Alcohol Use and Anxiety Disorders

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The relationship between anxiety and alcohol use is a topic of great theoretical and practical interest for both scientists interested in the nature and causes of psychopathology and practitioners working with anxious and/or alcohol abusing clients. Although it has been clearly established that anxiety disorders and alcohol use disorders are highly "comorbid" or co-occurring conditions (e.g., see Kushner, Abrams & Borchardt, 2000a for a review), the relationship between the symptoms or behaviors involved in each disorder (e.g., feelings of anxiety and levels of alcohol use) has not been as extensively reviewed. This chapter will review recent empirical evidence linking anxiety and alcohol at both the behavioral and disorder level to determine if similar conclusions can be derived regarding their relationship from data at both of these levels of enquiry. We will first briefly describe epidemiological studies linking anxiety disorders and alcohol use disorders. Then we will examine some of the etiological theories of the relationship between anxiety and alcohol use and their disorders, with a review of the empirical evidence supporting each theory. Next, some specific factors moderating and mediating the relationship between anxiety and alcohol use will be explored, with an emphasis on individual differences and specific processes involved in the relationship. A brief discussion of the differences between factors affecting onset, maintenance, and relapse in the anxiety and alcohol relationship will follow. The latest empirical evidence and thoughts about treating both alcohol use and anxiety related problems will also be reviewed. Finally, we conclude the chapter with some remarks about where the field stands and directions that future research in this area might profitably take.

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Symptomatic and Syndromal Levels of Enquiry in the Anxiety – Alcohol Relationship

Anxiety and alcohol use can both be characterized at two different levels: symptomatic and syndromal. An association at the former level would entail a clear relationship, for example, between feelings of anxiety and drinking behavior. That is, one would expect that higher levels of anxiety would be related to higher quantities and/or frequency of drinking behavior. In a classic paper, Persons (1986) describes the advantages of studying psychological phenomena at the symptomatic level rather than at the diagnostic category (or syndromal) level. The symptom approach allows for study of important phenomena that may be ignored by examining only the diagnostic category in question. For example, level of alcohol consumption is not considered in the diagnosis of alcohol abuse or dependence according to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association [APA], 1994), although there have been some recommendations for incorporating heavy drinking behaviors in the diagnostic definitions in future editions (Helzer, Bucholz, & Bierut, 2006). Nonetheless, level of consumption may be an important risk factor for alcohol problems (Dawson & Archer, 1993) and thus may be of interest as a "symptom" when considering the anxiety – alcohol relation from the symptom perspective. Also, the symptom approach recognizes the continuity of clinical phenomena and behaviors with normal phenomena and behaviors. This is a crucial point in the study of both drinking behavior and anxiety. For example, some argue that drinking problems are best viewed as lying on a continuum ranging from normal, non-problematic social drinking on one end to severe and pathological alcohol dependence on the other extreme (Sobell et al., 1996).

In addition to the arguments presented above in favor of focusing on symptoms rather than diagnostic categories, Chilcoat and Breslau's (1998) discussion of criteria for establishing causation between anxiety and alcohol abuse also demands a symptom-focused approach rather than a syndromal- focused approach. According to Chilcoat and Breslau, one of the criteria for causation includes a "gradient of effect", or dose response relationship between the two phenomena of interest. That is, as the level of exposure to the causal agent increases, a resulting increase in the level of the causal outcome should be expected. The gradient of effect relationship can be studied as it pertains to the relation of any anxiety-related symptom (e.g., number of panic attacks; severity of cognitive re-experiencing) with any alcohol-related symptom (e.g., severity of negative consequences resulting from alcohol use; usual number of alcohol beverages consumed per week).

The relationship between anxiety and alcohol can also be considered at the syndromal level. At this level, a relationship between alcohol and anxiety would be demonstrated if a diagnosis of one of the two disorders (i.e., anxiety disorder or alcohol use disorder) was associated with an increased likelihood of a diagnosis of the other disorder. The DSM IV (APA, 1994) distinguishes between two distinct types of alcohol use disorders: alcohol abuse and alcohol dependence.

Alcohol abuse is characterized by "recurrent and significant adverse consequences related to the repeated use of alcohol" (p. 198), whereas alcohol dependence must include "evidence of tolerance, withdrawal, or compulsive behavior related to alcohol use" (p.214). Alcohol dependence is considered more severe than alcohol abuse and always overrides the latter diagnosis. As was mentioned earlier, consumption levels are not considered in the diagnosis of either alcohol abuse or dependence. On the other hand, for anxiety disorders, both symptom levels (e.g., repeated panic attacks in the case of panic disorder) and/or negative consequences of the symptoms (e.g., distress about having another panic attack in the case of panic disorder) are considered in making a diagnosis.

Epidemiological Findings on the Anxiety – Alcohol Relationship

The relationship between anxiety and alcohol can be described using the concept of comorbidity, which can be defined as diagnosable, problematic alcohol use and anxiety symptoms that are both present at some point in a person's lifetime, but not necessarily at the same time (Kushner et al., 2000a). Comorbidity rates can be estimated using either clinical or community samples. Because individuals with more than one disorder are more likely to seek treatment, clinical samples may in fact inflate comorbidity estimates (Berkson, 1949). It is believed that community surveys provide more accurate reflections of the anxiety disorder – alcohol use disorder relationship. We will review the two most recent large-scale community surveys, which are representative of the results of these types of surveys.

In this chapter, we will present odds ratios (ORs) to quantify the comorbid relationship between anxiety disorders and alcohol use disorders. An OR reflects the odds that individuals will display a second disorder if a first disorder is present versus if it is not present. An OR of 1.0 reflects a lack of relationship, with higher ORs reflecting more significant relationships between two disorders. ORs of less than 1.0 reflect a decreased probability of having the second disorder given the presence of the first disorder.

