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ALCOHOL DOSE AND AGGRESSION: ANOTHER REASON WHY DRINKING MORE IS A BAD IDEA

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ABSTRACT OF THESIS

ALCOHOL DOSE AND AGGRESSION: ANOTHER REASON WHY DRINKING MORE IS A BAD IDEA

A wealth of studies have examined the impact of alcohol on violence; however, only a small number have addressed differences elicited by different doses of alcohol. Such studies are seriously limited by mixed findings, small sample sizes, inconsistent alcohol doses and control conditions, a bias toward studying only male participants, and the predominant use of only one particular measure to assess aggression. The present laboratory investigation was designed to elucidate and advance this literature by improving upon these limitations. Participants were 187 (95 men and 92 women) social drinkers. Following the consumption of one of 6 alcohol doses (i.e., 0.0g/kg; 0.125g/kg; 0.25g/kg; 0.5g/kg; 0.75g/kg; and 1.0g/kg), participants were tested on a laboratory task in which electric shocks were received from, and administered to, a fictitious opponent under the guise of a competitive reaction-time task. Aggression was operationalized as the intensity of shocks administered to their opponent. Analyses revealed that higher alcohol doses clearly elicited greater aggression in both genders consistent with a linear non-threshold dose-response model. Our data help to clarify a body of literature that has been afflicted with numerous limitations and will also help in the selection of alcohol doses for researchers conducting future laboratory-based aggression studies.

KEYWORDS: Alcohol, Aggression, Violence, Dose-response, Binge Drinking

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THESIS

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University of Kentucky

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ALCOHOL DOSE AND AGGRESSION: ANOTHER REASON WHY DRINKING MORE IS A BAD IDEA

THESIS

A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science in the
College of Arts & Sciences at the University of Kentucky

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The first draught serveth for health, the second for pleasure, the third for shame, and the fourth for madness. ~ Anacharsis

Chapter One

Introduction

Alcohol-related aggression, with its heavy societal burden and economic costs, is a well-researched, but complex phenomenon (Volavka & Nolan, 2008). The social and medical impetus to understand how, why, and when alcohol increases aggression is well justified. The availability of alcohol in inner-cities has been associated with increased homicide rates (Branas, Elliott, Richmond, Culhane, & Wiebe, 2009; Jones-Webb, et al., 2008). Cost estimates of the toll of violence directly attributable to alcohol exceed 78 billion dollars a year in the U.S. alone (Miller, 2006). Alcohol has repeatedly been linked to violent crimes, specifically, intimate partner violence (Foran & O'Leary, 2008; Hove, Parkhill, Neighbors, McConchie, & Fossos, 2010), homicide (Pridemore, 2002), sexual assault (Abbey, Zawacki, Buck, Clinton, & McAuslan, 2003) and violent assault (U.S. Department of Justice, 2008).

The fact that alcohol intoxication facilitates violence is no longer in dispute. At present, scientists are working on identifying risk factors and underlying mechanisms of the alcohol-aggression relation. However, other facets important to understanding the nature of intoxicated aggression are still understudied. One of these facets concerns the dose-response relation between the amount of alcohol consumed and the corresponding intensity of aggressive behavior. Available research on this topic falls into two main categories: a) naturalistic/survey and b) laboratory work. The data from both naturalistic/survey and laboratory researchers have revealed interesting insights but they tend to be mixed and incomplete thus providing an imporant impetus for further research

to help clarify the nature of the alcohol-aggression relation.

Naturalistic/Survey Research

A number of studies have investigated the relation between the quantity of alcohol consumed and the presence and intensity of violence. Some of these studies utilized event-based designs in which alcohol drinking patterns were examined in the context of past incidents of violent behavior (e.g., Phillips, Matusko, & Tomasovic, 2007) while others utilized *in vivo* observations of barroom behavior (e.g., Graham, Bernards, Osgood, & Wells, 2006; Graham, Osgood, Wells, & Stockwell, 2006). These reports suggest a close link between acute alcohol intoxication and aggression where larger quantities of alcohol are associated with increased aggression.

Large scale survey studies of college students have found that quantity of alcohol typically consumed on a drinking occasion is significantly associated with both aggression and hostility (Borders, Barnwell, & Earleywine, 2007; Neal & Fromme, 2007; Wells, Mihic, Tremblay, Graham, & Demers, 2008). Such findings are consistent across a wide range of populations. For example, one study found that prison inmates who considered themselves "substantially intoxicated" at the time of their offense were twice as likely to have engaged in lethal violence compared with those who considered themselves only "moderately intoxicated" (Phillips et al., 2007). A study of heavy drinkers found that number of heavy drinking days was significantly associated with an increased probability of aggression (Rolfe et al., 2006). Research on adolescents has also demonstrated positive relations between aggression and heavy drinking (Wells, Graham, Speechley, & Koval, 2006). *In vivo* barroom observations revealed that degree of alcohol intoxication was strongly associated with more severe aggression (Graham, Bernards, et

al., 2006; Graham, Osgood, et al., 2006; Graham & Wells, 2001). Moreover, similar findings have also been observed in general population studies (Wells & Graham, 2003; Wells, Graham, & West, 2000). Specifically, one such investigation determined that while the mere presence of alcohol was not significantly related to aggression, the number of drinks consumed six hours prior to an aggressive incident predicted the occurrence of physical violence (Wells & Graham, 2003). Another such study found that perpetrators of rape were most violent after very heavy drinking (Abbey, Clinton, McAuslan, Zawacki, & Buck, 2002). In summary, naturalistic/survey studies provide mounting support for a relation between the amount of alcohol consumed (i.e., higher doses) and violent behavior in real-world settings amongst a variety of populations (see also Tremblay, Graham, & Wells, 2008; Wells et al., 2006).

The studies reviewed above provide invaluable information to the field. However, the precise measurement of key factors such as blood alcohol content during the event, the dose consumed, time over which beverages were consumed, the intensity of ensuing violent behavior, as well as intervening environmental influences (e.g., provocation and other contextual factors) are often not available in such investigations. Moreover, many of these studies rely on self-reports of acts committed by persons (or victims) who were under the influence of alcohol and many of these reports are recorded months or even years after the commission of such crimes thus increasing the risk for distorted memories/recollections (see Greenfield & Kerr, 2008). As such, all of these hindering factors obfuscate the ability of this body of research to paint a fully accurate picture of the alcohol-aggression dose-response relation and its inability to allow for causal inferences about the effects of varying alcohol doses on aggression. The very

informative data provided by these studies should be complemented by *in vivo* laboratory investigations that can exert the necessary experimental controls that can address the above limitations.

