

Experimentally Induced Anxiety Attenuates Alcohol-Related Aggression in Men

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The purpose of this study was to test the hypothesis that state anxiety operates as moderator of the alcohol–aggression relation. Participants were 80 healthy male social drinkers between 21 and 33 years of age. They were randomly assigned to 1 of 4 groups: (a) alcohol + anxiety induction ($n = 20$), (b) placebo + anxiety induction ($n = 20$), (c) alcohol + no anxiety induction ($n = 20$), and (d) placebo + no anxiety induction ($n = 20$). Anxiety was induced by informing participants that they had to deliver a speech about what they liked and disliked about their body in front of a video camera. A modified version of the Taylor Aggression Paradigm (S. Taylor, 1967) was then used to measure aggressive behavior in a situation where electric shocks were administered to, and received from, a fictitious opponent under the guise of a competitive reaction time task. Results indicated that the anxiety induction was successful in suppressing aggression for participants who received alcohol equal to levels seen in placebo controls. Findings are discussed within the context of a number of theories of alcohol’s anxiolytic effects in relation to intoxicated aggression.

Keywords: alcohol, aggression, anxiety

The relation between acute alcohol consumption and aggressive behavior is well documented (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Ito, Miller, & Pollock, 1996). Alcohol has been implicated in approximately 50% of violent crimes worldwide (Murdoch, Pihl, & Ross, 1990; Pernanen, 1993). Archival crime statistics indicate that the prevalence of intoxicated aggression has increased steadily over the past 50 years (Pernanen, 1993; Spunt, Brownstein, Goldstein, Fendrich, & Liberty, 1995). An extensive body of correlational research also indicates that alcohol consumption is strongly associated with a number of aggressive behaviors, including violent threats (Bedford, Melzer, & Guralink, 2001), family violence (Lipsky, Caetano, Field, & Larkin, 2005), marital aggression (Leonard & Senchak, 1996), child abuse (Berger, 2005), firearm use (Price, Thompson, & Dake, 2004), sexual aggression (Testa, 2002), homicide (Pridemore, 2004), and suicide (Pirkola, Suominen, & Isometsa, 2004).

Although these investigations are compelling, they do not speak to whether there is a causal relation between alcohol and aggression. Useful complements to these correlational reports are experimental studies that examine the alcohol–aggression relation by manipulating alcohol consumption in

a laboratory setting and then recording in vivo aggressive reactions in response to provocation. Findings from such studies overwhelmingly demonstrate that alcohol increases aggression compared with placebo or nonalcohol beverages (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Taylor & Chermack, 1993). Meta-analytic studies conducted on laboratory investigations confirm these data by showing that alcohol has a “medium” effect size ($d = 0.49$ to 0.61) on aggression (Bushman, 1993; Bushman & Cooper, 1990; Ito et al., 1996). Importantly, it has also been shown that it is the acute, rather than the chronic, effects of alcohol intoxication that are most closely related to the expression of aggressive behavior (Chermack & Blow, 2002; Fals-Stewart, 2003).

Although alcohol’s effect size on aggression is in the medium range, it clearly does not increase aggression in all persons. Given this, researchers have focused on identifying variables that moderate the alcohol–aggression relation. Specifically, studies have shown that alcohol is more likely to facilitate aggression for persons with higher levels of dispositional aggressivity (Barnwell, Borders, & Earleywine, 2006; Heyman, O’Leary, & Jouriles, 1995), irritability (Giancola, 2002), rumination (Borders, Barnwell, & Earleywine, 2007), trait anger (Parrott & Zeichner, 2002), hostility, permissive beliefs about aggression (Leonard & Senchak, 1993), deviant attitudes (Zhang, Wiczorek, & Welte, 1997), sensation seeking (Cheong & Nagoshi, 1999), as well as lower levels of anger control (Parrott & Giancola, 2004), socialization, self-control (Boyatzis, 1975), dispositional empathy (Giancola, 2003), intelligence (Welte & Wiczorek, 1999), and executive cognitive functioning (Giancola, 2004b). Moreover, alcohol has also been found to potentiate aggression for persons with a difficult temperament (Giancola, 2004a), a desired image of power (Quigley,

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Corbett, & Tedeschi, 2002), and beliefs that alcohol causes aggression (Dermer & George, 1989), as well as high marital conflict (Quigley & Leonard, 1999) and dissatisfaction (Leonard & Senchak, 1993).

In addition, researchers have also identified contextual variables that moderate the alcohol–aggression relation. Zeichner and Pihl (1979) conducted a study to examine the effect of behavior contingencies on intoxicated aggression. In this investigation, intoxicated and sober participants completed a laboratory aggression task in which aversive tones were administered to the participant in response to electric shocks they delivered to a fictitious opponent. One group of participants received tone intensity levels that were matched to the intensity of electric shock they selected for their opponent, whereas another group received random tone intensity levels. Results indicated that matching shock levels reduced aggression in sober but not intoxicated participants (Zeichner & Pihl, 1979). Another study by these authors demonstrated that the lack of malicious intent by an instigator was sufficient to reduce aggression in sober but not intoxicated persons (Zeichner & Pihl, 1980). Taylor and colleagues conducted two studies examining the effects of social pressure on aggression also using a laboratory shock–aggression task (Taylor & Gammon, 1976; Taylor & Sears, 1988). In their first study, a confederate attempted to persuade participants to administer high-intensity shocks to a nonaggressive fictitious opponent (Taylor & Sears, 1988). This manipulation resulted in a significant increase in aggression for intoxicated but not for sober participants (Taylor & Sears, 1988). Perhaps more important, Taylor and Gammon (1976) manipulated social pressure in a similar manner, but attempted to dissuade participants from aggressive behavior. This manipulation was equally effective in suppressing aggressive behavior for both sober and intoxicated participants (Taylor & Gammon, 1976). Finally, in keeping with these findings, Giancola and Corman (2007) found that participating on a mentally taxing working memory task while simultaneously performing a laboratory shock–aggression task significantly suppressed aggression in intoxicated individuals, even below levels seen in sober controls.

A number of theories have been advanced to explain the mechanisms by which alcohol facilitates aggressive behavior. Three of the most well-accepted theories of alcohol-related aggression contend that alcohol's pharmacological properties facilitate aggression by (a) impairing anxiety and fear responses (Ito et al., 1996; Pihl, Peterson, & Lau, 1993), (b) disrupting higher order cognitive functions important in maintaining inhibitory control over behavior (Giancola, 2000; Steele & Josephs, 1990), and (c) increasing psychological and physiological arousal (Giancola & Zeichner, 1997; Graham, Wells, & West, 1997).

