THE INTERACTION OF ALCOHOL PLACEBO,

ATTENTION, AND SOCIAL ANXIETY

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Title of Study: THE INTERACTION OF ALCOHOL PLACEBO, ATTENTION, AND
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Abstract: The current study examined the interaction of alcohol placebo, attentional load, and anxious anticipation of an upcoming social stressor in terms of RSA, electromyographic startle response, tonic skin conductance, and self-reported anxiety. Results revealed increases in RSA, skin conductance, and startle response from the baseline condition to the experimental condition. Additionally, group differences were observed in terms of RSA but not in skin conductance or startle response. Pairwise comparisons based on *a-priori* hypotheses revealed that differences in change scores from baseline to experimental session were significant for the Placebo + Distract and Control + Distract groups but not for the Placebo + Anticipate or the Control + Anticipate groups. That is, the Placebo + Distract group evinced significantly more heart rate variability (indicative of lower anxiety) during the experimental session as compared to the Control + Distract group. However, the Placebo + Anticipate group did not demonstrate the same decreases in anxiety in comparison to the Control + Anticipate group. Taken together, it appears that there may be an anxiolytic benefit of alcohol placebo for individuals who are distracted from anticipating an upcoming social stressor but not for those who are attending to the anticipation of a social stressor. The current results suggest that individuals experiencing cognitive processes associated with social anxiety disorder (anticipatory processing) may experience less reduction in anxiety from the placebo effects of alcohol as compared to those not engaging in cognitive processes associated with social anxiety disorder.

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INTRODUCTION

THE RELATIONSHIP BETWEEN SOCIAL ANXIETY, ATTENTION, AND ALCOHOL

A growing body of evidence has identified an association between abnormal emotional reactivity and alcoholism; however the exact mechanisms causing this relationship remain unclear (Miranda, 2003). One aspect limiting the ability to identify these mechanisms is the tendency for individuals with alcohol use disorders to be treated as a homogenous group (Miranda, 2003). This is a common tendency despite previous evidence suggesting that this population represents a heterogenous sample with various etiological pathways of the disorder as well as a myriad of factors maintaining the disorder (Tartar & Vanyukov, 1994).

In order to accurately identify mechanisms leading to the association between abnormal emotional reactivity and alcohol use disorders, individual etiological pathways of alcohol use disorders must be examined. One etiological pathway of alcohol use disorders identified previously is comorbid anxiety (Merikangas, 1994). Specifically, approximately half (48%) of individuals with Social Anxiety Disorder (SAD) will also meet criteria for a co-morbid alcohol use disorder (Grant et al., 2005). This is markedly higher than the prevalence of alcohol use disorders in the general population (5.3%) (DSM-IV-TR; Lee, Chou, Cho, Park, Dawson, Grant, 2010).

Begin Furthermore, the onset of social anxiety typically precedes the onset of alcohol abuse (Davidson et al., 1993). These findings suggest that some characteristic of individuals with anxiety disorders makes them more likely to develop alcohol use problems. Individuals with co-occurring social anxiety and alcohol use disorders experience greater interference and impairment than either condition alone and often have a lower likelihood of overcoming the disorders than individuals who have either disorder alone (Falk, Yi, Hilton, 2008). Therefore, previous epidemiological studies have clearly identified that there is a link between social anxiety and alcohol, and that comorbid social anxiety and alcohol abuse leads to very poor outcomes. This necessitates an examination of the causal link between these two variables in order to develop effective interventions to treat these comorbid conditions.

In order to better understand the relationship between alcohol and anxiety reduction many moderators have been considered, and cognitive factors moderating the relationship have proved to be promising in elucidating this relationship. Moderators have been considered because the relationship between social anxiety and alcohol problems does not appear to be simple or linear (Sayette, 1999). Initial conceptualizations of a dose dependent relationship between anxiety reduction and alcohol (Conger, 1956) have not been found consistently (Levenson, Sher, Grossman, Newman, & Newlin, 1980). It has been noted that these mixed findings suggest that if alcohol reduces this kind of tension at all, it does so indirectly rather than directly through the pharmacological properties of the drug (Steele & Josephs, 1988). More recent studies have found that alcohol's effect on attention may moderate the relationship between alcohol and anxiety reduction (Steele et al., 1986; Steel & Josephs, 1988; Josephs & Steele, 1990; Sher et al., 2007). Specifically, The Attention Allocation Model suggests that alcohol is particularly reinforcing to individuals with symptoms of social anxiety because alcohol reduces the ability to attend to threatening social cues or to cognitively process upcoming social threats which are particularly salient to individuals with social anxiety (Steele at al., 1986). One study examined the Attention Allocation Model by assigning participants to an alcohol or alcohol placebo