Laboratory Research

Out of the voluminous amount of experimental studies on alcohol and aggression (for reviews see Bushman & Cooper, 1990; Chermack & Giancola, 1997; Exum, 2006; Ito, Miller, & Pollock, 1996), only a relatively small number have systematically tested the effects of different doses of alcohol on aggressive behavior. Of the subset of laboratory-based dose-response studies on alcohol-related aggression, most have assessed aggression with a task developed by Cherek (1981) known as the *Point Subtraction Aggression Paradigm (PSAP)* in which the participant has a set of response options that include 1) pushing a button repeatedly to earn money, 2) pushing a button repeatedly to take money away from a fictitious opponent, or 3) administering a blast of loud noise to a fictitious opponent (Cherek later removed this third option from his studies so that only the first two response options were available to participants). During the time allotted for selecting the response options, the participant would be intermittently "provoked" by having money removed from his/her total earnings by his/her fictitious opponent. Aggression was operationalized as either choosing to take money away from the opponent or choosing to deliver a loud blast of noise to this person.

Cherek and colleagues conducted at least seven separate studies in which the PSAP was utilized to measure the effects of different doses of alcohol on aggression (Cherek, Spiga, & Egli, 1992; Cherek, Steinberg, & Manno, 1985; Cherek, Steinberg, & Vines, 1984; Dougherty, Bjork, Bennett, & Moeller, 1999; Dougherty, Cherek, &

Bennett, 1996; Kelly, Cherek, & Steinberg, 1989; Kelly, Cherek, Steinberg, & Robinson, 1988). These scientists conducted their studies under a behavioral pharmacological model in which relatively few subjects were tested across multiple alcohol conditions using a repeated-measures design. Alcohol doses varied from study to study, with all but one investigation using three different doses, and only three of the studies using a dose of greater than 0.5g/kg (See Table 1 for dose equivalence in standard drinks).

Cherek's initial dose-response study utilized several very low doses of alcohol coupled with a placebo and found that alcohol increased aggression in three out of four participants at alcohol doses of 0.1 g/kg, 0.21g/kg, 0.42g/kg and 0.52g/kg with higher doses generally eliciting more aggression than lower doses (Cherek et al., 1984). A follow-up study found that under high provocation, alcohol doses of both 0.23g/kg and 0.46g/kg increased aggression relative to a placebo while a 0.12g/kg dose did not (Cherek, et al., 1985). Unexpectedly, this latter study found that the 0.23g/kg dose increased aggression above that of the 0.46g/kg dose, though the difference was not significant. Further studies by this group of researchers (Kelly et al., 1988; 1989) showed significant increases in aggression only at the highest doses administered (i.e., 0.5 g/kg and 0.75g/kg). Interestingly, findings from another experiment were unexpected, with doses of 0.25g/kg and 0.375g/kg actually decreasing aggression in three out of four participants (Cherek et al., 1992). Finally, Dougherty and colleagues were the only researchers from this group of scientists to examine the alcohol-aggression relation in women. Their first study used three alcohol doses: 0.25g/kg, 0.5g/kg, and 1.0g/kg with only the highest dose resulting in increased aggression relative to a placebo (Dougherty et al., 1996). Six out of ten participants evinced a quasi-positive increase between dose and

aggression (higher aggression at higher doses), while the remaining four participants counterintuitively exhibited the highest levels of aggression at low doses. In their second study using both men and women, three doses of 0.35g/kg were administered consecutively at one hour intervals (Dougherty et al., 1999). Results revealed a significant increase in aggression after the second drink to an equal extent in both men and women (cumulative dose = 0.7g/kg) compared with a placebo group. After the third drink (cumulative does of 1.05g/kg), aggression levels were maintained in men, but declined in women.

Apart from Cherek and colleagues' work, there has been scant research on dose-response effects in the alcohol-aggression relation. Taylor and Gammon (1975) compared the effects of two alcohol doses (0.34g/kg and 1.0g/kg) on forty young men using the *Taylor Aggression Paradigm (TAP;* Taylor, 1967), in which participants receive and administer electric shocks to/from a fictitious opponent under the guise of a competitive reaction-time task. The TAP measures aggression as the intensity of the shocks that the participant chooses to administer to his or her fictitious opponent. Taylor and Gammon (1975) found that a high alcohol dose (1.0g/kg) increased aggression significantly above a lower dose (0.34g/kg). Following the completion of their initial data collection they recruited an additional 10 participants to constitute a sober control group. Unexpectedly, these additional 10 participants exhibited levels of aggression intermediate to the two alcohol dose groups in spite of being sober. A follow-up study by these researchers used the same two alcohol doses in addition to a placebo group (which was not utilized in their first study). Their findings did not reveal a significant difference between the placebo and low dose (0.34g/kg) groups. Unfortunately, their follow-up

study did not use a sober control group making the nature of this lack of difference unclear. Aggression in the 1.0g/kg dose group was still found to be significantly higher than in the two other groups (Taylor et al., 1976). Finally, Bond and Lader (1986) compared a placebo condition with two alcohol doses (0.25g/kg, 0.75g/kg) in both men and women with a measure of aggression similar to the PSAP in which tone blasts were substituted for subtracting money from a fictitious opponent. These investigators found a linear pattern in which the high dose elicited more aggression than the low dose which, in turn, elicited more aggression than the placebo.