Studies attempting to subject these models to systematic empirical scrutiny are scarce. Some preliminary investigations have shown that alcohol leads to aggression by impairing information processing (Zeichner & Pihl, 1979) and attentional resources (Giancola & Corman, 2007; Zeichner, Pihl, Niaura, & Zacchia, 1982), as well as skills involving accessing alternative socially adaptive responses, hypothe-

sis generation and evaluation (Ogle & Miller, 2004; Sayette, Wilson, & Elias, 1993), goal selection (Ogle & Miller, 2004), outcome anticipation, and conflict resolution (Sayette et al., 1993). Finally, alcohol has also been shown to affect aggression by altering autonomic arousal (Assaad et al., 2003; Hoaken, Campbell, Stewart, & Pihl, 2003). However, to date, no published study has specifically examined the role of anxiety in the alcohol–aggression relation. Pihl et al. (1993) have argued that, among other factors, alcohol facilitates aggression via its anxiolytic properties by impairing the ability to adaptively act on threat- or danger-imparting (anxiety-provoking) cues.

There is a large literature on the effects of alcohol consumption on anxiety (reviewed in Greely & Oei, 1999; Sayette, 1993, 1999; Sher, 1987). However, these findings are inconsistent, with reports that alcohol increases, decreases, or has no effect on anxiety (Cappell & Greeley, 1987; Sher, 1987; Steele & Josephs, 1988). This body of research has focused on alcohol's hypothesized stress–response-dampening effects (Levenson, Sher, Grossman, Newman, & Newlin, 1980) by comparing anxiety in sober and intoxicated individuals following exposure to a stressor (e.g., tone blast, shock, or providing a self-disclosing speech). Whereas some researchers have reported stress–response-dampening effects using self-report measures of anxiety (Levenson, Oyama, & Meek, 1987; Levenson et al., 1980; Sayette, Breslin, Wilson, & Rosenblum, 1994; Sher, Bartholow, Peuser, Erikson, & Wood, 2007), others have not (Niaura, Wilson, & Westrick, 1988; Sayette, Contrada, & Wilson, 1990; Sayette & Wilson, 1991; Sayette, Wilson, & Carpenter, 1989). In addition to self-report measures, alcohol has been shown to have stress response–dampening effects when assessed with peripheral physiological indicators (i.e., cardiac interbeat interval, pulse transit time, muscle tension, heart rate, skin conductance, blood pressure, etc.; Finn & Pihl, 1987; Finn, Zeitouni, & Pihl, 1990; Levenson et al., 1980; Panknin, Dickensheets, Nixon, & Lovallo, 2002; Sayette & Wilson, 1991; Sher et al., 2007; Stewart & Pihl, 1994; Stritzke, Patrick, & Lang, 1995). Conversely, other researchers have reported no stress–response-dampening effects using these same physiological indicators (Abrams & Wilson, 1979; Levenson et al., 1980; Lindman, 1983; Sayette et al., 1994; Wilson, Brick, Adler, Cocco, & Breslin, 1989).

Recently, scientists have used fear-potentiated startle (Davis, 1989) as a measure of anxiety/fear response to examine alcohol's stress response–dampening effects. In one study, participants were shown unpleasant, neutral, or pleasant slides (to manipulate emotional valence) and received an unexpected intense tone to induce a startle response (Stritzke et al., 1995). Results demonstrated that alcohol reduced startle responses for all slide types. Two subsequent studies by these researchers demonstrated that alcohol reduced fear-potentiated startle, but only during a dual-task situation in which participants' attention was focused away (or distracted) from a threatening stimulus (Curtin, Lang, Patrick, & Stritzke, 1998; Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001). However, a more recent study examining the effect of alcohol dose on fear-

potentiated startle showed a reduction in startle responses for medium and high, but not low or no, alcohol doses; however, these findings occurred in the absence of distraction from threat (Donohue, Curtin, Patrick, & Lang, 2007).

Given these equivocal findings, theories have been put forth to attempt to reconcile these data. Contemporary models suggest that alcohol's presumed effects on anxiety are mediated via cognitive processes (see Wilson, 1988). Two of the most prominent models of this type are the appraisal-disruption model (Sayette, 1993) and the attention-allocation model (Steele & Josephs, 1990). The appraisal-disruption model postulates that acute alcohol intoxication produces anxiolysis by impairing the ability to appraise novel threat-imparting information by disrupting the spread of activation of stress-related information or representations stored in long-term memory so long as alcohol consumption temporally precedes the stressor. However, it is important to note that the model also predicts that anxiolysis is less likely to occur if the threatening or stressful information temporally precedes alcohol consumption, in which case the information will have been more adaptively processed (Sayette, 1993). The attention-allocation model contends that acute alcohol intoxication disrupts cognitive functioning, thus creating a "myopic" or a narrowing effect on attentional capacity. As a result, alcohol's effects on anxiety are moderated depending on where the remaining attentional capacity is allocated. Specifically, according to the model, an anxiolytic effect will occur if one's attention is distracted away from threatening cues, but an anxiogenic effect will occur if the opposite is the case (Steele & Josephs, 1990).

Studies have also shown that anxiety is negatively related to aggression. For example, low levels of self-reported fear during childhood significantly predict a diagnosis of conduct disorder during adolescence (Eaves, Darch, & Williams, 2004; Raine, Reynolds, Venables, Mednick, Farrington, 1998). Furthermore, psychiatric disorders typified by decreased fear (such as psychopathy, conduct disorder, and antisocial personality disorder) are associated with increased aggressive behavior (reviewed in Lorber, 2004). However, it is important to note that other disorders characterized by aggression, such as borderline personality disorder, intermittent explosive disorder, and posttraumatic stress disorder, are associated with high levels of anxiety (Coccaro, Kavoussi, Berman, & Lish, 1998; Crowson, Frueh, Beidel, & Turner, 1998; Orsillo, Heimberg, Juster, & Garrett, 1996; Silk, 1994). Laboratory studies have shown that anxiolytic drugs, such as diazepam (Cherek, Steinberg, Kelly, Robinson, & Spiga, 1990; Wilkinson, 1985), triazolam (Cherek, Spiga, Roache, & Cowan, 1991) and other benzodiazepines (Berman & Taylor, 1995), all tend to increase aggression. Laboratory researchers have also investigated the relation between anxiety sensitivity (i.e., fear of anxiety-related physiological sensations) and aggression (Broman-Falks, McCloskey, & Berman, 2006). Their results demonstrated an inverse relation between anxiety sensitivity and extreme aggression such that individuals with higher anxiety sensitivity were less aggressive in response to intense provocation. Finally, animal models have revealed that normally aggressive rats exhibit deficiencies in

anxiety and fear responses (Ferreira, Hansen, Nielsen, Archer, & Minor, 1989; Hard & Hansen, 1985). According to Gray (1982, 1987), decreased anxiety is associated with increased aggression because of a disruption of behavioral inhibition normally exerted by the septal-hippocampal system in response to exposure to threat or novelty.

