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TESTING THE ROLE OF ANXIETY AS AN UNDERLYING MECHANISM OF THE ALCOHOL-AGGRESSION RELATION

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ABSTRACT OF THESIS

TESTING THE ROLE OF ANXIETY AS AN UNDERLYING MECHANISM OF THE ALCOHOL-AGGRESSION RELATION

The purpose of this study was to test the hypothesis that acute alcohol consumption facilitates aggression through the reduction of adaptive anxiety/fear responses to danger/threat. Participants were 80 healthy male social drinkers between 21 and 33 years of age. They were randomly assigned to one of four groups: 1) Alcohol/anxiety induction (n=20), 2) Placebo/anxiety induction (n=20), 3) Alcohol only (n=20), and 4) Placebo only (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 (Taylor, 1967) was used to measure aggressive behavior in a situation where electric shocks were administered to, and received from, a fictitious opponent during a supposed competitive reaction-time task. Results indicated that the anxiety induction was successful in reducing aggression for participants who received alcohol. Results are discussed within the context of a number of theories specifying anxiety as a possible mediator of the alcohol aggression relation.

KEYWORDS: alcohol, aggression, anxiety, Taylor Aggression Paradigm, electric shocks

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THESIS

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2007

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THESIS

A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science in the
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Chapter One: Introduction

The relation between alcohol consumption and aggressive behavior has been well-established (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Ito, Miller, & Pollock, 1996). Recent FBI Uniform Crime Reports (2005, 2006) indicated that the rate of violent crimes has increased by 2.5% and 3.7% in the past two years. The latest Department of Justice report implicated alcohol use in over 30% of violent crimes (Department of Justice, 2002). Nevertheless, the precise number of violent crimes involving alcohol use is difficult to determine. Often, the offender is not present when the crime is reported or the victim has no knowledge of the offender's alcohol use. As such, the majority of crimes with reported offender alcohol use are cases of domestic abuse. Approximately 75% of reported domestic abuse cases specify alcohol consumption by the perpetrator (Department of Justice, 2002). In addition, alcohol is more closely related to cases of aggression and violence than the combined influence of all other drugs (Miczek, Weerts, & DeBold, 1993).

Alcohol and Aggression

Correlational studies have also linked alcohol use to violent crimes (reviewed in Moss & Tarter, 1993; Murdoch, Pihl, & Ross, 1990; Phil, Peterson, & Lau, 1993). Specifically, alcohol has been linked to cases of domestic violence (Quigley & Leonard, 2000; Sabia, 2004), sexual assault (Abbey et al., 2001; Ullman & Becklin, 2000), rape (Testa, 2002), assault (Martin & Bachman, 1997; Scott, Schafer, & Greenfield, 1999; Stevenson, Lind, & Weatherburn, 1999) and homicide (Lindqvist, 1991; Muzinic-Masle & Goreta, 2000; Pridemore, 2004).

The above studies are correlational in design and, although compelling, they do not speak to whether there is a causal relation between alcohol and aggression. Useful complements to these correlational investigations 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. These studies use a variety of competitive interpersonal tasks to measure aggression in the laboratory. One of the most common laboratory aggression tasks is the Taylor Aggression Paradigm (TAP) (Taylor, 1967). The TAP is presented as a reaction-time task between a participant and a confederate posing as an "opponent." Individuals are

told that the winner of each trial will administer an electric shock to the loser. However, the participant's performance on the reaction-time task is predetermined. After they "win" a trial, they select a shock intensity to administer to their "opponent." They also receive electric shocks supposedly from their "opponent," following losing trials, in order to induce provocation. Aggression is operationalized as the level of shock intensity selected. Variations of this task utilize the administration of tone blasts or the subtraction of points (redeemable for money) to operationalize aggression (reviewed in Giancola and Chermack, 1998).

In the past 35 years, over 60 studies using this methodology have been conducted to assess the effect of alcohol on aggression. Their findings overwhelmingly demonstrated that alcohol increases aggression compared with placebo or non-alcohol beverages (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Taylor & Chermack, 1993). Meta-analytic studies confirmed these data by showing that alcohol has a "medium" effect size (d = .49 to .61) on aggression (Bushman, 1993; Bushman & Cooper, 1990; Ito et al., 1996). Parenthetically, research also indicates 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; Collins & Schlenger, 1988; Wiley & Weisner, 1995). Specifically, in a sample of individuals with a history of violence, aggressive behaviors were overwhelmingly more likely to occur on days when alcohol was consumed as opposed to days when alcohol was not consumed (Fals-Stewart, 2003).

Risk Factors for Alcohol-Related Aggression

Although alcohol's effect size on aggression is in the "medium" range, it clearly does not increase aggression for all persons. Given this, researchers have focused on identifying variables that act as risk factors (i.e., moderators) of the alcohol-aggression relation. Specifically, studies have shown that alcohol only seems to facilitate aggression for persons with deviations on variables including, but not limited to, trait anger (Giancola, 2002a; Parrott & Giancola, 2004; Parrott & Zeichner, 2002), irritability (Giancola, 2002b), desired image of power (Quigley, Corbett, & Tedeschi, 2002), executive functioning (Giancola, 2004; Phil et al., 2003), antisocial personality disorder (Moeller et al., 1998), permissive beliefs about aggression (Leonard & Senchak, 1996),

dispositional empathy (Giancola, 2002c), dispositional aggressivity (Giancola, 2002d), intelligence (Welte & Wieczorek, 1999), degree of marital conflict (Quigley & Leonard, 1999) and sensation seeking (Cheong & Nagoshi, 1999). In contrast to the recent abundance of research focusing on risk factors for alcohol-related aggression, much less work has been directed at elucidating the mechanisms underlying the alcohol-aggression relation. Identifying risk factors is clearly important. However, equally important is determining exactly how alcohol causes aggression in persons at risk for such behavior.

Mechanisms of Alcohol-Related Aggression

A number of theories have been advanced to explain the mechanisms by which alcohol facilitates aggressive behavior. In general, these explanations fall under two broad categories of theoretical models of the alcohol-aggression relation: pharmacological and expectancy. One of the most basic pharmacological explanations is the disinhibition model. This model states that alcohol increases aggression directly by disrupting areas of the brain involved in the inhibitory control of behavior (Graham, 1980). However, this model cannot explain why acute alcohol intoxication does not result in aggressive behavior for all individuals (Bushman & Cooper, 1990; Giancola, 2003).

