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Literature review: connections between trauma and substance abuse

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Abstract

Over the last twenty years, extensive research in the areas of intimate partner violence (IPV), posttraumatic stress disorder (PTSD), and alcohol dependency has been conducted and recent research is exploring the relationship between PTSD and substance abuse. However, little research has examined the interrelationships among IPV and PTSD and alcohol dependence. In this chapter, I present an integrated review of literature, which lays the foundation for this book. Specifically, I summarize and critique the state of knowledge related to IPV, PTSD and alcohol dependence and the relationships between these phenomena by: a) defining each of these three constructs and reviewing theoretical and empirical literature that addresses the significance, prevalence, and current understanding of each, and, b) critically reviewing research in which the relationships between two or more of these constructs have been examined. This chapter concludes with a summary of current knowledge and identification of gaps that should be addressed in future research.

Search strategy

A search of CINAHL, Medline, PsycINFO, Proquest, and Cochrane data bases (1998-2008) was conducted using the key words of posttraumatic stress disorder (PTSD), violence, intimate partner violence (IPV), domestic violence, alcohol dependency, alcohol abuse, addiction, substance abuse, alcohol treatment, women and social support. The search was augmented by reviewing selected bibliographies with a focus on a combination of the keywords. Manual searches were done for classic references published prior to 1995. These papers were included if they were essential for understanding the concepts included in this review.

Given that most IPV is carried out on women [1] and alcohol studies have shown that alcohol reacts differently in female bodies than in male bodies [2], this review focuses on women. Although many individuals who are dependent on alcohol are also dependent on other psychoactive substances, this review focuses only on alcohol dependence to achieve clarity around both the biological and social factors related to alcohol dependence [3,4]

Prevalence and correlational studies [1,5-7] have documented the extent of IPV and association between IPV and PTSD. Dansky *et al.* found associations between IPV, PTSD and alcohol dependency [8]. However, there is little documentation or understanding of the mechanisms behind these relationships. There is some indication from the studies of alcohol dependence that PTSD plays a role in alcohol dependence but how this happens is not well explained. What are the interrelationships among these three factors and how do they influence the lives of women?

Intimate partner violence (IPV)

Definition, significance and prevalence

IPV refers to a pattern of physical, sexual and emotional violence by an intimate partner in the context of coercive control [7]. This violence may include hitting, pushing, name-calling, forced isolation from family and friends, criticism, non-consensual sexual activity or financial control [9]. IPV became part of the public discourse in the feminist movement during the 1960s and 70s when women began to speak openly about the barriers keeping them from having happy and productive lives. Over the last thirty years, major changes in the criminal justice system and the community support system have occurred [10]. Given this movement, it is surprising how secretive and stigmatizing IPV still is today. Researchers have made important contributions in understanding the prevalence, scope, nature and consequence of IPV. However, many other gaps in knowledge about this complex public health problem require in-depth exploration.

Until recently IPV has either not been recognized or was considered a private matter. In 1980, the Canadian Advisory Council on the Status of Women revealed, in the first national survey of spousal abuse, that 10% of Canadian women were victims of IPV. In 2004, the General Social Survey reported that 21% of Canadian women had been assaulted by an intimate partner within the previous five years. It is estimated that about two million American women experience IPV each year [11]. In a recent multi-country study, prevalence rates of IPV ranged from 15 to70% [12].

Although it is commonly believed that separation from the abusive partner ends the violence, 39% of female victims reported the abuse continued after separation [13]. Of those who reported post-separation abuse, 24% stated that the violence became more severe after leaving. Separated women were also at a greater risk of being murdered; with the risk of spousal homicides increasing after the woman had left her spouse (Hotton). Of the 846 Canadian women killed between 1991 and 2000 (80% of the total), 481 were killed by a current spouse, 185

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by an estranged spouse, 23 by a boyfriend and eight by the same sex partner. In 58% of all spouse homicides, there was a history of domestic violence [14]. At some point in their lives, nearly 25% of all women will be raped or physically assaulted by intimate partners [1,15]. More than 40% of the women who experience partner rapes and physical assaults sustain a physical injury [7]. As many as 324,000 women each year in the United States experience IPV during pregnancy [16].

In 1983, Federal and Provincial courts received a mandate to prosecute and charge all cases where there are reasonable and probable grounds of IPV. This led to the development of domestic violence courts, treatment programs and various task forces to study the issue. In 2000, a Federal Provincial task force was set up to review these policies and evaluate their effectiveness. The researchers had difficulty evaluating the effectiveness of the policies and interventions because of variation in the definition of an intimate partner, what constituted a violent act and methodological difference in sampling frames and survey instruments. The current prevalence of IPV and the dawning recognition of its consequences on society, has led researchers to search for a common definition and measurement tools for the field. These definitions are necessary for us to gain an understanding not only of the prevalence of IPV but also of associated risk factors [17]. Knowing the associated risk factors assists in theory building, and in the development of policy and programs.

The effects of violence can be devastating to a woman's mental and physical wellbeing including her reproductive health [18]. In addition to causing injury, violence increases women's long-term risk of many other health problems, including chronic pain, physical disability, drug and alcohol abuse, and depression [19-21]. Women with a history of physical or sexual abuse are also at increased risk for sexually transmitted infections, unintended pregnancy, and adverse pregnancy outcomes [18]. Three to 20 percent of women report abuse during pregnancy [21]. While researchers once thought that the prevalence of IPV increased during pregnancy [22], more recent research does not support the notion of an increase in number of assaults during pregnancy [23]. However, when it occurs, abuse during pregnancy presents significant health risks to both the mother and the unborn child Campbell *et al.*

To compound the health problems that women often experience, many victims of violence who seek care from health professionals often go inadequately treated [18]. These women have needs that health care providers often do not recognize, do not ask about, and do not know how to address (Parker *et al.*). In fact, health and social services providers are still debating whether they should or should not screen for this major health problem. Despite numerous recommendations for universal screening, most institutions do not want to mandate this practice as they are unprepared to handle disclosures [24] and there still insufficient evidence to show the benefits of universal screening in health care settings [15].

IPV is a social problem that has been associated with mental illness in general and with alcohol dependence [5,25]. The impact of IPV can be equal to being a hostage and subjected to torture [26,27]. Depression and PTSD have been identified as the most prevalent mental health disorders among survivors of IPV [28-30]. Exposure to life threatening situations increases the likelihood of PTSD [31,32].

Risk factors for of intimate partner violence

Many relational factors have been linked to an increased risk of IPV. These include: marital conflict, marital instability, and male dominance in the family, poor family functioning, emotional dependency and insecurity, belief in strict gender roles, desire for power and control in relationships, and exhibiting anger and hostility toward a partner [21,3334]. Although IPV occurs across all social, economic, religious and cultural groups, young women and those living below the poverty line are disproportionately affected [21]. Risk factors for IPV identified in the General Social Survey include Aboriginal heritage, youth (less than 24 years of age), alcohol dependence, living in a common-law relationship, having a physical or mental health impairment, or the presence of an emotionally controlling partner and trying to leave a relationship [14]. In addition, community risk factors for IPV include weak sanctions against domestic violence, strict gender roles and poverty, along with associated factors such as overcrowding, hopelessness, stress and frustration, and low social capital [1,21,35]. Individual factors associated with IPV include a history of physical abuse, prior injury from the same partner, having a verbally abusive partner, economic stress, partner history of alcohol or drug abuse, childhood abuse, and being under the age of twenty-four [7,36,37].

Theories of intimate partner violence

Various theoretical perspectives have been used to explain IPV. The first conceptual model developed by Walker [38,39] proposed three phases of abuse: tension building, the violent incident and honeymoon phase. Curnow identified a transition period between the end of phase two (the violent episode) and the beginning of phase three (honeymoon) where there was an *open window* for the women to receive help [18]. Numerous developmental theories, such as those proposed by Bowlby (1984) and Dutton (1998) have addressed some aspect of IPV and link abusive behaviors to an incident or incidents that disrupts the normal developmental process. Other major models explaining IPV are based on social learning theory and feminist theory. From a social learning perspective, violent behavior is learned; the experience of observing that relational violence is effective in resolving conflicts and in maintaining control over a partner reinforces that behavior [40]. As with other complex problems, single factor theories do not provide satisfactory explanations, as they provide simplistic ways of viewing complex phenomena. In fact, most researchers and counselors in this area utilize ecological and systemic models, which account for some of this complexity by considering multiple factors that influence IPV and women's responses to it in the context of their lives [41].

A feminist perspective maintains that IPV stems from the values of a patriarchal society that produce a culture of male domination [42,43]. Feminists view patriarchal systems as leading to the oppression and exploitation of women by men. They perceive the reluctance of society to take action to stop IPV and the unpaid work of women as symptomatic of this oppression [44,45]. Gender norms about the roles and responsibilities of men and women frequently provide justifications for violence. Men are to provide financially for the family, women are to tend to the house and children, and both women and children are to show obedience and respect to the man's view of what is appropriate behavior. If a man perceives that his wife has somehow failed in her role, or embarrassed him or his family and friends, he must correct her and he may act violently to do so. Consistent with this perspective, research on IPV has documented that men tend to use violence as a means of control [46,47].

In many cultures, men have the right to control their wives' behaviors, and, indeed are responsible for the behaviors of their wives; women who challenge this right *need* to be disciplined. In countries as different as India, Mexico, Bangladesh, Cambodia, Nigeria, Pakistan,

Tanzania, and Zimbabwe, the use of violence as discipline and the husband's responsibility to reform a wife who has not followed the expected customs and norms is common [1,48-51]. However, as prevalence surveys in both the United States and Canada show, despite the perception that western culture is very liberal, there are still roots of patriarchal beliefs that permeate our modern institutions and family structures.

Given that no single theoretical model is sufficient to fully explain IPV, a multiclausal, multifaceted model is needed [52-56]. Fortunately, research in the field has identified many relevant factors to help direct future theory development, while attending to women's experiences of abuse in context. For example, Coker *et al.* study of 191 women who had experienced IPV, tested whether the relationship between IPV and mental and physical health was mediated by social support. The conceptual model utilized in this study was based on a Stress

Process model, in which health status is negatively affected by chronic stressors and mediated by social support, perceived stress, coping and help seeking behaviors. Using structural equation modeling, they found that physical abuse was directly associated with poorer mental health and indirectly associated with poorer physical health. Higher levels of emotional support were found to moderate the impact of this abuse on health. The investigators suggested that improving social support available to abused women might improve their health. The sample size was close to minimum for this type of statistical analysis [57]. Current mental and physical health perceptions, anxiety, and PTSD were positively associated. The authors suggested that emotional support appears central to improved coping and. although social support may be hindered by alienation, this can be countered in ways that enhance psychological well-being and coping skills so that women can construct alternatives to abusive relationships.

A grounded theory study conducted by Wuest *et al.* [58] shines light on the factors producing stress and interference in the lives of mothers and children who have left abusive relationships. They identified *intrusion* as the central problem for these families which interferes with the ability of victims of IPV to maintain a consistent and harmonizing family life conducive for growth and development. Intrusion reflects real-life factors that intrude and take precedent in family life. These researchers did not specifically discuss the psychic intrusions from PTSD but considered chronic health problems, distressing symptoms and functional limitations to be aspects of intrusion.

Posttraumatic stress disorder (PTSD)

Definition, significance and prevalence

After experiencing a trauma, many people heal naturally over time. However, about one-third develop PTSD [59,60]. Results from the National Comorbidity Survey indicate that one in thirteen people in Canada will suffer from PTSD at some point in their life. In a sample of 83,000 military personnel, past year and lifetime rates of PTSD symptoms were 2.8% and 7.2%, respectively [61].

