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ANXIETY DISORDERS (DJ STEIN, SECTION EDITOR)

Exploring the Comorbidity of Anxiety and Substance Use Disorders

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Abstract Anxiety disorders and substance use disorders are highly comorbid, and such comorbidity complicates treatment and worsens prognosis. The mechanisms underlying the relationship between anxiety and substance use disorders are poorly understood. This paper reviews recent research attempting to explain these associations. Cognitive factors, such as attentional bias, expectancies, and anxiety sensitivity, appear to impact on the relation between anxiety and substance misuse. Temporality of the anxiety and substance use disorder may also indicate whether the substance use disorder is primary (anxiety may be a result of use) or secondary (substances may be used to self-medicate). Social phobia has been predominantly identified as a primary disorder preceding substance use, while the temporality of other anxiety and substance use disorders is less clear. The efficacy of concurrent treatment compared with separate treatment of either anxiety or substance use disorder is unclear and requires further research.

Keywords Anxiety disorders · Substance use disorders · Comorbidity · Temporality · Anxiety sensitivity · Expectancies · Attentional bias · Obsessive-compulsive disorder · Alcohol · Social phobia

Introduction

The high comorbidity of anxiety disorders (ADs) and substance use disorders (SUDs) has been repeatedly demonstrated among the general population [1, 2, 3•] and patients in treatment [4, 5]. A large-scale epidemiologic survey conducted in the United States found that having a SUD increased the risk of an AD by 1.7 to 2.8 times [1]. Similarly, Dutch data demonstrated that individuals with a pure AD were 2.4 times more likely to be diagnosed with alcohol dependence than controls [6]. Data from the National Comorbidity Survey-Replication in the United States (n=9,282) indicated a positive association between all ADs tested (social phobia, generalized anxiety disorder, panic disorder, agoraphobia) and alcohol abuse, alcohol dependence, drug abuse, and drug dependence [3•].

Comorbidity of ADs and SUDs tends to complicate treatment and contributes to a negative prognosis of both sets of disorders [7]. For example, people with comorbid alcohol use disorder and obsessive-compulsive disorder (OCD) were found to have an increased risk of suicide [8], while untreated ADs tend to result in poorer substance use outcomes and a greater risk of relapse [9].

Although the high comorbidity of ADs and SUDs and the resultant poorer prognosis have been established, the underlying mechanisms affecting the association between ADs and SUDs are not clear. This review highlights different explanations of these underlying mechanisms, with emphasis on recent research.

Theories of Association Between Anxiety and Substance Use Disorders

Although the majority of research indicates that SUDs and ADs are positively associated [3•, 6], the association between ADs and SUDs can be conceptualized in a number of ways. Lehman et al. (as reviewed in [10]) described these as follows: 1) the AD is the primary disorder, and substances are used to cope with the symptoms of the illness; 2) the

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SUD is the primary disorder, and anxiety symptoms develop as a consequence of substance use or withdrawal; 3) the AD and SUD are unrelated but may interact with each other; or 4) both ADs and SUDs share a common genetic or psychosocial cause.

Traditionally, theories that regard the AD as the primary disorder include the tension reduction theory [11], the stress response dampening model [12], and the self-medication hypothesis [13]. When combined, these theories argue that substances such as alcohol reduce both physiological and cognitive anxiety, thereby negatively reinforcing the use of these substances. Over time, internal anxiety and certain contextual situations (eg, social events in the case of individuals with social phobia) become conditioned cues for the use of substances [14]. However, these models have been criticized, as they fail to take into account differences in contextual situations, individual differences, and cognitive factors such as alcohol expectancies [14]. Martins and Gorelick [2] argue that analysis of data from the National Epidemiological Survey on Alcohol and Related Conditions in the United States (n=43,093) does not support the self-medication hypothesis. They reason that if individuals use substances to alleviate particular psychiatric symptoms, the choice of preferred drug should be fairly similar across disorders (ie, those individuals with anxiety should be more likely to use central nervous system [CNS] depressants, whereas those diagnosed with major depression would be more likely to use stimulants). However, respondents with mood disorders only and ADs only had similar adjusted odds ratios (ORs) of developing lifetime abuse or dependence for both depressants (alcohol, cannabis, sedatives) and stimulants (cocaine, amphetamine). However, Buckner et al. [15] suggest that although cocaine is a stimulant, it may still increase positive affect and allow a means to escape distress.

Temporality of the Association Between Anxiety and Substance Use Disorders

In order to test the self-medication hypothesis, it is important to establish the temporality of comorbid disorders. For a disorder to be considered primary, it needs to, by definition, occur prior to the comorbid disorder. It is also probable that the association between SUDs and ADs may differ by the type of AD. The research examining the relationship between various ADs and SUDs is limited, as many studies examine combined ADs and SUDs rather than separate diagnoses [3•]. Some research has, however, examined the temporality of comorbid ADs and SUDs [3•, 6, 16]. For example, Boschloo et al. [6] found that alcohol dependence was secondary to ADs in 71% of individuals with a pure AD. In comparison, alcohol dependence among individuals with a pure depressive disorder was equally likely to have a primary or secondary onset.