Thus, the purpose of the present study was to examine the hypothesis that state anxiety would interact with acute alcohol intoxication to affect aggression. As noted above, researchers have identified numerous dispositional and contextual variables that influence the alcohol-aggression relation. However, there have been no examinations of state anxiety as a possible moderator of this relation. Given the links among acute alcohol consumption, anxiety, and aggression, one could argue that anxiety is, in fact, both a mediator and a moderator of the alcohol-aggression association. It can be viewed as a mediator in the sense that alcohol reduces anxiety, which then increases the probability of aggression (Pihl et al., 1993). However, the role of anxiety in the alcohol-aggression relation can also be tested via its ability to suppress aggression. In other words, anxiety can be viewed as a moderator of the alcohol-aggression relation in that heightened anxiety, produced by a contextual stressor, should suppress aggression under alcohol similar to levels seen in sober individuals. It is this latter prediction that is the focus of the present study.

Method

Participants

Participants were 80 healthy male social drinkers between 21 and 33 years of age ($M = 23.76$, $SD = 2.8$). Social drinking was defined as consuming at least 3–4 alcoholic beverages per occasion at least twice per month. It is noteworthy that moderating influences on the alcohol-aggression relation tend to be observed primarily in men (Barnwell et al., 2006; Giancola, 2002, 2004b) and that alcohol is more likely to increase aggression in men than in women (e.g., Giancola et al., 2002; Gustafson, 1991; Hoaken & Pihl, 2000). As such, given that this is the first study to examine the moderating effects of anxiety on intoxicated aggression, we elected to conduct this preliminary investigation only on males. Participants were recruited through advertisements placed in newspapers in Lexington, Kentucky. Respondents were initially screened by telephone using a custom-developed interview (Giancola, 1999). Individuals reporting any past or present drug- or alcohol-related problems, serious head injuries, learning disabilities, or serious psychiatric symptomatology were excluded from participation. Individuals reporting abstinence from alcohol use or a condition in which alcohol consumption is medically contraindicated were also excluded. Respondents were screened for alcohol use problems using the Short Michigan Alcoholism Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975). Any person with a score of 8 or more on the SMAST was excluded from participation (Giancola, 2004b). Anyone with a positive breath alcohol concentration (BAC) reading or a positive urine drug test (i.e., cocaine, marijuana, mor-

phine, amphetamines, benzodiazepines, and barbiturates) result was also excluded (no participant was excluded for any of these reasons).

The ethnic composition of the participants was as follows: 60 Caucasians, 18 African Americans, 1 Hispanic, and 1 other. Other demographic indicators revealed that 92.5% of participants were never married, 20% had a high school degree and were not pursuing further education, 54% had a high school degree and were working on a bachelor's or an associate's degree, 21% had a bachelor's or an associate's degree, 4% had a graduate degree, and 1% did not graduate from high school. Furthermore, 55% supported themselves financially and earned an average of approximately \$25,000 per year; the remainder was supported by a parent or spouse.

Experimental Design

A 2 (Group: anxiety induction, no anxiety induction) \times 2 (Beverage: alcohol, placebo) \times 2 (Provocation: low, high) mixed-model design, with provocation as the repeated measure, was employed in this study. Participants were randomly assigned to one of four groups: (a) alcohol + anxiety induction ($n = 20$), (b) placebo + anxiety induction ($n = 20$), (c) alcohol + no anxiety induction ($n = 20$), and (d) placebo + no anxiety induction ($n = 20$).

Measures

Positive and Negative Affect Schedule—Extended (PANAS-X; Watson & Clark, 1994). The PANAS-X is a 60-item measure of state affect with 11 subscales (i.e., Fear, Hostility, Guilt, Sadness, Joviality, Self-Assurance, Attentiveness, Shyness, Fatigue, Serenity, and Surprise). The Fear, Hostility, Attentiveness, and Fatigue subscales were administered in a random order (total of 20 items). Participants rated the extent to which they experienced each adjective on a 5-point Likert scale (1 = *very slightly or not at all*, 2 = *a little*, 3 = *moderately*, 4 = *quite a bit*, and 5 = *extremely*). The Hostility, Attentiveness, and Fatigue subscales were included to mask the fact that anxiety was being assessed and to keep the measure at a length that could be completed quickly. For the purpose of this investigation, we used the Fear subscale to measure anxiety. Despite the name of this subscale, the items (i.e., afraid, frightened, scared, nervous, jittery, shaky) are more closely related to the concept of anxiety than fear. Anxiety has been defined as a long-lasting, general state of distress prompted by generalized cues such as an imminent unpleasant task or interaction (Lang, Davis, & Öhman, 2000). In contrast, fear can be defined as an emotional alarm system that is activated by specific cues such as imminent physical dangers, potential injury, or life-threatening situations (Barlow, 1988). As such, fear would be better conceptualized by terms such as *alarmed*, *dismayed*, *horrified*, *desperate*, or *terrified*. More important, Watson and Clark (1994) noted that the fear scale assesses the same basic affect as the Profile of Mood States (POMS) Tension–Anxiety scale. They reported correlations of .85, .74, and .56 with the POMS Tension–Anxiety scale,

the Hopkins Symptom Checklist Anxiety Scale, and the Spielberger State–Trait Anxiety Inventory, respectively. As a result of the strong correlations between the PANAS Fear subscale and other measures of anxiety, some researchers refer to this subscale as the Fear–Anxiety subscale and use it as a measure of anxiety despite the conceptual delineation of anxiety and fear (e.g., Gunthert, Cohen, & Armeli, 2002; Stice, 1997). Given the above correlations, the fact that this subscale has been likened to other measures of anxiety, and that it has been successfully employed as a measure of anxiety, we argue herein that the PANAS-X Fear subscale can be conceptualized as a measure of state anxiety.

Participants were asked to complete this state anxiety scale at different points throughout the study to assess alcohol's impact on anxiety as well as the effectiveness of the anxiety induction manipulation (see below). Specifically, the scale was administered following (a) obtaining informed consent (baseline), (b) the anxiety manipulation, (c) beverage consumption, and (d) the aggression task.

Taylor Aggression Paradigm (TAP; Taylor, 1967). A modified version of the TAP was used to measure aggressive behavior. This task placed participants in a situation where electric shocks were administered to, and received from, a fictitious opponent under the guise of a competitive reaction time task. Physical aggression was operationalized as the shock intensities selected by the participants. Given that aggression is highly influenced by provocation (Chermack & Giancola, 1997), shock selections were recorded under conditions of low and high provocation.

Participants were seated at a table in a small room. This room contained a table with a computer monitor and keyboard. White adhesive labels marked 1 through 10 were attached to the number keys running across the top of the keyboard. The labels *low*, *medium*, and *high* were placed above keys 1, 5, and 10, respectively, to indicate the subjective levels of shock corresponding to the number keys. The keyboard and monitor were connected to a computer located in an adjacent control room out of the participant's view.