The National Comorbidity Survey (NCS: e.g., Kessler et al., 1996) reported 12-month ORs for individuals with alcohol dependence and alcohol abuse of also having suffered from panic disorder (1.7 and 0.5 respectively), social phobia (2.8 and 2.3), generalized anxiety disorder (4.6 and 0.4), posttraumatic stress disorder (2.2 and 1.5), and specific phobias (2.2 and 1.2). The 12-month ORs associated with alcohol dependence are significant for all anxiety disorders with the exception of panic disorder (although the lifetime OR of alcohol dependence is significant for panic disorder). On the other hand, although having any anxiety disorder leads to a significant OR of developing alcohol abuse, the only specific anxiety disorder with a significant OR is social phobia. More recently, the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; e.g., Grant et al., 2004) reported 12-month ORs for individuals with alcohol dependence and abuse to also display any anxiety

disorder (2.6 and 1.1, respectively), panic disorder with agoraphobia (3.6 and 1.4) and without agoraphobia (3.4 and 0.8), social phobia (2.5 and 0.9), a specific phobia (2.2 and 1.1), and generalized anxiety disorder (3.1 and 0.9).[1]

The NCS (Kessler et al., 1997) also examined sex differences in comorbidity between alcohol use disorders and anxiety disorders. Anxiety disordered women and men do not differ significantly in their risk of developing alcohol dependence; however, women with social phobia, simple phobias or post traumatic stress disorder hive higher ORs of abusing alcohol than men with these anxiety disorders. Similarly, the NESARC (Smith et al., 2006) reported ORs of alcohol use disorders and anxiety disorders by race/ethnicity. Across all races/ethnic groups, there were significant ORs of any anxiety disorder with alcohol dependence but not with alcohol abuse. However, the pattern of comorbidity across specific anxiety disorders reveals significant racial/ethnic effects. For Whites and Blacks, the ORs for alcohol dependence and anxiety disorders were significant across almost all anxiety disorders. The only exception was that the OR for panic disorder with agoraphobia was significant for alcohol abuse but not for alcohol dependence among Blacks. On the other hand, for Native Americans and Hispanics, only a few of the anxiety disorders were significantly associated with alcohol dependence and none with alcohol abuse.

Two general conclusions can be made from the data reported across these large-scale community surveys. First, the relationship between anxiety disorders and alcohol dependence appears to be much stronger than between anxiety disorders and alcohol abuse, with the ORs for dependence much more likely to be significant than those for abuse. In other words, having a comorbid anxiety disorder increases the chances of displaying the more severe type of alcohol use disorder (dependence) moreso than the less severe type (abuse), suggesting a gradient of effect relationship. . Second, the relationship between alcohol use disorders and anxiety disorders differs between sexes and racial/ethnic groups. Although alcohol use is generally more common among men than women (Paavola, Vartiainen, & Haukkala, 2004), alcohol abuse and anxiety disorders are more closely related for women than for men. Furthermore, it appears that for Whites and Blacks, anxiety and alcohol dependence are more closely associated than for Native Americans and Hispanics. The following sections cover etiological models of the relationship between alcohol and anxiety, their maintenance, and their relapse, in an attempt to explicate the high level of comorbidity between anxiety disorders and alcohol use disorders observed in the epidemiologic surveys.

Etiological Models of the Relationship

In order to gain a better understanding of the relationship between anxiety and alcohol use, it is important to explore the mechanisms that may affect the etiology of the co-occurrence of symptoms of both of their disorders. There

[1] The NESARC did not specify significance of reported 12-month ORs.

are three main hypotheses that have been put forward, with some evidence supporting each hypothesis. First, there is some evidence that the associations between anxiety and alcohol arise from common underlying variables, such as common genetic or environmental factors, that cause both anxiety symptoms and problematic alcohol use. Second, some believe that certain aspects of problematic alcohol use, such as repeated experiences with alcohol withdrawal, cause anxiety symptoms and ultimately an anxiety disorder. Finally, others argue that anxiety symptoms cause alcohol misuse, culminating in an alcohol use disorder. Evidence examining these three hypotheses is presented below.

Common Underlying Variables

There is some evidence, provided by two main lines of research, to support the hypothesis that certain common, underlying factors are causing both anxiety symptoms and problematic alcohol use (Kushner et al., 2000a). First, family and twin studies have provided some evidence of possible common genetic contributions to the correlation between anxiety symptoms and alcohol consumption (e.g., Tambs, Harris, & Magnus, 1997). Family and twin studies have also examined the heritability of common underlying personality traits associated with both anxiety and alcohol use disorders. For example, several cross-sectional and longitudinal studies have linked the highly heritable personality trait of neuroticism (Jang, Livesley, & Vernon, 1996) with anxiety and its disorders (e.g., Jorm et al., 2000; Weinstock & Whisman, 2006). Neuroticism has also been linked to alcohol use disorders (Cox, 1987). Another personality dimension that is closely related to neuroticism, negative emotionality, has been associated with alcohol use disorders (Swendsen, Conway, Rounsaville, & Merikangas, 2002). In a study by Swendsen et al. (2002) examining heritability of negative emotionality, non-alcoholic individuals with alcoholic relatives did not differ significantly on scores of negative emotionality than those without alcoholic relatives. If negative emotionality were a heritable risk factor, non-alcoholic individuals with alcoholic relatives would score higher on this trait than those without alcoholic relatives. These findings suggest that negative emotionality may indeed be an individual risk factor rather than a heritable risk factor for alcohol use disorders. The inconsistent results between these two related personality traits (i.e. both associated with negative emotional states) demonstrates that common heritability for some of the personality traits relevant to the anxiety – alcohol relationship is still in need of further investigation.

Conversely, another personality risk factor for both anxiety disorders and alcohol use disorders, anxiety sensitivity (i.e. fear of anxiety; Stewart & Kushner, 2001), does have a strong heritable component that accounts for nearly half of the variance in scores on anxiety sensitivity measures (Stein, Lang, & Livesley, 1999). Therefore, it appears that some, but not necessarily all, underlying personality risk factors associated with both alcohol use disorders and anxiety disorders have a shared heritable component.

Second, results from some prospective studies suggest a possible common "third variable" contribution to the alcohol – anxiety relationship. For example, Zimmerman et al. (2003) found that remitted panic disorder and social phobia were as important as current panic/social phobia diagnoses in predicting future alcohol outcomes. That is, even individuals who were not currently experiencing sufficient symptoms to receive any anxiety disorder diagnosis were at higher risk of developing alcohol problems if they had *ever* been diagnosed with either panic or social phobia in the past. These findings can be interpreted to suggest that a third underlying factor (such as a common personality vulnerability or genetic predisposition) was driving both the alcohol problem and the past or current anxiety disorder.