Limitations. Unfortunately, a review of the above studies does not provide a clear picture of the dose-response relation between alcohol and aggression. First, in general, laboratory studies have shown that larger doses tend to increase aggression relative to placebo or sober controls. However, a number of notable exceptions have also demonstrated that higher doses actually elicited *less* aggression compared with low or moderate doses (i.e., Cherek, et al., 1985; Cherek et al., 1992) and that *higher levels* of aggression were observed in a knowingly sober control group compared with a low dose group (Taylor & Gammon, 1975). Second, doses have been relatively inconsistent across studies (see Table 2), with many investigations failing to utilize an alcohol dose greater than 0.5g/kg. Third, there is a lack of consistency in the use of control groups: some studies have utilized either a knowingly sober group or a placebo group, but not both. Fourth, it is not clear whether there are consistent differences in the dose-response relation between men and women. Apart from three studies (Bond & Lader, 1986; Dougherty et al., 1996; 1999) in which a combined total of *only* 46 women took part, no other reports have explicitly assessed the dose-response relation in women. Fifth, as is

evident in Table 2, many dose-response studies employed very small sample sizes. In fact, four out of the ten laboratory studies reviewed above used sample sizes of *six participants or less.* Given this, it is noteworthy that the study in which higher alcohol doses were found to decrease aggression (Cherek et al., 1992) *utilized only four participants*. A sixth reason justifying a new investigation into the dose-response effects of alcohol on aggression is that the majority of past studies have been conducted in one laboratory and by one group of researchers, utilizing one measure of aggression: the PSAP (Cherek and colleagues). Finally, *and most importantly,* our review of the existing research in this area highlights our lack of understanding of the shape of the alcohol-aggression dose-response relation.

Dose-Response Models

While it is beyond the scope of this article to describe all possible dose-response functions that a chemical substance can have on behavior, an understanding of the different plausible alternative models is essential prior to making experimentally derived inferences. In the case of alcohol, which is classified as a central-nervous system depressant, there is an inherent ceiling effect on how much aggression can be elicited; consuming high enough doses of alcohol will eventually lead to stupor, unconsciousness, coma, and even death. This physiological fact (Baldacchino, 2002) precludes a strictly rectilinear dose-response relation. In other words, there is a point at which increased consumption of alcohol will attenuate aggressive behavior. Whether there is a sudden precipitous drop in aggressive behavior due to physical incapacitation or whether this point of decreasing returns occurs gradually is not clear. Because ethical concerns limit the maximum alcohol doses that can be administered to human participants in research

settings, the effects of extremely high doses of alcohol on aggressive behavior will most likely remain uncertain.

Within the range of alcohol doses that are subject to ethical, experimental inquiry, a number of possible dose-response functions are possible, including a strictly rectilinear relation known as a linear non-threshold model (see Calabrese & Baldwin, 2003). Another possibility is what is termed a linear threshold model, which is indicated when a relation only exists after a certain minimum threshold of exposure is met (i.e., if alcohol is unrelated to aggression below a certain dose but is related to aggression above that dose). Another alternative, the disruption model, is a type of threshold model in which the effect is only present after a certain threshold is met but unlike the normal threshold model, the effect does not vary in strength with further increases of the substance. A disruption model of alcohol-related aggression would suggest that at a certain dose, alcohol leads to increases in aggression, but that consuming more alcohol above and beyond that threshold dose would have a minimal impact.

A number of hormetic or curvilinear dose-response relations are also possible. The inverted-U shaped dose-response curve would be indicated if increasing alcohol doses led to diminishing increases in aggression with a point at which higher alcohol doses led to decreasing levels of aggression. As mentioned earlier, extremely high doses of alcohol will always lead to diminished capacity to aggress and thus this model is likely the most descriptive of the entire spectrum of alcohol doses and aggression. Lastly, a J-shaped hormetic dose-response curve would be indicated if low doses of alcohol actually decreased aggression prior to aggression being increased with higher doses (e.g., Taylor, 1975).

All of these models: the linear non-threshold model, the linear threshold model, the disruption model, the inverted U-shaped model, and the J-shaped model, are all possible within the range of alcohol doses that are commonly investigated in both naturalistic and experimental settings. Knowing which type of model is truly representative of alcohol's impact on aggression is critical for both theoretical and practical reasons. Valid theory-development requires an accurate understanding of how variables of interest (i.e., alcohol and aggression) actually relate to each other. Public health interventions, similarly, are most effective when derived from accurate understanding of differential dose effects.

Current Study

The current study was designed to determine which of the above mentioned dose-response models is most representative of alcohol's influence on aggression. This was to be achieved by addressing each of the main limitations mentioned above, specifically, a) a well-established alternative to the PSAP was used to assess aggression (i.e., TAP); b) in an alternate laboratory setting; c) six different alcohol doses were administered including a high dose of 1.0g/kg along with both sober and placebo control conditions; d) both men and women where included; and e) the sample size is significantly larger than previous dose-response studies.

Table 1: Standard Drink Equivalents of Laboratory Alcohol Doses

	Weight					
Dose (g/kg)	100 lbs (45 kg)	130 lbs (59 kg)	160 lbs* (73 kg)	190 lbs** (86 kg)	220 lbs (100 kg)	250 lbs (114 kg)
.125	0.4	0.5	0.7	0.8	0.9	1
0.25	0.8	1.1	1.3	1.5	1.8	2
0.50	1.6	2.1	2.6	3.1	3.6	4.1
0.75	2.4	3.2	3.9	4.6	5.4	6.1
1.00	3.2	4.2	5.2	6.1	7.1	8.1

*Average weight for women ages 20 and over in the U.S. is 163 lbs (74 kg; McDowell et al., 2005)

** Average weight for men ages 20 and over in the U.S. is 190 lbs (86 kg; McDowell et al., 2005)

Though the alcohol content of what is considered a "standard drink" varies considerably (see Turner, 1990), for comparison purposes we use the *National Institute on Alcohol Abuse and Alcoholism's* definition of 14g of pure alcohol (NIAAA, 2000) as the measure of a "standard drink." This is roughly equal to 12 oz (355 ml) of beer, 5 oz (148 ml) of wine, and 1.5 oz (44 ml) of liquor.

