Drinking motives in alcohol use disorder patients with and without social anxiety disorder

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Drinking motives in alcohol use disorder patients with and without social anxiety disorder

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Abstract

The high comorbidity of alcohol use disorders (AUD) and social anxiety disorder (SAD) is often explained by excessive drinking in social situations to self-medicate social anxiety. Indeed, the motive to drink alcohol to lower social fears was found to be elevated in socially anxious persons. However, this social anxiety specific motive has not been directly investigated in primarily alcohol dependent individuals. We explored social anxiety, the motivation to drink alcohol in order to cope with social fears, and social anxiety as a consequence of drinking in AUD with and without comorbid SAD.

Male AUD inpatients with (AUD+SAD group, N = 23) and without comorbid SAD (N = 37) completed a clinical interview and a questionnaire assessment. AUD+SAD patients reported higher levels of depression and an elevated motive to drink due to social anxiety but did not experience more social fears as a consequence of drinking.

Previous results concerning alcohol drinking motives in order to relieve social fears could be replicated in a clinical AUD sample. Additionally, our findings suggest comorbid AUD+SAD patients to be more burdened regarding broader psychopathological symptoms. Thus, accessibility to SAD-specific screening and treatment procedures may be beneficial for primary AUD patients.

Keywords: alcohol use disorder, social anxiety disorder, alcohol drinking motive, self-medication, coping, information processing, cognitive bias
Alcohol use disorders (AUD) and social anxiety disorder (SAD) are highly comorbid (e.g., Kessler, Chiu, Demler, & Walters, 2005). Moreover, social fears on subclinical (Crum & Pratt, 2001) and clinical levels (Buckner, Schmidt, Bobadilla, & Taylor, 2006) precede the onset of pathological drinking patterns, therefore constituting SAD as a risk factor for later AUD (Buckner & Schmidt, 2009) and alcohol associated problems (Gilles, Turk, & Fresco, 2006). This relationship is not surprising given that alcohol is used as a coping strategy for social fears (Schneier, Martin, Liebowitz, Gorman, & Fyer, 1989). Also, alcohol addicted SAD sufferers unanimously confirmed the use of alcohol to cope with anticipatory anxiety regarding social situations (Randall, 2000). Moreover, social anxiety among students undergoing a brief alcohol intervention was related to poorer outcomes (Terlecki, Buckner, Larimer, & Copeland, 2011), which highlights the need to understand potential targets for change, such as beliefs about drinking, for successful therapy. Such findings are often conceptualized within the framework of the so-called “self-medication hypothesis” (Quitkin, Rifkin, Kaplan, & Klein, 1972). This hypothesis explains the high comorbidity between social anxiety disorder and alcohol use disorders with the negatively reinforcing properties of alcohol on drinking when regularly used in order to cope with social anxiety. However, despite its ostensible plausibility, the actual validity of the self-medication hypothesis has been questioned repeatedly (e.g., Battista, Stewart, & Ham, 2010; Carrigan & Randall, 2003).

Surprisingly, the exact mechanisms of how (and even: if) alcohol reduces state anxiety are not well understood (Carrigan & Randall, 2003). Consequently, the motive to drink alcohol in order to relieve social fears has attracted empirical interest. Results are mixed concerning the association of social anxiety and broader coping or enhancement motives to drink alcohol (e.g.
Buckner, Schmidt, & Eggleston, 2006; Norberg, Norton, Olivier, & Zvolensky, 2010). However, relevant differences between socially anxious and not anxious individuals can be found when specifically drinking to cope with social anxiety is assessed (Thomas, Randall, & Carrigan, 2003). For the German language, Wagner and coauthors (2004) developed two questionnaires that focus on the mutually dependent processes in social fears and alcohol use in alcohol dependent patients: the Drinking Due to Social Anxiety Questionnaire (DDSAQ) and the Social Anxiety Due to Drinking Questionnaire (SADDQ). Considering the investigation of alcohol drinking motives in social anxiety, the DDSAQ is of special relevance since it asks whether alcohol has been used for anxiety reduction in diverse social situations. The SADDQ refers to social anxiety as a consequence of problematic alcohol consumption, such as fears to publicly show withdrawal symptoms (Kushner, Abrams, & Borchardt, 2000). The SADDQ allows distinguishing social fears as a consequence of being addicted to alcohol (i.e., because of symptoms of alcohol withdrawal such as trembling) from other social fears. Obviously, this is crucial when studying individuals with a primary alcohol addiction. For instance, some SAD patients report the development of pathological social fears after the onset of AUD (Buckner, Timpano, Zvolensky, Sachs-Ericsson, & Schmidt, 2008). Moreover, models of social anxiety and alcohol use disorders (e.g., Stewart & Conrod, 2008) suggest a forward-feeding cycle in which social phobics use alcohol to manage acute social fears and consequently increase their social fears in the long term.