PTSD is a severe and complex disorder defined first as a syndrome in the DSM-III [62]. PTSD is comprised of three symptom clusters: 1) reexperiencing, including recurrent intrusive thoughts about the trauma, traumatic nightmares, and "flashbacks"; 2) numbing, including feelings of detachment from others, loss of interest in activities, and constricted affect; and 3) hyperarousal, including symptoms such as exaggerated startle, sleep disturbance, and memory impairment or trouble concentrating. Initially, a diagnosis of PTSD was linked to a single traumatizing and life-threatening event. However, there is a now recognition that an accumulation of many smaller negative events and stressors can cause PTSD, and this is called complex PTSD [63]. Many researchers now believe that this is the most appropriate lens to study

IPV since this type of trauma is most often a long-term pattern. In addition to the main symptoms of PTSD, there are related symptoms such as depression, anxiety, anger, guilt, chest pain, headaches, gastrointestinal problems and generalized pain. As with IPV, differences in the definitions and the scope of PTSD has resulted in variation in prevalence rates and results of studies in which associations between PTSD and other factors have been examined. The inclusion of PTSD in the DSM-III stimulated a tremendous amount of research and resulted in the founding of the International Society for Traumatic Stress Studies and the establishment of the Journal of Traumatic Stress in 1988 [64].

The symptoms of PTSD contribute to the development of other general health problems [65,66]. Growing awareness of the role that PTSD can play in other types of mental health problems and alcohol dependence as outlined in recent research [67], has prompted the study of the physiological impact of severe PTSD [68,69]. The development of PTSD begins with a precipitating traumatic or terrifying event. The event is usually life threatening, or at least can produce bodily harm, and it typically involves either interpersonal violence or massive disaster including, rape, assault, torture, terrorism, car or plane crashes, earthquake, tornado, or flood. Such a traumatic event has the potential to elicit intense and immediate feelings of fear, helplessness, and distress. These subjective responses frequently lead to adverse reactions that can lead to the symptoms of PTSD and resultant disability that is associated with this condition [70]. The consequences of the initial traumatic event extend far beyond the period when the person encounters it. At any time after the event, specific environmental cues that are reminiscent of the traumatic event may trigger a reoccurrence of symptoms, and anniversaries of the trauma are often difficult. Biological changes that occur in some individuals after a traumatic experience have been documented [31,71,63].

Theories of risk of posttraumatic stress disorder

Exposure to violence is a necessary but not always a sufficient cause of PTSD. There are many theories postulating why some people can cope with severe trauma without developing PTSD and why, even among those who develop PTSD, some have the symptoms for only a short time and others have the symptoms for a lifetime.

Factors proposed to increase risk of PTSD include previous trauma [72], genetic predisposition [73], and lack of social support after trauma [5]. There is some evidence to support each of these theories even though only one study has addressed genetic predisposition in a sample of 7,375 identical twins (Xian *et al.*). Although social support has been the subject of considerable research, there is mixed empirical support for its importance in preventing or reducing PTSD symptoms. Lack of clarity regarding the definition of social support, rather than the lack of importance of this trauma intervention [74,75], has led to this variation [76]. There is support for the role of previous violence is the development of PTSD. This makes intuitive sense given the theories underlying the development of complex PTSD [77,63]. More research is needed to identify which individuals remain more susceptible to PTSD after the first trauma and which interventions provide some protection against the impact of repeated trauma.

As a function of the complexity of this research, it would seem that the best results could be obtained from a multifactorial model. This

model could also provide guidelines for intervention because, once PTSD is established, it has a negative impact on the person's physical and mental health. These negative outcomes have been documented as irritable bowel syndrome, autoimmune disorder [65], habit disorders, substance abuse, eating disorders, phobias, and multiple personality disorders [31,69], some of which are considered to be the result of permanent changes to the brain [78,79].

Theories addressing mechanisms underlying PTSD

Biological theories: A classic study by Perry and colleagues (1995) [78] led the beginning of the investigation into the physiological impact of trauma based on, but extending, Hans Selye's Stress Adaptation Model. According to this model, after trauma, when people feel threatened, the arousal system is activated and, as the threat or trauma continues, the body releases endogenous opioids and a dissociative response is activated. This decreases arousal, heart rate, blood pressure and awareness of pain, despite increases in circulating epinephrine [80]. In acute stress situations, cortisol enhances survival by depressing the body's reaction to assault. After the trauma, feedback mechanisms return the heart rate, blood pressure and other physiological responses to normal. However, if the stress is severe and/or chronic, compensatory mechanisms can become overactivated and incapable of restoring the body to its previous state of equilibrium. Chronically traumatized persons will often be in a state of low-level fear, reflected in their bodies' physiology such as increased heart rate, muscle tone, and rate of respiration. According to Perry, the more intense and prolonged the traumatic event, the longer the activation of the stress response systems, and the more likely there will be a change in these neural systems [80]. Experimental increases in cortisol have shown initially to increase alertness, activity levels, and feelings of well-being; however, prolonged elevations produce withdrawal, dysphoria, and feelings of tiredness [81].

Various neurobiological studies of persons with PTSD have produced conflicting results related to the role of cortisol. In persons with PTSD, cortisol levels have been both elevated, [82], reduced, [83] or normal [84] when compared to persons without PTSD. Disturbances found in cortisol regulation and synthesis suggests glucocorticoid negative feedback is present in PTSD [70]. The implications of these observations are still not clear and further study is needed before conclusions can be drawn [85]. Research suggests that dysregulation and functional alterations in specific cortical and subcortical areas of the brain may underlie many of the symptoms of patients with PTSD [31,86].

Individuals with PTSD show evidence of increased autonomic reactivity [87]. This autonomic reactivity is thought to be a sensitized feedback mechanism. Repeated exposure to threatening stimuli, whether actual or with the reexperiencing symptoms, causes a pattern of repetitive neural activation [63]. The nervous system comes to anticipate the trauma, and soon the same neural activation can be elicited by decreasingly intense stimuli such that minor stressors can elicit full-blown hyperarousal or dissociation [80]. The brain organized around this internalization of the fear response resulting in behavior that may be consistent with symptoms associated with some psychiatric conditions [75]. O'Donnell *et al.* [88] suggest that alterations in the neuroanatomical structures and networks in the central and peripheral networks play a role in the initiation and the maintenance of the PTSD symptoms of hyperarousal and reexperiencing.

Studies of serotonin, a neurotransmitter, also provide some evidence of the biological roots of PTSD [69]. Serotonin is involved in functions such as hunger, aggression, sleep, and fear response. The neurons that produce serotonin have nuclei in the brain stem and extend to other parts of the central nervous system, including the amygdala, a small, almond-shaped portion of the brain that controls fear response. Low levels of serotonin create an anxiety reaction and may act on the amygdala in some way to help produce the symptoms of PTSD. Norepinephrine is a neurotransmitter responsible for long-term memory that acts on the hippocampus and it and cortisol are thought to play a role in this process [70]. There is early research in this area [75] but further research is needed to understand the roles of neurotransmitters, including both serotonin and norepinephrine in PTSD.

In the early days of recognizing PTSD, a *dose-response model* of PTSD was proposed in which it was suggested that symptoms become more severe with the intensity of the stressor (March 1993). Based on a Pavlovian fear-conditioning model [89] and research with laboratory rats [90], daily events are thought to trigger the fear response and become conditioned stimuli. There is some research support for this model [91]. For example, wounded veterans were two to three times more likely to have PTSD than are those who returned unharmed [92]. Furthermore, proximity to the epicenter of an earthquake predicted severity of PTSD symptoms [93]. Other studies do not support this model [94,95]. In addition, researchers usually rely on retrospective self-reports of the survivors themselves as the basis for measuring stressor intensity. In several studies, the survivor's present emotional status has been shown to affect how he or she remembers the traumatic experience [96-99]. The context of retrieval, including emotional state, affects how these recollections occur [100,101]. A critique of this model is that it fails to consider the range of emotions associated with the trauma encoded in the brain along with the memories. Thus, Pavlovian animal conditioning models that reduce trauma to its biological basis cannot capture this uniquely human aspect of trauma [64]. In sum, although there is evidence for the biological basis of PTSD, it does not address the full complexity of the condition.

Psychosocial theories: Although biological theories that explain the development of PTSD have received considerable attention, theoretical perspectives that address psychological and social factors that contribute to PTSD have also been proposed and tested. Asmundson *et al.* [102], using structural equation modeling, found that PTSD symptoms had a direct influence on mental health and an indirect influence on physical health through depression but not alcohol. Given the positive association of PTSD and alcohol dependence this result is surprising because of alcohol's negative effect on women's bodies; however, all of the research participants in this study were male and the impact of alcohol use on females is different [2].

Fontana *et al.* [74] study of 327 female military personnel also used structural equation modeling to test a model of the impact of stress related to military duty and stress related to sexual abuse and harassment on the development of PTSD. The model was a chronological one that included variables related to premilitary, military and postmilitary experience. During military service, 63% of the participants reported experiencing physical abuse and sexual harassment and 43% reported rape or attempted rape. While both duty-related and sexual stresses contributed separately and significantly to the development of PTSD, sexual stress was four times as influential in the development of PTSD as duty-related stress. Postmilitary social support significantly moderated relationship between sexual stress during military service and the development of PTSD. The significance of the impact of sexual stress supports the link between sexual abuse and harassment on PTSD

and. therefore, may make this study relevant to women who experience abuse from intimate partners. In addition, the impact of social support on PTSD symptoms may be relevant to this population and their use of alcohol use, as alcohol use has been shown to be higher in women who are isolated [2].

Many researchers have reported variation in the influence of social support on PTSD. Regehr *et al.* [76] used structural equation modeling to develop a framework for understanding the individual predictors of PTSD. They recognized that some negative aspects of social relationships might account for the conflicting results of previous studies on social support and PTSD and posited that some victims of PTSD might suffer from limited relational capacity, the ability to develop and sustain interpersonal relationships, which could limit their perception of social support. Results showed that persons who had poor relational capacity perceived less support from their network immediately after a traumatic event, and, six months later, the study participants had higher levels of depression and PTSD.

Although this study provided a focused look at an aspect of perceived social support, it did not consider broader social factors, such as stigma and racism, which may influence perceived social support.

Higher uses of alcohol have been noted in people with a history of trauma than in those without trauma, particularly those with PTSD [69,86,103-105]. The association between PTSD and alcohol dependence is clinically significant not only because of its frequency, but also because persons presenting with these two diagnoses are more difficult to treat than are persons with alcohol dependence alone [72,106]

Alcohol dependency

Definition, significance and prevalence

People use psychoactive substances for a variety of reasons. For most people, alcohol use in itself is not problematic, yet alcohol dependence is still a major public health problem worldwide that results in high economic costs to society [107]. The Global Burden of Disease Project estimated alcohol use to be responsible for 3.5% of all deaths [108]. This burden includes physical disorders and injuries attributable to alcohol [108]. Alcohol dependence was a factor in 6,701 deaths in this country in 1992, including 2,372 in Ontario alone [109], where the percentage of the population who report drinking is highest at 79%. The recent Canadian Addiction Survey [110] of 13,909 persons over the age of 15 showed no decrease in alcohol use since 1996. Importantly, 22.4% of Canadians were found to exceed the drinking guidelines, 9.1% indicated causing harm to themselves because of alcohol use, and 35.4% reported suffering harm because of someone else's drinking. Seventeen percent were classified as dependent on alcohol based on the Alcohol Use Disorder Identification Test (AUDIT), 8.9% of whom were women. One in every ten Canadians reported that drinking was responsible for family and marriage problems [110]. Canadian researchers suggest that two-thirds of women with alcohol dependence have mental health problems that include PTSD and many of these women are survivors of IPV [111]

Alcohol dependence refers to a behavioral pattern of alcohol use characterized by compulsion, preoccupation and relapse [112]. The description of dependence syndrome (Edwards & Gross, 1976) [113] was a significant step in the evolving concept of dependence. This syndrome consists of a cluster of cognitive, behavioral and physiological phenomena and these elements have been incorporated in the internationally-used criteria for diagnosis of alcohol dependence in both the DSM-IV [68] and ICD 10 systems. [107]

The key features of alcohol dependence are impaired control, preoccupation, relapse, tolerance and withdrawal symptoms [114]. Impaired control, a sign of compulsion, is the tendency to drink larger amounts over a longer period than planned and the inability to predict consumption [114]. Preoccupation is the attention focused on acquiring and drinking alcohol, where it occupies a central role in life and other activities are diminished [114]. Relapse is unsuccessful efforts to cut back, with recurrent inability to control consumption [114]. Alcohol use becomes more important than the problems it is causing, and its use continues, despite the problems it creates. Physical dependence can also consist of tolerance and a withdrawal syndrome. Tolerance refers to the increased need to consume more alcohol to achieve the desired effect [114]. Withdrawal symptoms vary according to the level of physical dependency and the previous numbers of detoxification but may include the following: hyperarousal, tremor, tachycardia, anxiety, fever, anorexia, diaphoresis, disorientation, hallucinations, and seizures [115]. It has been argued that a heavy reliance on the presence of tolerance and withdrawal symptoms may lead to a failure to diagnose serious dependence [116-120]. Therefore, clinicians should not rely too heavily on the presence of tolerance and withdrawal symptoms before they make the diagnosis of physical dependence. Peele views dependence as a social experience, in that people become addicted to a state of mind and body, and, even at the extremes, these people can act in other than addicted ways. Once dependence is established, continued drinking is associated with significant recurring problems [121].

