Childhood trauma in treated alcoholics. Prevalence and relevance for clinical impairment
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Citation for published version (APA):
General Discussion

8.1 Introduction

In this final chapter, the major findings regarding the three central themes of this thesis are summarized, discussed, and placed within the context of recent scientific developments in the particular area of interest. Furthermore, methodological issues that are important for the interpretation and extrapolation of the results are addressed. Finally, suggestions for future research and possible implications of the results are given.

8.2 Prevalence of physical and sexual assault histories in treated adult alcoholics

Are experiences of physical and sexual abuse frequently reported in a mixed-gender sample of treated alcoholics? In section 8.2.1 the Addiction Severity Index (ASI) as a screening instrument to assess the prevalence of lifetime physical and sexual assault histories is evaluated. Subsequently (8.2.2), results on prevalence rates of physical and sexual assault in our sample obtained with the Structured Trauma Interview (STI) are summarized and put into perspective through comparison with prevalence rates reported in other populations.

8.2.1 The Addiction Severity Index

The frequency of reported lifetime physical (25%) and sexual assault (17%) experiences obtained with the (Europ)ASI fall within the range of frequencies reported from recent studies of mixed-gender samples of treatment-seeking alcoholics also utilizing the ASI to assess assault histories: 21% and 33% for physical assault; 6% and 17% for sexual assault (Pettinati et al., 2000; Rice et al., 2001). However, our findings (presented in Chapters 2 and 3) indicate that the single-item format of the ASI is not particularly useful for estimating the prevalence of assault histories in alcoholics admitted to treatment services. Due to a focus on assault experiences by familial perpetrators (relatives, neighbors, acquaintances), screening
with the ASI is particularly limited for the assessment of abuse in male subjects. This gender
specific limitation of the ASI has not been examined previously. Given the preponderance of
men admitted to alcohol treatment facilities and our findings indicating significant lifetime
assault-related distress (Chapter 6), the need for a broader perpetrator screening than
currently included in the ASI is of clinical importance. Suggestions for improvement of
sensitivity of the format of the ASI assault items have been provided in Chapter 3. It should
be noted that, in addition to the technical modifications in the ASI format, a more specific
training of interviewers regarding assessment of assault histories is generally recommended
to minimize threats to reliability and validity of the used measure (Draijer, 1990; Carlson,
1997).

To the extent that the validity of the assault reports rests on reliability, the marginal
test-retest reliability of the ASI physical and sexual assault items in male veteran alcohol-
and/or cocaine-dependent patients (Cacciola et al., 1999) requires some discussion. This latter
study showed a decrease in lifetime assault reports between two administrations of the ASI
with a test interval of 1 to 3 months. Although there are some indications that men may be
less consistent in self-reports of sexual assault experiences compared to women (Friedrich et
al., 1997; Goodman et al., 1999), the rather poor test-retest reliability of the ASI
physical/sexual assault items is striking. These findings are inconsistent with earlier studies
supporting the stability over time and in different conditions of retrospectively obtained
childhood abuse histories (see Brewin et al., 1993 and Maughan & Rutter, 1997). More
particularly, good test-retest reliability of childhood abuse items measured before and after
approximately 3 months of substance abuse treatment has been shown (Bernstein et al.,
1994). No comparison data are available concerning the stability of lifetime assault reports
over time in a substance abusing sample. Clearly, whether substantial post-treatment shifts in
mood, symptomatology, or in the subjective meaning of potentially traumatic experiences
appreciably affects recall on the ASI assault items in samples of substance abusers, needs to
be evaluated.

In conclusion, since the ASI has become one of the most widely used instruments in the
international addictions field regarding clinical decision-making, research studies and
treatment outcome evaluations, further research on the validity and reliability of the
(modified) ASI assault items is clearly warranted, taking into account possible gender
differences. Regarding the current ASI assault items, ASI users should not only be aware of the underestimation of the frequency of physical and sexual assault reports, but also of a possible test-retest unreliability of such reports.

8.2.2 The Structured Trauma Interview

In line with the literature indicating that the use of behavior-specific questions to detect trauma histories produce significantly higher prevalence rates than single-item screening questions (see Draijer, 1990; Briere, 1997; Carlson, 1997), the highest (and presumably more accurate) lifetime assault rates were observed in response to the STI: lifetime physical abuse 51%, lifetime sexual abuse 29% (Chapter 3). These data support previous work indicating that lifetime sexual assault histories are frequently reported in samples of treated adult alcoholics (Murphy et al., 1980; Lammers, 1991; Moncrieff et al., 1996). For lifetime physical victimization comparison data are lacking.

More importantly, the STI provided clinically relevant data on early victimization experiences: nearly a third (31.6%) of our study participants reported childhood sexual or physical abuse (Chapter 6). In general, notions of high prevalence of childhood abuse being pseudomemories have been discounted, since false positive reports are a relatively rare phenomenon compared to false negative reports (Williams, 1995; Widom & Shepard, 1996; Widom & Morris, 1997; Chu et al., 1999; see also Carlson, 1997). Several techniques, generally acknowledged to enhance accuracy of abuse reports (see Draijer, 1990; Briere, 1997; Carlson, 1997), have been incorporated in the STI. In addition, support for the validity of the childhood physical and sexual abuse ratings of the STI has been provided by neurobiological research among female borderline patients, showing for example, a strong correlation between sexual and physical childhood trauma and hyperresponsiveness of the serotonergic system (Rinne et al., 2000, in press). Given the lack of a nonalcoholic control group in our study, our findings (presented in Chapter 6, Table 1) are compared with the childhood abuse rates from other studies among treated alcoholics. Besides, the observed rates are put into perspective through comparison with prevalence rates reported in other populations, namely psychiatric patients and adults in the general population.

The observed rate for childhood sexual abuse among treated female alcoholics (45%) falls within the range of earlier reported rates in this population (13% to 74%; Hayek, 1980;
Wilsnack, 1984; Covington, 1986; Scherotzki-Hanninger et al., 1986; Lammers, 1991; Windle et al., 1995; Hall, 1996; Horgan et al., 1998). The prevalence rate of 18% for childhood sexual abuse reports in men is higher than previously reported for male alcoholic inpatients (12%: Windle et al., 1995). Due to our rather restricted operational definition of childhood physical abuse, observed rates for both women (12%) and men (15%) are low compared to those earlier reported among treated alcoholics (females 40% to 71%: Downs et al., 1987; Miller et al., 1987, 1993; Windle et al., 1995; males 13% to 31%: Kroll et al., 1985; Schaefer et al., 1988; Windle et al., 1995). The observed rate for childhood dual abuse for men (5%) is similar to that reported by Windle et al. (1995) among male alcoholic inpatients. However, the dual abuse rate found for women (12%) is low compared to the one (23%) earlier reported among female inpatient alcoholics (Windle et al., 1995).