Cludius and coauthors (in press) found that the motivation to drink in order to cope with social anxiety, but not social anxiety itself, is related to increased self-reported problematic alcohol use as measured by the Alcohol Use Disorders Identification Scale (AUDIT, Allen, Litten, Fertig, & Babor, 1997) in a nonclinical student sample. This result is much in line with the common finding that social anxiety per se is not related to problematic drinking (Stevens & Gerlach, 2009). Indeed, the motive to drink to cope with social fears mediates the relation between social anxiety
and alcohol related problems in groups of high and low socially anxious participants (Buckner & Heimberg, 2010). Other studies also support drinking motives as moderators or mediators between social fears and alcohol use (Blumenthal, Leen-Feldner, Frala, Badour, & Ham, 2010; Windle & Windle, 2012). However, the motive to drink alcohol to cope with social fears and its relationship to social anxiety has not been directly investigated in a sample with primary alcohol use disorders, which may limit the generalizability of these results.

Thus, we investigated the relationship between social anxiety and alcohol drinking motives due to social anxiety in an inpatient AUD sample. Specifically, we tested whether AUD patients with versus without SAD had higher motives to drink due to social anxiety and experienced more social anxiety due to drinking. Additionally, because we hypothesized comorbid AUD and SAD patients to be more burdened regarding anxiety specific and broader psychopathological symptoms, we expected comorbid subjects to report higher scores on both social anxiety and depression measures.

Method

Participants

Participants were recruited from two inpatient rehabilitation clinics in Germany in which they were admitted to undergo long-term therapy for AUD. AUD patients were asked by one member of the study staff (S. H.) either at a pre-treatment diagnostic assessment or at the beginning of group therapy to participate. All patients were approached when they began therapy (i.e. within the first 3 weeks) and all patients had completed detoxification previously in another institution. Due to the possibly confounding effects of gender, the lower prevalence of AUD in
women, and because of the finding that comorbid AUD and SAD is more frequent in men (Buckner, Timpano, et al., 2008), only male patients were approached. Participants were not compensated for their contribution. All consecutive patients were approached and all gave consent and finished the investigation. Recruitment was conducted from July 2011 until February 2012. The study was approved by the Institutional Review Board.

The sample consisted of 60 male AUD inpatients of which 23 were comorbid for SAD (AUD+SAD group) while the remaining 37 patients did not show clinically relevant social fears (AUD-SAD group). Diagnostic statuses were assessed via structured clinical interviews (SCID; First, Spitzer, Gibbon, & Williams, 1996; German version: Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997). Additional sample characteristics can be depicted in Table 1 and 2.

**Questionnaires**

**Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996; German version: Hautzinger, Keller, & Kühner, 2006).** The BDI-II is a 21-item measure of depression symptoms. For the German version, adequate reliability and validity has been shown for clinical and nonclinical samples (Kühner, Bürger, Keller, & Hautzinger, 2007).

**Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998; German version: Stangier, Heidenreich, Berardi, Golbs, & Hoyer, 1999).** The SIAS consists of 20 items and assesses fears of more general social situations. Sufficient internal consistency and adequate convergent validity has also been shown for the German SIAS (Heinrichs et al., 2002). For our sample, we obtained a Cronbach’s α of .91.
Drinking Due to Social Anxiety Questionnaire (DDSAQ; Wagner et al., 2004). The DDSAQ is a 28 item measure to capture the motive to drink alcohol in order to relieve social fears. The items, such as “I drank alcohol so I could talk more freely and be more relaxed with other people”, were rated on a five point Likert scale. While originally developed based on data from AUD patients, Stevens and Gerlach (2009) also reported adequate psychometric properties of the instrument in a sample of SAD sufferers (in our sample: Cronbach’s α = .97).

Social Anxiety Due to Drinking Questionnaire (SADDQ; Wagner et al., 2004). To assess social anxiety as a consequence of alcohol consumption, the SADDQ was developed for alcohol dependent patients. It consists of 25 items (e.g. “I think I was more afraid of other people because of my alcohol-related problems”) and has high internal consistencies and good factorial and construct validity (Wagner et al., 2004). A Cronbach’s α of .97 was found for our sample.