Developing alcohol dependence after repeated use is not usually the intent of the individual, but the outcome of a complex combination of genetic, physiological, psychological, cultural, and environment factors. It is very difficult to distinguish exactly when a person becomes dependent on a substance as there is a continuum of use from first contact with the drug to a state of addiction. However, once a person becomes dependent, unless intervention occurs, the level of dependence typically escalates toward severe dependence with physical, mental and socioeconomic consequences [110]. Effective recovery programs and barriers to access are important areas of study [110].

Theories of alcohol dependency

Many theories regarding for alcohol dependence have been developed. However, few theories can provide an adequate explanation as to why dependency occurs [122]. The Moral Model was the first North America theory and still has a strong influence today. This model attributes the use of alcohol to personal choice, with dependence resulting from weak moral character. In this model, character building, personal will power and removal of temptations overcome dependency [117].

In the 1930's, the first biological theory, the disease model, gained prominence through support from the lay movement, Alcoholics Anonymous (AA). A radical departure from the moral model, in the disease model, the individual is viewed as ill rather than of weak character. This approach reduced some of the stigma associated with alcohol dependence and started the first substantive humanitarian movement in the treatment of alcohol dependent individuals. Recent advances in genetic studies have given more weight to biological theories, especially the Collaborative Study on the Genetics of Alcoholism (COGA), an innovative, multidisciplinary program launched by the National Institute on Alcohol Abuse and Alcoholism. This study involved nine research centers across the United States collecting DNA samples, questionnaires, electrophysiological measurements, and other data obtained from nearly 3,000 people from families with at least three alcohol dependent members. The analyses from this study identified

several chromosomal regions, particularly on chromosomes 1 and 4 that appear to be linked to several alcohol-related phenotypes, which are now the targets of more detailed analyses [123-125]

Recent research regarding alcohol cravings has provided new evidence of the biological bases of alcohol dependence. Neurobiological and brain-imaging studies have identified brain chemicals and regions [126] and neurotransmitter pathways involved in cravings [6]. The brain, seeking homeostasis, adapts to the prolonged or excessive presence of alcohol by making changes in certain brain cells, called neuroadaptation. This neuroadaptation is thought to occur more quickly in persons who are at risk of alcohol dependence because of an inherited vulnerability, or severe and repeated stresses such as trauma [127]. Differences in the brains of dependent and nondependent persons have been identified, some of which are permanent and others that disappear sometime after cessation of alcohol use [6]. Once these changes take place in the brain mechanism, it has been proposed that a process of classical conditioning is responsible for the cravings [126]. Understanding these occurrences may help with relapse prevention in dependent persons. This is especially so if there are other neuroadaptive patterns taking place, such as in PTSD [78,79].

In addition to classical conditioning, social learning theory, personality and rational theory have been used to understand alcohol dependence. Other researchers [128,129] have combined social learning and psychological theories to examine how individuals learn, modify and reinforce their own behavior, and what psychological characteristics predispose them to alcohol dependence. From a social learning theory perspective, people tend to repeat behaviors that bring reward and discontinue behaviors that bring punishment [40]. However, persons with alcohol dependence continue to drink even in the face of very negative consequences; it is posited that negative attention is a strong reinforcement in the absence of positive attention. In addition, most persons with alcohol dependence have very strong positive feelings associated with their initial intake of alcohol, which, in their memory, remains a positive reinforcement. Understanding reinforcement is a critical issue in dependency [130].

Personality theories focus on the role of an alcoholic personality in developing alcohol dependency. Although AA supports this belief, there is little empirical evidence to support this theory. Miller [130] demonstrated that persons with alcohol dependence have as broad a range of personalities as do non-alcoholic persons. In contrast, rational theory suggests that most people begin to use alcohol without realizing the potential harm. Individuals with alcohol dependence suffer from cognitive impairment and develop destructive thinking patterns. Empirical research supports this belief and most health promotion and treatment programs have a strong educational basis, which not only educates people about the impact of alcohol on the body but also identifies destructive thinking patterns and teaches skills to help change these thinking patterns.

Wide acceptance of biological and psychological theories along with the inability to fully move away from the Moral Model has prevented alcohol dependence from being seen as a social problem. Sociological theories allow alcohol dependency to be examined within the context of the entire society [131,132]. These theories, which include Marxist theory, alienation theory and availability control theory, acknowledge that alcohol use does not occur in a vacuum but is influenced by sociocultural factors, including cultural norms, ceremonies, politics, media and the beliefs about alcohol in our society. These theories move us beyond the individual to examine the purpose that alcohol use and dependency serves in our society and how social and cultural context may contribute to the use of alcohol. There are also subculture theories that focus on the role of broader environmental determinants in whether people from certain groups use alcohol in excess or not. In these theories, the culture must permit heavy drinking at least occasionally before the individual is able to become dependent on alcohol. These theories take the full responsibility off the individual and focus attention on external environmental factors.

Women and alcohol

Alcohol dependence poses special concerns for women. A complex blend of biological and psychosocial factors affects female alcohol use. Women are more susceptible to the physiological consequences of alcohol use because male and female bodies respond differently to alcohol [2,133]. These responses are explained by the fact that alcohol is more soluble in water than in fat. Women's bodies contain more fatty tissue proportionately than do men's bodies so that the same quantity of alcohol consumed by both a man and a woman of equal size will result in a higher blood alcohol level for the woman. In addition, women produce less alcohol dehydrogenase, the stomach enzyme that breaks down alcohol, and, therefore, break down less alcohol in the digestive process, leading to higher blood alcohol concentrations than men. Consequently, women are more susceptible to liver disease and other alcohol-related diseases [2].

The psychosocial realities of women who are dependent on alcohol also differ significantly from those of men with the same level of dependency, and this is especially true for women with children. Women suffer special social and emotional consequences for drinking, including discrimination due to sex-biased attitudes [131], social stigma, and double standards. In addition to gender-based oppression that exists in numerous cultures, a relational model makes the impact of this stigma more fully understood. Research evidence supports the premise that a woman's development and experiences throughout her lifetime are structured around relationships with others [130,134]. Relationships are highly valued and it is within this context that societal attitudes toward women's use of alcohol and women's views of themselves are formed [135]. Therefore, if society devalues women with alcohol dependence, these women may also think less of themselves.

Recovery from alcohol dependency

Although 85% of persons who are dependent on alcohol in Canada do not seek formal treatment [136], not these people remain dependent on alcohol. Some research has examined natural recovery from alcohol dependence. Bischof *et al.* [137] utilized cluster analysis to identify subgroups of natural remitters to build a conceptual framework for understanding the process of natural recovery. Common success factors identified in three subgroups included having few social problems and having a high degree of social support. This research is important because it highlights the importance of social support and provides information that could be useful in improving the low success rates of alcohol treatment programs. Studies on the impact of successive detoxifications have shown that each session of detoxification increases the possibility of seizures and anxiety responses. In addition, successive detoxification has been found to increase obsessivecompulsive cravings in one study [138], although, this finding was not supported in another study [139]. These studies support the need for more precise understanding of the factors that contribute to a person's ability to move from dependency to recovery.

Lack of social support is a risk factor for relapse [89]. It is unclear, however, whether a lack of social support slows recovery from the

acute stress symptoms, if these symptoms alienate possible sources of social support, or if these factors influence each other to affect recovery. Social support has been found to positively influence women's progress toward regaining health [67,76,137]. In addition, an unstable family environment during childhood [140], preexisting mood or anxiety disorder, and a family history of anxiety or mood-disorder are related to poorer outcomes for recovery [141,142].

None of these theories fully explains alcohol dependence. This supports the growing recognition of the need for a broad theoretical lens [122] to answer the questions that arise in attempting to prevent and treat this complex public health problem. Vellman [143] proposes such a theory that considers the role of internal factors such as vulnerability or predisposition (biology, genetics, personality, and socialization) and external factors (culture, politics, economics and advertising) in alcohol dependency.

Alcohol treatment for women

Substance abuse treatment for women received little attention until the 1970s when research on the different impact that alcohol had on women's bodies became prominent [144]. This increase in research also highlighted other factors in women's lives that are important in treatment for alcohol dependence, including access, child care, transportation and financial resources [144]. Researchers report that, despite recent research, women do not have access to a broad range of treatment services, the coordination of care is lacking and screening for women's suitability for treatment is frequently not carried out.

Harm reduction *versus* abstinence

The most significant debate in alcohol treatment is the controversy between a harm reduction model and complete abstinence, an indication of which is that several sessions of the American Academy of Addiction Psychiatry conference in 2006 were focused on this debate. Harm reduction is a philosophy of care that has widespread empirical support [145] and is based on the belief that treatment must start from the client's needs and personal goals. Therefore, any change that reduces the harms associated with substance use can be regarded as valuable. Harm reduction first emerged as a public health strategic for active substance users who were unable or unwilling to stop using (Marlatt). Harm reduction embraces the full range of harm-reducing goals, including, but not limited to, abstinence. Many people who use alcohol do not wish to stop and some are unable or unwilling to embrace abstinence. There is evidence that people may avoid seeking help altogether because they do not have life-long abstinence as their objective [146]. However, many treatment centers offer only abstinence-based programs. By accepting goals other than abstinence as reasonable starting places for treatment, harm reduction opens the door to this group of people in a way that traditional abstinence-oriented approaches cannot.

The tension between an abstinence model of care and one that focuses on harm reduction makes it difficult to compare research findings across studies but also contributes to lack of continuity in treatment provision [146]. People with alcohol dependence vary widely in the severity of substance use, personal goals regarding substance use (i.e., safer methods of using, moderation, or abstinence), motivation and stage of readiness to change [147], emotional and mental health status [148], personality strengths and vulnerabilities [149]and socioeconomic variables. This range of issues often works against abstinence, possibly explaining why an abstinence-only, one-sizefits-all approach runs the risk of failure with many clients. A key challenge is determining who may need programs emphasizing complete abstinence and who could benefit from controlled drinking, yet a better understanding of pathways to recovery is needed in inform these decisions. The inability to determine the best treatment for each person may contribute to the low success rate of treatment programs. Fueling the controversy in alcohol treatment is the lack of understanding of both philosophies at the point of care, such that advocates are often divided on the issue and perceive that those who hold opposing views disregard the other perspective.