Studies based on samples of psychiatric patients generally report (somewhat) higher childhood abuse rates than observed in our sample of treated alcoholics (see Herman, 1992a; Read, 1998). Comparison of our childhood abuse rates with those observed in a mixed-gender sample of 160 Dutch psychiatric inpatients obtained also with the STI (Draijer & Langeland, 1999) supports this observation: childhood physical or sexual abuse 32% vs. 42%, childhood physical abuse 14% vs. 26%, childhood sexual abuse 24% vs. 34%, and childhood dual abuse 7% vs. 10%. Community-based studies, however, generally report lower childhood abuse rates (based on similar definitions of such abuse) than found in our sample of treated alcoholics: childhood sexual abuse 7% to 33% for women, 2.5% to 8% for men (see Langeland & Hartgers, 1998; Bensley et al., 2000; Molnar et al., 2001); childhood physical abuse 9% to 34% for women (Draijer, 1988; Römkens, 1989; MacMillan et al., 1997; Bensley et al., 2000), 11% to 12% for men (MacMillan et al., 1997; Bensley et al., 2000); and childhood dual abuse 6.6% for women, 2.2% for men (Bensley et al., 2000). Obviously, increased childhood abuse rates are reported among women and men seeking help for their alcohol problems, relative to those found among women and men in the general population.

In addition to the relatively frequent reports of early physical and sexual victimization, adult assault experiences were also frequently reported in our sample of treated alcoholics: Physical assault was reported by 64% of females and 36% of males; sexual assault was reported by 33% of females and 5% of males. From a clinical perspective it is important to
acknowledge that childhood physical and sexual abuse reports were strongly associated with having been victimized during adult life. The significant increase in frequency of reports of adult sexual victimization among subjects reporting childhood physical abuse (a fourfold increase) or childhood sexual abuse (an almost eightfold increase) supports the literature indicating that revictimization is a rather robust phenomenon, particularly among adult women (see Roodman & Clum, 2001). Whether childhood physical abuse by itself is related to adult sexual victimization or that this relation is an artifact of the co-occurrence between childhood physical and sexual abuse (in our sample particularly among females), as recently shown in a female sample (Merrill et al., 1999), is not clear. Moreover, for interpretation of the data on adult (re)victimization the nature of our sample is of importance, because alcohol problems may be related to the adult victimization rates. The direction of this relationship, however, remains uncertain (see Testa & Parks, 1996). The only study to date that investigated the relationship longitudinally, found that alcohol abuse follows from victimization, but does not increase risk of later assault (Kilpatrick et al., 1997). Other data support the vicious cycle hypothesis assuming that assault and alcohol problems might each increase the risk of experiencing the other (Burnam et al., 1988; Merrill et al., 1999). Studies in this area usually focus on women, therefore, it remains to be elucidated whether there are gender differences regarding the role of alcohol problems in (re)victimization.

Based on our data it can be concluded that histories of both early and more recent victimization were frequently reported in our sample, regardless of gender. Compared to community-based rates, the childhood abuse experiences among persons seeking help for alcohol problems are rather high and warrant routine assessment. In addition, our results on the frequency of multiple trauma exposure argue for a comprehensive pre-treatment trauma assessment, accounting for complex trauma histories.

8.3 Childhood trauma and alcohol use disorders

Is violent victimization in childhood associated with the development of alcohol problems in adult life and is information on childhood trauma, particularly sexual and physical abuse, relevant for understanding the severity of drinking problems in treated alcoholics? In the
following subsections major findings regarding these two issues are summarized and discussed.

8.3.1 The relevance of childhood abuse for the development of alcohol use disorders

The key issue addressed in our review (Chapter 4) was whether childhood victimization places persons at increased risk of later alcohol problems when controlling for the effects of other family pathology or dysfunction. It was argued that conclusions about the relevance of childhood abuse in the risk of the development of alcohol problems should be drawn cautiously given the methodological limitations of the reviewed studies such as small sample size, retrospective assessment of abuse histories implying for instance, possible recall bias regarding childhood abuse in adults with alcohol problems ("effort after meaning"), and the potential role of confounders.

Based on our review (published in 1998) we concluded that the evidence was insufficient to draw conclusions about a child abuse-alcoholism relationship in men. At present, more data are available, particularly on the role of childhood sexual abuse in the development of problem drinking in men (see for a comprehensive review Simpson & Miller, 2002). Three of the four recent methodological more rigorous studies (all cross-sectional/retrospective) suggest a relationship between childhood sexual abuse and the development of alcohol problems independent of other background family factors (Galaiif et al., 2001; Molnar et al., 2001; Nelson et al., 2002). In contrast, however, results of the fourth study, a population-based study of male twins, suggest that childhood sexual abuse is not independently related to the onset of alcohol problems when taking into account other family factors such as parental alcoholism (Dinwiddie et al., 2000). Far less research attention has been paid to a possible link between childhood physical abuse and later alcohol problems in men. Although two studies found that, compared to childhood sexual abuse, childhood physical abuse is more strongly related to later alcohol problems in men (Kinzl & Biebl, 1997; Bensley et al., 2000), possible confounders of this relationship were not included in the analysis.

For women, the empirical evidence suggested that exposure to childhood physical or sexual abuse may increase the risk of alcohol problems in adult life (see also reviews of
Recent retrospective studies almost consistently replicated the prior evidence that childhood sexual abuse is associated with a substantial risk of alcohol problems in women when controlling for background family factors (Sher et al., 1997; Spak et al., 1997, 1998; Dinwiddie et al., 2000; Kendler et al., 2000; Galaif et al., 2001; Molnar et al., 2001; Nelson et al., 2002). The role of childhood physical abuse in the development of problem drinking by women has received much less research attention. One population-based survey showed that combined childhood sexual and physical abuse was associated with heavy drinking, whereas childhood physical or sexual abuse alone was not (Bensley et al., 2000). Another community-based prospective study among women with documented histories of sexual victimization in childhood indicated that childhood physical abuse was a significant predictor of binge drinking behavior but not of heavy alcohol consumption, even after controlling for the effects of parental drinking behavior, negative parental relationship, and childhood sexual abuse characteristics (Jasinski et al., 2000).

