it is established that alcohol administration reduces or controls intrusive symptoms in patients with PTSD, it remains to be established that these effects actually reinforce their alcohol consumption. The stress-induced drinking methodology, using the alcohol taste-rating task as an unobtrusive measure of alcohol consumption (see Higgins & Marlatt, 1973, 1975), could elucidate whether elicitation of PTSD symptoms actually leads to increased drinking in the laboratory. Similarly, this paradigm could be used to determine whether priming of trauma-related concepts in memory (e.g., McNally et al., 1994) leads to increased drinking in the laboratory.

Fifth, this area of research could benefit from an increased focus on comparison of comorbidity rates across a variety of traumatic events. Data suggest that some forms of trauma are more likely to lead to PTSD than others (APA, 1994). Similarly, it is possible that some forms of trauma may be more likely to result in alcohol abuse or comorbid PTSD and alcohol abuse. The many dimensions of traumatic experience (e.g., duration of traumatic exposure, exposure severity, interpersonal trauma vs. acts of God, and age at which the exposure took place) need to be considered in future comorbidity research. Exposure to atrocities and grotesque death appears to be one type of traumatic experience that is strongly associated with chronic PTSD (Yehuda, Southwick, & Giller, 1992). Preliminary data with combat veterans and individuals exposed to a disaster (e.g., Green et al., 1985, 1989) also suggest a strong link between exposure to grotesque death and posttraumatic alcohol abuse. These findings should be followed up with studies of individuals exposed to other traumatic events to determine their generalizability.

Sixth, this area of research would be enhanced by an increased focus on individual differences, such as gender and personality factors. For example, although overall men appear to be at greater risk for the development of alcohol abuse problems than women (Robins et al., 1984), women have been shown to be at heightened risk for coping-related alcohol use—abuse when compared with men (Cooper et al., 1992). No researchers have directly examined gender differences in rates of comorbid PTSD and alcohol abuse. This may be due to the fact that most comorbidity studies have focused on either individuals exposed to war-related combat (the large majority of whom are men) or individuals exposed to sexual abuse—assault (the large majority of whom are women). Examination of gender differences in comorbidity of alcohol abuse and PTSD diagnoses following traumatic events that occur more equally across genders might assist in addressing this issue.

Seventh, although there is a significant overlap between PTSD and alcohol abuse, not all individuals with PTSD develop drinking problems. An increased focus on individual differences that might predict which individuals develop posttraumatic drinking problems, given the presence of PTSD, would be instructive. Investigation of potential protective factors (e.g., social support and early disclosure of the traumatic event) might also be a useful avenue for research pursuit.

Finally, with regard to treatment, comparative efficacy studies on the various interventions designed to treat comorbid PTSD and alcohol abuse should be conducted. When evaluating outcome, reduction in both symptoms of PTSD and abusive drinking should be assessed. Furthermore, it remains to be determined whether approaches that focus on only one disorder might also reduce symptoms of the other disorder (e.g., determining whether a treatment that focuses on only treating PTSD symptoms might also have concomitant effects in reducing alcohol abuse symptoms; Gottheil, 1988). Moreover, it remains to be established which individuals might benefit most from which types of treatment approach.

Conclusions

This article reviews extant studies on the occurrence of alcohol abuse—dependence in individuals exposed to a wide variety of traumatic events. In general, the data suggest that individuals exposed to trauma are at greater risk for the development of alcohol problems than are individuals not exposed. Data indicate that trauma severity is positively associated both with alcohol consumption following the trauma and with severity of alcohol-related problems. Even more compelling is the large body of evidence supporting a relationship between PTSD and alcohol problems: Individuals with PTSD appear to be at significantly elevated risk for the development of alcohol-related problems when compared with both individuals exposed to trauma without PTSD and those who have never been exposed to a traumatic event. The most common notion proposed to account for the significant degree of comorbidity between PTSD and alcoholism is the idea that these comorbid individuals are abusing alcohol to self-medicate their PTSD symptoms. Self-medication in patients with PTSD might be achieved through alcohol's effects in reducing physiological reactivity, behavioral avoidance, negative affect, or cognitive symptoms. Alternatively, alcohol abuse—withdrawal might serve to trigger or exacerbate PTSD symptoms. The contrasting notions of self-medication and alcohol-induced intensification of PTSD symptoms in patients can be reconciled if data can be obtained that both processes may be operative in a cyclical fashion. This area of research could be improved through studies using superior methodologies for assessment of PTSD and alcoholism, a greater emphasis on comorbid patients' perceptions of the functional connection between their two disorders, emphasis on individual differences, and the use of experimental methods to assess the effects of alcohol in patients with PTSD, including the application of experimental cognitive paradigms and the alcohol challenge and stress-induced drinking methodologies. The large body of data supporting a link between PTSD and alcoholism has important assessment and treatment implications. Clinicians are cautioned against viewing either diagnosis as primary in terms of treatment. Emphasis should be placed on therapy approaches designed to address both problems. As greater knowledge is gleaned on the nature of the relationship between PTSD and alcoholism, assessment and treatment techniques can be further improved to better serve the needs of individuals simultaneously afflicted with these two behavioral disorders.

References


TRAUMA AND ALCOHOL ABUSE