Research appears to indicate that social anxiety disorder (social phobia) often has an onset prior to that of SUDs [3•, 4, 6, 16, 17]. Marmorstein [3•] found that among patients with both social phobia and an SUD, social phobia occurred first in at least 90% of the participants, while Boschloo et al. [6] found that alcohol dependence was four times more likely to be secondary to social phobia than to other ADs. Phobic disorders, including social phobia, have also been shown to precede the onset of opiate dependence [4]. However, it should be noted that social phobia tends to have a much earlier age at onset than other ADs [6], which may explain the temporality of the association. To examine the temporality of ADs and alcohol use disorders more clearly, Buckner and Turner [17] used a prospective dataset in which participants were assessed again after a 3-year period. They found that social phobia at time 1 was predictive of alcohol use disorder at time 2, while alcohol use disorder at time 1 did not predict social phobia at time 2, even after controlling for other SUDs and mental disorders at time 1. However, this relationship was only evident for women. Family cohesion and adverse family relations were identified as important moderators among women, with individuals with low family cohesion and adverse family relations having greater rates of alcohol use disorder at time 2 [17].

Although the evidence for social phobia as a primary disorder is relatively strong, the temporality of other ADs and SUDs is less clear. There is less research examining the comorbidity of other ADs and SUDs, and much of the available research also tends to focus on alcohol use disorders. Buckner and Turner [17] found that besides social phobia, other ADs (panic disorder, generalized anxiety disorder, posttraumatic stress disorder) did not significantly predict the development of alcohol use disorders. Marmorstein's [3•] review also indicates that generalized anxiety disorder tends to occur after the onset of the earliest SUD. In contrast, in a prospective study with Finnish adolescents, anxiety was predictive of a higher incidence of frequent alcohol and cannabis use. However, anxiety was measured by a single question, and alcohol and cannabis use were not measured with clinically significant indicators [18]. The temporal relationship between panic disorder and SUDs appears to vary according to the type of SUD. Panic disorder tends to occur prior to the onset of alcohol dependence, but temporality varies with other SUDs with about equal frequency [3•]. Research examining the comorbidity of OCD and alcohol use disorders appears to indicate that OCD often precedes the development of alcohol use disorders [8, 19]. About 70% of patients with OCD indicated that OCD preceded the onset of an SUD by at least 1 year [19], while in another study, 80% of participants reported the emergence of obsessive-compulsive symptoms during childhood, whereas alcohol misuse appeared primarily during early-adulthood [8]. Agoraphobia has been found to occur prior to most SUDs [3•].

It also has been suggested that the temporal sequencing of disorders is differentially associated with the characteristics of individuals with these disorders [6, 17]. In a Dutch study examining alcohol use disorders among anxious or depressed individuals, those with secondary alcohol dependence were described as more neurotic, single, and lonelier than those individuals with primary alcohol dependence, who were more often male and extroverts [6]. This may provide some support for the self-medication hypothesis, with neurotic and lonely individuals being more prone to use alcohol to cope.

Cognitive Explanations of Comorbidity

Cognitive processes, such as attentional bias, expectancies, anxiety sensitivity (AS), and drinking motives, have been explored in an effort to understand the impact of alcohol and other drugs on the processing of anxiety-related cues. Attentional bias refers to "the degree to which threatening or anxiety-related cues are preferentially attended to in the presence of stimuli with less motivational significance" [10]. Recently, Bacon and Ham [10] proposed an avoidance-coping cognitive model that identifies the role of alcohol in reducing the attentional biases to social threat as the link between SUDs and ADs. The model makes a distinction between controlled and automatic processing of social threat, with alcohol tending to automatically reduce the attentional bias to social threat, thereby leading to a reduction in anxiety. However, this model tends to focus on the development of alcohol problems among individuals with ADs rather than the maintenance thereof.