Alternatively, the expectancy model holds that beliefs about the outcome of a behavior [i.e. drinking alcohol] influence that behavior (Goldman, Del Boca, & Darkes, 1999). This model holds that it is not alcohol's pharmacological effects that are responsible for intoxicated aggression, but rather, expectancies about alcohol. Specifically, that the belief that alcohol increases aggressive behavior results in intoxicated aggression (MacAndrew & Edgerton, 1969). Investigations on expectancies about alcohol and aggression have focused on placebo effects and dispositional expectancies. Reviews of the literature on placebo effects and alcohol-related aggression have concluded that placebo manipulations do not facilitate aggression (see Bushman & Cooper, 1990; Ito et al., 1996). In addition, research on the relation between dispositional expectancies (beliefs that alcohol consumption produces aggression) and subsequent intoxicated aggressive behaviors is inconsistent at best (Rohsenow & Bachorowski, 1984; Chermack & Taylor, 1995).

Given the equivocal evidence for the expectancy model, alternatives to the disinhibition model's direct pharmacological explanation of alcohol-related aggression are receiving more attention. One such alternative is a set of indirect cause models. These models postulate that alcohol pharmacologically impairs psychological and/or physiological mechanisms which in turn result in aggressive behavior (Graham, 1980). Three of the most well-accepted theories of alcohol-related aggression are derivatives of the indirect cause model; they contend that alcohol's pharmacological properties facilitate aggression by a) impairing anxiety/fear responses (Ito et al., 1996; Phil, 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). Researchers have investigated the role of both cognitive impairment (Hoaken, Assaad, & Phil, 1998; Lau, Pihl, & Peterson, 1995) and increased arousal as mechanisms of alcohol-related aggression (Hoaken, Campbell, Stewart, & Pihl, 2003).

Specifically, researchers have used neuropsychological measures of prefrontal functioning to examine the effects of alcohol on aggressive behavior for individuals with low and high cognitive functioning (Lau, et al., 1995). The low and high cognitive functioning groups were given either an alcohol or placebo beverage and completed a laboratory aggression task. Persons that consumed alcohol were more aggressive than those who consumed the placebo beverage. In addition, individuals in the low cognitive functioning group displayed significantly more intoxicated aggression than those in the high cognitive functioning group. However, no interaction was observed between cognitive functioning and alcohol consumption.

This same group of researchers have also investigated the alcohol-aggression relation in a sample of individuals with high executive functioning (Hoaken et al., 1998). Participants received an alcohol or placebo beverage and completed a laboratory aggression task. It was hypothesized that sober individuals would be able to inhibit aggression for monetary reward while intoxicated individuals would not. Results indicated that when participants were not given the option of monetary reward for inhibiting their behavior, alcohol intoxication resulted in higher levels of aggression. However, when participants were asked to refrain from aggressive behavior for monetary

compensation, intoxicated individuals displayed significantly less aggression (Hoaken et al., 1998). This finding suggests that despite intoxication, individuals with high levels of prefrontal functioning still retained a sufficient amount of cognitive functioning to inhibit aggression when they were motivated to do so.

Alternatively, researchers have investigated physiological arousal as a possible mechanism of alcohol-related aggression. Alcohol has been shown to increase physiological arousal and subjective feelings of power and energy (Pihl & Peterson, 1995). Specifically, alcohol consumption can result in increased heart rate (Rush et al., 1989; Conrod et al., 1997). It has also been noted that this effect is more prominent for individuals with a high risk for alcoholism, such as sons of male alcoholics (Assaad et al., 2003; Steward, Finn, & Pihl, 1992). In addition, alcohol's effect on heart rate in this population has been linked to increased sensation-seeking and approach behavior (Finn, Earlywine, & Pihl, 1992; Pihl & Peterson, 1995). Increases in these types of behaviors can result in a greater likelihood of risk-taking, confrontations, or provocation thereby increasing the possibility of intoxicated aggression. In one investigation of this possible mechanism, male and female participants were given either an alcohol or placebo beverage and completed a laboratory aggression task (Hoaken et al., 2003). Intoxicated individuals displayed an increased heart rate after drinking and were more aggressive than individuals who did not consume alcohol. Despite these investigations of arousal and cognitive functioning, anxiety has never been tested as a possible mechanism of the alcohol-aggression relation.

Anxiety

It is important to note that conceptually, anxiety can be differentiated from 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). It is also possible to distinguish these two constructs in terms of the neurological substrates that subserve each construct. There is considerable agreement in the literature that the hippocampus and the limbic system are involved in both anxiety and fear (Gray, 1987; LeDoux, 1992). However, fear has been

hypothesized to be a basic emotion based in the amygdala (Mathews & Mackintosh, 1999). Anxiety can be conceptualized as a more complex emotional reaction activated by both the limbic system and the prefrontal cortex (Mathews & Mackintosh, 1999). Specifically, Mathews and Mackintosh (1999) argue that anxiety involves the symbolic representation of a prospective danger. In addition, anxiety and fear have been theorized to be distinct psychological constructs. Gray and McNaughton (2000) argue that, in terms of Gray's revised theory of behavioral inhibition and activation, fear and anxiety differentially activate specific psychological systems. They maintain that fear is the result of activation of the Flight-Fight-Freezing System (FFFS). The FFFS is activated when an individual encounters any aversive stimuli, either unconditioned, innate, or conditioned. When the FFFS is activated by aversive stimuli, the individual may flee, attack, or freeze depending on the situation. Anxiety is produced by the activation of both the Behavioral Inhibition System (BIS) and the Behavioral Activation System (BAS). The BAS is activated when an individual is motivated to attain a goal. The BIS is activated when an individual is motivated to avoid a stimulus. In Gray's revised theory, the BIS is only activated when there is simultaneous activation of the BAS. As such, Gray and McNaughton (2000) postulate that anxiety occurs only when the individual is motivated to attain a goal that requires approaching some danger.

Based on this conceptualization of anxiety and fear, it is important to examine the proposed mechanism of reduced fear responses put forth by Pihl et al. (1993). They argue that alcohol's anxiolytic effects can facilitate the expression of aggression by suppressing the inhibition normally exerted on behavior. The inhibitory control typically exerted by fear in this model is theorized to occur when there is motivation to aggress, but that behavior could result in punishment or retaliation. Gray and McNaughton's (2000) revised theory states that anxiety is produced when there is motivation to attain a goal that requires approaching some danger. Phil et al.'s (1993) description of motivated aggression and the potential for retaliation undoubtedly meets Gray and McNaughton's (2000) criteria for anxiety. In addition, fear of retaliation or harm implies awareness of possible future consequences of an interpersonal interaction. As such, this anticipation of possible retribution involves activation of the prefrontal cortex, in accord with Mathews and Mackintosh's (1999) description of anxiety. More specifically, Phil et al. (1993)

state that "Fear produced in response to threat (to cues of imminent punishment) play an adaptive role – that of helping individuals to avoid harm" (p.131). However, they continue by stating that, "If the anxiety (behavioral inhibition) such cues evoke is pharmacologically reduced, then the chance for harm inevitably increases" (Pihl et al., 1993, p. 131). The specific reference to cues of imminent punishment evoking *anxiety* and *fear* suggests that despite the delineation of the two constructs, in some circumstances the authors use the terms interchangeably. However, Pihl et al. (1993) clearly state that it is alcohol's pharmacological anxiolytic effects that suppress the inhibition of behavior. Thus, Pihl et al. (1993) argue that alcohol consumption reduces anxiety, thereby decreasing anxiety's inhibitory effects on behavior. As such, it is reduced anxiety and not fear that is the theoretical mechanism of alcohol-related aggression. The purpose of this thesis study is to focus on anxiety as a potential mechanism of the alcohol-aggression relation.