condition prior to an impending speech task (Steele & Josephs, 1988). While participants were waiting they were assigned to either a distraction task (rating art slides) or a task in which they were not distracted from anticipating the upcoming event. Those who consumed alcohol and engaged in the distraction task showed the greatest reduction in self-reported anxiety. Those who consumed alcohol, but did not engage in the distraction task, showed an increase in self-reported anxiety. Those who consumed placebo and engaged in distraction evinced no decrease in self-reported anxiety symptoms. The authors concluded that alcohol only had a diminishing effect on social anxiety symptoms if the participants were distracted from anticipating the upcoming social stressor (Steele & Josephs, 1988). This is presumably due to alcohol's ability to limit attention, which resulted in participants not engaging in anxiety provoking anticipation of the upcoming social interaction while in the distraction condition. Similar studies examining the basic tenants of the Attention Allocation Model have found results congruent with the original theory (Curtin, Patrick, Lang, Cacioppo, and Birbaumer, 2001; Josephs & Steele, 1990; Curtin, Patrick, Lang, Stritzke, 1998). However, other studies (e.g., Sayette, 1993a; Sayette, Smith, Breiner, & Wilson, 1992; Sher & Walitzer, 1986; Sayette, Martin, Perrott, Wertz, & Hufford, 2001) have reported alcohol-related reductions in stress and or anxiety without divided attention. This suggests that competing task demands may not be pre-requisite to alcohol's anxiolytic effects, that other variables may moderate the relationship, or that a wider range of outcome variables is needed to fully understand mechanisms of this relationship.

In order to better understand these findings, more recent studies have examined the interaction of alcohol, attention, and anxiety utilizing psychophysiological outcome variables (e.g., Sher, et al, 2007). Examining the physiological changes associated with these variables is important for several reasons. First, as previously mentioned the literature examining the relationship between social anxiety and alcohol use has been largely inconsistent in that many results are either mixed or have failed to be replicated. This necessitates a wider range of outcome variables in order to draw reliable conclusions. Second, studies have found that self-reports of anxiety and physiology related to anxiety

can be discordant (De Los Reyes et al., 2012; Sher, et al. 2007). Therefore, examining multiple indices of anxiety is important to obtain a clear understanding of the mechanisms moderating this relationship.

Researchers examining this relationship in terms of heart rate, skin conductance, and self-reported anxiety found that attention (participation in a continuous performance task) partially mediated alcohol's effects on reducing state social anxiety when participants were anticipating a speech task as measured by skin conductance. However, this mediation was not supported when social anxiety was measured in terms of heart rate (beats per minute) or self-reports (Sher et al. 2007). Therefore, this study shows partial support for the theory that attention mediates alcohol and anxiety but also demonstrates the need for multiple outcome measures. Taken together, it appears as though more information is needed to draw strong conclusions regarding attention's role within the alcohol and anxiety relationship.

Utilizing a wider range of psychophysiological outcome variables may lead to a better understanding of the relationship between alcohol, attention, and anxiety as the range of psychophysiological outcome variables used to examine the relationship between these variables has been relatively limited. The majority of studies have focused on heart rate acceleration and skin conductance, which have noted limitations in relation to drawing conclusions about anxious responding (Barry & Maltzman, 1985; Wilson & Abrams, 1977). Heart rate acceleration is utilized as a proxy for sympathetic nervous system activity. However, the interpretation of this measure in relation to anxiety is somewhat limited with some research indicating that it is not a defensive reflex (Barry & Maltzman, 1985). Skin conductance is taken as a measure of sympathetic nervous system activity and thus general physiological arousal. However, the direction (negative or positive) of arousal cannot be distinguished, only intensity of arousal, and thus the ability of this measure to be informative within the current literature is also somewhat limited.