AS is the fear of anxiety-related bodily sensations, such as increased heart and muscle tension, that is caused by the belief that these symptoms will result in negative physical, mental, or social outcomes [15]. AS has been identified as a risk factor for SUDs and has been linked to coping and conformity motives for risky drinking [20]. Individuals with high AS appear to be more vulnerable to using substances to manage their anxiety [20]. Although not a focus of this paper, it is notable that the neural circuitry in anxiety and SUDs overlaps, with, for example, serotonergic dysregulation evident in comorbid mood, anxiety, and addictive disorders [21–23]. Targeting the serotonin system has been suggested as a possible strategy in developing substance abuse treatment, particularly due to its importance in regulating impulsivity [22]. However, the relationship among AS, drinking motives, and alcohol consumption may vary by gender, and this may be because men and women experience different situations as anxiety provoking [20]. Although it is therefore thought that high AS is related to greater use of substances with anxiolytic properties, such as CNS depressants, Buckner et al. [15] found that individuals with cocaine dependence had higher AS than those without cocaine dependence, even after controlling for other comorbidities. Buckner et al. [15] suggest that although cocaine is a stimulant, it may still increase positive affect and allow a means to escape distress. It is also possible that AS is a consequence of cocaine use.

In their review of social phobia and alcohol use disorders, Morris et al. [14] highlight the importance of expectancies for explaining drinking behavior of people with comorbid social phobia. Expectancies refer to the belief or expectation that alcohol or other drugs will produce a desired effect. This notion is supported in the review by Morris et al. [14], in which individuals with comorbid social phobia and alcohol use disorders held more positive alcohol outcome expectancies (eg, tension reduction, social assertiveness) compared with individuals with low anxiety or without alcohol use disorders. That is, the belief in the effect of alcohol on anxiety symptoms may be more important than whether alcohol actually has an anxiolytic effect. This viewpoint is supported by the Carrigan and Randall [24] review, in which they concluded that individuals consume alcohol to reduce their social phobic anxiety, but that the evidence for the premise that alcohol actually does reduce social anxiety is less clear. Context may also interact with expectancies in its impact on substance use [25]. Social anxiety was positively related to hazardous drinking among college students for those endorsing higher positive (eg, "I would act sociable") or lower negative expectations (eg, "I would be clumsy) for alcohol use in social convivial contexts, while there was a negative association between social anxiety and hazardous drinking for students reporting low positive or high negative expectancies in convivial contexts. However, alcohol expectancies were not found to moderate the association between social anxiety and hazardous drinking for intimate (eg, on a date) or coping (eg, when sad) contexts [25].

Similarly, drinking motives play an important role in delineating the relationship between ADs and SUDs. Cooper (as reviewed in [14]) describes four drinking motive styles that may affect the relationship between anxiety and drinking, namely coping (to reduce anxiety), conformity (to reduce social censure), social (for affiliative reasons), and enhancement (to increase positive affect).

Drinking refusal self-efficacy, the expectation an individual has to be able to successfully refuse a drink, has also been linked to alcohol use outcomes. Among young adult drinkers with generalized anxiety, those with low drinking refusal self-efficacy and high expectancies regarding tension reduction were more likely to suffer from negative consequences of alcohol use [26].

Due to the cross-sectional and retrospective nature of the majority of the studies mentioned [3•, 6, 8, 26, 27], causality cannot be implied. It is possible that developmental differences account for some of these patterns. For example, although social phobia overwhelmingly occurs prior to SUDs, it is not clear whether this is because social phobia

increases the risk of SUDs or whether social phobia tends to have an onset in early-adolescence.

Other Factors Impacting on the Association Between Anxiety and Substance Use Disorders

Anxiety can be a manifestation of withdrawal or intoxication [4], and this may confound the relationship between ADs and SUDs. For example, individuals using cannabis often report a reduction in anxiety as a motivation for use, yet acute anxiety is often associated with cannabis use. However, it is critical to consider the dose taken as well as the context, as anxiety following cannabis use is often associated with high doses, drug-naïve subjects, and when it is taken in novel environments. In contrast, chronic users report relief from tension and anxiety as a common effect of using the drug. Nevertheless, chronic cannabis use is associated with a high prevalence of ADs [28].

Age may also impact on the association between ADs and SUDs. For example, a prospective study with Finnish adolescents reported that social phobia was associated with a lower incidence of frequent alcohol and cannabis use [18]. It is possible that adolescents with social phobia may not have the requisite social skills to acquire substances, or may be too withdrawn to spend much time in social situations that reinforce substance use [3•, 18].

ADs have also been found to act as mediators between adverse childhood events (eg, abuse, domestic violence, and household dysfunction) and SUDs, whereas adverse childhood events appear to lead to an increase in anxiety and mood disorders, which in turn lead to an increased risk of SUDs [27].