Table 2: Summary of Dose-response Studies of Alcohol-related Aggression

	Alcohol Doses (g/kg)[a]	Subjects	Gender	Paradigm
Taylor & Gammon, 1975	0[b], 0.34, 1.0	50	Male	TAP
Taylor et al., 1976	P[c] , 0.34, 1.0	30	Male	TAP
Cherek et al., 1984	P, 0.05, 0.1, 0.21, 0.42, 0.52	4	Male	PSAP
Cherek et al., 1985	P, 0.12, 0.23, 0.46,	11	Male	PSAP
Bond & Lader, 1986	P, .25, .75	45	Male/Female	PSAP
Kelly et al., 1988	P, 0.25, 0.5, 0.75	4	Male	PSAP
Kelly et al., 1989	P, 0.125, 0.25, 0.5	6	Male	PSAP
Cherek et al., 1992	P, 0.125, 0.25, 0.375	4	Male	PSAP
Dougherty et al., 1996	P, 0.25, 0.5, 1.0	10	Female	PSAP
Dougherty et al., 1999	P, 0.35[d]	26	Male/Female	PSAP
Present Study	**0.0, 0.125[e], 0.25, 0.5, 0.75, 1.0**	**187**	**Male/Female**	**TAP**

[a] Doses not originally given in g/kg were converted using conversion ratios consistent with other reviews (see Turner, 1990).
[b] Taylor & Gammon, 1975 added a sober control (n=10) post-hoc.
[c] P = Placebo beverage.
[d] Dougherty et al., 1999 used a cumulative dosing procedure in which 0.35g/kg was administered three times at one hour intervals.
[e] The 0.125g/kg dose is considered an "active placebo" (Ross & Pihl, 1989) in the present investigation.

Chapter Two

Methodology

Participants

Participants were 187 healthy male (n = 95) and female (n = 92) social drinkers between 21 and 34 years of age (M = 22.47; SD = 2.60) recruited from the greater Lexington, KY area through newspaper advertisements and flyers. Ethnic affiliations included 173 who identified themselves as Caucasian (88 men; 85 women), 10 as African American (7 men; 3 women), and 1 woman from each of the following groups: Hispanic, Asian, Indian, and "Other." All participants were screened for problem drinking using the *Short Michigan Alcoholism Screening Test (SMAST;* Selzer et al., 1975). Anyone scoring an "8" or more on the SMAST was excluded. Participants were also screened and excluded for serious mental illnesses (e.g., psychosis, bipolar disorder, current mood disorders etc.) and any medical condition in which receiving alcohol or mild electric shocks would be contraindicated (e.g., liver cirrhosis, stomach cancer, heart condition, pacemaker, epilepsy, etc.). Anyone who tested positive on a urine drug test, breath alcohol concentration (BrAC) test, or a pregnancy test was also excluded.

Pre-Laboratory Procedure

Persons eligible for the study were scheduled for an appointment to come to the laboratory. They were told to not consume any alcohol 24 hours prior to testing, to refrain from using recreational drugs from the time of the telephone interview, and to avoid eating four hours prior to testing. Due to hormonal variations associated with menstruation which may affect aggressive responding (Volavka, 1995), women were not tested between one week before menstruation and the beginning of menstruation.

Participants were informed that they would receive $15 per hour as compensation for their time.

Experimental Design and Beverage Administration

Six different alcohol dose conditions were utilized in a 6 (dose) X 2 (gender) independent-groups design. Participants were randomly assigned into "sober" (16 women, 17 men), "active placebo" [0.125g/kg (16 women, 13 men)], 0.25g/kg (16 women, 16 men; "low dose"), 0.5g/kg (17 women, 16 men; "medium dose"), 0.75g/kg (14 women, 18 men; "medium-high dose"), or 1.0g/kg (13 women, 15 men; "high dose") beverage conditions. In order to achieve similar BrACs among both men and women, women were given doses containing 10% less alcohol to account for differences in body fat and gastric alcohol dehydrogenase activity (Watson et al., 1981).

All dose conditions, with the exception of the sober dose (no alcohol), were administered 95% alcohol mixed in a 1:5 ratio with Tropicana orange juice. Participants rinsed their mouths with water following beverage consumption in order to obtain accurate BrAC readings. Depending on the dose consumed, drinking times ranged between 1 and 20 minutes in order to accommodate the time necessary to consume the different beverage volumes (see Table 3). In order to achieve optimal BrACs during the TAP, post-drinking wait times following alcohol consumption and the beginning of the TAP varied between dose conditions from 5 minutes for the placebo dose to 40 minutes for the 1.0g/kg dose (see Table 3). Participants in the sober condition were administered 473ml (16 oz) of orange juice and did not have a post-drinking wait prior to beginning the TAP. They were informed that they were not consuming alcohol, while participants in all other conditions were informed that they were consuming alcohol, but were not told

how much.

One could argue that all beverages should have been equated for volume and that post-drinking wait times should have been standardized. The rationale for such design choices were reasoned and based on maximizing both internal and external validity. First, if beverages were equated for volume, participants receiving the lower doses, especially the placebo, would have tasted almost no alcohol which could have confounded the aggression data by leading those individuals to believe that they drank only trace amounts of alcohol rather than the lower doses they actually consumed. Equating volumes standardizes stomach contents and visual cues pertaining to how much alcohol one might consume. However, debriefing data from previous work conducted by Giancola and colleagues show that even in the 1.0g/kg group, the amount of liquid administered is not enough to produce any stomach bloating, or discomfort, that might conceivably increase aggression. Moreover, although visual cues might produce some expectations about how much alcohol one is about to receive, once persons actually begin drinking their beverages, gustatory cues are far better indicators of how much alcohol one might be consuming. As with other researchers, Giancola et al. have used the 1.0g/kg dose in many previous studies and never found stomach bloating to be a variable that participants reported as uncomfortable inasmuch as all of our participants are accustomed to regularly drinking a 1.0g/kg dose that made them quite familiar with the stomach contents of this dose. Given these reasons, an argument can be made for not equating beverage volumes. Second, if post-drinking wait times were standardized, the 40 minute wait used for the 1.0g/kg condition would need to be used for all beverage groups. This would completely invalidate the placebo manipulation in that such manipulations are

only effective shortly after beverage consumption (Bradlyn and Young, 1983; Martin and Sayette, 1993) in addition to invalidating some of the lower doses by virtue of assessing aggression on different points of the descending limb of the BrAC curve rather than around the peak. Importantly, aggression has been demonstrated to reach maximal levels around the peak of the BrAC curve (Giancola and Zeichner, 1997). Thus, using a 40 minute post-drinking wait period for all dose groups would have allowed us to measure aggression on the ascending limb or the peak of the BrAC curve only for the 1.0g/kg group. All other groups would have been assessed at different points on the descending limb of the BrAC curve which would have produced a serious confound. In defense of this design, one could argue that the longer waiting times for the higher dose groups might have produced frustration thus leading to increased aggression. However, in response, it is very important to highlight previous work conducted which has demonstrated that even markedly large variations (20 minutes to 3 hours) in post-drinking wait times have been shown to not affect aggressive responding on the TAP (Giancola and Zeichner, 1997). For these reasons, a strong argument can be made that standardizing post-drinking wait times would lead to a less valid experimental design.