Alcohol and Anxiety

Acute alcohol consumption has been shown to have profound effects on anxiety. Researchers have theorized that γ-aminobutyric acid (GABA) receptors in the hippocampus underlie alcohol's anxiolytic effect (Gray, 1982, 1987). Research has implicated alcohol's action on the neurotransmitters serotonin, dopamine, norepinephrin, prostaglandin, GABA, and GABAergic neurosteroids in this anxiolytic effect (LaBuda, & Fuchs, 2002; Liljequist & Engle, 1984; Lovinger, 1997; Nestoros, 1980). However, a recent review of the literature suggests that it is alcohol's influence on GABA and GABAergic neurosteroids that are the major contributors to its anxiolytic and inhibitory effects (see Criswell & Breese, 2005). Alcohol is known to act as a GABA agonist, directly acting at the $GABA_A$ receptor thereby increasing its inhibitory effects in the brain, similar to anti-anxiety drugs such as the benzodiazepines (Warneke, 1991). Additionally, alcohol has been shown to increase the plasma precursors of GABAergic neurosteroids, thus increasing the level of GABAergic neurosteroids in the brain (Barbaccia et al., 1999; Morrow et al., 2001). These GABAergic neurosteroids enhance the responsiveness of the GABA released by alcohol binding at the $GABA_A$ receptors (Sanna et al., 2004).

There is also a wealth of self-report data indicating that acute alcohol consumption produces reductions in subjective anxiety following a laboratory induced stressor (Eddy, 1979; Levenson, Oyama, & Meek, 1987; Levenson, Sher, Grossman, Newman, & Newlin, 1980; Lindman, 1983; Sayette, Breslin, Wilson, & Rosenblum, 1994). In the above studies, intoxicated individuals were exposed to a stressor such as a tone blast, shock, or a self-disclosing speech. Subjective ratings of anxiety were obtained using questionnaires or continuous measures such as an anxiety dial throughout the experiment. These studies concluded that alcohol intoxication produced an anxiolytic effect in healthy social drinkers. In addition, acute anxiety is highly correlated with measures of physiological arousal such as heart rate, blood pressure, and galvanic skin conductance (see Hoehn-Saric & McLeod, 2000). Acute alcohol consumption has been shown to impact these physiological indicators (Carpenter, 1957; Sayette, Shiffman, Niaura, Martin, & Shadel, 2000) and galvanic skin response. Specifically, alcohol significantly attenuates heart-rate (Lehrer & Taylor, 1974; Levenson et at., 1980; Panknin, Dickensheets, Nixon, & Lovallo, 2002; Sayette & Wilson, 1991) and electrical skin conductance (Greenberg & Carpenter, 1957; McGonnel & Beach, 1968; Stewart & Pihl, 1994; Stritzke, Patrick, & Lang, 1995) in both alcoholics and normal volunteers subsequent to experiencing an acute anxiety provoking stressor such as an electric shock, an abrupt noise, or delivering a self-disclosing speech.

Despite the wealth of research on alcohol's anxiolytic effects, there are inconsistencies in the literature. Specifically, researchers have reported that alcohol increases, decreases, or has no effect on anxiety (Cappell and Greeley, 1987; Sher, 1987; Steele & Josephs, 1988). Therefore, cognitive models have been put forth to explain this discrepancy in the data. In general, cognitive models hypothesize that alcohol's effect on cognitive processes alter anxiety (Wilson, 1988). One major cognitive model is Hull's (1981) self-awareness model. This model states that alcohol consumption interferes with cognitive processes such that self-relevant information is not properly encoded. As a result, self-awareness is decreased. This model predicts that alcohol will reduce negative self-relevant information, particularly in highly self-aware individuals, thereby decreasing anxiety. Alcohol has been shown to decrease self-awareness (Hull, Levenson, Young, & Sher, 1983). However, researchers have also reported that alcohol's anxiolytic

effects are unrelated to high dispositional self-awareness (Niaura, Wilson, & Westrick, 1988; Sher & Walitzer, 1986; Wilson, 1988). In addition, data also suggest that alcohol decreases negative self-evaluation responses to negative self-relevant information independent of its effects on self-awareness (Yankofsky, Wilson, Adler, Hay, & Vrana, 1986).

A more recent attempt to clarify how alcohol might increase or decrease anxiety is Steele and Josephs' (1990) attention-allocation model. The attention-allocation model postulates that alcohol's effect on information processing capacity mediates alcohol's effects on anxiety. Specifically, alcohol intoxication interferes with an individual's ability to effectively allocate attention to multiple aspects of a situation. Consequently, alcohol consumption results in a narrowing of attention such that only the most immediate and salient cues can be attended to. Therefore, Steele and Josephs (1990) hypothesized that alcohol has anxiolytic effects when there is some concurrent distracting activity. Alcohol consumption prior to an anxiety-inducing event presented in conjunction with a neutral or pleasant distraction resulted in decreased anxiety (Josephs & Steele, 1990). In addition, Steele and Josephs (1988) have also reported that when intoxicated individuals are presented with an anxiety-inducing event and no distraction, alcohol does not display its typical anxiolytic effects. In some cases, alcohol actually increased anxiety. Without a distraction, the anxiety-inducing event was the most salient cue in the environment. As such, alcohol's narrowing effect on attention resulted in an increased awareness of the anxiety related cues.