Specifically, the addition of heart rate variability (Respiratory Sinus Arrhythmia) and startle response to the battery of psychophysiological outcome measures may provide a more comprehensive understanding of the relationship between social anxiety, attention, and alcohol.

A measure of heart rate variability called Respiratory Sinus Arrhythmia (RSA) is considered to be more closely linked to problematic responses in individuals with anxiety disorders than simple heart rate acceleration (Llera & Newman, 2010; Diamond, Fagundes, & Butterworth, 2012; Lyonfields et al., 1995; Thayer et al., 1996). This measure of heart rate variability reflects parasympathetic nervous system activity (Katona & Jih, 1975) and is mediated by the vagal nerve. The inclusion of startle response analysis, which is a physiological index of emotional responding mediated by the amygdala, will also be beneficial to this literature. Within the startle paradigm emotion is organized into two fundamental systems: an appetitive system and a defensive system (M. M. Bradley, Codispoti, Cuthbert, & Lang, 2001). It is hypothesized that these systems have evolved to process elements in the environment that either nourish or jeopardize physical survival. The defensive system is primarily activated in contexts involving threat, whereas the appetitive system is activated in contexts that promote survival. Physiological research of these systems has determined that activation of the defensive system is characterized by a potentiated startle response. Conversely, the appetitive system is characterized by inhibition of the startle response. Thus, this paradigm can serve as an objective biological indication of affect during an acute temporal period.

Aside from the addition of these important psychophysiological outcome measures, additional cognitive processes should be examined as well. Previous psychophysiological studies examining the relationship between attention, anxiety, and alcohol have not included a true control (no expectancy of the effects of alcohol) (Steele & Josephs, 1998, Josephs & Steele, 1990; Sher et al, 2007; Curtin et al. 1998), due primarily to economic resources necessary for the additional sample size. This variable has not been isolated despite previous studies demonstrating that the most reliable effects of alcohol consumption on symptoms of anxiety appear to be related to alcohol expectancy or placebo rather

than the pharmacological properties of alcohol use (Knight & Godfrey, 1993). Therefore, isolating the effects of alcohol placebo by comparing placebo directly to a true control condition may help to better understand the relationship between alcohol, attention, and anxiety.

The current study aimed to examine the relationship between attention, social anxiety, and alcohol use in light of the important considerations listed above. Specifically, the current study focused on the effects of alcohol placebo and attention on a more comprehensive battery of psychophysiological outcome measures associated with symptoms of social anxiety in order to gain a more comprehensive understanding of this complex relationship.

Several key hypotheses were expected in the current study. An interaction was expected between the beverage condition and the activity condition such that individuals consuming placebo beverage and engaging in a distraction task would evince 1) greater heart rate variability as assessed by RSA (indicating a decrease in anxiety), 2) comparatively lower skin conductance levels (indicating less anxiety), and 3) comparatively reduced startle magnitude (indicating less defensive affect), as compared to all other conditions. The rationale behind these main hypotheses is that alcohol placebo may reduce fear of the impending social interaction and that there is also a benefit of distraction or attentional load preceding an impending social interaction. That is, the current study posits that there is an anxiolytic benefit of alcohol placebo combined with distraction from anticipation that is not due to the pharmacological properties of alcohol limiting attentional capacity.

The current study will also discuss the manipulations to attentional load in terms of their relation to Social Anxiety Disorder. Specifically, attention towards anticipation will be conceptualized as a cognitive process (anticipatory processing) associated with Social Anxiety Disorder while distraction from anticipation will be conceptualized as a cognitive process associated with individuals without Social Anxiety Disorder (Clark & Wells, 1995; Hinrichsen & Clark, 2003). It was hypothesized that the placebo effect of alcohol may be different for individuals prompted to engage in a cognitive

process associated with social anxiety (anticipatory processing) as compared to those distracted from anticipating the social stressor. That is, we expected the effects of alcohol placebo to be limited in individuals prompted to attend to anticipation of a social stressor (a cognitive process associated with increased levels of social anxiety in social situations; Hinrichsen & Clark, 2003), as evidenced by 1) less heart rate variability as assessed by RSA (indicating a decrease in anxiety), 2) comparatively higher tonic skin conductance levels (indicating less anxiety), and 3) comparatively increased startle magnitude (indicating less defensive affect), as compared to individuals distracted from anticipation of a social stressor.