Aggression Task

A modified version of the *Taylor Aggression Paradigm (TAP;* Taylor, 1967) was used to measure aggression. This task places participants in a situation where electric shocks are received from, and administered to, a fictitious opponent during a supposed competitive reaction-time task. Physical aggression was operationalized as the shock intensities and durations selected by the participants. They were seated at a table in a small room. On the table facing the participant was a computer screen and a 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. The TAP has been repeatedly shown to be a safe and valid measure of aggressive behavior for men and women (Anderson and Bushman, 1997; Giancola and Chermack, 1998).

Aggression will be operationalized in two different ways. The first of these measures, *Unprovoked Aggression,* will be comprised of the average shock intensity selection ("1" through "10") and duration (in milliseconds) administered on the first trial of the 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. The score will be calculated by transforming the first trial shock intensity and duration variables into *z*-scores and then summing them. The rationale for doing this is to increase the reliability of both indices inasmuch as a meta-analytic investigation demonstrated that shock intensity and duration are significantly related to one another and are considered to be part of a more general construct of aggression (Carlson et al., 1989). For this reason, more recent studies using the TAP, and its modified versions, have adopted, and successfully used, similar combinatory techniques involving shock intensity and duration (Bartholow et al., 2005; Carnagey and Anderson, 2005; Giancola et al., 2009; Parrott and Zeichner, 2001; Ward et al., 2008). The second dependent measure, *Overall Aggression,* will be represented by the mean responses for shock intensity and duration (in milliseconds) across all trials of the TAP. These scores

will also be *z*-transformed and then summed.

Procedure. In order to disguise the fact that the TAP is a measure of aggression, participants were given a fictitious cover story. They were informed that the study was aimed at understanding the effects of alcohol on reaction-time in a competitive situation. Participants were told that they were about to compete against a person of the same gender in an adjacent room on a reaction-time task. In actuality, there was no opponent. Instructions for the TAP were given as participants began drinking their beverages. They were informed that shortly after the words "Get Ready" appeared on a computer screen, the words "Press the Spacebar" would appear at which time they had to press, and hold down, the spacebar. Following this, the words "Release the Spacebar" would appear at which time they had 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 viewed the shocks they selected and received on a "volt meter" and by the illumination of one of 10 "shock lights" [(ranging from 1 (low) to 10 (high)] on the computer screen.

Prior to beginning the TAP, participants' pain thresholds and tolerances were assessed in order to determine the intensity parameters for the shocks they would receive. This was accomplished via the administration of short duration shocks (.5 seconds) 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 non-dominant 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, participants 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.

Immediately before beginning the TAP, participants provided subjective ratings of their level of intoxication. This was done using a specially constructed scale, the *Subjective Intoxication Scale*, which ranged 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." 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 would not confound any potential beverage group differences in aggression.

The entire TAP procedure consisted of 34 trials. 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. Following a losing trial, they received 1 of 10 shock intensities that lasted one second. Shock intensities were administered in a random pattern. Immediately following the TAP, BrACs 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.

Table 3: Drinking and Post-Drink Wait Times in Minutes

Dose	Drinking Time	Post-Drink Wait
0.00g/kg (Sober)	5	0
0.125g/kg (Placebo)	1	5
0.25g/kg (Low)	2	5
0.5g/kg (Medium)	5	20
0.75g/kg (Medium-High)	15	25
1.0g/kg (High)	20	40

Chapter Three

Results

Manipulation Checks

TAP Checks. In order to assess the success of the TAP deception, participants were asked about their opponent, the reaction-time task, and their performance on the task. Participants were asked whether their opponent "played fair," whether they tried their best, and whether they believed the task was a good measure of reaction-time. They responded in such a way as to convince the researchers that the TAP deception was successful. For example, typical responses were that the opponent was "competitive," "cool," and "typical." In some cases, participants expressed ire at their opponents through inappropriate language, antagonistic remarks, and obscene hand gestures, strongly suggesting that they felt aggressive and angry toward this person and indeed believed that they were competing against someone.

Subjective Intoxication / Placebo Checks. All individuals in the placebo condition indicated that they believed they had consumed alcohol. Subjective intoxication ratings are presented in Table 4 for all doses except the sober condition in which no alcohol was administered. Overall, Post-TAP subjective intoxication ratings were significantly higher than Pre-TAP subjective intoxication ratings $t(186) = 3.05$, $p < .01$. No gender differences were found.

BrAC Levels. As noted above, all participants were assessed to ensure a BrAC of 0.0% prior to administering alcohol. BrACs were measured immediately prior to beginning the TAP, immediately after its completion, and then two more times at four minutes Post-TAP and ten minutes Post-TAP. Pre and post-TAP BrACs are presented in

Table 5 for all doses except the sober condition in which no alcohol was administered. Figure 1 provides a visual representation of the same data. BrACs taken immediately following the completion of the TAP were significantly *higher* than those taken immediate prior to the TAP $t(186) = 5.38$, $p < .001$. However, BrACs taken four minutes after the TAP was completed were significantly *lower* than BrACs taken immediately after the TAP was completed $t(186) = 1.99$, $p < .05$. After another six minute (10 minutes Post-TAP), BrACs continued to descend and were significantly *lower* than the previous readings $t(186)= 4.99$, $p < .001$. No gender differences were found indicating that gender-adjusted doses were appropriate.

Aggression Data

Unprovoked Aggression. A 6 (dose) x 2 (gender) between-groups design analysis of variance (ANOVA) revealed a significant main effect for dose, $F(5, 175) = 2.65$, $p < .05$ and gender, $F(1, 175) = 23.5$, $p < .001$. Post-hoc analyses detected significant differences between the 1.0g/kg group and all other dose groups except for the 0.75g/kg dose. The 0.75g/kg group only differed significantly from the placebo and the sober groups. Consistent with meta-analytic studies, the sober and active placebo doses did not differ significantly from one another, nor did either differ from the 0.25g/kg or 0.5g/kg dose conditions. The 0.25g/kg and the 0.5/kg groups differed significantly only from the 1.0g/kg group. All significant post-hoc *p*-values were < .05. No dose X gender interaction was found. Polynomial contrasts did not reveal any significant higher order effects (i.e., quadratic or cubic). Furthermore, segmented regression analysis identified a single sloping line without breakpoint as the best fitting function (Oosterbaan , 1994). See Figure 2 (panel A) for a graphic representation of mean TAP unprovoked aggression

scores for each dose condition.