Reviews of the literature have concluded that the TAP is a safe measure of aggression that boasts good construct validity (Anderson & Bushman, 1997; Giancola & Chermack, 1998). Convergent validity has been established by positive associations with self-report measures of aggression and by the fact that persons characterized as being physically violent have been found to be more aggressive, on this and similar tasks, than persons characterized as being nonviolent (Cherek, Moeller, Schnapp, & Dougherty, 1997; Giancola & Zeichner, 1995b; Hammock & Richardson, 1992). Discriminant validity has been established by a lack of significant associations with measures that are theoretically unrelated to aggression (Bernstein, Richardson, & Hammock, 1987; Giancola & Zeichner, 1995b).

First trial shock intensity. This measure comprises the shock intensity selection (1 through 10) participants administered on the first trial of the aggression task. It represents a measure of unprovoked aggression inasmuch as participants win the first trial and thus have no information about the ostensible aggressive intentions of their opponent.

Mean shock intensity. This measure comprises the mean shock intensity selection (1 through 10) within each provocation condition in the aggression task. As such, two separate dependent variables were calculated: (a) mean shock intensity under low provocation and (b) mean shock intensity under high provocation. These variables represent measures of aggression in response to provocation.

Procedure

Following the telephone screening interview, we scheduled individuals eligible for participation for an appointment to come to the laboratory. They were told to refrain from drinking alcohol 24 hr prior to testing, to refrain from using recreational drugs from the time of the telephone interview, and to refrain from eating 4 hr prior to testing. Participants were told that they would receive \$50 at the completion of the study as compensation.

On entering the laboratory, the experimenter explained the procedures of the study to the participants, who were asked to sign an informed consent form. The experimenter then assessed their BACs to ensure sobriety. If the BAC test was negative, participants underwent a urine drug test (On-Trak Teststik; Varian Inc., Lake Forest, CA). BACs were measured using the Alco-Sensor IV breath analyzer (Intoximeters Inc., St. Louis, MO). Demographic data were then collected. Participants were then escorted into a testing room and completed the baseline state anxiety measure (i.e., Fear subscale of the PANAS-X).

Anxiety induction. For those participants in the anxiety-induction groups, the manipulation was carried out as follows: Prior to beverage consumption, participants were told that their "thinking style" would be assessed by their ability to prepare and deliver a short speech in front of a video camera. They were read the following script:

You will be taking part in a portion of the study that deals with testing your thinking style. We are interested in your ability to think quickly with limited time for preparation. Research has shown that these skills are related to your reaction time. For this task you must quickly prepare and then deliver a short speech about what you like and dislike about your body while standing directly in front of this video camera that will record your speech. You will have 6 minutes to prepare a 3-minute speech. This will take place immediately after you complete the reaction time task. It is very important that you think about the speech you are about to give. This has been shown to improve performance on the reaction time task. This clock will now give you a 6-minute countdown. You will have this time to prepare your speech in your mind. When the 6 minutes are up, you will begin the reaction time task.

In actuality, the participants who received this manipulation did not have to deliver a speech. The manipulation was simply used to induce anxiety. This manipulation was a modified version of Sayette, Martin, Perrott, Wertz, and Hufford's (2001) speech stressor. Similar well-established and well-validated anxiety induction manipulations have been used in the alcohol research literature (see Levenson et al., 1980; Sayette, Smith, Breiner, & Wilson, 1992; Sher et al., 2007). Following the anxiety induction, the PANAS was

given a second time to assess the effectiveness of the anxiety manipulation. To ensure proper experimental control, the group that did not receive the manipulation also completed the state anxiety scale following a 6-min waiting period.

Beverage administration. Following the completion of the second state anxiety scale, participants were administered their beverages. Those who received alcohol were administered a dose of 1 g/kg of 95% alcohol USP mixed at a 1:5 ratio with a brand-name orange juice. Beverages were poured into the requisite number of glasses in equal quantities. Three ccs of alcohol were added to each glass with orange juice. The dosing procedure was also calculated for the placebo groups; however, they received an isovolemic beverage consisting of only orange juice (i.e., the missing alcohol portion was replaced with orange juice). Immediately prior to serving the placebo beverages, the rims of the glasses were sprayed with alcohol. Participants were not given any information regarding what to expect from their beverages.

We could have used a sober control group, in which participants received a nonalcoholic beverage and were told that they consumed no alcohol, in addition to the two beverage groups in this study (i.e., alcohol and placebo). Overall, research has shown that the vast majority of investigations have indicated that, whereas alcohol groups display significantly greater levels of aggression compared with sober control groups, placebo and sober controls do not differ significantly from one another (reviewed in Chermack & Giancola, 1997; Bushman & Cooper, 1990). In recognition of that previous research, we employed only an alcohol group and a placebo group.

Twenty minutes were then allotted for beverage consumption. To allow the alcohol to be sufficiently absorbed into the bloodstream, persons receiving alcohol had their pain threshold and tolerance tested (described below) 20 min after they finished their drinks. Specifically, immediately after beverage consumption (which took 20 minutes) participants waited 10 min and then completed the third state anxiety assessment (approximately 5 mins) and then received the aggression task instructions (approximately 5 min) in order to allow for a total of 20 min for the alcohol to be absorbed into the bloodstream (i.e., 20 minutes for beverage consumption and 20-minute wait period prior to pain threshold testing). Those in the placebo group did not have a 10-min wait time prior to completing the state anxiety scale and pain threshold and tolerance testing to avoid missing the short-lasting effects of placebo manipulations (Bradlyn & Young, 1983; Martin & Sayette, 1993). This study complied with the National Institute on Alcohol Abuse and Alcoholism's guidelines for the use of alcohol administration with human participants (National Advisory Council on Alcohol Abuse & Alcoholism, 2005).

Aggression task explanation. Following completion of the third state anxiety assessment, the aggression task was explained to the participants. They were informed that shortly after the words "Get Ready" appeared on the screen, the words "Press the Spacebar" would appear, at which time they would have to press, and hold down, the spacebar.

Following this, the words "Release the Spacebar" would appear, at which time they would have to lift their fingers off of the spacebar as quickly as possible. A "win" was signaled by the words "You Won. You Get to Give a Shock," and a "loss" was signaled by the words "You Lost. You Get a Shock." A winning trial allowed participants to deliver a shock to their opponent and a losing trial resulted in receiving a shock from this individual. Participants were not allowed to administer a shock to their opponent. That is, following a winning trial, the task paused until a shock was selected. However, participants were told that the "#1" shock button delivers a very low-intensity shock that is best characterized as "very mild" and "definitely not painful." This procedure has been widely used and is accepted to reflect a nonaggressive response option (Giancola & Chermack, 1998). Following a winning trial and pressing a shock button, participants were able to view their shock selection on a specially designed "volt meter" on the computer screen and by the illumination of 1 of 10 "shock lights" (ranging from 1 [low] to 10 [high]) on the computer screen. Both of these indicators displayed readings that corresponded with the selected shock level. These images were used to reinforce participants' beliefs that they were actually administering shocks. On losing a trial, participants received a shock and were given feedback regarding the level of that shock in the form of a signal on the volt meter and the illumination of 1 of the 10 "shock lights" on the computer screen.