A 21-year longitudinal study (Goodwin, Fergusson, & Horwood, 2004) found that once other factors were controlled (i.e., prior substance dependence, concurrent major depression, and affiliations with deviant peers), the ability of anxiety disorders to predict the development of alcohol dependence was no longer significant. The study points to a number of possible third variables including prior substance dependence which could contribute both to the development of anxiety disorder (see Norton, Norton, Cox, & Belik, in press) and of alcohol dependence (e.g., alcohol is consumed in larger quantities when combined with other substances; Barrett, Darredeau, & Pihl, 2006). Unfortunately the study did not test which of these factors was most important in explaining the link between anxiety and alcohol dependence.

Some factors that have emerged as possible contributors to the increased vulnerability of developing comorbid anxiety and alcohol use problems include either common genetic pre-dispositions (e.g., anxiety sensitivity), biological environment risk factors (e.g., fetal alcohol syndrome), or non-biological environmental risk factors (e.g., disruptive familial environment; Merikangas, Stevens, & Fenton, 1996). Unfortunately, no research has yet confirmed one or more of these candidate factors. More research needs to be conducted exploring these additional underlying mechanisms before one can make conclusions about their influence.

Alcohol Use Causes Anxiety

The second hypothesis dealing with the relationship between anxiety and alcohol use posits that prolonged drinking is actually a causal factor in anxiety symptoms and disorders. This alcohol-induced anxiety can occur through either psychosocial or physiological mechanisms. Psychosocially, it is hypothesized that alcohol may interfere with normal adaptation to stressful stimuli or that negative consequences produced by problematic drinking (e.g., loss of job or relational problems) can lead to anxiety symptoms and increased vulnerability of developing anxiety disorders (Kushner et al., 2000a).

Physiologically, alcohol withdrawal can often produce anxiety symptoms such as shakiness (see Kushner et al., 2000a) or increased startle, a common symptom of PTSD (Stewart et al., 1998). In addition, neural adaptation occurs

with frequent and excessive alcohol use over time such that repeated alcohol withdrawals actually sensitize this withdrawal-induced anxiety (Breese, Overstreet, & Knapp, 2005). This has often been referred to as the "kindling-stress hypothesis"; that is, repeated withdrawals from chronic heavy drinking are thought to worsen, or "kindle" withdrawal-induced anxiety.

A number of studies have also demonstrated increased norepinephrine activity as well as hyperexcitability of the central nervous system, especially of limbic structures, during alcohol withdrawal (Kushner et al., 2000a; Marshall, 1997). These are the same neural systems that have been implicated in panic attacks and panic disorder, providing a possible physiological explanation for the link between panic disorder and alcohol use disorders (Marshall, 1997).

A final area of research supporting the hypothesis that alcohol problems cause anxiety involves prospective studies. One such study by Kushner, Sher, and Erickson (1999), for example, found that a diagnosis of alcohol dependence at baseline quadrupled the risk of developing an anxiety disorder three to six years later. Prospective studies have also examined the relationship between PTSD and alcohol abuse to ascertain whether heavy alcohol use can be a risk factor for developing PTSD. It has been hypothesized that physiological and neurochemical changes due to prolonged heavy alcohol use and/or past reliance on alcohol to deal with life stressors at the expense of developing other coping mechanisms may increase an individual's susceptibility of developing PTSD after a traumatic experience (Brown & Wolfe, 1994; Stewart et al., 1998). A prospective study by Acierno, Resnick, Kilpatrick, Saunders, and Best (1999) found that a history of alcohol abuse increased the risk of developing PTSD in rape victims almost three-fold (OR = 2.65) when compared to the absence of this factor.

Anxiety Causes Alcohol Use

It has been hypothesized that anxiety symptoms and anxiety disorders promote alcohol use, as individuals drink to self-medicate their anxiety. The "self-medication hypothesis" (and the related tension reduction hypothesis) as applied to the understanding of the relationship between anxiety and alcohol posits that the pharmacological and/or psychological effects of alcohol lead to decreases in aversive anxiety symptoms, thereby motivating anxious individuals to increase their quantity and/or frequency of alcohol use via the process of negative reinforcement (Kushner et al., 2000a). Although the self-medication and tension reduction hypotheses clearly do not account for all drinking behavior (Greeley & Oei, 1999), there has been a good deal of empirical evidence to support these hypotheses as they apply to the understanding of comorbid anxiety and alcohol use disorders (Kushner et al., 2000a).

Anxiety disordered individuals do in fact self-report using alcohol to manage their anxiety (Kushner, Abrams, Thuras, & Hanson, 2000b; Thomas, Randall,

& Carrigan, 2003; see also Kushner, et al., 2000a for a review). In addition, Thomas and colleagues (2003) found that socially anxious individuals not only reported that they drank to feel more comfortable in social situations, but that they would actually avoid social situations if alcohol were unavailable.

As introduced earlier in this chapter, one possible criterion for establishing causation (Chilcoat & Breslau, 1998) is a dose response, or gradient of effect relationship: if anxiety causes alcohol use, one would expect that higher levels of anxiety would be associated with higher levels of alcohol use. Studies have found positive correlations between severity of PTSD arousal symptoms and severity of alcohol use disorder symptoms (McFall, Mackay, & Donovan, 1992; Stewart et al., 1998). Because correlation does not determine causation, one must rely on laboratory-based studies, such as a study by Abrams, Kushner, Medina, and Voight (2002), for evidence that induction of anxiety symptoms causes heavier drinking. The study found that participants with social phobia consumed more alcohol following an anxiety provoking activity (speaking in front of a group) than a control activity (reading a book), presumably in an effort to dampen the anxious feelings caused by the anxiety provoking activity.

Prospective research on non-clinical populations also supports a dose-response relationship between anxiety and alcohol use. In a diary-based study by Swendsen and colleagues (2000), moderate drinkers documented their daily drinking and mood states for a one-month period. The study revealed that only anxious feelings and not sadness or other negative affective states preceded and predicted increased alcohol consumption. As can be observed above, findings from correlational, laboratory-based experimental, and diary-based prospective research conducted with both clinical and non-clinical populations converge to provide some evidence for a relationship between anxiety symptoms and alcohol use where anxiety precedes and contributes to increased alcohol use.