Procedure

Upon giving informed consent, participants underwent the clinical interview (SCID; First, Spitzer, Gibbon, & Williams, 1996; German version: Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997). Afterwards, participants completed the questionnaire package. Upon finishing the questionnaires, the participants were debriefed about the investigation.

Data analyses

We chose to investigate a sample of n = 60 AUD inpatients by considering the following: We expected roughly 25% of AUD inpatients to suffer from additional SAD based on the NESARC and NCS-R results. Thus, by implementing a sample size of N = 60, we aimed at n = 15
for the AUD+SAD and n = 45 for the AUD only group. Albeit the n for the subgroups is small, we chose to investigate the presence of clinically significant differences between groups, for which sample sizes that allow for the detection of moderate to high effect sizes are sufficient: With an alpha error probability of .05 and a statistical power of .85, an effect of $f = .4$ can be detected in a sample of $N = 60$ (for calculation and software, see Faul, Erdfelder, Lang, & Buchner, 2007).

Considering our hypotheses, we examined whether AUD+SAD and AUD-SAD group differences on intensity and relationships between social anxiety and drinking behavior existed by conducting a MANOVA with group (AUD+SAD and AUD-SAD) as the between-group factor on the respective questionnaire scores.

Concerning missing data, a member of the study staff (S. H.) checked the completeness of the assessment directly upon finishing the questionnaires and asked participants who omitted an item to answer it to their best guess. Consequently, no data points are missing for our analyses.

**Results**

As summarized in Table 3, a between-subjects MANOVA revealed significant group differences between AUD+SAD and AUD-SAD patients ($F (4, 54) = 9.1; p < .01; \eta^2_p = .40$). Main group effects were found for the BDI-II, SIAS and DDSAQ. All scores were higher in AUD+SAD as compared to AUD patients without pathological social fears. Surprisingly, we found no differences concerning the SADDQ between the two groups.

**Discussion**

To explore the motivation to drink in order to cope with social anxiety and the experience of social anxiety due to drinking in a group of patients with primary alcohol addiction, we compared AUD patients with and without comorbid SAD. As expected, the AUD+SAD group scored higher on social anxiety and depression measures and also reported an elevated motive to
drink due to social anxiety as compared to AUD-SAD patients. Interestingly, no difference between groups was found on experiencing social fears as a consequence of alcohol consumption. To explore the possibility of both samples showing enhanced levels of social fears due to pathological alcohol consumption, an inspection of the respective questionnaire scores found for the validating samples (Wagner et al., 2004) is helpful. The authors report mean SADDQ scores for two primary AUD inpatient samples of 45.02 (SD = 23.73) and 43.99 (SD = 23.23). Both our samples’ SADDQ scores (see Table 3) range well within one standard deviation of the respective means. Consequently, the magnitude of social fears experienced as a consequence of alcohol consumption is not different compared to this previous sample.

In addition, comorbid patients showed substantially elevated scores on a depression measure. Although the frequency of Major Depression diagnoses did not differ between the groups, depressive symptoms were more prevalent in AUD+SAD participants, suggesting substantially higher levels of distress in this group. Moreover, the lack of relationship between groups and Major Depression diagnoses suggests that our study may have been underpowered given prior studies that showed comorbid AUD+SAD patients to suffer more frequently from additional Major Depression as compared to unimorbid patients (Buckner, Timpano, et al., 2008). However, Buckner and colleagues investigated patients with *primary* SAD with and without additional AUD, while we focused primarily on AUD inpatients. Also, depression rates are generally higher in women (Kessler et al., 2003). In consequence, the use of an all-male sample may also have caused these comparably low rates of comorbid depression.

We found AUD patients with additional SAD to report increased drinking motives to relieve social fears as compared to AUD patients without SAD, indicating that previous results concerning social fears and related alcohol use motives actually do extent to patients with pathological alcohol addiction. Somewhat surprisingly, we did not find enhanced social fears as a
result of pathological alcohol consumption. However, the lack of significant differences between groups may also stem from power issues given that our power of .85 and our sample size of N = 60 were too small for the detection of small to medium effects. Nonetheless, this result is consistent with the idea that social fears regularly are not the emotional consequence of suffering from an alcohol use disorder but rather frequently precede its onset (Buckner, Schmidt, & Eggleston, 2006; Carrigan & Randall, 2003; Morris, Stewart, & Ham, 2005). For instance, Schneier and coauthors (2010) found almost 80% of comorbid AUD and SAD patients to develop pathological social fears before the onset of clinical drinking patterns. Detoxification is often the first step in treatment of AUD and it is customary to complete detoxification in specialized inpatient clinics before entering rehabilitation treatment. This was also the case for all individuals participating in the present study. Thus, it is very unlikely that the present sample was still suffering from withdrawal symptoms at the time of testing.