Participants were told that they had a choice of 10 different shock intensities to administer at the end of each winning trial for a duration of their choosing. Regardless of beverage group assignment, all participants were informed that their opponent was intoxicated. This was done to ensure that the "drinking status" of the opponent did not confound any potential beverage group differences in aggression.

Pain threshold and tolerance testing. Participants' pain thresholds and tolerances were then assessed to determine the intensity parameters for the shocks they would receive. This was accomplished via the administration of short duration shocks (1 s) that increased in intensity in a stepwise manner from the lowest available shock setting, which was imperceptible, until the shocks reached a subjectively reported "painful" level. All shocks were administered through two finger electrodes attached to the index and middle fingers of the nondominant hand using Velcro straps. Participants were instructed to inform the experimenter when the shocks were "first detectable" and then when they reached a "painful" level. Later, during the actual testing, they received shocks that ranged from 1 to 10. These shocks were set at 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, and 100%, respectively, of the highest tolerated shock intensity. The threshold and tolerance determination procedure was conducted while the participant was seated in the testing room and the experimenter was in the adjacent control room. They communicated through an intercom. The experimenter secretly viewed the participant through a hidden video camera.

Aggression task. The aggression-potentiating effects of alcohol have been shown to be more likely to occur on the

ascending limb of the BAC curve (Giancola & Zeichner, 1997) and that a BAC of at least 0.08% is required to elicit robust levels of aggression (Giancola & Zeichner, 1997; Gustafson, 1992; Pihl, Smith, & Farrell, 1984). As such, the alcohol group began the task after they reached an ascending BAC of at least 0.09%. One could argue that the duration between the end of beverage consumption and beginning the aggression task should have been standardized for both beverage groups. This was not done because it would have reduced the effectiveness of the placebo manipulation (noted above) and would have produced undesirable large individual differences in BACs during the aggression task. Pertinently, following a standardized 30-min wait after beverage consumption, Giancola and Zeichner (1995a) found a BAC range between 0.06 and 0.14 using the same alcohol dose administered in the present investigation. More important, they also found a significant positive relation between this BAC range and aggression. Interestingly, these researchers also determined that even a long delay (approximately 3 hr) between the end of alcohol consumption and the measurement of aggression does not affect responses on the TAP (Giancola & Zeichner, 1997). Therefore, it is very unlikely that the extra 15–20 min alcohol participants had to wait before beginning the aggression task adversely affected their results. Given these data and arguments, it is clear that more experimental control is gained by standardizing BACs at the time aggression is assessed rather than simply waiting a predetermined time following beverage consumption. Following beverage administration, BACs were monitored as they increased. Participants began the pain threshold and tolerance testing procedure and the aggression task after reaching an ascending BAC of at least 0.09%. Those participants who did not receive alcohol began the pain threshold and tolerance testing and aggression task following the third state anxiety assessment. Immediately before beginning the aggression task, participants provided subjective ratings of their level of intoxication. This was done using a specially constructed scale ranging from 0 to 11 on which 0 was labeled *not drunk at all*, 8 was labeled *drunk as I have ever been*, and 11 was labeled *more drunk than I have ever been*.

The entire procedure consisted of two successive blocks of trials. During the first block, participants received shock intensities between 1 and 4 (mean intensity = 2.5) after they lost a trial. This denoted the low-provocation condition. During the next block, they received shock intensities between 7 and 10 (mean intensity = 8.5) after they lost a trial. This denoted the high-provocation condition. Each block consisted of 16 trials (8 wins and 8 losses). There were 2 transition trials between the blocks. Participants lost both trials and received respective shock intensities of 5 and 6. These trials were added to give the appearance of a smooth transition between the low- and high-provocation blocks. The two provocation conditions were not counterbalanced. Taylor and Chermack (1993) have argued that using the low-high sequence adds an increased degree of external validity to the task because this ordering best reflects how an escalation in interpersonal provocation leads to increased violence in "real-life" situations. Thus, there was a total

of 34 trials. All shocks delivered to the participants were of a 1-s duration. In actuality, reaction times were not measured; the competitive task was used to lead participants to believe that they were engaged in an adversarial interaction with another individual. The win-loss sequence was predetermined and controlled by the computer program that executed the task. The sequence was presented in a fixed random order with no more than three consecutive wins or losses. The trials were interspersed by 5-s intervals. The initiation of trials, administration of shocks to the participants, and the recording of their responses were controlled by a computer. The experimenters, other electronic equipment, and the computer that controlled the task were located in an adjacent room out of the participant's view. The experimenter secretly viewed and listened to the participant through a hidden video camera and microphone throughout the procedure. Participants were observed to ensure their compliance with beverage consumption and aggression task instructions.

Postaggression task measures. Immediately following the testing procedure, BACs were measured and participants were again asked to rate their subjective state of intoxication. In addition to this, they were asked whether the alcohol they drank caused them any impairment on a scale ranging from 0 to 10 on which 0 was labeled *no impairment*, 5 was labeled *moderate impairment*, and 10 was labeled *strong impairment*. Participants were then asked a yes/no question regarding whether they believed that they had consumed alcohol. They were also asked a variety of questions to indirectly assess the credibility of the aggression task deception manipulation (see below). The state anxiety scale was given a final time to measure state anxiety following the aggression task. All individuals who received alcohol were required to remain in the laboratory until their BAC dropped to 0.04% and they could pass a field sobriety test.

Deception manipulation. To disguise the task as a measure of aggression, we gave participants a fictitious cover story. As noted above, they were informed that the purpose of the study was to determine how different types of thinking styles influence reaction time in a competitive situation. To convince participants that they were actually competing against another person, a confederate was seated in a room adjacent to the testing room. As the experimenter led the participant into the testing room, s/he identified the confederate as the "opponent." No opportunity for an interaction between the participant and the confederate was allowed. Furthermore, immediately before assessing their pain thresholds and tolerances, participants were informed that their opponent would undergo the threshold and tolerance assessment procedure first. Participants were also informed that they would be able to hear their opponent's responses to the procedure over an intercom that ostensibly served the two testing rooms and the control room. In actuality, the confederate acted as the fictitious opponent and answered the experimenter's questions regarding the testing of their pain threshold and tolerance in accordance with a list of predetermined responses. All participants heard the same

experimenter-confederate verbal exchange. Of course, in reality, there was no actual opponent.