On the basis of the currently available retrospective studies and within the methodological limitations which also applied to the previously reviewed studies, it can be concluded that childhood physical and sexual abuse are associated with the development of alcohol problems or alcohol use disorders in adult life in both genders. In this regard, childhood abuse should be interpreted as a non-specific risk factor. Meta-analytic reviews of research on childhood abuse effects (mostly sexual abuse) underline the assumption of a spectrum of posttraumatic symptoms rather than a specific child abuse disorder (e.g. Weaver & Clum, 1995; Neumann et al., 1996; Paolucci et al., 2001). In addition, epidemiological research indicates that other psychiatric disorders (e.g. depression, PTSD) may bear an even stronger association with childhood abuse than alcohol use disorders (Molnar et al., 2001). Nonetheless, there is compelling and consistent evidence that early sexual traumatization is an independent risk factor for later alcohol problems among women. The consistency of the findings indicates that this type of childhood trauma is, in some way, of importance for the development of alcohol problems in women. Findings regarding an independent role of childhood sexual abuse for developing alcoholism in men have been less consistent, indicating the need for additional research in this area. Although the available data generally show a higher likelihood of alcohol problems among adults who were physically abused as children, it seems premature to conclude that childhood physical abuse is an independent risk
factor for the development of alcohol use disorders. Future studies should, therefore, include the assessment of childhood physical abuse beside childhood sexual abuse in order to enhance our knowledge regarding the role of this type of abuse in the etiology and pathogenesis of alcohol use disorders in both genders.

Regarding the interpretation of the data, to infer from retrospective data alone that childhood abuse increases the risk of the onset of alcohol use disorders is problematic. Until prospective research demonstrates that persons with documented histories of childhood abuse are at increased risk of alcohol problems, it cannot be firmly established that childhood abuse plays a role in the onset of alcohol use disorders. The reviewed studies demonstrated increased risk, not causation per se. Although the evidence so far suggests that in most cases childhood physical or sexual abuse preceded the onset of alcohol problems (Miller et al., 1993; Moncrieff et al., 1996; Spak et al., 1997; Nelson et al., 2002), future research needs to establish the temporal sequence of events to be able to make inferences about a possible causal direction.

Various mechanisms have been hypothesized to underlie the childhood trauma-alcoholism relationship, mostly assuming that alcohol use serves as a form of self-medication to mitigate negative psychological consequences of the abuse (see Chapters 1 and 4, as well as reviews of Widom & Hiller-Sturmhofel, 2001 and Simpson & Miller, 2002). As an explanatory framework, "self-medication models" - which can be considered diagnosis or symptom specific modifications of the more general "tension- or stress-reduction" models (see Shiffman & Wills, 1985; Chaudron & Wilkinson, 1988) - are preferred. Specifically the self-medication hypothesis of Khantzian (1985, 1997) is appealing as a possible explanation for the development of alcohol problems in traumatized individuals, because the model is partly based on knowledge concerning early deprivation in addicts. Some support for self-medication of abuse-related symptoms can be found in the more recent literature (Kessler et al., 1997b; Epstein et al., 1998; Fleming et al., 1998; Kunitz et al., 1998; Schuck & Widom, 2001; Teusch, 2001; De Graaf et al., 2002). For example, Kessler et al. (1997b) reported that the relationship between childhood interpersonal traumas and substance use disorder seems to be indirect and mediated by prior (abuse-related) lifetime comorbid disorders, such as major depression, bipolar disorders, generalized anxiety disorder, panic disorder and conduct disorder. Similar findings have been reported by De Graaf et al. (2002), showing a lack of
association between childhood trauma and pure substance use disorders, but a strong association between childhood trauma and psychiatric comorbidity in substance use disorders. In addition, a study among women indicated that the effect of childhood rape on alcohol abuse severity was mediated by the occurrence of PTSD (Epstein et al., 1998). These latter findings are in line with the more extensive literature suggesting that PTSD, rather than the traumatic exposure itself, might be a causal factor in substance use disorders (see Stewart, 1996; Chilcoat & Breslau, 1998; McFarlane, 1998; Stewart et al., 1998; Triffleman, 1998; Jacobsen et al., 2001). Most studies on substance use disorders in subjects with PTSD indicate that PTSD precedes development of substance abuse (see Stewart, 1996; Jacobsen et al., 2001).

Overall, these preliminary findings emphasize the need for further study regarding the potential mediating role of trauma-related symptoms in the relationship between childhood abuse and later alcohol problems. Khantzian (1997) argues that it is not necessarily a psychiatric condition that is self-medicated. Rather, a range of painful affects and subjective states of distress, which may or may not be associated with a psychiatric disorder, are more likely the important operatives that govern self-medication, such as for example, agitation, anger, rage, sadness, psychic numbing or insomnia. Regarding future research in this area, particularly worthwhile may be studies in developmental traumatology that attempt to link the neurobiology of childhood trauma-related negative affect symptoms with the neurobiology of alcohol use disorders, as suggested by De Bellis (2002). These studies may be critical in elucidating whether abuse-related dysregulations in biological stress response systems which may lead to an increased vulnerability for psychopathology, particularly mood and anxiety disorders (De Bellis & Putnam, 1994; De Bellis et al., 1999; Heim & Nemeroff, 1999; Pynoos et al., 1999; Kaufman & Charney, 2001), contribute to a substantially increased risk of developing alcohol use disorders in individuals due to self-medicating primary psychiatric disorders or subjective states of distress with alcohol. Additionally, dysregulated biological stress response systems associated with early trauma and neglect can have adverse influences on brain development, in particular in the frontal and prefrontal cortex, which may lead to impaired capacities for self-regulation and a greater incidence of impulsive behaviors such as alcohol abuse (see e.g. Van der Kolk & Fisler, 1994; Perry & Pollard, 1998; De Bellis, 2001, 2002). Studies in developmental traumatology may produce some further
updates to current influential psychobiological models or typologies of alcoholism (e.g. Cloninger, 1987; Babor et al., 1992; Schuckit et al., 1995b; Cloninger et al., 1996) and enhance our understanding of alcoholism, especially regarding the relevance of environmental-related mechanisms in the development of alcohol use disorders (see Higley & Bennett, 1999).

8.3.2 The relevance of childhood trauma, neglect, and trauma-related distress for the severity of drinking problems

Do traumatized individuals who seek alcoholism treatment report more severe alcohol problems than their non-traumatized counterparts? Our study extended previous research regarding this topic by examining associations of a broad spectrum of environmental factors and trauma-related distress with alcohol problem severity in a sample of treatment-seeking alcoholics (Chapter 5). In order to provide useful information for clinical practice, two frequently clinically applied severity indices were included, namely the ASI interviewer’s rating of the level of a client’s alcohol use problem severity and alcohol dependence severity categories based on DSM-III-R diagnostic criteria (CIDI). Consistent with the literature (e.g. Grant, 1996), in our consecutive series of 155 treated adult alcoholics the vast majority of patients (n = 150) were dependent on alcohol. Furthermore, the proportions of subjects in the mild (18.7%), moderate (36.7%), and severe (44.6%) dependence categories according to DSM-III-R criteria, are comparable with previously reported rates in treated alcoholics (e.g. Caetano, 1993; Andreatini et al., 1994). Findings indicating no significant gender differences in the severity levels of dependence corroborate one earlier report (Caetano, 1993).