Studies of alcohol treatment

There are numerous research studies on the characteristics of persons with alcohol dependence, but too few outcome studies addressing the impact of treatment. In a comprehensive analysis of research on alcohol treatment, Yalisove [114] identified a number of limitations and challenges in this body of work: (a) lack of a control group makes interpretation of results difficult because persons with alcohol dependence sometimes go into remission without treatment, (b) the heterogeneity of persons with alcohol dependence, and the resulting samples recruited for treatment studies, makes generalization difficult, (c) lack of consistency in identifying and measuring outcomes, including the length of time after treatment that outcome measures should be administered, (d) no agreement on how dropout rates should be calculated or how to handle discrepancies between laboratory results of blood or urine testing and selfreports of abstinence, (e) whether success should be based on abstinence rates or a reduction in consumption, and, (f) no standard measure for reduction in use. Yalisove [114] suggested that clinical trials should employ more specific standardized criteria.

Identifying the types of outcome domains required for adequately determining alcohol treatment outcomes has also created debate, with researchers focusing primarily on alcohol consumption outcomes [150]. However, there is growing support for the importance of assessing both alcohol consumption and alcohol-related problems. Furthermore, alcohol treatment researchers have begun to address other non-alcohol-specific measurement domains, including psychosocial functioning and quality of life [151]. The rationale is that using multiple measures to assess multiple outcome domains leads to a more complete assessment of treatment outcomes. This perspective is important as individuals report that many factors affect relapse [114].

The Rand (1978) report was the first large study that followed 1,340 persons with alcohol dependence treated in eight residential and follow-up programs for 18 months (completion rate of 62%) and compared them to a with a group of 400 persons who attended AA and had other forms of treatment. At 18 months, the clients who attended treatment programs had a remission rate of 67% compared to 54% for the comparison group. In the remission group, 27% of participants were drinking asymptomatically and another third had drunk during the 18 months but not during the last 6 months. Eighty five percent of the original participants were interviewed four years later and 28% of the treatment program group were still abstaining, with 22% of these having abstained for one year or more, 8% drinking low quantities with no problem, and another 10% drinking moderately without symptoms (Yalisove) [114]. In the comparison group, there was a 20% improvement (Yalisove) [114]. The results of this study suggest that professional treatment program may be more effective than self-initiate peer support groups (AA) in reducing alcohol consumption.

In contrast to the focus on professional versus lay model of care, the MATCH study (1997) compared three types of professional treatment for alcohol dependence: Twelve Step Facilitation, Cognitive Behavioral

Treatment and Motivational Enhancement Treatment. Five outpatients and five aftercare programs were used to recruit participants (N=1,726) who were randomly assigned to each one of the treatment conditions. Treatment duration was 12 weeks and efforts were made to ensure program integrity. One year after treatment ended, interviews were conducted with 90% of the participants and 75% of their relatives assess amount of alcohol intake and measure blood alcohol using laboratory sampling. Results indicated that 35% of outpatient and 19% of aftercare clients were abstinent one year after completing treatment. Additionally, 12.4% of outpatient and 7.3% of aftercare were drinking safely. There were no significant differences between treatment groups. Lack of a control group prevents any conclusion about whether these treatments were better than no treatment. Three year outcomes were obtained from 84.7% of the sample with similar results [114].

Miller *et al.* [152] conducted a meta-analysis of seven multisite studies that tested interventions for alcohol dependence, including the Rand report and the MATCH study. The analysis showed that 24% of patients in outpatient treatment maintained abstinence and over 11% of maintained low level drinking without symptoms for more than a year after completing the treatment. In summary, there is some evidence that persons with alcohol dependence improve after treatment, although these improvements are modest and the quality of evidence is limited by the lack of a true control group (no treatment). No single treatment has been superior to other treatments [152]. Furthermore, a focus on outcomes to the exclusion of what happens during treatment makes if difficult to know which elements of treatment are responsible for positive effects [114].

Clinicians and researchers studying the barriers to alcohol treatment advocate the need for a flexible, inclusive, and comprehensive treatment model that addresses a myriad of needs, fits with this diverse group of people, and is open to a variety of treatment goals aside from abstinence [153]. Within such a model, treatments could be matched to the unique needs of the individual to maximize overall success. Denning (2000) suggests that interventions be matched to the client's current stage of change in relation to a specific behavior. Tatarsky [154] argued that many of the issues commonly associated with substance use problems, such as early trauma and mental health disorders, are signs that clients may want to self-medicate. He proposes that these issues be must be identified and addressed before it is possible to consider modifying substance use patterns. However, we know that many alcohol treatment centers do not make resolution of trauma, or other underlying issues, part of their plan of care, despite knowing that this factor makes it more difficult to maintain abstinence.

Before Najavits *et al.* [72] conducted their landmark study, little research had connected PTSD with substance abuse. After this initial connection, Najavits developed the program, *Seeking Safety*. Seeking Safety is a manual-based, integrated program that establishes safety as the most urgent clinical need for women with PTSD and substance abuse problems and teaches coping skills to work toward discontinuing use, letting go of dangerous relationships, gaining control over PTSD symptoms and asking for help [155]. This program is the most empirically studied treatment thus far for trauma, PTSD and substance abuse. Positive treatment outcomes, including decreases in substance use, PTSD symptoms, suicidal thoughts and risk, and depression and increased social adjustment, family functioning and problem solving, have been documented in seven studies, including two randomized control trials. To assist in comparison of findings across studies, Najavits not only documented the details of her program and the underlying philosophy [156] but clearly documented her research methodology so that others could replicate it [157].

Relationships among IPV, PTSD and alcohol dependence

The association between IPV, PTSD and alcohol dependence has been the subject of several studies, although few studies have considered all three constructs. For a disorder to be a risk factor for alcohol dependence, its onset must precede that of the alcohol dependence. The ideal method to determine the order of onset of two disorders is to use a prospective, longitudinal design in which healthy persons are followed over time and monitored for the development of the disorders under investigation. However, these studies are difficult and costly. Consequently, most studies of the relationships between IPV, PTSD and alcohol dependence are retrospective and cross-sectional.

Intimate partner violence and post-traumatic stress disorder

The Trauma Recovery Project was a large, prospective epidemiologic study designed to examine multiple outcomes of major trauma, including quality of life and PTSD [158]. This study enrolled 1,048 eligible patients triaged at four trauma centers who were age 18 years and older, had admission Glasgow Coma Scale score of 12 or greater, and who stayed 24 hours or longer in the center. A well-being scale was used to measure quality of life after injury; patient outcomes were assessed at discharge and at six-month time intervals. PTSD was diagnosed in 32% of patients 6 months post treatment. PTSD was more frequent in women (39%) than in men (29%) and more frequent in younger, low-income patients. Perceived threat to life predicted PTSD onset. This study raises the question of why women, why younger and why lower income persons were more affected. Perhaps access to social support may partially explain these findings if these groups of women had lower levels of social support and resources to draw on in recovering from trauma. Although this study dealt with trauma in general, it offers some insight into IPV since many women who leave abusive partnerships often perceive a threat to their lives. It also provided some evidence that more disadvantaged women who have less access to social support and resources may be at greater risk of PTSD. Alcohol use was not studied in this project, nor was any other maladaptive coping mechanism.

The prevalence of PTSD in women who have experienced IPV has been estimated in several studies. Both Stein and Woods [159,160] found that 50% of women who had experienced IPV had symptoms of PTSD. In the latter study, women had been out of the relationship for 6 to 9 years. These studies included relatively small, convenience samples of women; however, Golding's [28] metaanalysis showed that between 31% and 84% (weighted mean 63.8%) of female survivors of IPV women who experience IPV also experience PTSD. Most IPV research has been cross-sectional, yielding information about the emotional and mental health effects of exposure to trauma. However, little is known about how physiological factors evolve over time or how they might influence health. In an ongoing longitudinal study being carried with a community sample of adult Canadian women who have recently left an abusive partner, 51.6% of women has symptoms of PTSD, yet only 7.1% had a formal diagnosis of PTSD [161]. This demonstrates not only a high prevalence of PTSD but also a lack of recognition of it within health care settings.

Intimate partner violence and alcohol dependency

Research on IPV and alcohol use has frequently focused on the role that alcohol use plays in the escalation of violence [162,163]. Results of a few studies suggest the survivors of IPV are more likely to have alcohol dependence [162-165]. Often overlooked is the issue that women with IPV and alcohol dependence who seek treatment for alcohol dependence have difficulty benefiting from present alcohol

treatment programs [166,167]. In two studies [168,169], 50% to 65% of women seeking treatment for alcohol dependence were victims of IPV in the past year. Although several other studies have shown similar prevalence rates [20,42,170], little research has been conducted to enhance our understanding of why these women are so vulnerable. Testa *et al.* [171] did not find an association between heavy episodic drinking and subsequent victimization at the hands of an intimate partner. Furthermore, little is known about other roles that alcohol use might play in a survivor's experiences of IPV, and the impact, if any, that alcohol dependence might have on the presence and severity of depressive symptoms in abused women [172].

Chase *et al.* [167] studied 103 women who were seeking treatment for alcohol dependence and who reported experiencing IPV in the previous year. This study did not demonstrate the expected findings of a greater history of childhood trauma than the general population or higher levels of stress or marital conflict. However, all the participants in this study were partnered, abstaining from alcohol, receiving marital therapy and engaged in ongoing research. Although social support was not specifically addressed in this study, it points to the importance of social support in recovery from alcohol dependence. Results of other studies [42,160,173] suggest that the relationship between IPV and alcohol dependence is complex. We do not fully understand the role that alcohol plays in the lives of women who have experienced IPV; however, given the review of research, we can hypothesis that women may use alcohol to self-medicate after an IPV incident or to regulate PTSD symptoms. In addition, we know that reasons for using alcohol may change over time and, given what we understand about women's physiological response to alcohol and trauma, we can surmise that some women will become alcohol dependent.

Regrettably, in many alcohol treatment settings, trauma is not investigated or treated, and clinical staff members are generally not requested to assess for PTSD [174-176].

Post-traumatic stress disorder and alcohol dependency

Although there is no single theory that relates trauma to alcohol dependency, higher uses of alcohol have been noted for people with a history of trauma than those without trauma, particularly those with PTSD [31,69,103-105]. These histories of trauma can include physical or sexual assault [164].

One theory regarding the role of PTSD and traumatic life events as risk factors for alcohol dependence posits that the person uses alcohol to cope with the traumatic experiences [177]. Results of Clark *et al.* [178] study of adolescents with alcohol dependence provides support for this theory. In this study, adolescents with alcohol dependence were 6 to 12 times more likely to have a history of physical abuse and 18 to 21 times more likely to have a history of sexual abuse compared with adolescents in the control group. These findings suggest that the relationship of PTSD to alcohol dependence in adolescents might be gender-specific because the association between these factors was stronger in females than in males [178]. High rates of co-morbidity between PTSD and alcohol dependence in women have been documented in several studies [106, 175,179]. Women's high risk of comorbid PTSD and alcohol dependence may be related to a higher incidence of childhood physical and sexual abuse in women than in men [180]. In comparison to women with alcohol dependence or PTSD alone, those with PTSD and alcohol dependence were found to present with a more severe clinical profile and experience poorer treatment outcomes [181].

The comorbidity problems associated with PTSD are extensive. In the National Comorbidity Survey, Kessler *et al.* [60] found that approximately 80% of patients with PTSD met the criteria for at least one other DSM disorder. In another study, Breslau *et al.* [141] found 3036% persons with PTSD had a lifetime prevalence of major depressive disorder, 15% percent had obsessivecompulsive disorder, and 10-13% had a panic disorder. Comorbidity between PTSD and depression is associated with greater symptom severity and higher risk of suicidal behaviors [6]. Importantly, when the comorbid disorders are identified and treated, the intensity of PTSD symptoms usually decreases [182]. Perhaps the decrease in symptom severity is due to the social support the participants receive as they were being treated for the other conditions.