Results

Manipulation Checks

Task deception. Participants were asked about their subjective perceptions of their opponent, whether their opponent tried hard to win, whether they thought the task was a good measure of reaction time, how well they believed they performed on the task, and so forth. Typical responses included that their opponent was competitive, did well, played fairly, tried hard, did better or about the same as them, and that the task was a good test of reaction time. In sum, their descriptions indicated that the deception manipulation was successful.

Placebo checks. All participants in the placebo group indicated that they believed that they consumed alcohol. In response to the questionnaire inquiring about how drunk they felt, persons in the alcohol group reported average pre- and post-TAP ratings of 4.43 ($SD = 2.26$) and 4.60 ($SD = 1.98$; scale range: 0–11). Persons in the placebo group reported average ratings of 1.65 ($SD = 1.29$) and 2.08 ($SD = 1.64$), respectively: alcohol versus placebo pre-TAP ratings: $t(79) = -6.75, p < .001, d = 1.53^1$; post-TAP ratings: $t(79) = -6.15, p < .001, d = 1.40$. In response to a question about whether the alcohol they drank caused any impairment, individuals in the alcohol group reported an average rating of 4.85 ($SD = 2.52$) and those in the placebo group reported an average rating of 2.05 ($SD = 1.77$), $t(79) = -5.75, p < .001, d = 1.30$ (scale range: 0–10). The success of a placebo manipulation is evidenced by reports that participants had consumed alcohol, not that they believed that they were equally intoxicated as those who had received a high dose of alcohol (Martin & Sayette, 1993; Sayette, Martin, Wertz, Perrott, & Peters, 2005). This has been demonstrated repeatedly in previous studies using placebo manipulations (Chermack & Taylor, 1995; McCloskey & Berman, 2003; Pihl & Zacchia, 1986).

Anxiety induction checks. Data for the anxiety induction were analyzed using a 2 (Beverage: alcohol, placebo) \times 2 (Group: anxiety, no anxiety) \times 4 (Anxiety Assessment Time: baseline, postinduction, postbeverage, post-TAP) mixed-model analysis of variance (ANOVA), with anxiety assessment time as the repeated measure, which revealed a significant Anxiety Assessment Time \times Group interaction, $F(3, 77) = 4.67, p = .005, \eta^2 = .167^2$. Results indicated that there were no significant differences in state anxiety between the anxiety induction and no anxiety induction groups at the baseline measurement. Following the anxiety induction, the anxiety induction group

¹ Cohen's d values of 0.2, 0.5, and 0.8 indicate small, medium, and large effect sizes, respectively (Cohen, 1992).

² Eta squared (η^2) values can be interpreted in the same way R^2 values are interpreted. An η^2 of .04 indicates that 4% of the variability in the dependent variable can be accounted for by the independent variable.

showed a significant increase in state anxiety from baseline: baseline, $M = 1.15$, $SD = .23$; postanxiety induction, $M = 1.24$, $SD = .33$; $t(39) = -2.57$, $p = .01$, $d = 0.32$, whereas the no anxiety induction group demonstrated a significant decrease from baseline: baseline, $M = 1.19$, $SD = .23$; postanxiety induction, $M = 1.11$, $SD = .23$; $t(39) = 2.55$, $p = .01$, $d = 0.36$. In addition, the anxiety induction group had significantly higher anxiety ratings than their no anxiety induction group counterparts after the anxiety induction: anxiety induction group, $M = 1.24$, $SD = .33$; no anxiety induction group, $M = 1.11$, $SD = .21$; $t(79) = -2.00$, $p = .049$, $d = 0.48$. Following beverage administration, self-reported anxiety increased from the postanxiety induction measurement for those in the placebo + no anxiety induction group. However, this increase was not significant. In addition, anxiety ratings significantly decreased for the alcohol + no anxiety induction group, $t(19) = 3.87$, $p = .001$, $d = 1.10$ (see Table 1). There was no significant difference between placebo groups following beverage administration. However, after beverage administration, the difference between alcohol groups approached significance, $t(39) = -1.89$, $p = .06$. Following the aggression task, the anxiety ratings for the alcohol + anxiety induction group were still significantly higher than the alcohol + no anxiety induction group, $t(39) = -2.44$, $p = .025$, $d = 0.83$. However, there were no significant differences in anxiety ratings between the alcohol + anxiety induction group and the placebo groups. The alcohol + no anxiety induction group had lower postaggression anxiety task ratings than the placebo groups, but this difference was not significant (see Table 1).

Aggression Data

First trial shock intensity. Data for first trial shock selection were analyzed using a 2 (Beverage: alcohol, placebo) \times 2 (Group: anxiety, no anxiety) ANOVA, which revealed a significant Beverage \times Group interaction, $F(1, 79) = 5.29$, $p = .024$, $\eta^2 = .073$ (see Figure 1). The main effects for group and beverage were not significant. However, it is worth noting that the main effect for beverage approached significance, $F(1, 79) = 3.19$, $p = .078$, $\eta^2 = .041$. Unprovoked aggression was significantly lower for the alcohol + anxiety induction group compared with the alcohol + no anxiety induction group: alcohol + anxiety induction, $M = 3.42$, $SD = 2.36$; alcohol + no anxiety induction,

$M = 5.21$, $SD = 3.12$; $t(39) = 1.85$, $p = .036$, $d = 0.60$. The placebo groups did not differ significantly from one another. In addition, there was no significant difference between the alcohol + anxiety induction and placebo + anxiety induction groups: alcohol + anxiety induction, $M = 3.42$, $SD = 2.36$; placebo + anxiety induction, $M = 3.80$, $SD = 2.98$; $t(39) = 0.438$, $p = .664$. However, there was a significant difference between the alcohol + no anxiety induction and the placebo + no anxiety induction conditions: alcohol + no anxiety induction, $M = 5.21$, $SD = 3.12$; placebo + no anxiety induction, $M = 2.70$, $SD = 1.89$; $t(39) = -3.056$, $p = .006$, $d = 0.99$.