No significant relations between trauma, childhood neglect (operationalized as perceived parental dysfunction), and trauma-related distress (operationalized as the presence of a DSM-III-R PTSD diagnosis) and the two severity indices of drinking problems were observed in alcoholic men. In female alcoholics, reports of childhood dual abuse were associated with lower EuropASI alcohol use severity ratings, whereas perceived dysfunctioning of mothers was associated with more severe DSM-III-R dependence levels. Based on these findings it can be concluded that having information about (childhood) trauma and neglect may not be relevant for understanding drinking problem severity in treated male alcoholics. For treated female alcoholics, however, both trauma and neglect may
be associated with drinking problem severity, but due to the rather small number of women (n = 33) our findings remain speculative. Recently, gender differences in associations between childhood trauma and drinking problem severity (including both alcohol abuse and dependence) were found in treated addicts mostly reporting primarily alcohol problems (Chermack et al., 2000), stressing the need for further study of this topic in a larger sample. Results of this latter study suggest that a family history of violence (i.e. a combination of moderate/severe childhood physical abuse and interparental violence) has a greater impact on the clinical course of alcohol problems in women compared to men.

The replication in our study of nonsignificant associations of dependence severity with childhood sexual traumatization (Molnar et al., 2001) and childhood physical abuse (Kroll et al., 1985; Schaefer et al., 1988), despite variations in operationalizations of dependence severity across studies, is striking and suggests that alcohol dependence symptoms are indeed little affected by such experiences. Additionally, no “dose effect relationship” was observed with more intrusive and frequent childhood abuse being associated with more severe levels of dependence. Thus childhood abuse, while associated with an increased risk of alcohol dependence (particularly sexual abuse in females), seems not to be associated with the severity of the alcohol problems once dependence developed. This pattern of findings is similar to the findings regarding affective disorders (also disorders with a large genetic component like alcohol dependence), suggesting that the risk of onset of the disorder is more likely to be associated with major psychosocial stressors than the subsequent course of the illness (Post, 1992; Bernet & Stein, 1998; Harris et al., 1999; Kendler et al., 2001). Research findings suggest that a more severe course of alcohol dependence is probably related to personal dispositions, such as genetic vulnerability (e.g. Yates et al., 1996; Schuckit & Smith, 2001; Connor et al., 2002), neuroadaptations in brain systems due to the effects of chronic alcohol use (see Schmidt et al., 2000; Schoffelmeer et al., 2000), age of onset of alcohol problems (Svanum & McAdoo, 1991), and psychiatric comorbidity (Schaefer et al., 1987; Nelson et al., 1996; Hallman et al., 2001). For example, early-onset cases of alcohol use disorders, which are frequently associated with conduct problems or antisocial behavior (e.g. Watson et al., 1997), are often more persistent and severe than later-onset cases. In fact, antisocial personality disorder is considered one of the most significant forms of comorbid psychopathology in treated alcoholics and is related to an earlier onset, a more severe course, and poorer outcomes (see Verheul, 1997).
Our nonsignificant findings regarding a diagnosis of PTSD and ASI alcohol use severity ratings are consistent with those of Moylan et al. (2001) in treated pregnant drug-dependent women. Our negative results regarding comorbid posttraumatic stress disorder and dependence severity could be due to the examination of PTSD as a dichotomous rather than as a dimensional variable, since particularly Cluster D hyperarousal symptoms have been linked to the degree of alcohol dependence (Stewart et al., 1998). However, whether these latter findings reflect substance-induced intensification of PTSD arousal symptoms or intensification of the use of alcohol motivated by symptoms of hyperarousal remains to be elucidated. Most likely, with the evolution of alcohol dependence, a vicious circle comes into play where one disorder serves to sustain and/or exacerbate the other (see e.g. Lacoursiere et al., 1980; Stewart, 1996). It has been suggested that the close resemblance of PTSD arousal symptoms with symptoms of the alcohol abstinence syndrome (see Stewart et al. 1998 and Jacobsen et al., 2001) contributes to a more severe course of alcohol dependence in traumatized individuals with PTSD. Autonomic physiological arousal that occurs in alcohol withdrawal may serve as a conditioned reminder of traumatic events, leading to an increase of PTSD symptoms, which prompts the need for further self-medication through increasing use of psychoactive substances (see e.g. Jacobsen et al., 2001). Noteworthy, preliminary research on cue reactivity does suggest that exposure to trauma reminders in individuals with PTSD and alcohol dependence leads to a significantly higher increase in drug craving ("pathological wanting") compared to individuals with PTSD and cocaine dependence (Coffey et al., 2002). In future research more emphasis should be placed on understanding the potential relationship between the PTSD symptoms and the course of alcoholism.

Clearly, additional research is needed to elucidate whether childhood trauma, neglect, and trauma-related symptoms are relevant for the clinical course of alcohol use disorders. In the DSM-IV the subtyping of dependence according to severity has been replaced by subtyping alcohol dependence as having developed with or without accompanying tolerance or withdrawal (physiological component), a distinction for which some clinical validity has been reported (Schuckit et al., 1998b). Further research may address whether there are differences in trauma and neglect histories between alcohol-dependent individuals with and without a physiological component.
8.4 Childhood trauma and psychiatric comorbidity

Are childhood trauma and neglect, in particular physical and sexual abuse, important factors in understanding clinical impairment in treated alcoholics, specifically regarding comorbid affective and anxiety disorders (including PTSD), suicide attempts and dissociation? Two important statements concerning the data on psychiatric symptoms need to be made before discussing the results. First, due to practical limitations the data are restricted to Axis I syndromes. However, factors that complicate the psychiatric profile of treated alcoholics are the frequent presence of personality disorders (Axis II) as well as conjoint chronic medical illness (Axis III). Therefore, the potential associations between childhood trauma and psychiatric comorbidity discussed below should be viewed as one part of a far more complex whole. Second, the administration of the instruments to assess symptomatology was postponed until 2 to 8 weeks after treatment admission in order to be able to distinguish comorbid psychiatric disorders from alcohol intoxication and withdrawal symptoms. It is generally acknowledged that many Axis I disorders or symptoms will "clear" after a period of abstinence. For example, in alcohol-dependent patients rates for a diagnosis of major depression dropped from 67% before admission to 13% in the detoxification period (7 to 10 days after cessation of drinking) (Davidson, 1995).