There has been substantial study of alcohol dependence in patients with PTSD [85], especially studies focused on the pathophysiology and clinical presentation of PTSD. In women, depression and other anxiety disorders are most common, followed by alcohol dependence [60,92]. Persons with PTSD and alcohol dependence have higher rates of other psychological and physical problems. This comorbidity makes it increasingly difficult to develop a model of the relationship between PTSD and alcohol dependence. Two pathways have been suggested to explain the high correlation between PTSD and alcohol dependence. The first one suggests that the alcohol dependence precedes the PTSD and that the subsequent impairment in judgment leads to high risk behaviors that lead to trauma [183]. The second pathway has PTSD preceding alcohol dependence, with alcohol used as a form of self-medication [177]. This hypothesis is supported by research in which central nervous system depressants have been linked to improvements in PTSD symptoms and by reports of an escalation of alcohol use with PTSD symptoms [177].

Symptoms of PTSD, reexperiencing, hyperarousal and numbing are signs of central nervous system stimulation. Alcohol being a central nervous system depressant, calms stimulation. If the person experiences release from the PTSD symptoms, increased use of alcohol is likely and overuse may occur. Overuse of alcohol produces withdrawal symptoms when the alcohol blood level drops in the body. One of the prominent signs of alcohol withdrawal is hyperarousal because of the central nervous system is stimulated. This hyperarousal can trigger reexperiencing and even greater hyperarousal, which can lead to alcohol consumption and if the person is try to abstain, relapse. A study of 1,007 adults documented that PTSD preceded and increased the incidence of alcohol dependence and not vice versa [184]. This may make detoxification and recovery more difficult [185]. Researchers suggest that persons with PTSD and alcohol dependence may find the physiological arousal resulting from substance withdrawal extremely difficult or impossible to cope with, and inpatient admission with pharmacological support may be necessary. However, little research has examined the outcomes of these detoxification procedures [186,187]. Because of the evidence of noradrenergic dysregulation in PTSD and alcohol withdrawal, clinical trials of different medication during detoxification are recommended [85,106,188].

Persons with alcohol dependence and PTSD are more difficult to treat because they are more impaired and have more social problems [106]. These individuals are reported to be challenging, have difficulty maintaining relationships with therapists and treatment staff, and evoke strong negative emotional responses from therapists [72,106,179,189-193]. In response to this research, Najavits *et al.* [155] has developed a special program for women with PTSD and alcohol dependence called Seeking Safety (www.seekingsafety.org). This program, which calls for both trauma and substance abuse to be treated concurrently, is being evaluated in different client populations. Najavits has provided

guidelines for evaluation so that future meta-analyses will be possible [155].

Najavits has made significant contributions to both research and practice in this area. One study [72] compared a group of dually diagnosed women (PTSD and alcohol dependence) to a single diagnosis group of women with either PTSD or alcohol dependence. Women in the dual diagnosis group were found to have more severe clinical profiles that those in the single diagnosis groups as well higher levels of PTSD symptoms, substance use, general symptomatology, and suicidal ideation; poorer coping; and more substantial personal histories of trauma and family histories of alcohol dependence. The high lifetime rate of suicide attempts (78.6%) was of major concern.

Research examining treatment for women with co-occurring PTSD symptoms and alcohol dependence is in the initial stages. Clinical trials comparing the results of different concurrent treatment programs need to be completed to examine the long-held belief that once alcohol dependence has been treated, other problems with be easier to resolve. Additional research, which seeks to understand the complex interrelationship between PTSD and alcohol dependence, is needed as a foundation for such studies.

Interrelationships among intimate partner violence, post-traumatic stress disorder and alcohol dependency

Few studies have examined the interrelationship among IPV, PTSD and alcohol dependence. In a retrospective, correlational study, Dansky *et al.* [170] examined the associations among IPV, PTSD and substance abuse in a study of 39 participants who were being treated for alcohol or cocaine dependence. Female and male subjects did not differ significantly by race, age, marital status, education, or employment. Eighty-five percent of the participants had experienced IPV and indicated that they feared death or serious harm. Approximately 25% of the individuals met the diagnostic criteria for PTSD. Consistent with previous research, participants who had experienced IPV were four times more likely to meet criteria for PTSD, and twice as likely to meet criteria for lifetime PTSD as those who had no history of IPV. These findings provide further evidence that IPV can increase the risk of PTSD. However, research addressing the interrelationships between IPV, PTSD and alcohol dependence has focused primarily in only two areas, biological and sociological.

Social dimensions of the relationships among IPV, PTSD and alcohol dependence

The experience of IPV tends to happen in situations of social isolation and abusive partners tend to increase that isolation as part of coercive control [7]. Even after leaving an abusive relationship, social support can be limited because of stigma, decreased financial resources, impaired relationship ability, discrimination and lack of appropriate resources [15,36]. IPV is a key context in which trauma occurs and trauma is a necessary condition for PTSD [104]. Social support is reported to reduce the occurrence of PTSD and reduce the symptoms of PTSD [77]. It has been hypothesized that PTSD sufferers self-medicate with alcohol (Dube *et al.*) [104] but that this is less likely to happen if the person has access to a confidante [5,137]. Women who have alcohol dependence tend to isolate themselves socially and research shows that they have increased PTSD symptoms. Social support reduces alcohol dependence and the incidence of relapse [89,137].

Social support is a complex phenomenon that has many dimensions that are influenced by internal and external factors. Examples of internal factors are: previously experience, PTSD symptoms, and self-esteem. Some examples of external factors are: financial resources, prejudice, and social problems [5]. Therefore, it is important to study how support is offered and perceived and the barriers to access.

Social stigma has an impact on social support and IPV, PTSD and alcohol dependence are each associated with social stigma [37,194,195]. Stigma reduces an individual's likelihood of reaching out for support and decreases the likelihood of being accepted by groups. Individuals from stigmatized groups are *less than.* Such persons tend to hide those factors that separate them from others, providing more power to the stigma. PTSD symptoms and cravings for alcohol and relapse may occur more often under the pressure of secrecy yet talking with other individuals honestly about their symptoms, cravings and relapse makes it easier to cope with these difficulties [71,196]. Sharing challenges and feelings is the strength of all self-help groups. However, social stigma and its associated secrecy serve to alienate individuals from their social support networks and community resources [5].

Recent studies have shown that individuals do not always perceive social relationships as supportive [3,4,15,74,137,]. Women with alcohol dependence, especially women with children and women who are seeking help for IPV, are often cautious of offers of social support [2,197]. This was documented in the research by Wuest *et al.* [58] in their grounded theory study focused on intrusion. Women who are also financially dependent on social agencies may experience increased fear and negative perceptions of "help" provided by such agencies. Is the high occurrence IPV, PTSD and AD in low-income populations due to high rates of precipitating events or because of a lack of resources to recover from or cope with these events?

Biological dimensions of the relationships among IPV, PTSD and alcohol dependence

Recent research in both PTSD and alcohol dependence has demonstrated changes in the brain mechanism, neurological and, neurohormonal pathways [6,126,139] due to severe or repeated trauma. Trauma creates these changes [69,104] and the over-consumption of alcohol adds to these changes [6]. Once these changes occur, the lower brain system is activated, which results in cravings [126] or intrusions [164] When the person responds to these cravings or intrusions in a manner that temporarily satisfies the lower brain, a situation of classical conditioning is established. It would be easy for a person to confuse the hyperarousal from alcohol withdrawal with the hyperarousal from PTSD and, thereby, reinforce or condition their relationship by using the alcohol. Relief from hyperarousal after using alcohol would then increase the desire for more alcohol. Although triggered by biological processes, many believe that classical conditioning is a biopsychological condition. The changes created by both PTSD and alcohol dependence interfere with both serotonin and cortisol. Serotonin is involved in functions such as hunger, aggression, sleep and fear response [69]. Low levels of serotonin create an anxiety reaction and may act on the amygdala to produce PTSD symptoms. This is interesting as AA teaches people to handle their cravings using "HALT", (*i.e.,* take appropriate action when you become Hungry, Angry, Lonely, or Tired). It would seem that the connection to the cravings and the amygdala were intuitively recognized by the founders of AA.

Conclusion

Although there is extensive research regarding the biological basis of alcohol dependence, there remains controversy as to whether or not alcohol dependence is a disease, and whether abstinence or harm

reduction is the best treatment protocol. While some research continues to focus on identifying single factors to explain alcohol dependence, much of current research attempts to consider complexity and most of the biomedical community now consider alcohol dependence to be a disease of the brain with contributing psychosocial factors and consequences (DSM-IV-TR). Similarly, IPV is considered a major health and social problem with extensive physical, psychological and social contributing factors and consequences, the most severe of which can be linked to PTSD and complicated by alcohol dependence.

Research on the interrelationships among IPV, PTSD and alcohol dependence has focused primarily on prevalence and co-morbidity. In some studies, structural equation modelling has been used to test models or explanations that could be a foundation for further research and policy development. The most promising efforts attend to complexity in more fully exploring the interrelationships of social support, IPV, PTSD and alcohol dependence. Other areas for research that have been identified in this paper are the role of cortisol [70,85]; the role of neurotransmitters [75]; impact and practices of detoxification [187] and recovery and relapse prevention programs, and barriers to access to treatment, especially for women [110]. Despite the difficulties of clinical trials, in the days of evidence-based practice, treatment centers need to be more precise in their description of treatment components and their underlying philosophy and both outcomes and process measures included in evaluating treatment effects. Without this precision, we cannot discover which interventions work and which do not or why they work.

Given the complexity of each of these areas, it is interesting to note the lack of qualitative research. The interrelationships among IPV, PTSD and alcohol dependence presents a puzzling pattern that has led to some interesting hypotheses. At the same time, low rates of recovery and high rates of relapse are a reality for the women and children caught in this maze. In this situation, the best intentions of both expert and client appear thwarted by apparently irrational choices. This situation is one that is ideal for examination through qualitative research [198] so that new insights into the experiences of recovery from alcohol dependence in the context of women's lives can be developed. The value of eliciting and analyzing women's stories about the dynamics, experiences and challenges of IPV, PTSD and, alcohol dependence may be a fruitful approach to gaining a more nuanced understanding of the issues faced by these women as they access treatment and how these experienced are shaped by the woman and her social context. Given the commonality of risk factors and the strong link between social support and IPV, PTSD and alcohol dependence, participatory action research and community development appear ideally suited for this area. As a foundation for this type of research, a better understanding of the women who are experiencing all three of these conditions and how they would perceive as supportive is needed.