Overall Aggression. A 6 (dose) x 2 (gender) between-groups design ANOVA was conducted utilizing the overall measure of aggression. Again, significant main effects were found for dose, $F(5, 175) = 4.50$, $p < .05$ and gender, $F(1, 175) = 36.0$, $p < .001$. Post-hoc analyses confirmed the data observed with the unprovoked aggression variable with the addition of the 0.25g/kg group being significantly different from the 0.75g/kg group. A dose X gender interaction was not found. Higher order effects (quadratic and cubic) were also not significant. As in the case of the Unprovoked Aggression variable, segmented regression analysis identified a single sloping line without breakpoint as the best fitting function. See Figure 2 (panel B) for a graphic representation of mean TAP overall aggression scores for each dose condition.

Table 4: Subjective Intoxication Scale Scores

Rating	**Placebo**	**0.25g/kg**	**0.5g/kg**	**0.75g/kg**	**1.0g/kg**
Pre-TAP	1.34 (1.14)	1.59 (1.21)	3.45 (1.82)	3.17 (1.02)	3.86 (1.21)
Post-TAP	1.56 (1.13)	2.53 (1.46)	3.33 (1.94)	3.27 (1.16)	4.04 (2.45)

Table 5: Breath Alcohol Content (Means and Standard Deviations)

Dose	*Pre-TAP*	*Post-TAP*	*4 Min. Post-TAP*	*10 Min. Post-TAP*
0.125g/kg	.014 (.008)	.010 (.006)	.009 (.006)	.008 (.006)
0.25g/kg	.023 (.011)	.028 (.010)	.027 (.008)	.026 (.007)
0.5g/kg	.051 (.016)	.056 (.010)	.055 (.009)	.053 (.009)
0.75g/kg	.074 (.021)	.085 (.018)	.083 (.016)	.076 (.016)
1.0g/kg	.104 (.013)	.112 (.014)	.110 (.015)	.103 (.013)

Figure 1: Breath Alcohol Content Readings Before and After the TAP for Each Dose Condition

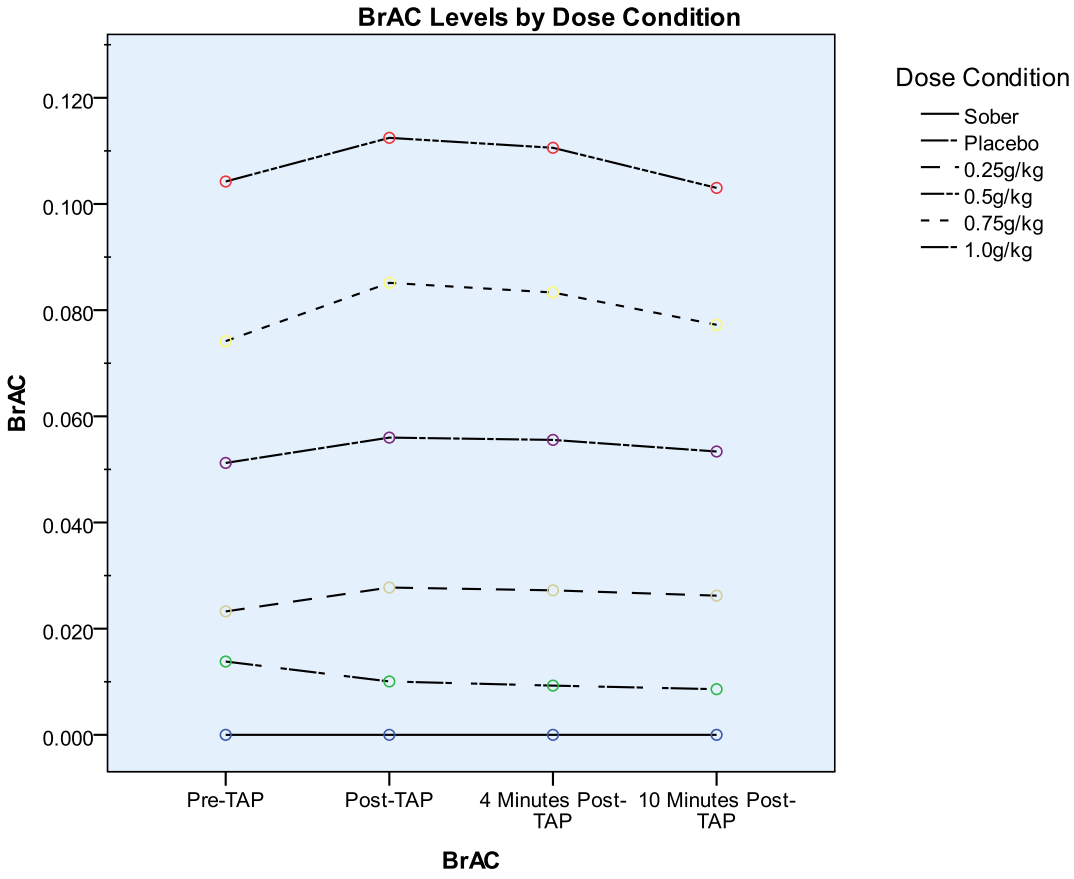
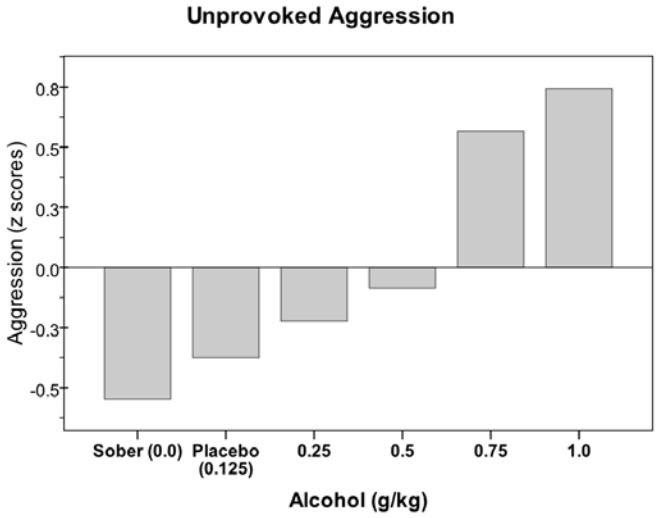
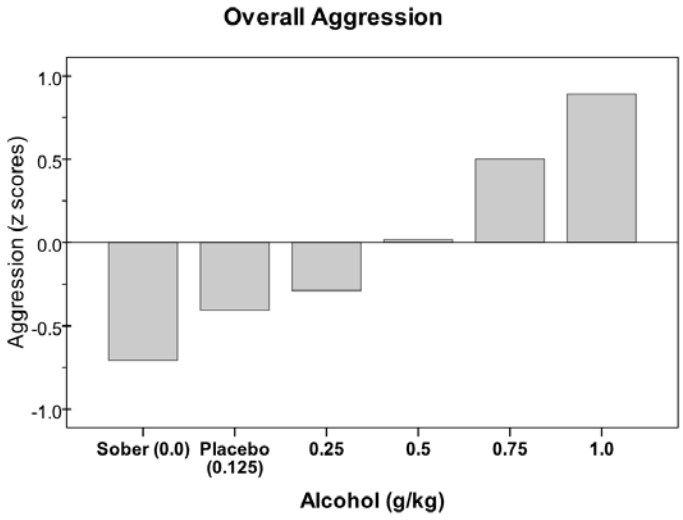