8.4.1 The relevance of childhood trauma for comorbid affective disorders, anxiety disorders, and suicide attempts in treated alcoholics

Is childhood trauma, especially physical and sexual abuse, relevant for understanding comorbid affective and anxiety disorders as well as suicide attempts in treated alcoholics? Previous investigations, with the exception of Windle et al. (1995), have typically considered only one form of abuse, with one gender, and one type of symptom or syndrome rather than a range of psychiatric disorders. Through the inclusion of a broad range of environmental hazards, severity indicators of childhood abuse, and a broader spectrum of affective and anxiety disorders, our findings (Chapter 6) represent an important advance in specifying the relationship of childhood trauma and psychiatric comorbidity in treated alcoholics.

Before discussing the significance of childhood physical and sexual abuse histories in psychiatric comorbidity, we will briefly discuss our data regarding the observed prevalence
of comorbid lifetime Axis I symptoms. Comorbid mood and/or anxiety disorders were observed in 62.3% of our patients and 16.8% met criteria for a posttraumatic stress disorder. This is in general agreement with the literature about psychiatric comorbidity among treatment-seeking alcoholics (e.g. Hesselbrock et al., 1985; Black et al., 1987; Ross et al., 1988; Herz et al., 1990; Tómasson & Vaglum, 1995). Also, the observed rate for suicide attempts (24.5%) is high and in line with previous research (Roy & Linnoila, 1986; Roy, 2000). Overall, the anxiety disorders and among them the phobic disorders accounted for the most frequent diagnoses in our sample. The observed rate for Axis I lifetime comorbid mood and anxiety disorders is higher than that reported among individuals with an alcohol use disorder in a community-based study (Regier et al., 1990: 19.4% for anxiety disorders, 13.4% for affective disorders), confirming that treatment-seeking alcoholics have more severe and complex symptom patterns. However, given the preponderance of alcohol-dependent patients in our sample, comparing prevalence rates of individual diagnoses with those observed in individuals with a lifetime alcohol dependence diagnosis in the general population may be more revealing. In fact, comparison of our data with those of Kessler et al. (1997a) based on assessment with the same instrument (CIDI DSM-III-R) demonstrates a more severe clinical profile in treatment-seeking alcohol-dependent males, but not in females: Rates for the examined individual comorbid diagnoses were consistently higher among the treated male alcoholics than those reported by alcohol-dependent men in the community, except for depression (22.1% vs. 24.3%) and generalized anxiety (4.9% vs. 8.6%). In contrast, for the treated female alcoholics, only the observed rates for PTSD (27.3% vs. 26.2%) and dysthymia (33.3% vs. 20.9%) were (somewhat) higher than reported among alcohol-dependent women in the general population.

Focusing on the question whether childhood physical/sexual abuse histories are important for a better understanding of other psychiatric symptoms in alcoholics patients, our results can be summarized as follows: Controlling for the potential confounding effects of other childhood adversities and adult assault histories, childhood sexual abuse was independently associated with the presence of comorbid social phobia (total sample and male patients), agoraphobia (male patients), and PTSD (male patients). Childhood dual abuse was independently related to the presence of PTSD (total sample). Childhood abuse (physical and/or sexual) was not related to the presence of comorbid affective disorders nor to suicide attempts, instead for the
presence of these symptoms maternal dysfunctioning was particularly important. And, childhood sexual abuse and dual abuse independently contributed to the number of comorbid diagnoses compared with the other risk factors. Besides, more severe and intrusive forms of early sexual abuse as well as multiple traumatization were associated with complex comorbidity patterns, including PTSD, agoraphobia, social phobia, dysthymia, and suicidality.

Based on these findings, reflecting that childhood abuse (particularly sexual abuse and combinations of sexual and physical abuse) may be associated with a complex set of clinical features, one may conclude that these experiences are clinically important. Particularly meaningful are the findings indicating that childhood abuse reports independently predicted the number of comorbid mood and/or anxiety conditions as well as some individual diagnoses (PTSD, social phobia, and agoraphobia) after controlling for the effects of other childhood stressors and adult victimization experiences. This has not been previously demonstrated in treated alcoholics. Taken together, the present findings seem to confirm an abuse-related posttrauma symptom spectrum, particularly with chronic and pervasive trauma (Type II trauma: Terr, 1991) in treated alcoholics and concur with studies suggesting chronic or complex PTSD rather than simple PTSD in individuals exposed to sexual and/or physical abuse (e.g. Roth et al., 1997). It may well be that our findings on specific abuse-related disorders do reflect symptom overlap (i.e. panic attacks and avoidance) between PTSD and phobic symptoms rather than the presence of separate disorders (see e.g. Deering et al., 1996; Orsillo et al., 1996). For example, in community samples it has been demonstrated that phobic symptoms are more prevalent in chronic PTSD than in acute PTSD (Davidson et al., 1991; Rothbaum et al., 1992). The latter would be in line with suggestions of Van der Kolk and colleagues (1996) to view PTSD and its comorbid conditions as a complex spectrum of adaptations to trauma. However, additional research on symptom patterns in alcoholic patients with PTSD is necessary to confirm this suggestion concerning complexity of clinical outcome.

An important question is how to interpret the distinct clinical profile observed in early sexually/physically traumatized alcoholic patients. An explanation for such a distinct pattern can be found in (neuro)biological research demonstrating that genetic predisposition coupled with stress in early, critical phases of development induce biological alterations in stress
response systems which may increase the risk for an individual to develop mood and anxiety disorders when exposed to further stress (De Bellis & Putnam, 1994; De Bellis et al., 1999; Heim & Nemeroff, 1999, 2001; Pynoos et al., 1999; Heim et al., 2000; Kaufman & Charney, 2001). Neurobiological systems that might be altered by traumatic experiences include the hypothalamic-pituitary-adrenal (HPA) axis, the sympathetic nervous system, central catecholamines, serotonin systems, and the endogenous opiates. Alternatively, methodological explanations involve the possibility of reporting bias such as for example, that subjects with lifetime PTSD symptoms and/or mood and anxiety symptoms might overreport traumatic exposure, that the different clinical manifestations found between abused and nonabused patients may likely be the result of mood state effects, or that reporting bias may be present in posttrauma symptoms due to the recall status of abuse histories. The notion of possible mood state effects has generally not been empirically supported (Robins et al., 1985; Brewin et al., 1993; Ferguson et al., 2000; Edwards et al., 2001). However, in a general population study, subjects who had recently recalled sexual abuse memories reported increased posttraumatic distress compared to those with continuous recall or more distant recovery of abuse memories (Elliott & Briere, 1995). Besides, although the veracity of abuse reports is unknown due to an absence of independent corroborating evidence of these reports, even if these reports are only subjective perceptions, the present study found a distinct pattern of psychiatric morbidity associated with childhood abuse that might still be of clinical interest to those who treat patients with alcohol use disorders.