References

1. Counts D, Brown JK, Campbell JC (Eds.) (1999) To Have and To Hit. (2nd ed.) Chicago, IL, University of Chicago Press.
2. Deal SA, Galaver J (1994) Are women more susceptible than men to alcohol induced cirrhosis? Alcohol, Health and Research World 18: 189-191.
3. McKay JR (1999) Studies of factors in relapse to alcohol, drug and nicotine use: a critical review of methodologies and findings. *J Stud Alcohol* 60: 566-576. [Crossref]
4. Hanna F, Guindon MH (2002) Women and alcoholism: a biopsychosocial perspective and treatment approaches (Practice and Theory). Journal of Counseling and Development 80: 145-153.
5. Aranda MP, Castaneda I, Lee PJ, Sobel E (2001) Stress, social support, and coping as predictors of depressive symptoms: Gender differences among Mexican Americans. Social Work Research 25(1): 37-48.
6. Chandler LJ (2003) Ethanol and brain plasticity: receptors and molecular networks of the postsynaptic density as targets of ethanol. *Pharmacol Ther* 99: 311-326. [Crossref]
7. Tjaden P, Thoennes N (2000) Full report of the prevalence, incidence, and consequences of violence against women: findings from the national violence against women survey. Washington, DC: National Institute of Justice. Report NCJ 183-781.
8. Pico-Alfonso MA (2005) Psychological intimate partner violence: The major predictor of posttraumatic stress disorder in abused women. *Neurosci Biobehav Rev* 29(1): 181-193. [Crossref]
9. Paluzzi PA, Houde-Quimby C (1996) Domestic violence: Implications for the American college of nurse-midwives. *J Nurse Midwifery* 41(6): 430-435. [Crossref]
10. Kilpatrick DG (2004) What is violence against women: defining and measuring the problem. *J Interpers Violence* 19: 1209-1234. [Crossref]
11. Campbell JC (2002) Health consequences of intimate partner violence. *Lancet* 359: 1331-1336. [Crossref]
12. Garcia-Moreno C, Jansen HA, Ellsberg M, Heise L, Watts CH (2006) Prevalence of intimate partner violence: findings from the WHO multicounty study on women's health and domestic violence. Lancet 368(9543): 1260-1269. [Crossref]
13. Hotton T (2001) Spousal violence after marital separation. Juristat, Canadian Centre for Justice Statistics 21(7): 1-19.
14. Besserer S, Brzozowski J, Hendrick DO, Ogg S, Trainor C (2001) A profile of criminal victimization: Results of the 1999 General Social Survey. Ottawa, ON: Statistics Canada.
15. Coker AL, Watkins KW, Smith PH, Brandt HM (2004) Intimate partner violence and physical health consequences: Policy and practice. *J Interpers Violence* 19: 1296-1323. [Crossref]
16. Gazmararian JA, Petersen R, Spitz AM, Goodwin MM, Saltzman LE et al. (2000) Violence and reproductive health: current knowledge and future research directions. *Matern Child Health J* 4(2): 79-84. [Crossref]
17. Saltzman LE (2004) Issues related to defining and measuring violence against women: response to Kilpatrick. *J Interpers Violence* 19: 1235-1243. [Crossref]
18. Parker B, McFarlane J, Soeken K, Silva C, Reel S (1999) Testing an intervention to prevent further abuse to pregnant women. *Res Nurs Health* 22: 59-66. [Crossref]
19. Campbell JC, Garza M, Gielen AC, O'Campo P, Kub J, et al. (2003) Intimate partner violence and abuse among active duty military women. Violence Against Women 9: 1072-1092
20. Coker AL, Smith P, Bethea L, King M, McKeown R (1997) Physical Health Consequences of Physical and Psychological Intimate Partner Violence. *Arch Fam Med* 9(5): 451-457. [Crossref]
21. Heise L, Garcia-Moreno C (2002) Violence by intimate partners. World Report on Violence and Health. Geneva: World Health Organization.
22. Hoge CW, Lesikar SE, Guevara R, Lange J, Brundage JF, et al. (2002) Mental disorders among U.S. Military personnel in the 1990's: Association with high levels of health care utilization and early military attrition. *Am J Psychiatry*159: 1576-1583. [Crossref]
23. Jasinski JL (2001) Explanations for violence against women. In C, M. Renzetti, J. L. Edleson, R. & Kennedy. Sourcebook on violence against women. Newbury Park CA: Sage
24. Plichta SB, Falik M (2001) Prevalence of violence and its implications for women's health. *Womens Health Issues* 11: 244-258. [Crossref]
25. Campbell JC (2004) Helping women understand their risk in situations of intimate partner violence, *J Interpers Violence* 19: 1464-1477. [Crossref]
26. Dutton MA (1992) Empowering and healing the battered woman. New York: Springer.
27. Herman JL (2001) Trauma and Recovery: From Domestic Abuse to Political Terror, London: Penguin.
28. Golding JM (1999) Intimate partner violence as a risk factor for mental disorders: a metaanalysis. Journal of Family Violence 14(2): 99-132.
29. Laffaye C, Kennedy C, Stein MB (2003) Post-traumatic stress disorder and health-related quality of life in female victims of intimate partner violence. *Violence Vict* 18: 227-238. [Crossref]
30. Ratner C (1993) Review of D'Andrade and Strauss, Human motives and cultural models. Journal of Mind and Behaviors 14: 89-94.
31. Bremner JD, Narayan M, Staib LH, Southwick SM, McGlashan T, et al. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *Am J Psychiatry* 156: 1787-1795. [Crossref]

32. Paslow R, Jorm A, Christensen H, Jacomb P, Rodgers B (2004) Gender differences in factors affecting use of health services: an analysis of a community study of middle aged and older Australians. *Soc Sci Med* 59(10): 2121-2129. [Crossref]

33. Harway M, O'Neil JM (Eds.) (1999) What causes men's violence against women? Thousand Oaks, CA: Sage.

34. Kantor GK, Jasinski JL (1998) Dynamics and risk factors in partner violence. In J. L. Jasinski & L. M. Williams (Eds.). Partner violence: A comprehensive review of 20 years of research. Thousand Oaks, CA: Sage.

35. Wathen CN, MacMillan H (2003) Violence against women: integrating the evidence into clinical practice. Canadian Medical Association Journal 169: 570-571.

36. Rennison C (2000) Intimate Partner Violence, Special Report 1993–2000. Washington DC: Bureau of Justice Statistics, U.S. Department of Justice. Publication No. NCJ 178-247.

37. MacMillan HL, Wathen CN (2005) Family violence research: Lessons learned and where from here? *JAMA* 294: 618–620. [Crossref]

38. Leiner AS, Compton MT, Houry D, Kaslow NJ (2008) Intimate partner violence, psychological distress, and suicidality: A path model using data from African American women seeking care in an urban emergency department. Journal of Family Violence 23(6): 473-481.

39. Walker R, Logan TK, Jordan CE, Campbell JC (2004) An Integrative Review of Separation in the Context of Victimization Consequences and Implications for Women. *Trauma Violence Abuse* 5(2): 143-193. [Crossref]

40. Bandura A (1979) The social learning perspective: Mechanism of aggression. In A. Toch (Ed.). Psychology of crime and criminal justice (pp. 298-336). New York: Holt, Rinehart & Winston.

41. Feldman CM, Ridley CA (1995) The etiology and treatment of domestic violence between adult partners, Clinical Psychology: Science and Practice 2: 317-348.

42. Cunradi CB, Caetano R, Schafer J (2002) Alcohol-related problems, drug use, and male intimate partner violence severity among US couples. *Alcohol Clin Exp Res* 26: 493-500. [Crossref]

43. Kaufman M (2001) Building a movement of men working to end violence against women. Development (Society for International Development) 44(3).

44. Dobash RE, Dobash R (1992) Women, violence and social change. New York: Routledge.

45. Kwesiga E, Bell M, Pattie M, Moe A (2007) Exploring the Literature on Relationships Between Gender Roles, Intimate Partner Violence, Occupational Status, and Organizational Benefits. Journal of Interpersonal Violence 22(3): 312-326.

46. Dutton DG (2007) The abusive personality: violence and control in intimate relationships. New York: Guildford.

47. Gottman JM (1999) The marriage clinic. New York: Norton.

48. Armstrong A (1998) Culture and choice: Lessons from survivors of gender violence in Zimbabwe. Harare, Zimbabwe: Violence Against Women in Zimbabwe Research Project,

49. Gonzalez-Montes S (1999) Domestic violence in Cuetzalan, Mexico: Some research questions and results. In Center for Health and Gender Equity (CHANGE), (Ed.)

50. Osakue G, Hilber AM (1998) Women's sexuality and fertility in Nigeria. In R. Petchesky & K. Judd. Negotiating Reproductive Rights (pp. 180-216). London: Zed Books.

51. Michau L (1998) Community-based research for social change in Mwanza, Tanzania. Center for Health and Gender Equity (CHANGE), (Ed.). Proceedings of the Third Annual Meeting of the International Research Network on Violence Against Women (pp. 49). Washington DC

52. Gelles RJ, Loseke DR (1993) 'Issues in causes', in R.J. Gelles & D.R. Loseke (Eds) Current Controversies on Family Violence. Newbury Park CA: Sage.

53. Johnson D, Westermeyer J, Kattar K, Thuras P (2002) Daily charting of posttraumatic stress symptoms: A pilot study. *J Nerv Ment Dis* 190: 683-92. [Crossref]

54. McKenry PC, Julian TW, Gavazzi SM (1995) Toward a biopsychosocial model of domestic violence. Journal of Marriage and the Family 57: 307-320.

55. Mrazek PJ, Haggerty RJ (Eds.) (1994) Reducing risks for mental disorders: Frontiers for preventive intervention research. Washington, DC: National Academy.

56. Wells A, Matthews G (1996) Anxiety and cognition. Current Opinion in Psychiatry 9(6): 422-426.

57. Pedhazur E, Pedhazur-Schmelkin L (1991) Measurement, Design, and Analysis: An Integrated Approach. London: Lawrence Erlbaum.

58. Wuest J, Ford-Gilboe M, Merritt-Gray M, Berman H (2003) Intrusion: The central problem for family health promotion among children and single mothers after leaving an abusive partner. *Qual Health Res* 13(5): 597-621. [Crossref]]

59. Breslau N, Peterson EL, Kessler RC, Schultz LR (1999) Short screening scale for DSM-IV posttraumatic stress disorder. *Am J Psychiatry* 156(6): 908-911. [Crossref]

60. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB (1995) Posttraumatic stress disorder in the national comorbidity survey. *Arch Gen Psychiatry* 52: 1048-1060. [Crossref]

61. Statistics Canada (2003) Canadian community health survey: Canadian forces supplement on mental health. Ottawa.

62. American Psychiatric Association (1980) Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, DC: Author.

63. van der Kolk BA, Saporta J (1992) The biological response to psychic trauma: Mechanism and treatment of intrusion and numbing. Anxiety Research (UK). 4: 199-212.

64. McNally L (1998) On the linguistic encoding of information packaging instructions. In P. Culicover & L. McNally (Eds.). Syntax and Semantics. The limits of syntax 161-184.

65. Freidman M, Schnurr P (1995) The relationship between trauma, post-traumatic stress disorder, and physical health, (p. 518). In M. Friedman, D. Charney & A Deutch, (Eds.). Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to PTSD. Philadelphia, PA: Lippincott-Raven.

66. Wagner AW, Wolfe J, Rotnitsky A, Proctor SP, Erickson DJ (2000) An investigation of the impact of posttraumatic stress disorder on physical health. *J Trauma Stress* 13: 41-55. [Crossref]

67. Rothchild AJ, Bates KS, Boehringer KL, Syed A (1999) Olanzapine response in psychotic depression. *J Clin Psychiatry* 60(2): 116-118. [Crossref]

68. American Psychiatric Association (2000) Diagnostic and statistical manual of mental disorders (4th ed., text revision). Washington, DC: Author.

69. Southwick SM (2003) Salivary cortisol responses to dexamethasone in adolescents with posttraumatic stress disorder. *J Am Acad Child Adolesc Psychiatry* 42: 1310-1317. [Crossref]

70. Yehuda R, Teicher MH, Trestman RL, Levengood RA, Siever LJ (1996) Cortisol regulation in posttraumatic stress disorder and major depression: A chronobiological analysis. *Biol Psychiatry* 40: 79-88. [Crossref]

71. Ehlers A, Clark DM (2000) A cognitive model of posttraumatic stress disorder. *Behav Res Ther* 38: 319-345. [Crossref]

72. Najavits LM, Weiss RD, Shaw SR (1998) "Seeking safety": Outcome of a new cognitivebehavioral psychotherapy for women with posttraumatic stress disorder and substance dependence. *J Trauma Stress* 11: 437-456. [Crossref]

73. Xian H, Chantarujikapong S, Scherrer J, Eisen S, Lyons M, et al. (2000) Genetic and environmental influences on posttraumatic stress disorder, alcohol, and drug dependence in twin pairs. *Drug Alcohol Depend* 61: 95-102. [Crossref]

74. Fontana A, Rosenheck R (1998) Focus on women: Duty related and sexual stress in the etiology of PTSD among women veterans who seek treatment. *Psychiatr Serv* 49: 658-662. [Crossref]

75. Zlotnick C, Warshaw M, Shea MT, Allsworth J, Pearlstein T, et al (1999) Chronicity in posttraumatic stress disorder (PTSD) and predictors of course of comorbid PTSD in patients with anxiety disorder. *J Trauma Stress* 12(1): 89-100. [Crossref]

76. Regeher C, Hemsworth D, Hill J (2001) Individual predictors of posttraumatic distress: A structural equation model. *Can J Psychiatry* 46: 156-161. [Crossref]

77. LeBlanc VR, Regehr C, Jelley RB, Barath I (2007) Does Posttraumatic Stress Disorder (PTSD) affect performance? *J Nerv Ment Dis*195(8): 701-704. [Crossref]

78. Perry B, Pollard RH, Blakley TL, Baker WL, Vigilante D (1995) Childhood trauma, the neurobiology of adaption and use-dependent development of the brain: How states become traits. Infant Mental Health Journal 16(4): 271-291.