Social anxiety appears to have a more complicated relationship with alcohol use than do other types of anxiety, however. Specifically, some studies examining the relationship between social anxiety and alcohol consumption show a positive relationship, whereas other studies show either no linear relationship or even a negative relationship (Ham & Hope, 2005; Stewart, Morris, Mellings, & Komar, 2006; Tran, Haaga, & Chambless, 1997). The negative relationship between social anxiety and alcohol consumption may exist because socially anxious individuals actually avoid the types of social situations that involve drinking *because of their social anxiety*, thus leading to lower levels of alcohol consumption (Stewart et al., 2006). Nonetheless, social anxiety has been found to predict alcohol dependence, as well as problems caused by alcohol (Gilles, Turk, & Fresco, 2006; Stewart et al., 2006). Thus, social anxiety does appear related to alcohol-related consequences, even if it does not always predict increased alcohol use.

Another criterion discussed by Chilcoat and Breslau (1998) as necessary for causation is temporality. If anxiety causes increased or problematic alcohol use, then anxiety symptoms should predate alcohol-related problems, and anxiety

disorder diagnoses should precede alcohol disorder diagnoses. In the cases of social phobia, panic disorder, and PTSD diagnoses, the anxiety disorder usually preceded the onset of the alcohol use disorder in comorbid individuals (Cox, Norton, Swinson, & Endler, 1990; Kushner, Sher, & Beitman, 1990; Stewart & Conrod, 2002).Furthermore, a cross-national investigation by Merikangas et al. (1998) confirmed that anxiety disorders preceded alcohol use disorders for the majority of participants. Assessing temporality or relative order of onset of symptoms of alcohol use disorders and anxiety disorders among comorbid cases can also help elucidate the anxiety – alcohol causal relationship. For example, a recent study by Bernstein, Zvolensky, Sachs-Ericsson, Schmidt, and Bonn-Miller (2006) found that the symptom of panic attacks predated the onset of heavy drinking behaviors for the vast majority of participants with both panic attacks and heavy drinking in a community sample. Although many anxiety disorders and their symptoms predate alcohol use disorders and heavy drinking, this relationship is normally inverse for individuals with generalized anxiety disorder (GAD). For the majority of individuals with GAD, alcohol use disorder predated the GAD (Kushner et al., 1990), suggesting that, for those cases, anxiety could not have been the cause of the alcohol use disorder. Thus, as can be seen from the findings above, for most comorbid individuals (excluding a majority of GAD individuals), the order of onset supports the possibility that anxiety may play a causal role in the development of alcohol use disorders. And conversely, for those with GAD, the alcohol use may play a causal role in the development of the anxiety disorder. However, Chilcoat and Breslau (1998) clarify that the temporality criterion is necessary but not sufficient for determining a causal association between disorders.

Finally, the prospective study by Kushner and colleagues (1999) mentioned earlier in the chapter that found an increased risk of future anxiety disorders for individuals with alcohol dependence also found the converse. That is, having a diagnosis of anxiety at baseline increased the risk three- to five-fold for a new onset of alcohol dependence three to six years later (see also Goodwin et al., 2004). The study demonstrates that the causal relationship between anxiety and alcohol is potentially bi-directional in nature. Overall, the evidence presented above does seem to support the fact that, at least in some instances, anxiety symptoms and anxiety disorders do precede and possibly promote problematic alcohol use.

Moderating and Mediating Variables in the Anxiety – Alcohol Relationship

More recent work has focused on finding specific variables that either moderate or mediate the causal relationship between anxiety and alcohol use. A moderator variable is a qualitative (e.g., sex) or quantitative (e.g., anxiety level) variable that affects the direction and/or strength of the relation between two

other variables (Baron & Kenny, 1986). A mediator variable, on the other hand, explains how or why the relationship between a predictor and given criterion (e.g., between anxiety and alcohol use) exists. That is, the mediator actually accounts for the relationship between the two variables (Baron & Kenny).

Alcohol expectancies. A potential moderator variable between anxiety and problematic alcohol use includes certain "alcohol outcome expectancies" (i.e., beliefs about the consequences of drinking alcohol). For anxious individuals who self-medicate to avoid anxiety, an important aspect of the self-medication hypothesis involves the notion that self-medicators anticipate anxiety, and that they expect that alcohol will actually decrease their feelings of anxiety (e.g., Kushner, Sher, Wood, & Wood, 1994; Tran et al., 1997). Studies have shown that tension reduction expectancies predict drinking frequency and quantity in non-alcoholic drinkers with panic disorder (Kushner et al., 2000b) and comorbid problem drinking in women with PTSD (Ullman, Filipas, & Townswend, 2005). These results support the role of tension reduction alcohol expectancies as a moderator: increased or problematic drinking occurs among anxiety disorder patients only when tension reduction alcohol expectancies are present.

Another methodology that has been employed to investigate the role of alcohol outcome expectancies in the anxiety – alcohol relationship is the experimental manipulation of expectancies via the placebo-controlled design. If expecting alcohol were to induce a cognitive or placebo-induced anxiety reducing effect among anxious individuals, such an effect would provide additional evidence for the contribution of alcohol expectancies in explaining the anxiety – alcohol relationship. The empirical evidence provided thus far has found mixed results for this placebo anxiolytic effect. Some studies have found that the belief that one was consuming alcohol, even when one was actually consuming a placebo, was enough to lower feelings of anxiety among anxiety-disordered patients (Abrams, Kushner, Lisdahl, Medina, & Voight, 2001; Lehman, Brown, Palfai, & Barlow 2002). On the other hand, research by MacDonald, Stewart, Hutson, Rhyno, and Loughlin (2001) conducted with participants high in anxiety sensitivity did not support a cognitively-mediated tension reduction effect of alcohol. The researchers actually found a "reverse placebo" effect, where high AS participants in a placebo condition, who had expectations of alcohol-induced tension reduction, but did not benefit from alcohol's physiological tension-reduction properties, appeared to have even higher levels of anxiety than participants in a control condition where they neither received nor expected alcohol. Regardless of the direction of the placebo effect, all of these findings do suggest a role for cognitive expectancy variables in accounting for the effects of alcohol among anxious individuals.