Steward and Conrod’s (2008) integrated model of substance use and anxiety disorders offers another explanation for the lack of significant differences regarding social anxiety due to alcohol use between our groups. The model suggests a forward-feeding vicious cycle for the simultaneous maintenance and exacerbation of pathological fears and substance use disorder symptomatology: Once both anxiety and substance use disorders are established, they aggravate each other through self-medication for anxiety on the one hand and a substance-induced increase in anxiety on the other hand. Our results suggest that especially the self-medication motive distinguishes comorbid and unimorbid AUD patients and that the anxiety-relieving and thus negatively reinforcing properties of alcohol consumption may be of special importance.
Finding evidence of enhanced drinking motives to cope with social fears in comorbid patients with primary alcohol use disorder also sheds some light on the underlying functionality of alcohol consumption, despite the inconclusive results concerning anxiety-relieving properties of acute alcohol intoxication (for an overview, see Stevens, Rist, & Gerlach, 2008): regardless of the actual fear-relieving effects of alcohol, comorbid AUD and SAD patients seem to believe in these effects which in turn may enhance the motive to consume alcohol in social situations.

Nonetheless, some limitations of our investigation have to be acknowledged. First, our overall sample size was comparably small which may question the generalizability of our findings. Second, we included only male patients, which may have had a significant impact on the results, especially given the reported gender differences in drinking to cope with negative affect, alcohol-related problems due to drinking to cope with negative affect (Norberg et al., 2010), and comorbidity rates (Kessler et al., 1997). Third, replication and further investigations on this topic are clearly needed in order to draw stronger conclusions. Fourth, we used pre-existing groups within a cross-sectional design which increases the likelihood of participant-related confounds. Additionally, time- and history-related confounds may have affected our results. In order to minimize the possible effects of confounding variables, longitudinal investigations of this topic should be conducted in the future. Fifth, we did not incorporate any measures of past drinking history or frequency, so that based on our results, no inferences can be drawn from alcohol drinking motives to concrete behavior. Nonetheless, a recent investigation (Cludius et al., in press) found the motive of drinking due to social anxiety to be related to self-reported hazardous drinking in a student sample. Future research should further investigate this important link between alcohol drinking motives and self-reported and observed drinking behavior, and also examine the relationship between the motive to drinking alcohol due to social fears on the one hand and the severity of alcohol-related problems on the other hand.
Taken together, our results show that AUD patients with comorbid SAD report an elevated motive to drink to relieve social fears in comparison to unimorbid AUD patients, and that comorbid patients are more burdened regarding other psychopathological symptoms. Our findings suggest that careful diagnostic procedures or additional social anxiety screenings as well as accessibility to SAD specific treatment procedures may be beneficial in patients undergoing long term therapy for alcohol addiction. Indeed, Buckner and coauthors (2008) showed the efficacy of combining motivation enhancement therapy for AUD and cognitive-behavioral therapy for SAD in a preliminary case study. Concerning comorbid substance use disorders and depression, a meta-analytic review (Hesse, 2009) showed the superiority of targeting both disorders directly in contrast to treating substance use disorders alone on both depressive and substance use disorder symptoms. Consequently, although further research is clearly needed, treating both AUD and SAD simultaneously may as well be advantageous for comorbid patients.
References


Footnotes

1. Sociodemographic variables that differed between groups (having own children, age) significantly correlated with one or more dependent variable ($r$ (having own children, SIAS) = -.32, $p = .013$; $r$ (having own children, DDSAQ) = -.38, $p = .002$; $r$ (age, DDSAQ) = -.34, $p = .008$). However, incorporating these two variables as covariates in the MANOVA did not effect the results significantly. We thus report the results of the analysis without covariates.”
### Table 1