Anxiety and Aggression

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 et al., 1998). Similarly, research has shown that psychiatric disorders typified by decreased fear, such as psychopathy, conduct disorder, and antisocial personality are associated with increased aggressive behavior (reviewed in Lorber, 2004). In addition, laboratory studies demonstrate that drugs with anxiolytic effects such as diazepam (Cherek et al., 1990; Wilkinson, 1985), triazolam (Cherek et al., 1991), as well as other benzodiazepines (Berman and Taylor, 1995) all tend to increase aggression. Finally,

animal models have revealed that normally aggressive rats exhibit deficiencies in anxiety and fear responses (Ferreira et al., 1989; Hard & Hansen, 1985). According to Gray (1982, 1987), decreased anxiety results in a lessening of the inhibition the septal/hippocampal system normally exerts on behavior when an individual is exposed to threat or novelty. As such, there is an increased likelihood of an aggressive response to the threatening or novel cue.

Purpose and Hypotheses

Given alcohol's well established effect on attenuating anxiety, coupled with the association between lowered anxiety and aggression, the purpose of this investigation was to examine the hypothesis that anxiety would serve as an underlying mechanism of the alcohol-aggression relation. Researchers have noted the possibility that alcohol increases the likelihood of aggressive behavior through reductions in anxiety (Ito et al., 1996; Phil, Peterson, & Lau, 1993). However, as noted above, despite investigation of other theorized mechanisms (Hoaken, Assaad, & Phil, 1998; Hoaken, Campbell, Stewart, & Pihl, 2003; Lau, Pihl, & Peterson, 1995), there are no examinations of anxiety as a potential mechanism of the alcohol-aggression relation. Clearly, further understanding of the mechanisms of alcohol-related aggression is needed. Intoxicated aggression is a complex relation involving multiple mechanisms, interactions, and causal pathways. As such, it is essential to assess hypothesized mechanisms put forth by the theoretical models of this relation. Without a thorough understanding of these mechanisms, research cannot speak to furthering knowledge of etiology or impacting clinical interventions.

It is hypothesized that anxiety reduction is a mechanism by which alcohol facilitates aggression. In fact, it is expected that intoxicated participants exposed to an anxiety inducing manipulation will demonstrate a significant reduction in aggression compared with intoxicated participants in whom anxiety is not induced.

Chapter Two: Methodology

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. Participants were recruited through advertisements placed in newspapers in Lexington, Kentucky. 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 on average approximately $25,000 per year; the remainder were supported by a parent or spouse.

Experimental Design

A 2 (Group: anxiety induction, no anxiety induction) × 2 (Beverage: alcohol, placebo) × 2 (Provocation: high, low) mixed model design, with provocation as the repeated measure was employed in this study. Participants were randomly assigned to one of four groups: 1) Alcohol/Anxiety (n=20), 2) Placebo/Anxiety (n=20), 3) Alcohol/No Anxiety (n=20), and 4) Placebo/No Anxiety (n=20).

Six meta-analytic reviews have been conducted on the effect of alcohol on aggression finding that it has an effect size ranging from d = .49 to d = .61 (see Bushman, 1993; Bushman & Cooper, 1990; Ito et al., 1996). For the purpose of conducting a power analysis, a median value was used for the effect size (d = .55). In reviewing the literature it became apparent that there is no meta-analysis for the effects of anxiety on aggression and very few studies in general. However, our reading of this literature leads us to estimate a "medium" effect size. Using an alpha level of .05, Cohen's formulas (Cohen, Cohen, West, & Aiken, 2003) indicated the need for at least 20 subjects per cell to achieve a power level of .80.

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, Surprise). The Fear, Hostility, Attentiveness, and Fatigue subscales were administered in a random order. Participants rated the extent to which they experienced each adjective on a five 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 intent of the study and keep the measure at a length that could be completed quickly. For the purpose of this investigation, the Fear subscale was utilized in order to measure anxiety. Despite the name of this subscale, the items (6 items: afraid, frightened, scared, nervous, jittery, shaky) are more closely related to the conceptualization of anxiety than fear. Specifically, Watson and Clark (1994) note that the fear scale assesses the same basic affect as the Profile of Mood States (POMS) Tension-Anxiety scale. They report 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 (Watson & Clark, 1994).

The PANAS-X Fear subscale was administered to measure state anxiety. Participants were asked to complete the state anxiety scale at different points throughout the study in order to assess alcohol's impact on anxiety as well as the effectiveness of the anxiety induction manipulation. Specifically, the scale was administered following: 1) obtaining informed consent, 2) the anxiety manipulation, 3) beverage consumption, and 4) the aggression task.

Aggression Task

A modified version of the TAP (Taylor Aggression Paradigm) was used to measure aggressive behavior (Taylor, 1967). This task placed participants in a situation where electric shocks were administered to, and received from, a fictitious opponent during a supposed 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, 1995; 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, 1995).

Measures of Aggression

First Trial Shock Intensity. This measure comprises the shock intensity selection ("1" through "10") participants administered on the first trial of the task. It represents a measure of unprovoked aggression inasmuch as participants won the first trial and thus had 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. As such, two separate dependent variables were calculated: 1) mean shock intensity under low provocation and 2) mean shock intensity under high provocation. These variables represent measures of aggression in response to provocation.

Procedure

Respondents were initially screened by telephone using a custom in-house interview (Giancola, unpublished manuscript). 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 scoring an "8" or more on the SMAST was excluded from participation. Following the telephone screening interview, individuals eligible for participation were scheduled for an appointment to come to the laboratory. They were told to refrain from drinking alcohol 24 hours prior to testing, to refrain from using recreational drugs from the time of the telephone interview, and to refrain from eating for four hours prior to testing. Participants were told that they would receive $50 at the completion of the study as compensation.

Upon entering the laboratory, the procedures of the study were explained to participants and they were asked to sign an informed consent form. The experimenter then assessed their breath alcohol concentration (BAC) to ensure sobriety. BACs were measured using the Alco-Sensor IV breath analyzer (Intoximeters Inc, St-Louis, MO). Anyone with a positive BAC reading was excluded. If the BAC test was negative, participants underwent a urine drug test. Anyone with a positive urine drug test (i.e., cocaine, marijuana, morphine, amphetamines, benzodiazepines, and barbiturates) result was also excluded. Demographic data were then collected; followed by the administration of the self-report anxiety scale.

Participants were then escorted into a testing room. Prior to receiving their beverages, they completed a baseline test of state anxiety using the state anxiety scale.

Anxiety Manipulation

The anxiety 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 count down. 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 that received this manipulation did not have to deliver a speech. The manipulation was simply used to induce anxiety. This manipulation is a well established and validated anxiety induction used in the alcohol literature (see Levenson et al., 1980; Sayette, et al., 1992). Following the administration of the anxiety induction, the state anxiety scale was given 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 minute wait period.

Beverage Administration

Participants who received alcohol were administered a dose of 1g/kg of 100% alcohol USP mixed at a 1:5 ratio with Tropicana® orange juice. Beverages were poured into the requisite number of glasses in equal quantities. 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). Three c.c.s of alcohol were added to each placebo beverage and 3 c.c.s were layered onto the juice in each glass. 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.