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METHOD

PHYSIOLOGICAL DATA ACQUISITION

Ag – Agl 4 mm surface electromyography electrodes (In Vivo Metric, E220 – LS) were attached to the skin above the orbicularis oculi of the left eye in a bipolar configuration in order to measure startle magnitude. Skin above the orbicularis oculi was abraded with Nuprep abrasive skin prepping gel, and electrodes were filled with Signa electrode gel. An amplification setting of 50,000 on a Biopac MP150 bioamplifier was used to collect raw electromyography data. Rectification of the raw signal using a bandpass filter setting of 28 – 500 hz, and a time constant of 10 ms, was completed on a MP150 EMG 100c integrator (T. D. Blumenthal et al., 2005). The rectified signal was then manually scored using Acqknowledge scoring software. Startle scores were calculated by subtracting the baseline eye blink magnitude (data point just before startle reflex) from the peak electromyographic magnitude between 21 and 200ms after the acoustic probe was administered. Consistent with previous procedures published in affective modulation of the startle response literature (Dempsey, Cohen, Hobson, & Randall, 2007), trials were rejected if they included (1) excessive noise (> 19 µV) during baseline or (2) non-startle blink activity occurring at zero to 21 ms after probe administration.

Begin Participants were excluded if they failed to show a reliable startle response (no regular increase (< 5 μv) in startle magnitude 21 – 200 ms after probe onset). Impedance was measured with a Checktrode model 1089 impedance meter, and was below 10 kΩ for all physiological measurements.

Biopac EL507 skin conductance electrodes were placed on the medial phalanges of the second and fourth digits of the left hand in order to measure skin conductance. Skin was prepped with distilled water and electrodes were pre-filled by the manufacturer with isotonic gel. A transduction range of 0-20 microsiemens on a Biopac MP150 GSR100C skin conductance amplifier was used to collect tonic skin conductance data. The resulting skin conductance signals were then manually scored using Acqknowledge scoring software. Mean skin conductance level in microsiemens was measured for 3 minute periods at habituation, baseline, and immediately following the experimental manipulation of attention to or distraction from anticipation.

Biopac EL503 EKG electrodes were placed above the right clavicle and on rib T10 of the left thoracic cage in order to measure heart rate variability. Skin was prepped with alcohol, and electrodes were filled with Signa electrode gel. An amplification setting of 1000 on a Biopac MP150 bioamplifier was used to collect heart rate data.

PROCEDURE

Sixty individuals were recruited from an online research recruiting system (SONA) at a large Midwestern University. Participants endorsing a range in severity of symptoms of SAD on the Social Interaction Anxiety Scale were included and the four experimental groups (placebo + distraction, placebo + attention, control + distraction, and control + attention) had equal distributions of SAD symptom severity. The decision to control for this variable was made based on results in the literature indicating that this variable often affects results, sometimes positively (Buckner, Schmidt, Eggleston 2006; Kushner & Sher 1993; Lewis & O'neill, 2000), and

sometimes negatively (Eggleston, Woolaway-Bickel, Schmidt, 2004; Ham, Bonin, Hope, 2007; Holle, Heimberg, Sweet, Holt, 1995). Sex (male or female) and alcohol use were also randomized amongst the four experimental groups. Upon entering the lab and signing consent, individuals completed a battery of demographic, anxiety, and substance use related self- report measures. Physiological equipment was then connected to the participants.