79. Roberts TE, Berridge K (1993) The neural basis of drug craving: An incentive–sensitization theory of addiction. *Brain Res Brain Res Rev* 18: 247-291. [Crossref]

80. Perry BL, Jones H, Tuten M, Svikis DS (2003) Assessing maternal perceptions of harmful effects of drug use during pregnancy. *J Addict Dis* 22(1): 1-9.

81. Plihal W, Krug R, Pietrowsky R, Fehm HL, Born J (1996) Corticosteroid receptor mediated effects on mood in humans. *Psychoneuroendocrinology* 21: 515-523. [Crossref]

82. Maes M, Lin A, Bonaccorso S, van Hunsel F, Van Gastel A, et al. (1998) Increased 24 hour urinary cortisol excretion in patients with posttraumatic stress disorder and patients with major depression, but not in patient with fibromyalgia. *Acta Psychiatr Scand* 98: 328-335. [Crossref]

83. Mason JW, Giller E, Kosten T, Ostroff R, Podd L (1986) Urinary free cortisol levels in posttraumatic stress disorder patients. *J Nerv Ment Dis* 174: 145-149. [Crossref]

84. Baker DG, West SA, Nicholson W, Ekhator N, Kasckow J, et al. (1999) Serial CSF corticotrophin-releasing hormone levels and adrenocortical activity in combat veterans with posttraumatic stress disorder. *Am J Psychiatry* 156(4): 585-588. [Crossref]

85. Jacobsen LK, Southwick SM, Kosten TR (2001) Substance use disorders in patients with posttraumatic stress disorder: a review of the literature. *Am J Psychiatry* 158: 1184-1190. [Crossref]

86. Bremner JD, Staib LH, Kaloupek D, Southwick SM, Soufer R (1999) Neural correlates of exposure of traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder. *Biol Psychiatry* 45: 806-816. [Crossref]

87. Metzger LJ, Orr SP, Berry NJ, Ahern CE, Lasko NB, et al. (1999) Physiologic reactivity to startling tones in women with posttraumatic stress disorder. *J Abnorm Psychol* 108(2): 347-352. [Crossref]

88. O'Donnell T, Hegadoren KM, Coupland NC (2004) Noradrenergic mechanisms in the pathophysiology of post-traumatic stress disorder. *Neuropsychobiology* 50: 273-283. [Crossref]

89. Keane TM, Zimering RT, Caddell, JT (1985) A behavioral formulation of posttraumatic stress disorder in Vietnam veterans. Behavior Therapist 8: 9-12.

90. Foa DW (1992) Treating PTSD: Cognitive-behavioral strategies. New York: Guilford.

91. Jones E1, Wessely S (2001) Psychiatric battle casualties: an intra- and interwar comparison. *Br J Psychiatry* 178: 242-247. [Crossref]

92. Kulka RA, Schlenger WE, Fairbank JA, Hough RL, Jordan BK, et al. (1990) Trauma and the Vietnam War Generation: Report on findings from the National Vietnam Veterans Readjustment Study (p. 54). New York: Brunner/Mazel.

93. Pynoos RS, Goenjian A, Tashjian M, Karakashian M, Manjikian R, et al. (1993) Post-traumatic stress reactions in children after the 1988 Armenian earthquake. *Br J Psychiatry*163: 239-247. [Crossref]

94. Bowman ML (1997) Brain impairment in impulsive violence, In C. Webster & M. Jackson (Eds.). Impulsivity: Perspectives, principles, and practice (116-141). New York: Guilford.

95. Bowman ML (1999) Individual differences in responding to adversity with posttraumatic distress: Problems with the DSM-IV model. *Can J Psychiatry* 44: 21-33. [Crossref]

96. Harvey AG, Bryant RA (2000) Two-year prospective study of psychophysiological arousal, acute stress disorder and posttraumatic stress disorder. Journal of Abnormal Psychology 109: 341-344.

97. Roemer I, Reik W, Dean W, Klose J (1997) Epigenetic inheritance in the mouse. *Curr Biol* 7: 277-280. [Crossref]

98. Schwarz JC, Barton-Henry ML, Pruzinsky T (1985) Assessing child-rearing behaviors: A comparison of ratings made by mother, father, child, and sibling on the CRPBI. *Child Dev* 56(2): 462-479. [Crossref]

99. Southwick SM, Morgan CAIII, Nicolaou AL, Charney DS (1997) Consistency of memory for combat-related traumatic events in veterans of Operation Desert Storm. *Am J Psychiatry*154: 173–177. [Crossref]

100. LeDoux JE, Xagoraris A, Romanski LM (1989) Indelibility of subcortical emotional memories. *J Cogn Neurosci* 1(3): 238-243. [Crossref]

101. Morrison PD, Allardyce J, McKane JP (2002) Fear knot. Neurobiological disruption of long-term fear memory. *Br J Psychiatry* 180: 195-197. [Crossref]

102. Asmundson GJ, Stein MB, McCreary DR (2002) PTSD symptoms influence health status of deployed peacekeepers and non-deployed military personnel. Journal of Nervous and Mental Disease 190: 807-15.

103. Diaz A, Simatov E, Rickert VI (2000) The independent and combined effects of physical and sexual abuse on health, results of a national survey. Journal of Pediatric and Adolescent Gynecology 13: 89.

104. Dube SR, Felitti V, Dong M, Chapman D, Giles W, et al. (2003) Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use: The adverse childhood experiences study. Pediatrics 111: 564-572.

105. Hall J (1996) Geography of childhood sexual abuse: Women's narratives of their childhood environments. (Environment and Health). *ANS Adv Nurs Sci* 18: 29-47. [Crossref]

106. Brady KT, Sonne S, Anton RF, Randall CL, Back SE, et al. (2005) Sertraline in the Treatment of Co-occurring Alcohol Dependence and Posttraumatic Stress Disorder. *Alcohol Clin Exp Res* 29(3): 395-401. [Crossref]

107. World Health Organization (Ed.) (1992) International statistics classification of diseases and related health problems, (10th Revision). Geneva: Author.

108. Murray C, Lopez A (Eds). (1996) Summary: The global burden of disease. Cambridge, MA: Harvard University Press.

109. Xie X, Rehm J, Single E, Robson L (1996) The economic costs of alcohol, tobacco, and illicit drug abuse in Ontario: 1992. Toronto, ON: Addiction Research Foundation.

110. Adlaf EM, Begin P, Sawka E (Eds.) (2005) Canadian Addiction Survey (CAS): A national survey of Canadians' use of alcohol and other drugs: Prevalence of use and related harms: Detailed report. Ottawa, ON: Canadian Centre on Substance Abuse.

111. Ziberman ML, Tavares H, Blume SB, el-Guebaly N (2003) Substance use disorders: Sex differences and psychiatric comorbidities. *Can J Psychiatry* 48(1): 5-13. [Crossref]

112. Jaffe JH (1983) Drug addiction and drug abuse. In L. S. Goodman. & A. G. Gilman (Eds.) The Pharmacological Basis of Therapeutics (6th ed.). New York: Macmillan.

113. Edwards G, Gross MM (1976) Alcohol dependence: provisional description of a clinical syndrome. *Br Med J* (1): 1058-1061. [Crossref]

114. Yalisove D (2004) Introduction to alcohol research, Implications for treatment, prevention and policy, Boston, MA: Pearson.

115. Naegle M, D'Avanzo C (2000) Addictions and substance abuse; Strategies for advanced practice nursing. Princeton, NJ: Prentice Hall.

116. Peele S (1983) Through a glass darkly. Psychology Today 17: 38-42.

117. Peele S (1984) The cultural context of psychological approaches to alcoholism: Can we control the effects of alcohol? American Psychologist 39: 1337-1351.

118. Peele S (1987) The limitations of control-of-supply models for explaining and preventing alcoholism and drug addictions. *J Stud Alcohol* 48: 61-89. [Crossref]

119. Peele S (1987a) Why do controlled drinking outcomes vary by investigator, by country and by era? Cultural conceptions of relapse and remission in alcoholism. Drug and Alcohol Dependence 20: 173-201.

120. Peele S (1990) Personality and alcoholism: Establishing the link. In D.A. Ward (Ed.), Alcoholism: Introduction to Theory and Treatment (3rd Ed. pp. 147-156). Dubuque, IO: Kendall/Hunt.

121. Schuckit MA (1995) Drug and alcohol abuse, New York: Plenum.

122. Csiernik R (2003). Introduction to substance use and abuse, Theory & practice. Toronto, ON: Alcohol & Drug Recovery Association of Ontario.

123. Foroud T, Edenberg HJ, Goate A, Rice J, Flury L, et al. (2000) Alcoholism susceptibility loci: Confirmation studies in a replicate sample and further mapping. *Alcohol Clin Exp Res* 24: 933-945. [Crossref]

124. Reich T, Edenberg HJ, Goate A (1998) Genome-wide search for genes affecting the risk for alcohol dependence. *Am J Med Genet* 81: 207-215. [Crossref]

125. Schuckit MA, Edenberg HJ, Kalmijn J, Flury L, Smith TL, et al. (2001) A genome-wide search for genes that relate to a low level of response to alcohol. *Alcohol Clin Exp Res* 25: 323-329. [Crossref]

126. Anton R (1999) What is a craving? Models and implications for treatment (alcohol craving). *Alcohol Res Health* 23(3): 165-173. [Crossref]

127. Koob G, LeMoal M (1999) Brain reward circuits in alcoholism. CNS Spectrums 4: 23-38.

128. Morgenstern J, Mckay J (2007) Rethinking the paradigms that inform behavioral treatment research for substance use disorders. *Addiction* 102(9): 1377-1389. [Crossref]

129. Noar SM, Zimmerman RS (2005) Health Behavior Theory and cumulative knowledge regarding health behaviors: are we moving in the right direction? *Health Educ Res* 20(3): 275-290. [Crossref]

130. Miller EM (1986) Street women. Philadelphia: Temple University Press.

131. Carter CS (1997) Ladies don't: A historical perspective on attitudes toward alcoholic women. Affillia, 12: 471-478.

132. Goode E (1999) Drugs in American Society. New York: McGraw-Hill.

133. Wilsnack RW, Wilsnack SC, Miller-Strumhofel S (1994) How women drink: Epidemiology of women's drinking and problem drinking. Alcohol Health and Research World 18: 173-181.

134. Gilligan C (1982) In a Different Voice. Cambridge, MA: Harvard University Press.

135. Finkelstein N (1993) The Relational Model. In D. Kronstadt, P.F. Green & C. Marcus, (Eds.). Pregnancy and exposure to alcohol and another drug use (pp. 126-163). Rockville, MD Department of Health and Human Services, Center for Substance Abuse Prevention.