In addition, whether our findings provide support for the self-medication hypothesis remains unclear, our data do not permit conclusions on causal links between childhood trauma and later psychopathology. It may be noted, however, that the individual diagnoses found to be specifically associated with childhood abuse usually predate alcohol problems (Kushner et al., 1990; Schneier et al., 1992; Stewart, 1996; Kessler et al., 1997a; Jacobsen et al., 2001), findings which at least do not exclude self-medication as a possible mechanism. In addition, in PTSD the use of alcohol and heroin may alleviate intrusive and arousal symptoms, whereas marijuana and benzodiazepines seem to dampen hyperarousal (Bremner et al., 1996; see also Jacobsen et al., 2001); findings supporting the biologically based assumption of using numbing substances (central nervous system depressants) for self-medicating trauma-related hyperarousal symptoms (Van der Kolk et al., 1985; Kosten & Krystal, 1988; Charney et al., 1993). Prospective studies tracking the temporal relationship
between the onset of PTSD, mood disorders, anxiety disorders and alcohol use disorders are needed to unravel the interrelationships between the disorders, whereas neurobiological studies could explore how negative affect symptoms in alcoholics may arise from a common vulnerability (see De Bellis, 2002). Clearly, understanding comorbidity of psychiatric disorders requires a rigorous integration of biological, psychological and environmental factors.

Finally, our findings indicate that, not only childhood abuse, but also other negative childhood or adult experiences or circumstances are relevant in understanding psychiatric comorbidity in treated alcoholics. This underscores the need for greater clinical attention to the role of a much broader range of childhood and adult stressors in the evaluation and treatment of alcoholic patients.

8.4.2 Childhood trauma and dissociation in treated alcoholics

Our original rationale for conducting this study was based on the hypothesis that severe psychological trauma would be linked to higher levels of dissociation in treated alcoholics. Dissociation has long been conceptualized as a reaction to trauma, particularly trauma in childhood when the capacity for dissociation may be at its highest. The early writings of Pierre Janet have underscored the importance of dissociation as a primary psychological process in response to overwhelming experiences (Van der Hart & Horst, 1988; Nemiah, 1989; Van der Kolk & Van der Hart, 1989). The relationship between dissociation and trauma is well established, in particular with posttraumatic stress disorder (e.g. Spiegel et al., 1988; Carlier et al., 1996; Bremner & Brett, 1997), childhood sexual and physical abuse (e.g. Chu & Dill, 1990; Draijer, 1994; Draijer & Langeland, 1999), disruptions of parental care (e.g. Irwin, 1994; Brodsky et al., 1995; Zlotnick et al., 1995), and lack of parental care (e.g. Modestin et al., 2002), especially maternal neglect (Draijer & Langeland, 1999; Van den Bosch et al., in press). However, reviewing the literature on this issue in addicted samples resulted in a somewhat different study aim, namely to assess the relationship of trauma and dissociation in alcoholic patients accounting for potential reasons for an absence of such a link. In Chapter 7 a series of hypotheses were tested assuming that the absence of the relation results from (1) underreporting of child abuse experiences leading to an underestimation of effect on dissociation, (2) reporting substance-related symptoms on the DES implying
psychometric weakness of the dissociation measure in a population of treated alcoholics, or (3) a low prevalence rate of psychological dissociation in substance abusers due to the potential existence of substance abuse as a form of chemical dissociation.

Based on the pattern of findings it was argued that the absence of a trauma-dissociation link in our study is not very likely to be due to methodological issues as suggested in the first two hypotheses: e.g. substantial childhood physical and sexual abuse rates were observed, the DES showed good psychometric properties, and a higher mean DES score might be expected than that observed (i.e. 11.4) if substance-related symptoms would have contributed to the score. Rather, findings seemed to suggest that in alcoholic patients the absence of a childhood abuse-dissociation relation is likely to be due to a low dissociative capacity or diminished tendency of particularly male alcoholics to use psychological dissociation. Based on gender differences in diagnosed cases of dissociative disorders (preponderance of women) and alcohol use disorders (preponderance of men) and on research indicating sex and age differences in adaptive response patterns of traumatized children (e.g. Perry et al., 1995; Chandy et al., 1996; Garnefski & Arends, 1998; Perry & Pollard, 1998), it is speculated that chemical dissociation in response to trauma is more common in men, whereas psychological dissociation as response to trauma is more common in women. Dissociation-related symptoms such as anxiety, somatic complaints, withdrawal, dissociation and disorders such as anxiety disorders, major depression, and dissociative disorders are more often found in females, whereas posttrauma hyperarousal-related symptoms (e.g. poor impulse control, aggression, hypervigilance) and disorders such as conduct disorder, attention deficit hyperactivity disorder (ADHD), and adult antisocial personality disorder are more often found in males (Perry et al., 1995; Perry & Pollard, 1998). Perry and colleagues argue that stress may cause those children with externalizing reactive response styles to manifest disturbances in different ways such as more heavy use of alcohol or other psychoactive substances, but not by developing a dissociative disorder. Gender differences in alcohol use among sexually traumatized adolescents have been reported, with boys exhibiting more (extreme) use of alcohol than girls (Chandy et al., 1996; Garnefski & Arends, 1998). Furthermore, alcohol use disorders are frequently associated with conduct problems or antisocial behavior (e.g. Watson et al., 1997; Kunitz et al., 1998), and in treated male alcoholics childhood conduct problems are frequently reported (Verheul, 1997). It has also been suggested that dissociative symptoms and antisocial behavior symptoms may be
different expressions of comparable etiological processes (e.g. Putnam, 1997). Because the neurobiology of hyperarousal is distinct from the neurobiology of dissociation (Perry et al., 1995), neuroimaging studies are needed to further examine gender differences in trauma response patterns.