Following baseline data collection individuals were randomized into one of two beverage conditions. In one condition individuals consumed an alcohol free cranberry flavored soft-drink which they were led to believe contained alcohol. The glass in this condition was swabbed with alcohol (80 proof vodka) to increase believability. The other half of the sample consumed an equivalent amount of the cranberry flavored soda with no deception of the alcoholic content of the beverage. Following this period of beverage consumption all participants were told that they would be engaging in a social interaction task in a manner similar to previous studies (Steele & Josephs, 1988; Josephs & Steele, 1990). Individuals were then randomized into one of two conditions manipulating attention to anticipation. One condition consisted of a five minute period in which participants were prompted to attend to anticipation of the upcoming social interaction. Specifically, a standardized procedure aimed at increasing attentiveness to anticipatory processing which has previously been demonstrated to decrease RSA and increase startle magnitude, was utilized to ensure that attention to anticipation occurs (Mills, Grant, Judah, & Lechner, Under Review). Conversely, the other half of participants were randomized into a condition in which they were distracted from anticipating the social interaction. Specifically, participants were asked to rate art slides; a manipulation which has been used as a distractor in previous research examining the relationship between alcohol, attention, and anxiety (Steele & Josephs, 1988; Josephs & Steele, 1990).

 Startle response was elicited 12 times throughout each five minute period (baseline and anticipatory periods) in order to assess state affect. Heart rate variability and tonic skin

conductance were also measured during baseline and anticipatory periods. The interaction of the beverage condition and attentional conditions was inherent due to the temporal precedence of these conditions (beverage condition always occurred prior to attentional condition). Individuals completed self-report forms of state affect and state anxiety following the experimental component of the study. Individuals were then debriefed, and the nature of the study including the alcohol deception was disclosed.

MEASURES

Social Interaction Anxiety Scale (SIAS) & Social Phobia Scale (SPS); (Mattick & Clark, 1998) The SIAS and SPS are both 20 item self-report measures of symptoms of social anxiety. The SIAS measures severity of fears regarding social interactions while the SPS measures anxiety related to social performance situations. Responses for both scales range from 0 (not at all characteristic of me) to 4 (extremely characteristic of me).

State Trait Anxiety Inventory (STAI); (Spielberger, Gorssuch, Lushene, Vagg, & Jacobs, 1983). The STAI is a 40 item self-report measure of state and trait anxiety symptoms. Identical items are assessed as experiences currently (right now) and generally (most of the time). Items such as "pleasant" are rated on a 4 point Likert scale from 0 (not at all) to 3 (very much so).

Positive and Negative Affect Scale (PANAS); (Watson, Clark, and Tellegen, 1988). The PANAS is a 20 item self-report measure assessing the experience of specific emotions. The measure uses a 5 point Likert scale to rate emotions such as irritability ranging from 0 (very slightly or not at all) to 4 (extremely).

Alcohol Use Disorder Identification Test (AUDIT); (Saunders, Aasland, Babor, et. al., 1993). The AUDIT is a 10 item self-report measure assessing the likelihood that an individual will meet criteria for an alcohol use disorder. Most items measure the frequency of problematic drinking

events such as "how often have you found you were not able to stop drinking once you started" on a scale ranging from 0 (never) to 4 (daily or almost daily).

PARTICIPANTS

Participants were undergraduate students enrolled at a large Midwestern university. The age of the sample ranged from 18 to 27 with a mean age of 19.49 (SD = 1.47). Females constituted 52.5% of the sample; 83.6% identified as Caucasian, 8.2% as African American, 3.3% as Native American, 1.6% as Asian American, and 3.3% as other. The average Alcohol Use Disorders Identification Test score was 7.93, SD = 6.58, which is just below the recommended cutoff score of 8 indicating hazardous or problematic drinking (Conigrave, Hall, Saunders, 1995). The mean beverage consumption of the sample was 21.53 (SD = 31.03) drinks in the month prior to testing, and 4.68 (SD = 4.51) days drinking in the month prior to testing. The mean Social Phobia Scale score for the sample was 17.47 (SD = 12.64), and the mean score on the Social Interaction Anxiety Scale was 21.97 (SD = 13.53).

Analysis of key variables revealed no differences between randomized groups. Specifically, the four experimental groups (Alcohol Placebo + Distraction, Alcohol Placebo + Anticipation, Control + Distraction, Control + Anticipation) did not differ in terms of Race χ^2 (12, N = 61) = 13.46, p = .337; Sex χ^2 (3, N = 61) = 2.12, p = .537, AUDIT Score $F(3, 58)$ = .58, p = .630; SIAS Score $F(3, 58)$ = .87, p = .461; or SPS Score $F(3, 58)$ = 1.23, p = .306. Despite the lack of significant differences between groups, important covariates identified in previous research were entered into analyses in order to decrease error between groups as indicated in the analytic strategy section.