In addition to the two beverage groups that were used in this study (i.e., alcohol and placebo), a sober control group, in which participants receive a nonalcoholic beverage and are told that they consumed no alcohol, could also have been used. 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 tend to differ significantly (reviewed in Chermack & Giancola, 1997; Bushman & Cooper, 1990). In recognition of previous research demonstrating that sober and placebo groups do not differ significantly in aggression, we only employed an alcohol and a placebo group.

Twenty minutes were then allotted for beverage consumption. Following beverage consumption, participants waited 10 minutes so that the alcohol could be absorbed into the bloodstream. The participants then completed the state anxiety scale (approx 5 minutes) to assess the effects of their beverage on state anxiety. Those in the placebo group did not have to wait before completing the state anxiety scale.

Aggression Task Explanation

Following completion of the state anxiety scale, 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 and at that 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 not 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 shock button "#1" delivers a very low intensity shock that is best characterized as "very mild" and "definitely not painful." This procedure has been widely used and accepted to reflect a non-aggressive 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 one of 10 "shock lights" [(ranging from 1 (low) to 10 (high)] on the computer screen. Both of these indicators displayed readings that correspond with the selected shock level. These images were used to reinforce participants' belief that they were actually administering shocks. Upon 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 one 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 Testing

Participants' pain thresholds were then assessed to determine the intensity parameters for the shocks they would receive. This was accomplished via the administration of short duration shocks (one second) 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 respectively set at 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, and 100% of the highest tolerated shock intensity. The threshold 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 et al., 1984). Following the pain threshold testing procedure, BACs were monitored as they increased. Participants began the aggression task after reaching an ascending BAC of at least 0.09%. Those participants that did not receive alcohol began the aggression task following the pain threshold testing.

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 loses). There were two "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 provocation conditions were not counterbalanced to accurately reflect a more natural simulation of provocation increasing over time. Thus, there were a total of 34 trials. All shocks delivered to the participants were of a one second 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 five second 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 controls the task were located in an adjacent control 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.

Post Aggression 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. Participants were then compensated. All individuals who received alcohol were required to remain in the laboratory until their BAC dropped to 0.04%.

Deception Manipulation

In order to disguise the task as a measure of aggression, participants were given 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. In order 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, participants were informed that their opponent would undergo the threshold 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 thresholds 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.

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, and how well they believed they performed on the task, etc. Typical responses included that the 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. 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 a 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 to 11). The placebo group reported average ratings of 1.65 (*SD* = 1.29) and 2.08 (*SD* = 1.64), respectively, [alcohol vs. placebo pre-TAP ratings: $t(78) = -6.75$, $p < .001$, $d = 1.53$; post-TAP ratings: $t(78) = -6.15$, $p < .001$, $d = 1.40$]. In response to a question about whether the alcohol they drank caused any impairment, persons 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), $t(78) = -5.75$, $p < .001$, $d = 1.30$, (scale range: 0 to 10).

Anxiety Manipulation Checks. Results indicate that there were no significant differences between the Anxiety and No Anxiety groups at the baseline measurement. Following the anxiety induction, results indicate a significant decrease from baseline for the No Anxiety group [baseline mean = 1.19, SD = .23; post anxiety induction mean = 1.11, SD = .23; $t(38) = 2.55$, $p = .01$, $d = .36$]. In contrast, following the anxiety induction, the Anxiety group had a significant increase from baseline [baseline mean = 1.15, SD = .23; post anxiety induction mean = 1.24, SD = .33; $t(38) = -2.57$, $p = .01$, $d = .32$]. In addition, the Anxiety group had significantly higher anxiety ratings than their No Anxiety group counterparts after the anxiety induction [Anxiety group mean = 1.24, SD = .33; No Anxiety group mean = 1.11, SD = .21; $t(77) = -2.00$, $p = .049$, $d = .48$] (see Figure 3.1). Immediately prior to the aggression task, self reported anxiety increased from the post anxiety induction measurement for those in the Placebo/No Anxiety group

only [post manipulation mean = 1.09, SD = .18; post beverage mean = 1.11, SD = .21]. However, this increase was not significant. Following the aggression task, the Alcohol/Anxiety group had significantly higher anxiety ratings than the Alcohol/No Anxiety group was no further increase [Alcohol/Anxiety mean = 1.08, SD = .14; Alcohol/No Anxiety group mean = 1.00, SD = 0.00; $t(38) = -2.44$, $p = .025$, $d = .83$].

Aggression Data

First Trial Shock Intensity. Data for first trial shock selection were analyzed using a 2 (Beverage: alcohol, placebo) X 2 (Group: anxiety, no anxiety) Analysis of Variance (ANOVA) which revealed a significant Beverage X Group interaction, $F(1,76) = 5.29$, $p = .024$ (see Figure 3.2). Unprovoked aggression was significantly suppressed for the Alcohol/Anxiety group compared with the Alcohol/No Anxiety group [Alcohol/Anxiety mean = 3.45, SD = 2.31; Alcohol/No Anxiety mean = 5.05, SD = 3.12; $t(38) = 1.85$, $p = .036$, $d = .60$]. However, there was no significant difference in first trial shock intensity between the placebo groups.

Mean Shock Intensity. Data for shock intensity were analyzed using a 2 (Beverage: alcohol, placebo) X 2 (Group: anxiety, no anxiety) X 2 (Provocation: low, high) mixed model Analysis of Variance (ANOVA) with Provocation as the repeated-measure which revealed a significant Beverage X Group interaction, $F(1,76) = 4.58$, $p = .036$ (see Figure 3.3) . Aggression was significantly suppressed for the Alcohol/Anxiety group compared with the Alcohol/No Anxiety group, [Alcohol/Anxiety mean = 4.79, SD = 1.55; Alcohol/No Anxiety mean = 6.05, SD = 2.15; $t(38) = 2.13$, $p = .04$, $d = .69$]. Furthermore, there was no significant difference in aggression between the two placebo groups. The main effect of provocation was also significant $F(1,76) = 170.13$, $p < .001$.