Alternatively, low dissociation levels as according to the DES may have been due to psychometric weakness of the DES in an alcoholic population. In our study, this hypothesis could not be fully rejected. Therefore, more attention to the psychometric properties of the DES in alcoholic samples is warranted. Until further research with a diagnostic instrument for dissociative disorders - which may be more sensitive than the self-report DES - has been conducted (see Steinberg, 1997), our findings of low dissociation level should be viewed with caution. Further study of dissociative comorbidity in treated alcoholics using the Structured Clinical Interview for Dissociative Disorders (SCID-D) (Steinberg, 1994), an interview excluding systematically both intoxication as well as withdrawal effects of substances in assessment of dissociative symptoms is needed. The use of such a diagnostic tool may unravel whether some alcoholics misinterpret independent dissociative symptoms as substance-related, resulting in a low dissociation level. For example, alcoholic blackouts – which are accepted as a normal part of drinking – have been used by dissociative identity disorder patients as a socially acceptable excuse for or to explain away amnesic symptoms (Braun, 1984). However, usually “blackouts” appeared before these patients started to use alcohol. It should be noted, though, that even with a clinician administered instrument like the SCID-D, it may still be difficult to differentiate independent and substance-related dissociative symptoms in treated alcoholics, particularly in patients with a chronic history of alcoholism who may be unaware of their symptoms because dissociative behaviors may have been covered up by intoxication for long periods of time (see Kluft, 1985; Boon & Draijer, 1995).

For a further differentiation between the hypothesis of measurement problems of dissociation and the chemical dissociation hypothesis, this area of research may benefit from studies examining dissociation levels and a trauma-dissociation relation in individuals with alcohol use disorders in different treatment settings and in nonclinical samples. Such research may be very revealing exploring possible gender differences. For example, in alcoholics treated in a psychiatric hospital (including a larger number of females compared to our study), a
significant gender difference has been reported in dissociative symptoms, namely higher rates in women (Cornelius et al., 1995). Studies as such in large mixed-gender samples may also evaluate whether the amount or type of childhood trauma experienced matters regarding the reported level of dissociation. In our study female and male alcoholics did not experience similar amounts of overall childhood trauma. Consistent with many other studies in different populations (e.g. Nicolai, 1990; Windle et al., 1995; Draijer, 1996; Bensley et al., 2000; De Graaf et al., 2002), women were more likely to report childhood sexual abuse. Evidence for a link with dissociation is more consistent for sexual abuse than for physical abuse (see Putnam, 1997), a type of trauma more often reported by male subjects. In fact, gender difference in type of trauma is an alternative reason as to why in particular male alcoholics may report low dissociation levels. In addition, possible gender differences in dissociative phenomena and trauma-related symptoms could be studied (Gershuny & Thayer, 1999). Based on the typological model of pathological dissociation, evidence for a subgroup with low dissociative capacity would include a lack of particularly profound psychogenic amnesia in the face of significant childhood trauma (Waller et al., 1996). At present, the DES symptoms most frequently reported in male alcohol-dependent patients were included in the factor absorption/imaginative involvement (Wenzel et al., 1996); symptoms which are assumed to represent nonpathological types of dissociation (Boon & Draijer, 1993, 1995; Waller et al., 1996). Additional research in alcoholic samples may determine the frequency of dissociative symptoms on the three DES subscales (absorption/imaginative involvement, amnesia, depersonalization/derealization) to increase our understanding of low dissociation levels. Besides, the possible existence of two forms of PTSD with respect to (pathological) dissociation has been indicated in a sample of PTSD patients (Putnam et al., 1996). If alcoholic individuals (particularly males) with a PTSD diagnosis do not report having experienced flashbacks or psychogenic amnesia, but report other avoidance symptoms and profound hyperarousal symptoms, this would be consistent with low DES scores.

Obviously, dissociative comorbidity in substance abuse populations deserves more scientific attention. Although our study did indicate the absence of a trauma-dissociation relation, it should not be interpreted to imply that the role of childhood trauma in dissociation among treated alcoholics can be discounted. The conservative alpha level used in the analyses might have caused us to overlook meaningful findings (a relation between extrafamilial sexual abuse and dissociation), however, the risks of making Type I errors and interpreting
miniscule correlations seemed to justify this decision. Furthermore, in a clinical report it has been argued that individuals with dissociative disorders may be particularly prone to early relapse (Kolodner & Frances, 1993), therefore, the exclusion of early dropouts in our study may have contributed to an artificially low dissociation level. Studies including larger mixed-gender samples using both the DES and a diagnostic interview to assess dissociative symptoms are needed to see whether our findings are replicated. Neurobiological research is indicated to increase our understanding of (the relevance of childhood trauma for) dissociative comorbidity in alcoholics.

8.5 Methodological issues: internal and external validity of results

In the Chapters 2 through 7 several methodological limitations of the study have already been outlined such as the cross-sectional nature of the sample, the retrospective assessment of abuse histories, the correlational nature of findings, the reduction of statistical power due to the complex study design and limited sample size (particularly of women), as well as more specific methodological remarks regarding the study design and instruments used in particular parts of the study. Some further methodological remarks were made in the discussion of the major findings (sections 8.2 – 8.4). Here, the focus is on the internal and external validity of the results.

The use of a sample of alcoholic patients limits external validity, because treated subjects are not representative of all individuals with alcohol use disorders. Furthermore, the results in the present investigation may be specific to the center studied and, therefore, even generalizability to other settings or treatment programs may be somewhat limited. The treatment center in which the study was performed is, however, the sole treatment provider for alcohol use disorders in the regional health catchment area. Data were collected over a period of 8 months (September 1994 - May 1995) at the following alcohol treatment programs: the outpatient program provided by the Consultation service for Alcohol and Drugs (CAD); three inpatient programs for longer residence: the Vrouwenkliniek (women only, this clinic has a supra-regional function), the Heinzestraat (men only), and the Jacob Obrechtstraat (mixed-gender); and, the day-treatment program of the Vrouwenkliniek. Given the regional function of the center and the focus of the included alcohol programs, admission bias toward traumatized patients can be neglected. In addition, we found no indication for
previous treatment bias in reporting abuse: childhood abuse reports were unrelated to past treatment for alcohol problems. These findings are in line with those of a prospective study based on documented cases of childhood sexual abuse in women, showing that non-recall of the abuse two decades later was unrelated to past treatment for alcohol or drugs (Williams, 1994).

Furthermore, the primary addiction for which the patients were seeking treatment was alcohol. Compared to alcohol abuse/dependence, hard drug abuse/dependence is more strongly related to childhood sexual abuse (specifically of a severe nature) (Schäfer et al., 2000) and lifetime sexual and physical assault (Grice et al., 1995). In addition, higher rates of trauma-related distress (PTSD, dissociation) are generally reported in drug abusing samples compared to samples of alcoholics (e.g. Dunn et al., 1995; Grice et al., 1995). Hence, findings may not directly apply to persons with substance use disorders other than primarily alcohol problems.