ANALYTIC STRATEGY

A series of 4 (group: Placebo + Distract, Placebo + Anticipate, Control + Distract, Control + Anticipate) x 2 (time: baseline, experimental) repeated measures Mixed ANOVAs were

conducted to examine group differences in RSA, Electromyographic Startle Magnitude, and tonic

Skin Conductance Level responding. Electromyographic data were standardized within subject

and then averaged by experimental condition (baseline, experimental), consistent with previous

research in the field (Blumenthal, 1998; Dempsey, et al., 2007). In order to reduce error caused

by differences between groups in physiological responding, habituation heart rate variability and

skin conductance levels were included as covariates in the repeated measures analysis; a

technique used in previous studies (e.g., Sher, et al., 2007). Similarly, in order to reduce error in

measuring the effects of the manipulation caused by group differences in reported social anxiety

(Miller & Chapman, 2002), total scores from the SIAS were included as covariates. Specifically,

RSA analyses included SIAS and Habituation RSA as covariates. SCL analyses included SIAS

score, and Habituation Skin Conductance Levels as covariates. EMG analyses included SIAS

total scores only. Habituation EMG magnitudes were not entered as covariates because this

potential source of between group error is already accounted for through z-transformations

conducted to compute the baseline and experimental EMG magnitudes.

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FINDINGS

MANIPULATION CHECKS

Several markers of the experimental manipulations were examined. The first manipulation tested was the induction of anxiety via anticipation of a social stressor, regardless of randomization to the attention or distraction conditions. Tonic skin conductance levels and startle response magnitude both suggest that anxiety increased upon anticipation of a social stressor, $F (1, 54) = 13.562$, $p = .001$, $\eta2 = .376$ and $F (1, 52) = 12.114$, $p = .001$, $\eta2 = .307$. A significant change in heart rate variability upon anticipation was also observed $F (1, 54) = 30.208$, $p < .001$, $\eta2 = .359$, however the direction of the change was contrary to the original hypotheses; these results are explained in the discussion section. The second manipulation tested was the believability of the alcohol placebo condition. A question examining participant's beliefs about the beverage they consumed revealed that individuals in the placebo condition reported that they believed the beverage they consumed contained alcohol whereas the control condition did not believe they consumed alcohol, $\chi2 (3, N = 58) = 58.0$, $p < .001$. Additionally the placebo group reported feeling significantly greater effects of alcohol than the control group (the control group reported no feelings of intoxication), $F (1, 56) = 10.564$, $p = .002$.

Consistent with increased anxiety, a significant decrease in positive affect as assessed by the Positive and Negative Affect Scale was observed over time $F(1, 54) = 8.867$, $p = .005$, $\eta2 = .138$. However, no significant change in Negative affect as assessed by the same scale was observed over time $F(1, 54) = 3.209$, $p = .08$, $\eta2 = .056$. No significant change in self-reported symptoms of general anxiety were observed over time as assessed by the state portion of the State Trait Anxiety Scale $F(1, 54) = .637$, $p = .43$, $\eta2 = .012$. No group differences were observed for these self-report measures.

PSYCHOPHYSIOLOGY

A significant increase in heart rate variability (RSA) over time was observed, $F(1, 54) = 30.208$, $p < .001$, $\eta2 = .359$. RSA was significantly higher in the experimental time period than in the baseline time period (mean difference = .514, $p < .001$). The group by time interaction was also significant, $F(3, 54) = 2.901$, $p = .044$, $\eta2 = .143$. Pairwise comparisons revealed that heart rate variability rose significantly from baseline to experimental condition for the Placebo + Distraction group (mean difference = .93; $F(1, 52) = 28.303$, $p < .001$, $\eta2 = .352$) and the Placebo + Anticipation group (mean difference = .54; $F(1, 52) = 9.070$, $p = .004$, $\eta2 = .149$), but not for the Control + Distraction or Control + Anticipation groups. No group differences were observed in overall heart rate variability, $F(3, 52) = 2.062$, $p = .117$, $\eta2 = .106$.