A number of studies have examined specific aspects of alcohol expectancies in individuals with social anxiety. For people high in social anxiety, expecting that alcohol would decrease social anxiety or increase social assertiveness was associated with both higher self-reported drinking quantities (Tran et al., 1997) and higher alcohol consumption in a laboratory setting (Kidorf & Lang, 1999). More general tension reduction expectancies, on the other hand, had no effect

on alcohol consumption. In addition, socially related alcohol expectancies have been associated with higher levels of alcohol dependence in socially anxious individuals (Ham, Carrigan, Moak, & Randall, 2005).

These studies demonstrate that different expectancies affect alcohol consumption depending on the particular type of anxiety-related psychopathology involved. For individuals with panic disorder or PTSD, the research until now appears to indicate that general tension reduction expectancies provide sufficient motivation for increasing alcohol consumption. However, for individuals with social anxiety, it appears that specific social-related alcohol expectancies, but not general tension reduction expectancies, tend to motivate these individuals to drink, resulting in greater risk of developing alcohol dependence. As has been done with social anxiety, increasingly directed research in other types of anxiety-related psychopathology is needed to determine more precisely the kinds of expectancies that help explain each group's increased risk for alcohol problems in order to target these more specifically in treatment.

Self-efficacy. Self-efficacy has been defined by Bandura (1977) as the conviction that one can successfully execute a behavior required to produce a certain outcome. Research conducted with socially anxious individuals suggests that self-efficacy may act as a link between alcohol expectancies, social anxiety and heavy drinking behaviors. Positive socially related alcohol expectancies and low self-efficacy to avoid heavy drinking interact with one another in increasing problematic drinking among socially anxious individuals (Burke & Stephens, 1997; Gilles, Turk, & Fresco, 2006). Thus, increasing individuals' self-efficacy in refusing alcohol or avoiding heavy drinking could be incorporated when developing treatment programs for comorbid individuals, at least in the case of social phobia comorbidity. More research is needed to determine the relevance of the self-efficacy construct to the comorbidity of alcohol use disorders with anxiety disorders other than social phobia.

Drinking motives. Problematic drinking can arise because of maladaptive drinking motives (reasons for drinking) even in the absence of particularly elevated levels of alcohol consumption (Stewart et al., 2006). For some individuals who experience fear or anxiety, avoidance behaviors, such as drinking to self-medicate, become negative reinforcement strategies used to attenuate or cope with anxious states. Because such avoidance strategies are negatively reinforcing through their effects in alleviating anxiety, the drinking behavior continues over time and the individual comes to rely on drinking as a primary coping strategy. For example, in many cases, social anxiety is not associated with higher drinking levels (Tran et al., 1997), but it is with problematic reasons for drinking, such as drinking to cope with negative emotional states, as well as with greater risks of developing alcohol problems (Thomas, Randall, & Carrigan, 2003; Stewart et al., 2006). A further problem with coping-related drinking is that, as an avoidance behavior, it may serve to maintain anxiety by preventing habituation of the anxiety response.

Research by Cooper (1994; see also Cooper, Russell, Skinner, & Windle, 1992) exploring drinking motives and their relation to problematic drinking

uncovered four factors explaining motivation for drinking. Two of these motives involve positive reinforcement from drinking: social (i.e. to obtain social rewards) and enhancement (i.e. to enhance positive mood or well being), whereas the other two motives involve negative reinforcement from drinking: coping (i.e. to cope with negative emotions), and conformity (i.e. to avoid negative social consequences such as social rejection). Only the negative reinforcement motives of coping and conformity predict alcohol problems after controlling for quantity and frequency of alcohol use.

A study by Stewart and colleagues (2006) also found that for undergraduate students, problem-drinking symptoms were positively associated with the negative reinforcement motives of coping and conformity drinking. The study also found that coping and conformity drinking motives mediated the relationship between social anxiety (specifically, fear of negative evaluation) and drinking problems. That is, individuals with social anxiety experienced drinking problems because they drank either to cope with negative emotions (i.e. coping drinking motives), or to avoid negative social consequences (i.e. conformity drinking motives). In addition, the study found either no association or even a negative direction association between social anxiety measures and drinking quantity and frequency. Together, research by Cooper and her colleagues and Stewart and her colleagues support the notion that drinking to cope with anxiety or to conform with peer pressure confers additional risk for problem drinking over and above the risks associated with level of alcohol consumption.

Additional support for coping and conformity drinking motives as important factors in the anxiety – alcohol relationship has been provided by studies conducted with non-clinically anxious participants. For example, Deacon and Valentiner (2000) found a significant association between scores on the Beck Anxiety Inventory (BAI, a widely-used measure of anxiety symptoms) and coping motivated drinking but not social or enhancement motivated drinking. In addition, Thomas et al. (2003) found that socially anxious individuals more often reported drinking before and during social situations in an effort to feel more comfortable (i.e. to cope with their social anxiety) than non-socially anxious individuals.

Anxiety sensitivity has been associated with greater drinking levels and argued to be a potential risk factor for alcohol use disorders (Stewart & Kushner, 2001), as we describe in greater detail in the next section. Results from a study by Stewart et al. (2001) supported coping and conformity motives as mediators in the relationship between AS and higher drinking levels. That is, high AS individuals' greater drinking behavior was at least partially explained by high coping and conformity motive scores.

Anxiety sensitivity. Anxiety sensitivity (AS) is an important individual difference that may mediate the anxiety – alcohol relationship. It is possible that anxiety disordered patients are at increased risk of alcohol problems because of their higher levels of AS (i.e., higher levels of fear of anxiety), which, in turn, promote greater motivation and behaviors (e.g., alcohol use) to escape or avoid the feared symptoms (Stewart & Kushner, 2001). Thus, consistent with the

self-medication hypothesis, it is possible that individuals high in AS use alcohol for its anxiolytic or arousal dampening effects. In fact, high AS individuals have been found to prefer alcohol and other "depressants" over "stimulant" type drugs (DeHaas, Calamari, & Bair, 2002; Norton, Rockman, & Ediger, 1997). Additionally, elevated levels of AS have been associated with increased drinking behavior, including increased typical weekly drinking frequency, and yearly excessive drinking frequency (Stewart, Peterson, & Pihl, 1995; Cox & Klinger, 1988; Stewart, Zvolensky, & Eifert, 2001). Moreover, Stewart, Conrod, Samoluk, Pihl, and Dongier (2000) found evidence for the mediating role of AS in explaining the association between PTSD symptoms and negative reinforcement drinking.