Figure 2: Aggression Levels

Panel A: Unprovoked aggression (first trial shock intensity and duration, standardized and then summed) as a function of alcohol dose.



Panel B: Overall Aggression (combined shock intensity and duration for all trials except trial 1, standardized and then summed) as a function of alcohol dose.



Chapter Four

Discussion

As can be seen in Figure 2, our results demonstrated that higher alcohol doses produced linear increases in aggression for both dependent variables. This pattern was evidenced in both men and women using an aggression paradigm (i.e., the TAP) that has never previously been used to assess the effects of alcohol dose on aggression in women or with the number and range of doses used herein. Moreover, we were able to show that the relationship between alcohol dose and aggression is best defined by a linear-shaped function up to a high dose of 1.0g/kg, as opposed to a curvilinear or threshold-shaped function, suggesting that in general, the more alcohol an individual consumes, the more likely that individual will exhibit aggressive behavior.

An important result that emerged from this experiment is that there were no statistically significant differences in aggression between any of the lower doses [i.e., Sober (0.0g/kg), Active Placebo (0.125g/kg), 0.25g/kg, and 0.5g/kg)]. Compared with the lower doses just mentioned, significant increases in aggression were only observed in the 0.75g/kg and the 1.0g/kg doses; particularly the 1.0g/kg dose which produced the greatest amount of aggression. These data confirm the findings of more naturalistic studies indicating that violent behavior tends to occur at higher BrAC levels by demonstrating that the highest levels of aggression occurred at the highest alcohol doses.

An important design element in this study was the decision to utilize different wait times following alcohol consumption prior to beginning the TAP for the different dose conditions in order to control for each group's location alcohol's biphasic absorption curve; a simpler methodology would have been to control for wait time, but not control

for the more serious confounds resultant from alcohol's pharmacological properties. All evidence indicates that we were able to successfully control for alcohol's biphasic effects in that we were able to maximize BrACs within each dose condition while remaining on the ascending limb of the biphasic curve (see Tables 4-5 and Figure 1); all dose conditions (except the Sober and Placebo control conditions) transitioned, on average, from the ascending limb to the descending limb of the absorption curve within four minutes following the completion of the TAP. Given such pronounced success at achieving optimal BrACs across diverse alcohol doses, we hope that the post-alcohol-consumption wait-times utilized in the present study might be informative to other researchers interested in controlling for alcohol's biphasic effects (see Table 3 for wait times for each dose condition).

Clarifying Previous Research

This study helped to clarify the mixed results of past studies by implementing a design that specifically addressed a number of previously limiting issues. The first issue addressed in the present study was the inconsistency of experimental alcohol dose conditions. As is evinced in Table 2, past studies have used either a sober or a placebo control group. The present study was the first to explicitly include *both* a sober and a placebo control condition, thus allowing us to use two different baselines from which to compare the effects of different alcohol doses. As is also evidenced in Table 2, previous studies differed considerably on both the number of alcohol doses administered (between three and six) as well as the highest dose administered (0.46g/kg to 1.0g/kg). The present study utilized six alcohol conditions (see Table 2) with a maximum dose of 1.0g/kg, making it only the second alcohol-aggression study to utilize six alcohol doses (Cherek

utilized six doses in one previous study with a maximum dose of 0.52g/kg; Cherek et al., 1984). Having more alcohol doses that span a wider range of alcohol amounts allows for stronger inferences regarding how alcohol affects aggression at different doses.

Cherek and colleagues have contributed substantially to understanding dose-dependent effects of alcohol on aggression and their work with the PSAP is laudatory. Studies from this group of scientists constitute the majority of the experimental literature on this subject (7 out of 10 studies reviewed above). However, the extent to which their findings generalize across other laboratory settings and other measures of aggression is a critical question. The current investigation's findings have, in general, supported their findings, while simultaneously providing clarification on past inconsistencies such as their 1992 study (Cherek et al.) which found that higher doses of alcohol unexpectedly decreased aggression. One possible reason for the mixed findings in previous experimental investigations of the dose-response relation between alcohol and aggression is the use of small sample sizes. For example, the study with unexpected results just mentioned used only four participants (Cherek et al., 1992). More importantly, the median number of participants for all 10 experimental studies reviewed in this paper is *only eight.* The current investigation provides a substantial improvement in sample size over past studies by examining dose-dependent effects of alcohol on aggression in 187 participants. Another limitation addressed by the present investigation concerns the tendency of most previous studies to use only male participants. Only three prior studies examined the alcohol dose-response effects on aggression in women (Bond and Lader, 1986; Dougherty et al., 1996; 1999) all of which used the PSAP or a modified version of this task.