FIGURE 3.1

Anxiety Induction Manipulation Check

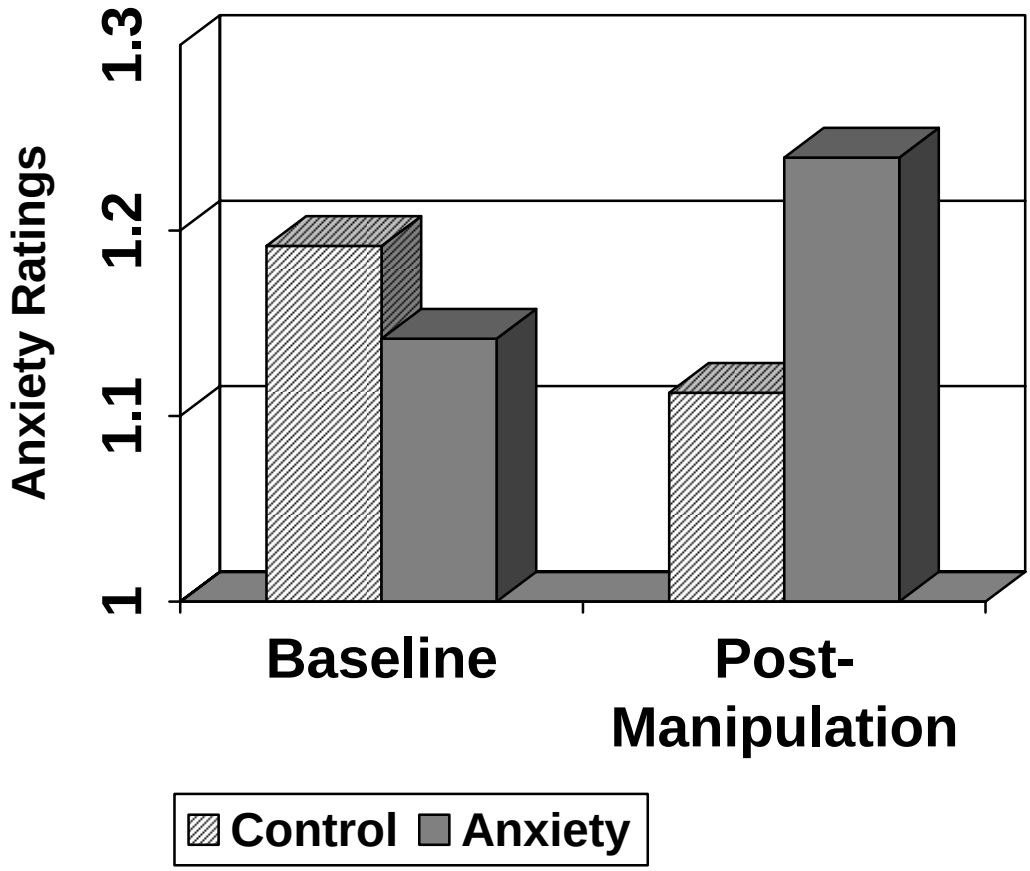


FIGURE 3.2

Unprovoked Aggression

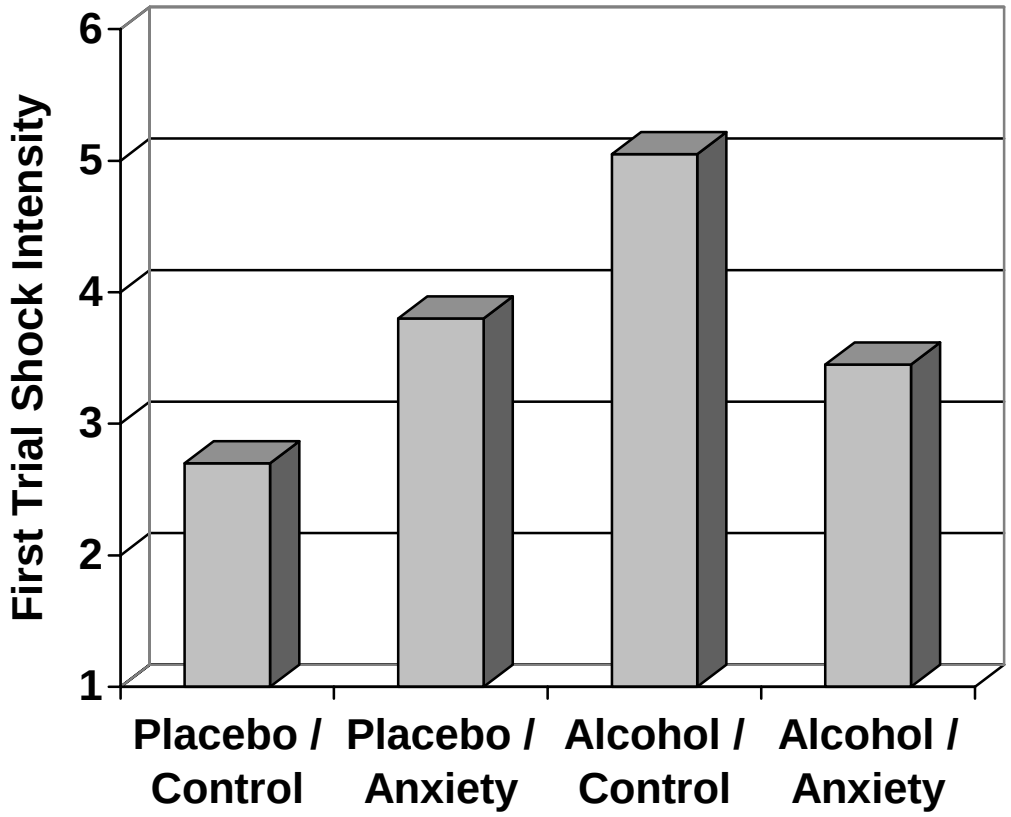
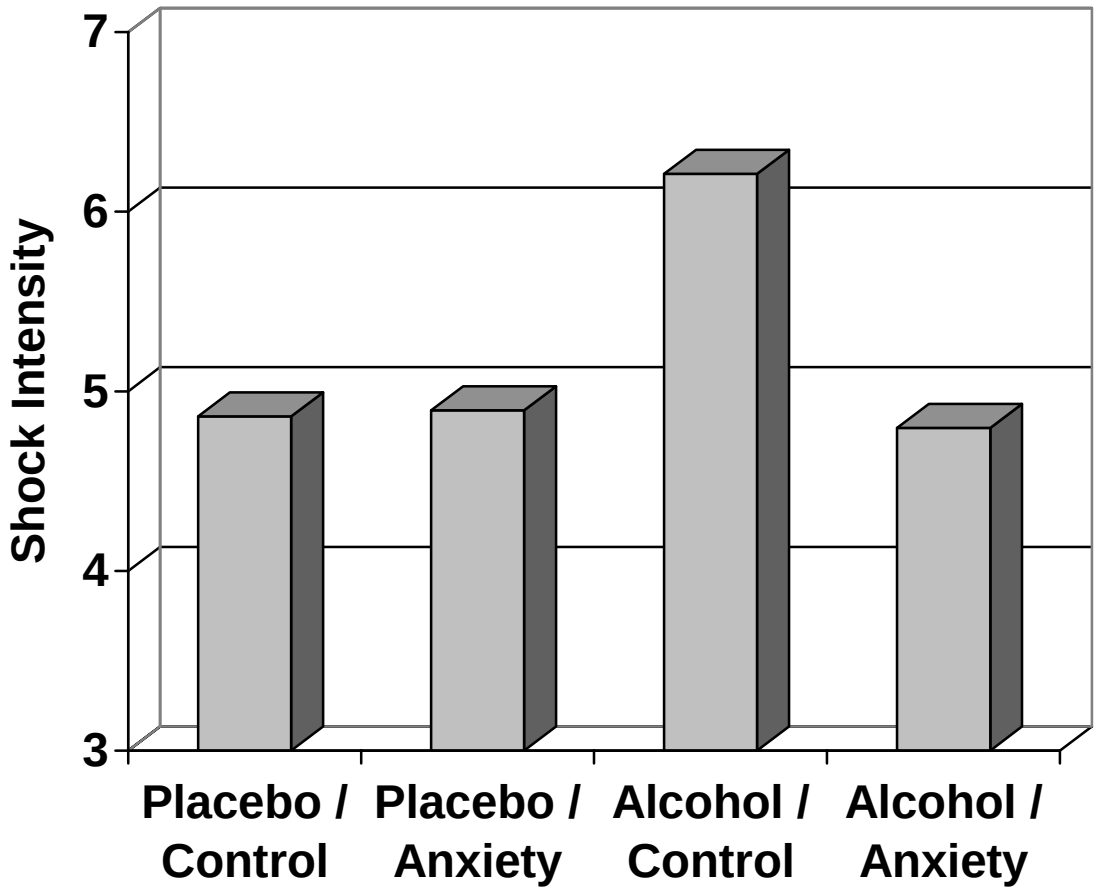