Stewart et al. (2001) also examined the relationship between lower-order components of AS and drinking behavior. AS consists of three lower-order factors: AS physical concerns (e.g., worrying that a rapidly beating heart is a sign of having a heart attack), AS psychological concerns (e.g., worrying that not being able to keep one's mind on a task is a sign of going crazy), and AS social concerns (e.g., worrying about appearing nervous in front of others; see Zinbarg, Mohlman, & Hong, 1999). After controlling for the other two factors, AS social concerns emerged as the only significant predictor for weekly drinking frequency and yearly excessive drinking frequency. Thus, it is possible that AS also plays an important role in the relationship between social anxiety and drinking problems given the elevation of AS social concerns among those with social phobia (Zinbarg et al., 1999).

Summary. Although these mediating and moderating variables are typically studied in isolation, as can be seen above, alcohol expectancies, self-efficacy, drinking motives, and anxiety sensitivity may interact with each other or intervene with one another in explaining problematic alcohol use in anxious individuals. For example, people with certain anxiety disorders may be more likely to drink to cope with their anxiety sensations (coping motives) because of their fear of these sensations (anxiety sensitivity), thereby increasing their risk for drinking problems. Additional research exploring the interplay between the different variables affecting the anxiety-alcohol relationship would provide important steps toward creating more effective treatments for comorbid individuals.

Anxiety, Alcohol Use, and Other Health Behaviors

Alcohol consumption has been shown to be highly related to risky health behaviors, such as smoking and illicit drug use (Paavola, Vartiainen, & Haukkala, 2004; Tolstrup et al., 2005). In a longitudinal study (Paavola et al., 2004), earlier alcohol use was associated with later smoking, and smoking in adolescence predicted alcohol use in adulthood. Despite this close association between alcohol use and smoking, combining the two behaviors does not

increase the rate of anxiety disorders above the rate associated with alcohol use only (Kandel, Huang, & Davies, 2001).

A diagnosis of an anxiety disorder combined with a drug use disorder constitutes a significant risk factor for developing alcohol dependence (lifetime OR = 5.81; Kessler et al., 1997). This increased risk appears to be even higher than the risk associated with being diagnosed with an anxiety disorder alone (i.e. without a drug use disorder; lifetime OR = 1.85). Unfortunately, like many epidemiological surveys, the National Comorbidity Survey does not break drug use disorders down by drug type/class, which could further elucidate the drug – anxiety – alcohol relationship. One possible explanation for this elevated risk for alcohol dependence among those with comorbid anxiety – drug use disorders is that problematic drug use, through the drugs' potentially anxiogenic effects, exacerbates the need to self-medicate with alcohol, resulting in increased risk for alcohol dependence relative to those with non-comorbid anxiety disorders.

Maintenance of Comorbid Anxiety and Alcohol Problems

Empirical findings supporting all three causal hypotheses suggest the possibility of multiple causal pathways involved in the etiology of comorbid alcohol use disorders and anxiety disorders. The specific causal pathway involved may vary across people or across anxiety sub-types. Regardless of the etiology of the comorbid anxiety symptoms and problematic alcohol use, there have been countless studies confirming that, once comorbid, anxiety and alcohol use do, in fact, exert important influences on each other (see Kushner et al., 2000a). Furthermore, processes involved in the initiation of the comorbidity may differ from those involved in the maintenance of problematic alcohol use and anxiety. A feed-forward model has been proposed (Kushner et al., 2000a) in which once both alcohol use and anxiety are present, each promotes the maintenance or exacerbation of the other. For example, anxious individuals may resort to alcohol to decrease feelings of anxiety, which might be an effective strategy in the short term, providing reinforcement for this pattern. However, alcohol, and especially withdrawal from alcohol, increases anxiety-like symptoms in the longer run via physiological mechanisms such as kindling. Alcohol may also worsen anxiety levels because of the negative familial, social or occupational consequences of heavy drinking. Individuals will then increase their drinking behavior in an attempt to alleviate these worsening feelings of anxiety because drinking has become a learned strategy for dealing with these symptoms, especially if there is a failure to recognize that the alcohol may actually be promoting the anxiety in the medium to long term.

Finally, in individuals with PTSD, alcohol that is used to cope with anxiety may prevent normal "habituation" of the anxiety symptoms following trauma exposure. On the other hand, for individuals who are not drinking, anxious

feelings caused by PTSD may naturally remit with time. Thus, when individuals drink in an attempt to numb or avoid these feelings, they may be preventing this natural recovery from taking place, leading to maintenance of the anxiety symptoms in the long run (Stewart et al., 1998).

Treatment Outcome and Relapse

When individuals who suffer from both anxiety disorders and alcohol use disorders enter treatment for either disorder, their treatment outcome is often negatively affected by their comorbidity. Alcohol use disorders have been found to predict poorer anxiety disorder treatment outcomes for patients with both PTSD (Forbes, Creamer, Hawthorne, Allen, & McHugh, 2003), panic disorder with agoraphobia, social phobia, and generalized anxiety disorder (Bruce et al., 2005).

Comorbid anxiety problems also increase the likelihood of relapse in treated or abstinent alcoholics (e.g., Driessen et al., 2001; Kushner et al., 2005; Willinger et al., 2002). For example, if a comorbid PTSD – alcoholic individual does not know how to cope with flashbacks and nightmares of the traumatic event in ways other than through drinking, then continued re-experiencing symptoms can serve as a major risk factor for return to problem alcohol use following initially effective alcohol abuse treatment. Not all studies have shown this relationship, however. In one study (LaBounty, Hatsukami, Morgan, & Nelson, 1992), alcoholics with comorbid panic disorder did not differ in their rates of relapse to drinking problems from non comorbid alcoholics. However, in a recent review, Bradizza et al. (2006) noted some methodological issues with this paper, including the absence of a valid and reliable diagnostic measure and the failure to define relapse, limiting the conclusions that could be drawn from the study. Moreover, despite the similar relapse rates, the study did find that more comorbid alcoholic and panic disordered patients reported relapsing to cope with negative emotions than non comorbid alcoholics. Thus, these observed differences in the relapse process might be useful for improving treatments for this group.