**Sample characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Alcohol use disorder with social anxiety disorder (N = 23)</th>
<th>Alcohol use disorder without social anxiety disorder (N = 37)</th>
<th>Test statistic ((df))</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (SD)</strong></td>
<td>42.6 (8.2)</td>
<td>49.0 (8.9)</td>
<td>(T_{(58)} = 2.8 ) (p = 0.008)</td>
<td>(\eta^2 = .12)</td>
</tr>
<tr>
<td><strong>Marital status (%)</strong></td>
<td></td>
<td></td>
<td>(\chi^2_{(4)} = 2.9) (n. s.)</td>
<td>(\phi = .22)</td>
</tr>
<tr>
<td>Single</td>
<td>10 (43.5 %)</td>
<td>11 (29.7 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In relationship</td>
<td>3 (13.0 %)</td>
<td>3 (8.1 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>6 (26.1 %)</td>
<td>12 (32.4 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Divorced</td>
<td>4 (17.4 %)</td>
<td>9 (24.3 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Widowed</td>
<td>0 (0 %)</td>
<td>2 (5.4 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Having own children (%)</strong></td>
<td>8 (34.8 %)</td>
<td>25 (67.6 %)</td>
<td>(\chi^2_{(1)} = 6.2 ) (p = 0.13)</td>
<td>(\phi = .32)</td>
</tr>
<tr>
<td><strong>Previous AUD therapies (%)</strong></td>
<td></td>
<td></td>
<td>(\chi^2_{(2)} = 0.4) (n. s.)</td>
<td>(\phi = .09)</td>
</tr>
<tr>
<td>None</td>
<td>12 (52.2 %)</td>
<td>21 (56.8 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>One</td>
<td>8 (34.8 %)</td>
<td>10 (27.0 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>More than one</td>
<td>3 (13.0 %)</td>
<td>6 (16.2 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Current use of antidepressants (%)</strong></td>
<td>12 (52.2 %)</td>
<td>11 (29.7 %)</td>
<td>(\chi^2_{(1)} = 3.0) (n. s.)</td>
<td>(\phi = .22)</td>
</tr>
</tbody>
</table>
Table 2

*Comorbid diagnoses*

<table>
<thead>
<tr>
<th></th>
<th>Alcohol use disorder with social anxiety disorder (N = 23)</th>
<th>Alcohol use disorder without social anxiety disorder (N = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Depression (%)</td>
<td>5 (21.7 %)</td>
<td>5 (13.5 %)</td>
</tr>
<tr>
<td>Dysthymia (%)</td>
<td>8 (34.8 %)</td>
<td>9 (24.3 %)</td>
</tr>
<tr>
<td>Panic Disorder (%)</td>
<td>3 (13.0 %)</td>
<td>0 (0.0 %)</td>
</tr>
<tr>
<td>Generalized Anxiety Disorder (%)</td>
<td>0 (0.0 %)</td>
<td>1 (2.7 %)</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder (%)</td>
<td>0 (0.0 %)</td>
<td>1 (2.7 %)</td>
</tr>
</tbody>
</table>

*Note.* Group differences regarding comorbid diagnoses did not reach significance ($\chi^2 (5) = 9.3; p = .1; \varphi = .39$).
Table 3

*Group differences on psychopathology and alcohol drinking motives*

<table>
<thead>
<tr>
<th></th>
<th>Alcohol use disorder with social anxiety disorder Mean (SD)</th>
<th>Alcohol use disorder without social anxiety disorder Mean (SD)</th>
<th>F (df₁, df₂)</th>
<th>η²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI-II</td>
<td>16.5 (11.6)</td>
<td>10.6 (9.3)</td>
<td>4.6 (1,57)</td>
<td>.07</td>
<td>.037</td>
</tr>
<tr>
<td>SIAS</td>
<td>33.7 (14.5)</td>
<td>14.7 (10.7)</td>
<td>33.5 (1, 57)</td>
<td>.37</td>
<td>&lt; .000</td>
</tr>
<tr>
<td>DDSAQ</td>
<td>41.2 (22.0)</td>
<td>19.1 (19.6)</td>
<td>16.3 (1, 57)</td>
<td>.22</td>
<td>&lt; .000</td>
</tr>
<tr>
<td>SADDQ</td>
<td>46.0 (28.0)</td>
<td>38.9 (27.1)</td>
<td>1.0 (1, 57)</td>
<td>.02</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

*Note.* BDI-II, Beck Depression Inventory II; SIAS, Social Interaction Anxiety Scale; DDSAQ, Drinking Due to Social Anxiety Questionnaire; SADDQ, Social Anxiety Due to Drinking Questionnaire.