Mean shock intensity. Data for shock intensity were analyzed using a 2 (Beverage: alcohol, placebo) \times 2 (Group: anxiety, no anxiety) \times 2 (Provocation: low, high) mixed-model ANOVA, with provocation as the repeated measure, which revealed a significant Beverage \times Group interaction, $F(1, 79) = 4.58$, $p = .036$, $\eta^2 = .068$ (see Figure 2). The main effect for group was not significant. However, there was a significant main effect for beverage, $F(1, 79) = 4.31$, $p = .041$, $\eta^2 = .055$. The main effect of provocation was also significant, $F(1, 79) = 170.13$, $p < .001$, $\eta^2 = .694$, indicating that high provocation engendered greater aggression than low provocation. Aggression was significantly lower for the alcohol + anxiety induction group compared with the alcohol + no anxiety induction group: alcohol + anxiety induction, $M = 4.80$, $SD = 1.59$; alcohol + no anxiety induction, $M = 6.21$, $SD = 2.07$; $t(39) = 2.13$, $p = .04$, $d = 0.69$. Furthermore, the placebo groups did not differ from one another. In addition, there was no significant difference between the alcohol + anxiety induction and placebo + anxiety induction groups: alcohol + anxiety induction, $M = 4.80$, $SD = 1.59$; placebo + anxiety induction, $M = 4.89$, $SD = 1.84$; $t(39) = .177$, $p = .86$. However, there was a significant difference between the alcohol + no anxiety induction and the placebo + no anxiety induction conditions: alcohol + no anxiety induction, $M = 6.21$, $SD = 2.07$; placebo + no anxiety induction, $M = 4.42$, $SD = 1.68$; $t(39) = -2.98$, $p = .005$, $d = 0.97$.

Discussion

Results demonstrated that the anxiety induction was successful in increasing subjective ratings of anxiety. Furthermore, alcohol decreased anxiety ratings for persons who did not receive the anxiety induction. These results suggest that

Table 1
State Anxiety Ratings

Group	Time 1		Time 2		Time 3		Time 4	
	Baseline		Postinduction		Postbeverage		Post-TAP	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Placebo + no anxiety induction	1.18	0.26	1.09	0.18	1.12	0.21	1.05	0.12
Placebo + anxiety induction	1.08	0.11	1.18	0.26	1.09	0.19	1.05	0.19
Alcohol + no anxiety induction	1.2	0.21	1.13	0.25	1.04	0.09	1.0	0.0
Alcohol + anxiety induction	1.2	0.29	1.29	0.39	1.18	0.3	1.07	0.14

Note. TAP = Taylor Aggression Paradigm.

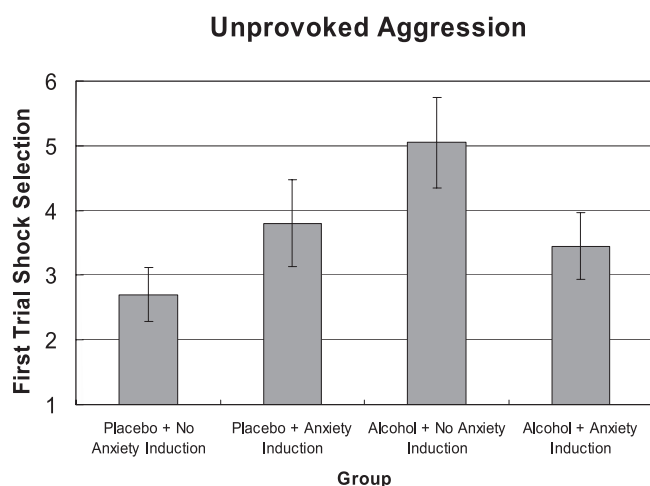


Figure 1. Effects of beverage and anxiety induction on unprovoked aggression.

the anxiety induction attenuated aggression in intoxicated individuals. In addition, intoxicated persons who underwent the anxiety induction were no more aggressive than their sober counterparts. It should also be noted that there were no significant differences between the two placebo groups. Those in the placebo + anxiety induction condition did not exhibit significantly less aggression than those in the placebo + no anxiety induction condition. However, it is likely that those individuals in the placebo + no anxiety induction group exhibited such little aggression that the participants who received the anxiety manipulation could not exhibit a significantly lower amount of aggression, essentially demonstrating a floor effect. This pattern of findings was consistent across both measures of aggression. Taken together, these data suggest that elevations in state anxiety may be effective in suppressing intoxicated aggression. It is also worth noting that no provocation interaction was found for either measure of aggression. Given that provocation is one of the strongest elicitors of aggression, it might be hypothesized that the anxiety manipulation would fail to lower aggression at higher levels of provocation. However, it could be argued that the lack of a significant interaction involving provocation is indicative of the strength of the anxiety induction manipulation.

Nevertheless, it remains unclear how a decrease in anxiety can lead to an increased likelihood of intoxicated aggression. Several models have been proposed that attempt to explain the causal mechanism by which alcohol's effect on anxiety may lead to increased aggressive behavior. One possibility is a purely biological explanation. Alcohol has well-established pharmacological effects, acting as a gamma-aminobutyric acid (GABA) agonist, directly affecting the GABA_A receptor (Criswell & Breese, 2005; Warneke, 1991). More specifically, it has been suggested that alcohol's pharmacological effects on GABA in the septal-hippocampal system are primarily responsible for anxiolysis (Gray, 1982, 1987). Moreover, Gray (1982, 1987) has argued that the septal-hippocampal system is

responsible for inhibiting behavior following exposure to threat. Alcohol enhances GABA's ability to open CL-ion channels, which results in more frequent GABAergic neuronal firing and increases GABA's inhibitory effects. This increase in GABAergic inhibition results in a decrease of the septal-hippocampal system's effect on behavioral inhibition. As a result, alcohol produces more aggression because the neurological pathways that would normally work to constrain aggressive responses are disengaged. Of course, other neurotransmitters such as serotonin have also been implicated in alcohol's anxiolytic effects (LeMarquand, Pihl, & Benkelfat, 1994).

Others have advanced more psychological explanations for alcohol's effects on anxiety and aggression. Pihl et al. (1993) argued that alcohol has an indirect effect on anxiety reduction. These theorists maintain that alcohol reduces anxiety, and thereby increases aggression, by disrupting cognitive abilities involving the perception and processing of cues that signal threat, danger, or punishment that typically engender inhibition. According to these theorists, the probability of aggression is increased as threat cues associated with interpersonal conflicts are effectively removed from a situation. One possible explanation for the data presented in this study is that the anxiety manipulation resulted in increased perception and processing of the interpersonal conflict inherent in the aggression task. If the anxiety induction resulted in increased attention to this conflict, it could have produced greater inhibition and thus an attenuating effect on aggression. However, the data from this investigation cannot speak to the participant's perception or processing of threat.