Another study compared relapse rates for alcoholic individuals with comorbid social phobia, or panic disorder with agoraphobia, or agoraphobia without a history of panic attacks to relapse rates for alcoholics without any comorbid anxiety disorder (individuals with other anxiety disorders were excluded) following treatment for alcoholism (Marquenie et al., 2006). The results suggested that the comorbid anxiety disorders did not have a significant impact on either relapse rates or days to relapse. Nonetheless, some methodological problems may account for this study's failure to support higher alcoholism relapse among treated alcoholic patients with comorbid anxiety disorders. First, the study used a retrospective design. Participants were contacted for the study an average of 20.3 months (and up to 42 months) after baseline assessment, even though the majority of participants who relapsed did so within the first few months post

baseline, raising issues concerning potential inaccuracy in the self-reports due to retrospective memory bias. Also, compared to the non comorbid group, the comorbid group had less chronic alcoholism and a shorter period between the initial and follow-up assessments, which could have led to underestimates of the rate of relapse in the comorbid group. Although the authors stated the failure to observe group differences in alcoholism relapse persisted when these group differences were statistically controlled in the analyses, statistical control of possible confounds is never definitive, (and in fact may even produce biased parameter estimates) especially when the confounding variables are correlated with the predictor of interest, again limiting any firm conclusions that can be drawn from the Marquenie et al. study.

Several studies, on the other hand, have found higher relapse rates in comorbid patients. Driessen et al. (2001) found that treated alcoholic patients with comorbid anxiety had 29% higher alcoholism relapse rates than did alcoholics without comorbid anxiety. Furthermore, a study examining the relationship between trait anxiety and relapse in a sample of abstinent alcohol dependent patients found that higher trait anxiety was significantly predictive of relapse to uncontrolled drinking (Willinger et al., 2002). Finally, in the best-controlled study to date, Kushner et al. (2005) found that alcoholic patients with an anxiety disorder (especially those with comorbid panic disorder or social phobia) were significantly more likely to relapse to problem drinking (using multiple criteria for drinking relapse) than alcoholic patients without an anxiety disorder. These finding are particularly convincing given the methodological soundness of the study. For example, the study used a prospective design where all participants were contacted between 90 and 120 days after the beginning of treatment, thus increasing reporting accuracy by reducing reliance on long-term retrospective memory. Also, all participants were given the same standardized treatment and assessed at a consistent time following treatment.

Taken together, it appears that comorbid alcohol and anxiety may have a negative effect on treatment outcome and relapse but with some mixed results. The methodologically superior studies do seem to suggest such a negative effect. In addition, although a couple of studies failed to show differences in relapse rates, one of these negative studies did provide some evidence for the self-medication hypothesis through highlighting the importance of coping with anxiety in explaining relapse to alcohol use among patients with comorbid alcohol and anxiety disorders. These differences could be useful in developing specific relapse prevention types of treatments (see Marlatt & Donovan, 2005) for comorbid patients.

Treatments of Comorbid Anxiety and Alcohol Use Disorders

Individuals who suffer from both anxiety problems and alcohol use problems present a special and challenging population with regards to treatment. As was shown above, this population often suffers worse anxiety and alcohol treatment

outcomes than populations experiencing symptoms in only one of the two domains. Although the study of specific treatments for anxiety – alcohol comorbidity is still in its infancy, this area is growing and there are now several promising approaches to treating comorbid anxiety and alcohol use disorders (Stewart & Conrod, in press).

There have been mixed findings regarding the effects of pharmacological treatment for anxiety on drinking outcomes with some studies finding improvements in alcohol outcomes and others finding more equivocal results (see review by Kushner et al., 2000a). One study (Randall et al., 2001) found that treating individuals with comorbid social phobia and alcohol use disorder with paroxetine (a selective serotonin reuptake inhibitor) did improve anxiety, but did not result in significant decreases in drinking frequency and quantity. Relative to placebo, paroxetine treatment did, on the other hand, lead to improvements on the Clinical Global Index for alcohol. Other studies have found that successful treatment of anxiety with buspirone was also associated with a reduction in alcohol use (Tollefson, Montague-Clouse, & Tollefson, 1992; Kranzler et al., 1994). These mixed findings are partially consistent with the self-medication hypothesis, although the paroxetine study does suggest that there is more to the maintenance of problematic drinking behavior in anxious individuals than just the self-medication process.

There have also been mixed findings for the effectiveness of cognitive behavioral therapy (CBT) to improve anxiety and problematic drinking symptoms in comorbid patients. Thevos et al. (2000) found that, for female patients with comorbid social phobia and alcohol use disorders involved in project MATCH (the largest treatment-matching trial to date), CBT treatment for alcohol was more effective in delaying return to drinking than Twelve-Step Facilitation (TSF). It was hypothesized that TSF, which encourages participation in Alcoholics Anonymous (AA), a group-based treatment modality that heavily involves public speaking as patients share their experiences, may be too intimidating for women with social phobia. Subsequently, Randall, Thomas, & Thevos, (2001) examined whether conducting parallel CBT treatments aimed at decreasing social anxiety and at addressing problematic drinking behaviors would have additional benefits for comorbid patients compared to treatment of the alcohol disorder alone. For patients receiving the parallel treatments, the sessions consisted of CBT treatment for alcohol followed immediately by CBT for social anxiety (i.e. the two treatments are offered simultaneously, but independently of each other). Surprisingly, they found that patients who participated in the parallel treatment had worse drinking outcomes, as assessed by drinking quantity and frequency measures, than did patients who participated in the alcohol only treatment. There are several possible explanations for these unexpected findings. It is possible that clients in the parallel treatment group engaged in more social situations as a consequence of their social phobia treatment, resulting in more opportunities to drink. Additional research needs to be conducted that would include other types of outcome measures that are not specifically linked to frequency or quantity of drinking. As was mentioned

earlier in the chapter, coping drinking motives and problematic consequences of drinking are useful therapy targets for comorbid individuals, particularly in the case of social phobia. It is also possible that the lack of integration of the two treatments or the excessive demands of combining two already intensive treatments may have affected results. The parallel treatment did in fact lead to somewhat higher drop out rates than the alcohol treatment alone, suggesting that the parallel treatment may have been too much for comorbid patients to handle (Conrod & Stewart, 2005).