136. Kessler RC, Berglund P, Zhao S (1996) The twelve-month prevalence and correlates of serious mental illness (SMI). In: R. W. Manderscheid & M. A. Sonnenschein (Eds.). Mental health, United States, 1996. (pp 59-70). Washington DC: U.S. Department of Health and Human Services

137. Bischof G, Rumpf J, Hapke U, Meyer C, John U (2003) Types of natural recovery from alcohol dependence: a cluster analytic approach. *Addiction* 98: 1737-1746. [Crossref]

138. Malcolm R, Herron J, Anton R, Roberts J, Moore J (2000) Recurrent detoxification may elevate alcohol craving as measured by the Obsessive-Compulsive Drinking scale. *Alcohol* 20: 181-185. [Crossref]

139. Duka T, Townshend J, Collier K, Stephens D (2002) Kindling of withdrawal: A study of craving and anxiety after multiple detoxifications in alcoholic inpatients. *Alcohol Clin Exp Res* 36: 785-795. [Crossref]

140. King GR, Xiong Z, Ellinwood EH (1997) Blockade of cocaine behavioural and tolerance by the co-administration of ondansetron, a 5-HT receptor antagonist, and cocaine. *Psychopharmacology (Berl)* 130(3): 159-165. [Crossref]

141. Breslau N, Davis GC, Andreski P, Peterson E (1991) Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Arch Gen Psychiatry* 48: 216-22. [Crossref]

142. Smith EM, North CS, Spitznagel EL (1993) Alcohol, drugs, and psychiatric comorbidity among homeless women: An epidemiologic study. *J Clin Psychiatry* 54: 82-87. [Crossref]

143. Velleman R (2001) Counselling for alcohol problems (2nd ed.). London: Sage.

144. Califano J (2006) Women under the influence. New York: The National Center on Addiction and substance abuse at Columbia University.

145. Tatarsky A (2003) Harm reduction psychotherapy: Extending the reach of traditional substance use treatment. Journal of Substance Abuse Treatment 25(4): 249-256.

146. Rotgers F (1999) Client-driven, research-guided treatment for substance users: Bringing harm reduction to clinical practice. In E.T. Dowd & L. Rugle (Eds.) Comparative Treatments of Substance Abuse. New York: Springer.

147. Prochaska JO, DiClemente CC, Norcross JC (1992) In search of how people change: Applications to addictive behaviors. *Am Psychol* 47: 1102-1114. [Crossref]

148. Carey K, Roberts L, Kivlahan D, Carey M, Neal D (2004) Problems assessment for substance using psychiatric patients: development and initial psychometric evaluation. *Drug Alcohol Depend* 75: 67-77. [Crossref]

149. Teichman M, Barnea Z, Ravav G (1989) Personality and substance use among adolescents: a longitudinal study. *Br J Addict* 84: 181-190. [Crossref]

150. Litten RZ, Allen JP (1992) Measuring alcohol consumption: psychosocial and biochemical methods. Totowa, NJ: Humana.

151. Foster JH, Powell JE, Marshall EJ, Peters TJ (1999) Quality of life in alcohol-dependent subjects--a review. *Qual Life Res* 8: 255-261. [Crossref]

152. Miller WR, Walters ST, Bennett ME (2001) How effective is alcoholism treatment in the United States? *J Stud Alcohol* 62: 211-220. [Crossref]

153. Boardman T, Catley D, Grobe JE, Little TD, Ahluwalia JS (2006) Using motivational interviewing with smokers: do therapist behaviors relate to engagement and therapeutic alliance? *J Subst Abuse Treat* 31: 329-339. [Crossref]

154. Tatarsky A (Ed.) (2002) Harm Reduction Psychotherapy: A New Treatment for Drug and Alcohol Users. Northvale, NJ: Aronson.

155. Najavits LM (2002) Seeking Safety: A treatment manual for PTSD and substance abuse. New York, NY: Guilford.

156. Najavits LM (2001) Seeking safety: Cognitive-behavioral therapy for PTSD and substance abuse. New York: Guilford.

157. Najavits LM (2003) How to design an effective treatment outcome study. *J Gambl Stud* 19: 317-337. [Crossref]

158. Holbrook TL, Hoyt DB, Stein MB, Sieber WJ (2001) Perceived threat to life predicts posttraumatic stress disorder after major trauma: Risk factors and functional outcome. *J Trauma* 51: 287-92. [Crossref]

159. Stein M (2001) Major depressive disorders and post traumatic stress disorder comorbidity in female victims of IPV. *J Affect Disord* 66(2-3): 133-138. [Crossref]

160. Woods SJ (2005) Intimate partner violence and post-traumatic stress disorder symptoms in women: what we know and need to know. *J Interpers Violence* 20: 394-402. [crossref]

161. Wuest J, Merrit-Gray M, Ford-Gilboe M, Lent B, Varcoe C, et al. (in press) Chronic pain in women survivors of intimate partner violence. *J Pain* 9(11): 1049-1057. [Crossref]

162. Kyriacou DN, McCabe F, Anglin D, Lapesarde K, Winer MR (1998) Emergency department based study of risk factors for acute injury from domestic violence against women. *Ann Emerg Med* 31(4): 502-506. [Crossref]

163. Murphy CM, Farrell TJ, Fals-Stewart W, Feehan M (2001) Correlates of intimate partner violence among male alcoholic patients. *J Consult Clin Psychol* 69: 528-540. [Crossref]

164. Kilpatrick DG, Acierno R, Saunders BE, Resnick HS, Best CL, et al. (2000) Risk factors for adolescent substance abuse and dependence: Data from a national sample. *J Consult Clin Psychol* 68: 19-30. [Crossref]

165. Testa M, Livingston JA, Leonard K (2003) Women's substance use and experience of intimate partner violence: A longitudinal investigation among a community sample. Addictive Behavior 28(9): 1649-1664.

166. Miller BA, Wilsnack SC, Cunradi CB (2000) Family violence and victimization: Treatment issues for women with alcohol problems. *Alcohol Clin Exp Res* 24: 1287-1297. [Crossref]

167. Chase KA, O'Farrell TJ, Murphy CM, Fals-Stewart W, Murphy M (2003) Factors associated with partner violence among female alcoholic patients and their male partners. *J Stud Alcohol* 64(1): 137-149. [Crossref]

168. Chermack ST, Walton M, Fuller B, Blow F (2001) Correlates of expressed and received violence across relationship types among men and women substance abusers. *Psychol Addict Behav* 15: 140-151. [Crossref]

169. Swingle JM, McCrady BS, Epstein E (2001) Couple violence in a clinical sample of female alcoholics. Poster presented at the annual scientific meeting of the Research Society on Alcoholism, Montreal, Canada.

170. Dansky BS, Byrne CA, Brady KT (1999) Intimate violence and posttraumatic stress disorder among individuals with cocaine dependence. *Am J Drug Alcohol Abuse* 25(2): 257-268. [Crossref]

171. Testa M, Fillmore MT, Norris J, Abbey A, Curtin JJ, et al. (2006) Understanding alcohol expectancy effects: revisiting the placebo condition. *Alcohol Clin Exp Res* 30: 339-348. [Crossref]

172. Paranjape A, Heron S, Thompson M, Bethea K, Wallace T, et al. (2007) Are alcohol problems linked with an increase in depressive symptoms in abused innercity African American women. *Womens Health Issues* 17(1): 37-43. [Crossref]

173. Najavits LM, Liese BS (1997) Cognitive therapy for substance abuse. In J. H.

174. Bollerud K (1990) A model for the treatment of trauma-related syndromes among chemically dependent inpatient women. Journal of Substance Abuse Treatment 7: 83-87.

175. Fullilove MT, Fullilove RE, Smith M, Winkler K, Michael C, et al. (1993) Violence, trauma, and post-traumatic stress disorder among women drug users. Journal of Traumatic Stress 6: 533-543.

176. Yandow V (1989) Alcoholism in women. Psychiatry Annual 19: 243-247.

177. Bremner JD, Southwick SM, Darnell AD, Charney DS (1996) Chronic PTSD in Vietnam combat veterans: Course of illness and substance abuse. *Am J Psychiatry* 153: 369-375. [Crossref]

178. Clark D, Lesnick L, Hegedus AM (1997) Traumas and other adverse life events in adolescents with alcohol abuse and dependence. *J Am Acad Child Adolesc Psychiatry* 36: 1744-1751. [Crossref]

179. Brown PJ, Recupero PR, Stout R (1995) PTSD substance abuse comorbidity and treatment utilization. *Addict Behav* 20: 251-254. [Crossref]

180. Bux D, Volpicelli J, Balaraman G, Hahn J, Wallace H (1999) The Role of Uncontrollable Trauma in the Development of PTSD and Alcohol Addiction. Alcohol Research and Health 23: 256. [Crossref]

181. Back SE, Sonne SC, Killeen T, Dansky BS, Brady KT (2003) Comparative profiles of women with PTSD and comorbid cocaine or alcohol dependence. *Am J Drug Alcohol Abuse* 29: 169-189. [Crossref]

182. Khouzam HR, Donnelly NJ (2001) Posttraumatic stress disorder. Safe, effective management in the primary care setting. Postgraduate Medicine 110: 60-62, 67-70, 77-78.

183. Cottler LB, Compton WM, Mager D, Spitnagel E, Janca A (1992) Posttraumatic stress disorder among substance users from the general population. *Am J Psychiatry* 149: 664-670. [Crossref]

184. Chilcoat HD, Breslau N (1998) Posttraumatic stress disorder and drug disorders: testing causal pathways. *Arch Gen Psychiatry* 55: 913-917. [Crossref]

185. Hutchison KE, Rohsenow D, Monti P, Palfai T, Swift R (1997) Prepulse inhibition of the startle reflex: Preliminary study of the effects of a low dose of alcohol in humans. Alcohol Clinical Experimental Research 21: 1312-1319. [Crossref]

186. Arai K, Ohata H, Shibasaki T (1998) Non-specific corticotrophin-releasing hormone receptor type 1 antagonist reverses restraint stress-induced shortening of sodium pentobarbital-induced sleeping time of rats: Evidence that an increase in arousal induced by stress is mediated through CRH receptor type 1. Neuroscience Letters 255: 103-106.

187. Shaham Y, Erb S, Leung S, Buczek Y, Stewart J (1998) A selective, nonpeptide antagonist of the corticotropin-releasing factor 1 receptors attenuates stressinduced relapse to drug seeking in cocaine and heroin trained rats. Psychopharmacology (Berl) 137: 184-190.

188. Greenfield S, Kolodziej M, Sugarman D, Muenz L, Vagge L, et al. (2002) History of abuse and drinking outcomes following inpatient alcohol treatment: A prospective study. *Drug Alcohol Depend* 67: 227-234. [Crossref]

189. Famularo R, Kinscherff R, Fenton T (1992) Parental Substance Abuse and the Nature of Child Maltreatment. *Child Abuse Negl*16: 475-483. [Crossref]

190. Fullilove MT, Lown EA, Fullilove RE (1992) Crack 'hos and skeezers': Traumatic experiences of women crack users. Journal of Sex Research 29: 275-287.

191. Nace EP, Davis CW, Gaspari JP (1991) Axis II comorbidity in substance abusers. *Am J Psychiatry* 148: 118-120. [Crossref]

192. Paone D, Chavkin W, Wilets I, Friedmann P, Des Jarlais D (1992) The impact of sexual abuse: Implications for drug treatment. Journal of Women's Health 1: 149-153.

193. Root MPP (1989) Treatment failures: The role of sexual victimization in women's addictive behavior. *Am J Orthopsychiatry* 59: 542-549. [Crossref]

194. Copeland J (1997) A qualitative study of barriers to formal treatment among women who selfmanage change in addictive behaviour. *J Subst Abuse Treat* 14(2): 183-190. [Crossref]

195. Lee J, Pomeroy E, Bohman T (2007) Intimate partner violence and psychology health in a sample of Asian and Caucasian women: The role of social support and coping. Journal of Family Violence 22(8): 709-720.

196. Daley D (1998) A psychoeducational approach to relapse prevention, relapse conceptual, research and clinical perspectives. Malibu, CA: Haworth.

197. Dutton DG (1998) Domestic assault of women: Psychological and criminal justice perspectives. Boston, MA: Allyn & Bacon.

198. Morse J, Richards L (2002) Readme first for a user's guide to qualitative methods. London: Sage.