FIGURE 3.3

Aggression



Chapter Four: Discussion

Results indicate that the anxiety induction was successful in attenuating aggression in intoxicated individuals. In addition, those who received alcohol and the anxiety manipulation did not significantly differ from those who did not receive alcohol. This pattern of findings was consistent across both measures of aggression. Taken together, these data support the hypothesis that the inhibition of anxiety may act as a potential mechanism for alcohol-related aggression.

One potential criticism of these findings is that this investigation induced fear rather than anxiety. As noted in the introduction, fear and anxiety are conceptualized as distinct psychological constructs. The anxiety induction utilized in this study was designed to be emotionally distressing without including the possibility of physical danger. In addition, the participants were motivated to complete the anxiety-inducing speech for monetary compensation. Based on Gray and McNaughton's (2000) description of anxiety as elicited when there is an approach avoidance conflict (simultaneous BIS and BAS activation), it is argued that the anxiety induction indeed produced anxiety. In contrast, in order to induce fear the aversive cue must be specific stimuli of imminent physical danger or potential injury (Barlow, 1988). Gray and McNaughton (2000) specify that fear is produced by activation of the FFFS alone. Therefore, in order to induce fear, the manipulation must be designed to present a specific aversive stimulus that signals imminent physical danger. For example, a fear induction within the context of this study would consist of informing the participants that after drinking, they would have the opportunity to compete against an opponent on a reaction-time task. The participants would be told that this is not required, but that their opponent had requested a way to pass the time while detoxifying. In this way, the participant is not motivated to complete the task for monetary compensation, thereby minimizing the likelihood of BAS activation. During the reaction-time task the participant would be able to observe a confederate on a video monitor. After several practice runs, the confederate would complain about the task and indicate that the computer was not functioning properly. After losing a reaction-time trial, the confederate would become visibly angry when shocked by the participant. The confederate would then tell the participant; "Now you've done it! I'm only going to give you the highest shocks for as long as I can!" The threat of

imminent pain and possible retaliation after the experiment is over should be sufficient to induce fear. When comparing this manipulation to the anxiety induction utilized in this study, it is arguable that each would elicit a distinctly different affective state.

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 that alcohol's anxiolytic effects have a pure biological explanation. As previously noted, alcohol has well established pharmacological effects, acting as a GABA agonist, directly affecting the $GABA_A$ receptor (Criswell & Breese, 2005; Warneke, 1991). In addition, it is alcohol's pharmacological effects on GABA in the septal/hippocampal system that produces anxiolysis (Gray, 1982; 1987). Moreover, Gray (1982; 1987) has argued that the septal/hippocampal system is responsible for inhibiting behavior following exposure to a 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 as the neurological pathways that would normally work to constrain aggressive responses are disengaged.

Similarly, Pihl et al. (1993) predict that alcohol's pharmacological effects on anxiety reduce the inhibition normally produced by exposure to a threatening cue. Instead, the probability of aggression is increased as the threat is effectively removed. Specifically, they hypothesize that alcohol eliminates the threat normally produced in interpersonal conflicts; thereby increasing the likelihood of aggression. One possible explanation for the data presented in this study is that the anxiety manipulation resulted in increased awareness of the interpersonal conflict inherent in the aggression task. If the anxiety induction resulted in increased attention to this conflict, it could result in greater inhibition and a suppressive effect on aggression. However, the data from this investigation cannot speak to the participant's perception of threat. Alternative theoretical explanations could also account for the observed effect of the anxiety induction on intoxicated aggression.

One such alternative explanation is provided by the Appraisal-Disruption Model (Sayette, 1993). 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 impairment of cognitive processes produced by alcohol (Sayette, 1993). Simply put, it states that acute alcohol intoxication impairs an individual's ability to appraise new information. Sayette (1993) argues that alcohol's impact on anxiety depends on when stressful information is presented. Alcohol is predicted to decrease anxiety when the stressful/anxiety-inducing information is presented after intoxication. Intoxication is theorized to impair appraisal of this information and thus the individual does not become anxious. If the stressful information is presented prior to intoxication, the Appraisal-Disruption Model predicts that alcohol will not reduce anxiety. In this situation, the stressful information is processed before intoxication without the interference of alcohol. Accordingly, alcohol will fail 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 increasing the likelihood of aggression.

To date, only one study has directly tested the Appraisal-Disruption Model (Sayette et al., 2001). That study found that when a stressor was presented following intoxication, alcohol attenuated anxiety on some, but not all measures. In addition, they noted that when the stressor was presented prior to intoxication, alcohol did not have a consistent effect on anxiety (Sayette et al., 2001). The present study provides indirect support for the Appraisal-Disruption Model in that the Alcohol/Anxiety group was exposed to the anxiety induction before beverage consumption and alcohol did not reduce self-reports of anxiety in those individuals. Nevertheless, this study did not manipulate beverage consumption and anxiety induction order. As such, the data cannot speak to alcohol's effects on appraisal or subsequent effects on anxiety.