On the other hand, another study (Bowen, D'Arcy, Keegan, & Senthilselvan, 2000) found that parallel CBT for panic disorder and standard alcohol treatment in a group of comorbid panic disordered and alcoholic patients did not result in significantly different treatment outcomes than standard alcohol treatment alone. Both treatments resulted in significant decreases in anxiety and in drinking behaviors. The authors noted that the relaxation training and stress management components of the standard alcohol treatment might have limited the ability to distinguish between treatments as these components may have been useful in targeting the comorbid anxiety.

A recent randomly-controlled study conducted by Schade and colleagues (2005) compared standard alcohol treatment alone to standard alcohol treatment with anxiety treatment consisting of CBT plus optional fluvoxamine (a selective serotonin re-uptake inhibitor) treatment (again, a parallel approach) in patients with a primary diagnosis of alcohol dependence and a comorbid diagnosis of panic disorder, agoraphobia, or social phobia. There were no differences in alcohol outcome measures between both groups of patients. The additional anxiety treatment did, on the other hand, improve anxiety symptoms. It can be speculated that improved anxiety scores are significant for this population, as decreased anxiety may serve as a protective factor for longer-term outcomes. The study examined outcome results 32 weeks after initial assessment, but did not look at longer- term outcomes (1 year or later) in these patients. Future studies should examine longer-term treatment outcomes in comorbid anxiety and alcohol patients.

Few studies, however, have reported outcomes for truly integrated treatments. Integrated treatment models recognize the complex relationship between anxiety disorders and alcohol use disorders and their possible mutual maintenance (Zahradnik & Stewart, in press). Furthermore, their aim is to create a hybrid of the treatments that work best for each disorder separately, and also include in the treatment strategy an understanding of the reciprocal influences each disorder has on the other (Zahradnik & Stewart). Integrated treatments have been developed and tested for certain anxiety disorder – substance use disorder combinations, but until now only one study (Kushner et al., 2006) has investigated an integrated treatment focusing on comorbidity with alcohol use disorders in particular. The treatment integrated CBT for panic disorder with content focusing on the interaction between alcohol use and panic symptoms. The integrated treatment was provided on top of treatment as usual (TAU) for the alcohol use disorder and compared to a group who received only the TAU.

The trial was conducted on a sample of comorbid panic disorder – alcoholic patients, with promising results. The group receiving the integrated treatment showed better anxiety and alcohol outcomes than the TAU alcohol only treatment group.

It is hoped that integrated treatments will provide a more effective strategy in treating comorbid patients. Integrated treatments appear to be the most recommended by "expert opinion"; however few of the recommendations are supported by randomized controlled trials, or even quasi-experimental designs (Watkins, Hunter, Burnman, Pincus, & Nicholson, 2005). More research needs to be conducted to develop and test integrated treatment strategies and to compare them against parallel or sequential approaches.

Prevention and/or Early Intervention

Another treatment approach receiving recent research attention involves targeting the vulnerability factors (e.g., AS) associated with problematic drinking in cases with, or at risk for, comorbid anxiety disorders (Conrod, Stewart, Comeau, & MacLean, 2006, Watt et al., 2006). A study by Watt et al. found promising results for brief CBT targeted at reducing AS in a sample of university women. Three 50-minute CBT sessions led to a decrease in the proportion of women with high AS who engaged in negative consequence drinking (based on elevated scores on the Rutgers Alcohol Problem Index; RAPI; White & Labouvie, 1989), as well as a decrease in conformity-motivated drinking (a high-risk drinking motive; Cooper et al., 1992; Cooper, 1994) and in emotional relief alcohol expectancies (i.e. a positive alcohol expectancy similar to tension reduction expectancies described earlier). The treatment also significantly reduced AS levels (Watt et al., 2006). Another similar AS-focused brief CBT delayed drinking onset in young at-risk adolescents (mean age = 14; Conrod, Castellanos, & Mackie, in press). Later drinking onset has been shown to be a factor protecting against the development of later alcohol use problems (Grant, Stinson, & Harford, 2001). The intervention also reduced panic attacks in high AS adolescents (Castellanos & Conrod, 2006). Together, these results suggest that brief CBT focused on reduction or management of AS may be a useful strategy for prevention of or early intervention with anxiety – alcohol use disorder comorbidity.

Conclusion

Although there is ample evidence supporting the existence of a strong relationship between anxiety and its disorders, and alcohol use and its disorders, much remains unknown regarding the nature of the relationship. More research examining particular circumstances in which anxious individuals are more

likely to self-medicate needs to be conducted, with a focus on potential differences in high-risk drinking situations across the various anxiety disorders. In addition, there has been little research about protective factors that can decrease anxious individuals' needs to self-medicate their anxiety symptoms. For example, a few studies have explored the role of self-efficacy in heavy drinking among individuals with anxiety disorders (Burke & Stephens, 1997; Gilles et al., 2006). Initial results suggest that it might be beneficial for treatments to incorporate specific strategies to increase anxious individuals' feelings of self-efficacy, especially about avoiding heavy drinking in particular high risk situations (e.g., those involving anxiety). Increased knowledge about this and other potential protective factors may be useful when designing specific treatments for comorbid populations (Burke & Stephens, 1997).

It has also been found that alcohol expectancies, drinking motives, and anxiety sensitivity all moderate or mediate the relationship between anxiety and alcohol use/abuse. In addition, these three variables have been found to interact with or intervene with each other when explaining reasons and circumstances for increased and problematic alcohol use in anxious individuals (e.g., Stewart et al., 2001). More research exploring the interplay between these and other moderating and mediating variables would provide deeper and more complete understanding of the precise mechanisms through which anxiety and alcohol use affect one another.

Finally, although research on treating and preventing comorbid anxiety disorders and alcohol use disorders is still in relatively early stages, promising approaches are emerging in this growing clinical research area. For example, in prevention/early intervention, recent studies have shown promising results for targeting personality risk factors directly (e.g., AS) in attempts to reduce both emergent problematic drinking (Conrod et al., 2006; Watt et al., 2006) and emerging anxiety disorder symptoms (Castellanos & Conrod, 2006). Recent research aimed at developing truly integrated treatments for comorbid patients that consider the interplay between anxiety symptoms and drinking behaviors (Kushner et al., 2006) has also provided positive preliminary results. Treatment research efforts should continue exploring ways to address the factors discussed in this chapter (e.g., moderating and/or mediating factors) linking anxiety and problematic alcohol use to improve the efficacy of treatments we have available for comorbid patients.

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