All subjects were consecutively enrolled in the study according to strict inclusion and exclusion criteria. Although the use of such criteria can optimize internal validity, the results of the study cannot be assumed to apply to excluded patients, i.e. individuals who had severe cognitive impairments as, for example an alcohol amnesic disorder (Korsakoff’s syndrome) or dementia associated with alcoholism, and individuals who did not have sufficient command of the Dutch language. Since study inclusion required persons to remain in treatment for several weeks, a bias inherent in the methodology is the exclusion of early treatment dropouts. We do not know whether early treatment dropouts differed from study subjects in their reporting of childhood abuse histories or in having comorbid disorders. Because early treatment dropout may be related to childhood abuse (Palmer et al., 1995) or trauma-related distress (Kovach, 1986; Young, 1990; Kolodner & Frances, 1993), the results on the prevalence of childhood abuse and trauma-related distress of the present investigation may not apply to alcoholics who drop out of treatment prematurely. The question remains whether the relationships between these variables are fundamentally different in early dropouts compared to the patients in the study sample.

Finally, the question remains whether our sample was representative of the target population. Although our participation rate of 53% has to be considered, analysis of differences between non-participants and participants revealed no major selection bias in terms of socio-demographic variables, drinking history, ASI physical and sexual assault
reports, and problem severity in the ASI domains including for instance, severity ratings of alcohol problems and of psychiatric problems. Although participants and non-participants did not differ in lifetime assault rates, it is not clear whether they differed in childhood abuse prevalence rates. Since outpatients and patients born outside the Netherlands were underrepresented in the sample, results should be considered with some reservation.

8.6 Further research

Throughout this general discussion as well as in the discussions included in Chapters 2 through 7 a number of suggestions for future research have been made. Some important remaining issues are addressed in this section.

At present, there are a number of studies available on the prevalence of childhood physical and sexual abuse histories in non-veteran treatment-seeking female alcoholics. However, information on the prevalence of such histories in non-veteran male alcoholics in alcoholism treatment is sparse. Further studies need to be conducted to investigate the prevalence of childhood physical and sexual abuse in male alcoholics. In addition, our research indicates that histories of physical and sexual assault are far more prevalent in treatment-seeking alcoholics, especially in men, than are reported on the basis of information gathered with the most widely used standard instrument in clinical and research settings, i.e. the (Europ)ASI. Until the necessary modifications concerning the assault items included in this instrument have been made, researchers should attempt to include a more sensitive tool for assessing assault histories.

Our study does suggest a distinct pattern of psychiatric comorbidity associated with childhood abuse in treatment-seeking alcoholics. Further studies need to be conducted to investigate trauma-related psychiatric comorbidity, including personality disorders in inpatient as well as outpatient populations. Research as such may provide more information on the complexity of clinical outcome associated with early trauma, especially regarding the presence of complex PTSD, since one limitation of our study was the focus on Axis I symptoms. Future comorbidity studies in alcohol treatment samples should explore the relationship between PTSD and its many comorbid conditions, an issue not addressed in our study.
Other areas of interest for the study of the prevalence and relevance of childhood trauma for clinical impairment in individuals with an alcohol use disorder may be treatment facilities other than alcoholism treatment centers as for example, psychiatric hospitals and treatment settings or programs for childhood abuse survivors. Studies conducted in alcoholism treatment centers are generally limited due to a small number of female participants, a limitation that may be less pronounced in other treatment settings.

From a clinical perspective, it is important to conduct studies examining whether alcohol use disorders preceded by childhood trauma are more treatment-resistant as well as whether trauma-related distress in treatment-seeking alcoholics increases the vulnerability to relapse, as has been suggested in the clinical literature (Root, 1989; Bollerud, 1990; Young, 1990). At present, systematic studies on these issues in non-veteran samples of individuals who seek help for a primary alcohol problem are lacking.

Finally, to advance our current understanding of self-medication of trauma-related distress in alcoholics, prospective and longitudinal studies are needed.

### 8.7 Clinical implications

Our findings underscore the need for routine assessment of childhood trauma histories in women and men presenting for the treatment of alcohol use disorders, and for considering the information on such histories in diagnostic and treatment formulations. In the choice of measures for routine intake assessments and diagnostic evaluations attention should be paid to instruments for trauma assessment and for assessment of comorbid conditions, including PTSD. Posttraumatic stress disorder may be underdiagnosed, since standard psychological test batteries usually do not include measures regarding these symptoms (Carlson, 1997). At present, a range of inventories and interviews for trauma(-response) are available (see: Stamm, 1996; Briere, 1997; Carlson, 1997; Draijer, in press).

For patients with both alcohol dependence (or broader substance use disorders) and PTSD no systematic treatment approach of proven efficacy has been developed yet (see e.g. Triffleman, 1998; Jacobsen et al., 2001). It has been argued that existing treatments for men who typically experienced trauma other than (multiple) childhood abuse (i.e. war trauma or criminal victimization) may not apply to patients who experienced repetitive childhood physical and/or sexual abuse (Najavits et al., 1998b). Controlled treatment trials for these
comorbid patients are lacking, though there are some reports on case series or detailed clinical approaches (see Triffleman, 1998). Nevertheless, there seems to be a consensus in the literature that an integrated treatment in which substance abuse and PTSD are simultaneously addressed should be preferred (e.g. Brems & Johnson, 1997; Najavits et al., 1997, 1998b; Stewart et al., 1998; Brady et al., 2000). An integration of principles from specialized treatment approaches developed for populations of substance abusers and nonaddicted traumatized populations has been suggested (e.g. Najavits et al., 1998b; Triffleman, 1998). It is, however, acknowledged that some existing treatments for both disorders alone may not be indicated in comorbid cases such as for example flooding- or exposure-based interventions for PTSD due to an additional risk of relapse to substance abuse.

In line with principles in the clinical management of patients with complex PTSD or disorders of extreme stress (DESNOS) (see e.g. Chu, 1998; Van der Hart & Nijenhuis, 1999; Draijer, in press) the emphasis should preferably be on stabilization (management of acute intoxication/withdrawal and psychiatric symptoms) and reduction of symptoms. Significant decreases in PTSD and substance dependence symptoms have been reported in an uncontrolled treatment trial among 17 women with substance use disorders and PTSD or DESNOS based on group therapy with a psychoeducational format using largely cognitive and coping-skills-based interventions without exposure therapy (Najavits et al., 1996; 1998b). However, further work needs to be done regarding the impact of treatment of this population.

Finally, since childhood abuse, particularly early sexual victimization, may be a significant risk factor for subsequent problems with alcohol, including substance abuse prevention in treatment programs for young victims of childhood trauma is suggested. Alternative coping mechanisms might be provided to them.