An alternative explanation than those provided by the above theoretical models is provided by Steele and Josephs' (1988) Attention-Allocation Model. The Attention-Allocation Model predicts that alcohol impairs information processing capacity (Steele &

Josephs, 1990). As previously noted, this model posits that alcohol intoxication interferes with an individual's ability to effectively allocate attention to multiple parts of a situation. 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 distracting event. The distracting event occupies all of the intoxicated individual's attentional capacity. Steele and Josephs (1990) also postulate that without a distraction from anxiety-inducing cues, alcohol will not have an anxiolytic effect. In this model, anxiety could function as a mechanism of alcohol-related aggression through alcohol's effects on attention. Specifically, in hostile situations, provocative cues are more predominant than anxiety-inducing cues, resulting in attention focused only on provocation. Instead of anxiety inhibiting the aggressive behavior, the individual is focusing only on the provocative cues thereby increasing the likelihood of aggression. However, the Attention-Allocation Model hypothesizes that alcohol will affect behavior depending on where an individual's attention is focused. Specifically, if attention is focused on inhibiting cues, intoxication will result in inhibited behavior.

MacDonald and associates (2000) conducted a series of studies to test this hypothesis. These studies examined the effect of intoxication on intention to engage in risky sexual behavior. Sober and intoxicated participants were presented with a vignette describing an interaction between two members of the opposite sex who want to engage in sexual intercourse but do not have access to a condom. Participants were the asked to put themselves in this situation and rate the likelihood of engaging in sexual intercourse. They were either asked if they would engage in sexual intercourse (impelling cue) or if they would engage in sexual intercourse *without a condom* (inhibiting cue). Intoxicated participants presented with the inhibiting cue were equally as cautious as their sober counterparts. More importantly, they also conducted an experiment to test a possible intervention to promote less risky decisions in intoxicated individuals. In this experiment individuals were stamped with one of three stamps as they entered a bar. The stamps contained either: a neutral cue (a smiley face), a moderate inhibiting cue ("SAFE SEX"), or a strong inhibiting cue ("AIDS KILLS"). Participants' BACs were measured and then they were given a similar vignette about having unprotected sexual intercourse. Intoxicated participants (BAC > .08) that received the strong inhibiting cue were more

likely to report the intention of safe sex choices than their sober (BAC < .08) counterparts (MacDonald, Fong, Zanna, & Martineau, 2000).

It can be argued that the results of the current investigation also support the Attention-Allocation model. It is possible that the anxiety induction functioned as a strong inhibitory cue, resulting in the suppression of aggression. In contrast with most naturally occurring anxiety and related inhibitory cues, the anxiety induction was designed to be salient. However, it is also possible that both the anxiety induction and the aggression task were salient cues. Participants in the Alcohol/No Anxiety group displayed the highest level of aggression due to the fact that their attention was focused solely on the aggression task. Likewise those in the Alcohol/Anxiety group displayed levels of aggression equal to the placebo groups because their attention was focused on the anxiety induction and the imminent "speech" throughout the aggression task. In this way, the results of this investigation provide support for the hypothesis that alcohol narrows attention to only the most salient cues. However, those in the Alcohol/Anxiety group were exposed to salient cues for both aggression and anxiety. It could also be argued that, throughout the aggression task, the provocative cues should have been more salient than the anxiety cues. If the provocative cues were the most salient, the Attention-Allocation model would predict that those in the Alcohol/Anxiety group would be equally aggressive as those in the Alcohol/No Anxiety group. However, since no measure of attention or distraction was included in this study, it is impossible to determine the degree to which the individuals in the Alcohol/Anxiety group focused their attention on the anxiety induction versus the aggression task.

Despite the fact that this study cannot speak to differentiating between the Appraisal- Disruption and Attention-Allocation models, it does significantly contribute to the existing literature on possible mechanisms of alcohol-related aggression. As previously noted, decreased anxiety, decreased cognitive functioning, and increased arousal have all been implicated as possible mechanisms of alcohol-related aggression (Ito et al., 1996; Giancola, 2000; Giancola & Zeichner, 1997; Graham, Wells, & West, 1997; Phil, Peterson, & Lau, 1993; Steele & Josephs, 1990). However, these theorized mechanisms need not be mutually exclusive. As this is the first study to examine anxiety in this role, it is important to note that it provides complementary evidence to

investigations pertaining to the other two theoretical mechanisms. Specifically, both cognitive functioning and arousal are closely related to anxiety and implicated in the theoretical models presented above. While Phil's model maintains that alcohol's direct pharmacological effect on anxiety is a mechanism of intoxicated-aggression, both the Appraisal-Disruption and Attention-Allocation Model posit that alcohol has indirect effects on anxiety. Specifically, that alcohol affects some aspect of cognitive functioning that in turn leads to decreased anxiety.

A potential limitation of this study is that this study included no measure of distraction. The anxiety manipulation could have simply distracted participants from the aggression task, thereby reduced aggressive responding. Future studies should attempt to control for these possibilities by testing several other anxiety induction manipulations and include measures of distraction to determine the distracting properties of each. This study cannot speak to how reductions in anxiety associated with alcohol consumption act as a mechanism for the increased likelihood of aggression. As such, a direct test of the Attention-Allocation Model and anxiety would be beneficial. Subsequent studies should focus on identifying how anxiety functions as a mechanism of this relation. Specifically, future studies might focus on identifying the differences between the biological and cognitive effects of anxiety on alcohol-related aggression. The association between decreased anxiety and the other theoretical mechanisms of the alcohol-aggression relation provide a theoretical framework for examining this conceptual difference. Specifically, the role of cognitive impairment and increased arousal should be investigated as possible mediators of the relation between anxiety and intoxicated aggression. Ultimately, anxiety should be tested against alternative theoretical mechanisms to further elucidate the etiology of alcohol-related aggression.

This is the first study to investigate the role of anxiety in alcohol-related aggression. As such, it is important to note that the anxiety induction utilized in this study was effective in attenuating aggression in intoxicated individuals. This finding lends support to the hypothesized role of anxiety as a mechanism of the alcohol-aggression relation. Furthermore, the anxiety induction was presented before alcohol consumption and produced increases in self-report anxiety, providing support for the Appraisal-Disruption Model. Overall, this methodology used in combination with other

measures of anxiety (i.e. physiological indicators) has the potential to further clarify the relation between anxiety and intoxicated aggression.

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