In order to examine differences between the magnitude of RSA change scores and groups, pairwise comparisons were conducted based on a-priori hypotheses. Analyses revealed that the difference between heart rate variability change scores for Placebo + Distract and Control + Distract were significant; $F(1, 26) = 6.618$, $p = .016$, $\eta2 = .203$. Conversely, heart rate variability change scores for Placebo + Anticipate and Control + Anticipate were not significant; $F(1, 26) = .985$, $p = .330$, $\eta2 = .037$.

A significant increase in electromyographic startle magnitude over time was observed, $F_{(1, 54)}$ = 13.562, p = .001, $\eta 2$ = .376. Startle magnitude was significantly higher in the experimental data collection period than in the baseline period (mean difference = .480, p < .001). However, the group by time interaction was not significant, $F_{(3, 54)}$ = 1.111, p = .353, $\eta 2$ = .058. No group differences were observed in overall electromyographic startle magnitude, $F_{(3, 54)}$ = .486, p = .694, $\eta 2$ = .026.

A significant increase in tonic skin conductance over time was observed, $F_{(1, 52)}$ = 12.114, p = .001, $\eta 2$ = .307. Tonic skin conductance was significantly higher in the experimental data collection period than in the baseline period (mean difference = .607, p < .001). However, the group by time interaction was not significant, $F_{(3, 54)}$ = 1.251, p = .301, $\eta 2$ = .067. No group differences were observed in overall tonic skin conductance, $F_{(3, 54)}$ = .689, p = .563, $\eta 2$ = .037.

CONCLUSIONS

CURRENT RESULTS

The purpose of the current study was to examine the interaction of alcohol placebo and attention to or distraction from anticipation of an upcoming social stressor in terms of RSA, electromyographic startle response, tonic skin conductance, and self-reported anxiety. In conducting this study, we were particularly interested in examining the ability of alcohol placebo compared to control to interact with attention or distraction from anticipation in reducing psychophysiological indices of anxiety because previous studies have typically focused on the pharmacological rather than psychological effects of alcohol. Furthermore, we aimed to examine these variables in terms of two measures of psychophysiology that have not been included in this specific literature: RSA and startle reflex. Results revealed increases in RSA, skin conductance, and startle response from the baseline condition to the experimental condition. Additionally, group differences were observed in terms of RSA but not in skin conductance or startle response. The observed increases in skin conductance over time were expected and reflect greater sympathetic nervous system activation indicative of heightened anxiety. Similarly, the observed potentiation in startle response over time was expected and reflects greater negative affect indicative of increased anxiety. The increases in skin conductance and startle response suggest that the manipulation of anticipating a social interaction was effective in increasing physiological indices of anxiety.

Conversely, the observed increases in heart rate variability over time, indicating decreased anxiety, were not originally expected. However, the direction of RSA may suggest a process of recovery that has been observed in previous studies (Hinrichsen & Clark, 2003), rather than a simple pattern of decreased heart rate variability during the experimental session. That is, heart rate variability was expected to decrease less for the most anxiolytic conditions and decrease more for the least anxiolytic conditions; however it appears to have increased more for the most anxiolytic condition and increased less for the least anxiolytic condition. Pairwise comparisons based on a-priori hypotheses revealed that differences in change scores from baseline to experimental session were significant for the Placebo + Distract and Control + Distract groups but not for the Placebo + Anticipate or the Control + Anticipate groups. That is, the Placebo + Distract group evinced significantly more heart rate variability (indicative of lower anxiety) during the experimental session as compared to the Control + Distract group. However, the Placebo + Anticipate group did not demonstrate the same decreases in anxiety in comparison to the Control + Anticipate group. Taken together, it appears that there may be an anxiolytic benefit of alcohol placebo for individuals who are distracted from anticipating an upcoming social stressor but not for those who are attending to the anticipation of a social stressor. That is, for individuals who were prompted to anticipate an upcoming social stressor in a manner similar to how individuals with social anxiety disorder anticipate an upcoming social stressor (Clark & Wells, 1995), alcohol placebo resulted in no significant reduction in anxiety. Conversely, for individuals who were distracted from anticipating an upcoming social stressor, alcohol placebo significantly reduced anxiety as compared to the control (no placebo) + distract group. The current results suggest that individuals experiencing cognitive processes associated with social anxiety disorder (anticipatory processing) may experience less reduction in anxiety from the placebo effects of alcohol as compared to those not engaging in cognitive processes associated with social anxiety disorder.