Another possible explanation of our findings can be derived from Sayette's (1993) appraisal-disruption model. Sayette (1993) developed this model, in part, to address inconsistencies within the literature regarding alcohol's effect on anxiety (see Cappell & Greeley, 1987; Steele & Josephs, 1988). The appraisal-disruption model suggests that alcohol's effects on anxiety are a result of the impair-

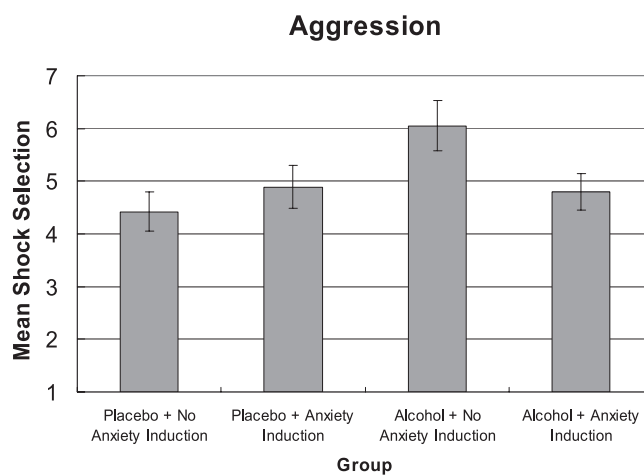


Figure 2. Effects of beverage and anxiety induction on average shock intensity selection.

ment of cognitive processes produced by alcohol (Sayette, 1993). Sayette (1993) argued that alcohol's impact on anxiety is dependent on the temporal occurrence of stressful information. Alcohol is predicted to decrease anxiety only when stressful or anxiety-inducing information is presented after intoxication. In this case, intoxication is theorized to impair appraisal of this information because the stressful information will not be properly processed and integrated into existing knowledge structures and thus the individual is not expected to become anxious. Alternatively, if the stressful information is presented prior to intoxication, the appraisal-disruption model predicts that alcohol is less likely to reduce anxiety. In this situation, the stressful information is processed before intoxication without the interference of alcohol. Accordingly, alcohol is less likely to reduce the anxiety associated with the stressful information. Within this model, it is expected that a potentially threatening situation that arises during intoxication may fail to produce anxiety, or any associated inhibitory effect, thereby possibly increasing the likelihood of aggression.

The present study provides indirect support for the appraisal-disruption model in that the alcohol + anxiety induction group was exposed to the anxiety manipulation before beverage consumption and alcohol did not reduce self-reports of anxiety in those individuals. Nevertheless, this study did not manipulate the temporal order of beverage consumption in relation to the anxiety induction. As such, the data cannot speak to alcohol's effects on information appraisal or subsequent effects on anxiety.

Another theory-based explanation of alcohol's effects on stress is provided by the attention-allocation model (Steele & Josephs, 1988). The model predicts that alcohol impairs information processing capacity (Steele & Josephs, 1990). This results in a narrowing of attention to the most immediate and salient cues in the environment. Thus, alcohol is predicted to have an anxiolytic effect only if there is a simultaneous, non-anxiety-provoking, distracting event or target. Because of the diminished cognitive resources resulting from alcohol intoxication, the distracting event purportedly occupies all of the intoxicated individual's attentional capacity. As such, Steele and Josephs (1990) postulated that distraction moderates the relation between alcohol consumption and anxiety. Evidence to support their model comes from studies demonstrating that following an anxiety induction manipulation, alcohol significantly decreased subjective anxiety for persons whose attention was distracted by performing a cognitive task. However, for those assigned to a no distraction condition, alcohol actually increased anxiety (Josephs & Steele, 1990; Steele & Josephs, 1988).

It can be argued that the results of the current investigation can also be explained by the attention-allocation model. One possibility is that the anxiety induction functioned as a strong inhibitory cue by activating information or representations of threat and danger, which would serve to suppress aggression. Another possibility is that the anxiety induction paired with the provocative nature of the TAP simply produced a dual task whereby the anxiety induction focused individuals' attention away from the TAP's provocative cues. Participants in the alcohol + no anxiety induc-

tion group displayed the highest level of aggression because their attention was presumably focused solely on the TAP. Likewise, those in the alcohol + anxiety induction group displayed levels of aggression equal to the placebo groups because their attention was focused presumably on the stress and worry elicited by the anxiety induction. These findings are consistent with recent research from our laboratory that shows that distraction from the TAP's instigatory cues using a working memory task also significantly suppressed aggression (Giancola & Corman, 2007).

Before concluding, some limitations of the present investigation should be addressed. First, given that we did not set out to test the attention-allocation model when designing this investigation, we did not include a measure to determine whether attention was actually distracted or focused away from the TAP during the anxiety manipulation. Second, the study would have benefited from a more thorough measurement of anxiety. Although the anxiety ratings used in this investigation demonstrated significant differences between the groups, the differences were small. One possible explanation is that the anxiety measure we used was not sensitive enough to assess anxiety accurately. Alternatively, the anxiety manipulation may not have produced large enough changes in state anxiety. By including additional measures of anxiety, it would have been possible to determine whether the anxiety induction produced larger changes in state anxiety or whether the measure used in this study was not sensitive enough to detect the changes. Third, it is possible that the anxiety induction could have served to increase self-awareness. Hull (1981) proposed a model of alcohol use that postulates that alcohol serves to decrease self-awareness. Specifically, alcohol reduces awareness of negative self-relevant information. As such, alcohol consumption is hypothesized to decrease anxiety, particularly in highly self-aware individuals, by minimizing attention to self-relevant aversive information.

Hull's (1981) hypothesis was tested by having sober and intoxicated subjects compete on the TAP either with or without a mirror and video camera in the room (Bailey, Leonard, Cranston, & Taylor, 1983). Results indicated that the self-awareness manipulation indeed served to attenuate aggression, yet it did not interact with alcohol consumption. In addition, numerous other studies, using similar self-awareness manipulations, have also demonstrated that increased self-awareness was effective in reducing aggression (Carver, 1975; Froming, Walker, & Lopyan, 1982; Scheier, 1976; Scheier, Fenigstein, & Buss, 1974). It could be argued that the manipulation used in the present investigation served to increase self-awareness rather than anxiety. The manipulation focused the individual's attention on things that they liked and disliked about their body, thus increasing awareness of negative self-relevant information. The use of an alternative anxiety induction that is less personally relevant might eliminate this possibility. Finally, the reliability of the measure of unprovoked aggression can be questioned because it was operationalized as the participants' shock selection on the first trial of the TAP. However, this measure has yielded positive findings in numerous studies (e.g., Berman & Taylor, 1995; Chermack & Taylor, 1995). At-

tempting to alter the reliability of this measure by instituting a sufficiently large number of initial consecutive wins runs the risk of decreasing the believability of the task.

In conclusion, this is the first study to demonstrate that state anxiety is an effective moderator of the alcohol–aggression relation. Specifically, the anxiety induction used in this investigation was effective in attenuating aggression in individuals who consumed alcohol. As such, these data further the understanding of conditions in which alcohol’s aggression-potentiating effects can be suppressed. The role of anxiety in the relation between alcohol intoxication and aggression is not well understood. As noted above, conceptual models have been advanced to explain the effect of anxiety on the alcohol–aggression relation. However, few studies have examined these models directly and none, other than this investigation, have assessed the impact of contextual anxiety on intoxicated aggression. Future studies may focus on other negative affective states, in addition to anxiety, as well as identifying how anxiety may function as a mechanism of the alcohol–aggression relation.

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