Implications for Psychological Treatment and Research

While psychological treatments such as cognitive-behavioral therapy have proven efficacious for the treatment of ADs and SUDs separately [29•, 30•], there is little research on the treatment of comorbid ADs and SUDs [29•]. The research conducted has yielded inconsistent results [31•], with some studies demonstrating no clear advantage of simultaneously treating ADs and SUDs [30•, 32]. A recent systematic review of randomized controlled trials compared manual-guided psychological interventions for alcohol misuse among individuals with comorbid depressive or anxiety disorders. Motivational interviewing and cognitive-behavioral therapy were both associated with significant improvements in alcohol and anxiety symptoms. Although the brief interventions were effective, longer interventions resulted in even better outcomes [29•]. A stepped or staged approach to treatment was, however, recommended by some of the studies reviewed [29•]. A meta-analysis of patients in alcohol treatment programs concluded that both cognitive-behavioral therapy and pharmacotherapy had small to medium effect sizes for anxiety- and alcohol-related outcomes. However, the effect on anxiety-related symptoms was smaller than among psychiatric populations, and improvements in alcohol-related outcomes were predominantly in terms of reduced frequency, intensity, and quantity of use rather than abstinence [31•]. In contrast, a systematic review by Hesse [30•] did not find that integrated psychotherapeutic treatment for ADs and SUDs yielded better outcomes than a program focused solely on the SUD. Despite the dearth of research into the pharmacotherapeutic treatment in individuals with co-occurring disorders [21, 23], similar pharmacotherapeutic agents work for ADs with or without comorbid SUDs [21]. When choosing medication for use in patients with comorbid ADs and SUDs, safety, toxicity, and abuse potential should be among the foremost considerations [23]. There is a need to develop new interventions for the treatment of comorbid ADs and SUDs and to examine the combination of both pharmacologic and psychosocial treatments [30•].

A study by Puleo et al. [33] suggests that early intervention for ADs may prevent the development of future SUDs. For example, successful cognitive-behavioral therapy for childhood anxiety was linked to less substance use and related problems at a 7.4-year follow-up than cognitive-behavioral therapy that was unsuccessful. This relationship held after controlling for other predictors of substance use outcome, such as comorbid attention-deficit/hyperactivity disorder, family history of SUDs, and negative life events [33].

As anxiety may be a result of withdrawal [4], it has been recommended that where possible, patients should be evaluated for anxiety symptoms after a period of abstinence. The anxiety symptoms associated with substance-induced ADs are likely to disappear following a period of abstinence, while those linked to independent ADs are likely to remain [4]. The period of abstinence would vary according to the type of SUD. For example, opiates may require several weeks of abstinence, while substances with a shorter half-life (eg, cocaine, alcohol) may require lesser periods of abstinence [4, 7].

The high comorbidity of ADs and SUDs should alert clinicians to screen all patients with ADs for the development of SUDs, and vice versa [2]. Considering temporality of comorbid disorders is critical in treatment. For example, if substance misuse is used to self-medicate anxiety, treatment of the AD may reduce the use of substances. Similarly, if anxiety arises as a result of chronic substance misuse, treatment of the substance misuse may alleviate symptoms of anxiety [34].

It would also be interesting to explore whether a dose–response relationship can be detected between the degree of SUDs and ADs. Subclinical levels of both may have different patterns of interaction and presentation. In addition, the

treatment required may also differ by the type of AD and SUD, as well as any other comorbidities. More research targeting groups with different SUDs and ADs would therefore be informative. The comorbidity between ADs and depressive disorders is also high and may have a reinforcing effect in terms of SUDs [6, 35]. For example, Boschloo et al. [6] found that while individuals with a pure AD were 2.4 times more likely to suffer from alcohol dependence than controls, these odds were raised to 4.4 times in those with both an AD and a depressive disorder. In a sample of adolescents, Fröjd et al. [18] found that depression appeared to mediate the association between anxiety and substance use.

Additional research is required to tease out temporality and effective interventions for comorbid disorders. Hall et al. [34] recommend more prospective studies as opposed to correlational or retrospective studies to determine causal and temporal relationships between these variables. Better evaluations of the effectiveness of treatment programs for comorbid SUDs and ADs, as well as evaluations of the effects of preventive interventions for ADs on the development of SUDs are also required [34].

Conclusions

The association between ADs and SUDs is complex, with many factors appearing to moderate and mediate this relationship. Cognitive processes, such as attentional bias, expectancies, and anxiety sensitivities, appear to play important roles in the development and prevention of SUDs among individuals with ADs. Self-medication may be a motivating factor in the development of SUDs for individuals with social phobia, with the majority of studies indicating that social phobia is the primary condition. Directionality is less clear for the comorbidity of other ADs and SUDs. Although psychotherapeutic treatment targeting ADs and SUDs separately is effective, research supporting the effectiveness of concurrent treatment for both types of disorders is inconsistent. The high comorbidity of ADs and SUDs should alert clinicians to screen for both disorders in patients presenting with an AD or SUD. Where possible, temporality should be established, as this may inform treatment. Clinicians should also be aware that withdrawal from substances may mimic the anxiety symptoms of ADs and ideally should screen for anxiety symptoms after a period of abstinence. Finally, research should focus on associations between particular ADs and SUDs, as well as on the development of effective treatments for comorbid ADs and SUDs.

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