CURRENT FINDINGS IN CONTEXT OF EXTANT LITERATURE

These findings may help to explain some of the mixed results regarding the relationship between social anxiety and alcohol use disorders. The Attention Allocation Model suggests that alcohol is particularly reinforcing to individuals with SAD because alcohol reduces the ability to attend to threatening social cues or to cognitively process upcoming social threats which are particularly salient to individuals with SAD (Steele at al., 1986). This is presumably due to alcohol's deleterious effects on attentional capacity. However, the current study suggests that the placebo effect of alcohol also plays a role in the relationship between social anxiety, alcohol consumption, and attention. Specifically, alcohol placebo appears to effectively reduce physiological indices of anxiety when individuals are distracted from anticipating a social stressor but not when they are attending to anticipation of a social stressor. The moderating role of attention to or distraction from anticipation on the anxiolytic effects of alcohol placebo is a new finding within the literature. These results indicate that alcohol placebo does play a significant role in the relationship between symptoms of social anxiety and alcohol, and that the effect of alcohol placebo may be contingent upon attention to the upcoming social interaction.

This may help to clarify some of the mixed findings present within the literature. For one, some studies have found that symptoms of social anxiety reduce risk of developing an alcohol use disorder (Buckner, Schmidt, Eggleston 2006; Kushner & Sher 1993; Lewis & O'neill, 2000), whereas others find that it increases risk (Eggleston, Woolaway-Bickel, Schmidt, 2004; Ham, Bonin, Hope, 2007; Holle, Heimberg, Sweet, Holt, 1995). It is possible that individuals with social anxiety may receive little or no anxiolytic benefit of alcohol placebo and therefore follow one of two paths. One in which alcohol is consumed and offers little to no immediate reinforcement (due to lack of placebo), ultimately making their symptoms of social anxiety a buffer to problematic drinking. Conversely, individuals with social anxiety may learn that more alcohol consumption is required to be reinforcing (in order to receive the pharmacologically

induced anxiolytic benefit) and thus are more likely to engage in heavier, more problematic drinking. However, these hypotheses cannot be directly tested within the current study and must be examined in a follow up study designed to test this question specifically.

Additionally, the effects of alcohol placebo may help to explain why studies comparing alcohol to alcohol placebo have often failed to find differences in physiological responding across multiple indices of physiology (Sher, 2007). By making comparisons of the pharmacological effects of alcohol to the placebo effects of alcohol previous studies have really only able to examine the pharmacological effects in isolation (because both conditions contain active placebo). The most ecologically valid comparison may be between alcohol and a true control condition. This improvement in ecological validity may produce results that better explain results found in epidemiological studies of the relationship between social anxiety and alcohol.

LIMITATIONS

While the results of the current study may provide new insight into the relationship between social anxiety, attention, and alcohol use they must be considered in light of several noteworthy limitations. One potential limitation of the present study is the lack of an alcohol condition in order to make comparisons between placebo, true control, and alcohol. While this study aimed to examine alcohol placebo versus true control specifically, the inclusion of an alcohol condition would have provided more comprehensive results. A second limitation is the lack of inclusion of individuals diagnosed with social anxiety disorder. While the current study aimed to examine a specific cognitive process associated with social anxiety (anticipatory processing), this process was manipulated experimentally in individuals with a range of social anxiety symptoms. The effects of this process in individuals diagnosed with Social Anxiety Disorder may differ from the effects observed in the current study.

FUTURE DIRECTIONS

Future studies would improve on the current study by examining all three conditions (alcohol, alcohol placebo, and true control) in individuals anticipating a social interaction who have been diagnosed with Social Anxiety Disorder versus individuals with average to low level of social anxiety symptoms. This would allow for a comprehensive test of the current suggestion that individuals with Social Anxiety Disorder may experience less anxiolytic benefit from alcohol placebo than individuals without Social Anxiety Disorder. Additionally, future studies could expand upon the current results by examining the moderating effect of lack of placebo on development of Alcohol Use Disorders. If these studies produced results supporting the implications of the current results it would be informative to the treatment of this comorbid condition.

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