The Dose-Response Shape Between Alcohol and Aggression

As explained earlier, the positive linear relation observed in this experiment between alcohol dose and aggression is only one of several theoretically possible relations. One possible dose-response shape could have been that alcohol only produced aggression above a particular "dose threshold" after which aggression either remains stable or continues to increase with higher alcohol doses. Conversely, some researchers have suggested that lower doses of alcohol may actually decrease aggression by producing a state of affective tranquility (Taylor and Gammon, 1976). Our results argue against such hypotheses. However, it is important to note that our conclusions pertain only to blood alcohol concentrations within a certain range. Given the ethical limitations of how much alcohol one can administer in experimental settings, we cannot definitively determine what dose or BrAC constitutes the point at which aggression will begin to decline. Animal analogue studies pose one possible source of information concerning this issue. For example, one study with mice demonstrated that the aggressive-eliciting effects of alcohol begin to attenuate somewhere between doses of 1.0g/kg and 3.0g/kg of alcohol (Miczek et al., 1998). Unfortunately, animal studies have reached inconsistent results concerning this question, and it is unclear how such findings would generalize to humans and different alcohol tolerance levels.

Accurate awareness of the shape of the dose-response function between alcohol and aggression is critical for developing and advancing theoretical causal explanations as well as for identifying factors that put individuals at risk for alcohol-related violence. For example, an interesting, but not unexpected finding of this study is that women are less aggressive than men after consuming similar doses of alcohol, and yet still demonstrate

the same basic dose-response pattern (i.e., more alcohol = more aggression; see Giancola et al., 2009 for a review of gender differences in alcohol-related aggression). While it is possible that different mechanisms are responsible for alcohol's aggression eliciting effects in men and women, the fact that they both exhibit a similar dose-response shape suggests that a similar mechanism might be underlying the relation in both genders and that this mechanism has a more pronounced effect on men than women.

Implications for Public Policy

The title of this article alludes to a very important public health implication derived from the results of this investigation: that alcohol-related violence (and all of its societal burdens) can be attenuated by limiting the amount of alcohol consumed on any one occasion. While this suggestion may appear to be commonsensical, it is worth noting that the number of "commonsense" interventions with little or no empirical support is staggering and that many of these interventions have been shown to actually do more actual harm than good (e.g., Landman, Ling, & Glantz, 2002; Mann et al., 1997; Patel, 1996). Knowing that both men and women behave more aggressively the more they drink, provides justification for policies designed to target excessive drinking as a means of reducing violence.

Unfortunately there is no clear threshold or cutoff of when alcohol begins to elicit aggression. Despite our findings that a statistically significant increase in aggression over baseline levels did not occur until a 0.75 g/kg or higher dose, segmented-regression analyses revealed that the dose-response models that best fit the data are not threshold models; rather, they are linear models without lower or upper breakpoints. As is apparent in Figure 2 there were progressively increasing mean levels of aggression for every

increase in alcohol dose, which finding is consistent with a linear relation. The ultimate implication of this finding for public health interventions is that the only sure way to eliminate alcohol-related violence is to reduce alcohol consumption to zero. Of course, a linear relation also implies that less comprehensive harm reduction efforts to attenuate the amount of alcohol consumed by any one individual per occasion are still likely to be beneficial in terms of reducing alcohol-related violence.

Conclusion

A wealth of experimental studies have investigated the impact of alcohol on aggression. However, only few of these have examined the effects of different doses of alcohol on aggression and a clear picture of this dose-response relation has not been forthcoming. We believe that our findings help to clarify this literature by demonstrating a positive linear relation between alcohol and aggression in both men and women up to a high dose of 1.0g/kg. Our findings also have clear public health implications: violence and excessive drinking go hand in hand. Aggression is just one more reason why drinking more is a bad idea. Understanding the dose-response relation between alcohol and aggression is a critical step on the road to understanding exactly *how* alcohol influences aggression.

References

Abbey, A., Clinton, A. M., McAuslan, P., Zawacki, T., & Buck, P. O. (2002). Alcohol-involved rapes: Are they more violent? *Psychology of Women Quarterly, 26*(2), 99-109.

Abbey, A., Zawacki, T., Buck, P.O., Clinton, A.M., & McAuslan, P. (2003). Sexual assault and alcohol consumption: What do we know about their relationship and what types of research are still needed. *Aggression and Violent Behavior, 9*, 271-303.

Anderson, C. A., & Bushman, B. J. External validity of "trivial" experiments: The case of laboratory aggression. (1997). *Review of General Psychology, 1,* 19-41.

Baldacchino, A. (2002). Systemic effects of excess alcohol consumption. In W. Caan(Ed.), *Drink, drugs and dependence: From science to clinical practice* (pp. 17-25). New York: Routledge.

Bartholow, B. D., Anderson, C. A., Carnagey, N. L., & Benjamin, A. J. (2005). Interactive effects of life experience and situational cues on aggression: The weapons priming effect in hunters and nonhunters. *Journal of Experimental Social Psychology, 41,* 48-60.

Bond, A., & Lader, M. (1986). The relationship between induced behavioral aggression and mood after the consumption of two doses of alcohol. *British Journal of Addiction, 81*, 65-75.

Borders, A., Barnwell, S. S., & Earleywine, M. (2007). Alcohol-aggression expectancies and dispositional rumination moderate the effect of alcohol consumption on alcohol-related aggression and hostility. *Aggressive Behavior, 33*, 327-338.

Branas, C. C., Elliott, M. R., Richmond, T. S., Culhane, D. P., & Wiebe, D. J. (2009). Alcohol consumption, alcohol outlets, and the risk of being assaulted with a gun. *Alcoholism-Clinical and Experimental Research, 33,* 906-915.

Bushman, B., & Cooper, H. (1990). Effects of alcohol on human aggression: An integrative research review. *Psychological Bulletin, 107,* 341-354.

Calabrese, E. J., & Baldwin, L. A. (2003). Ethanol and hormesis. *Critical Reviews in Toxicology, 33*(3), 407-424.

Carlson, M., Marcus-Newhall, A., & Miller, N. (1989). Evidence for a general construct of aggression. *Personality and Social Psychology Bulletin, 15,* 377-389.

Carnagey, N. L., & Anderson, C. A. (2005). The effects of reward and punishment in violent video games on aggressive affect, cognition, and behavior. *Psychological Science, 16,* 882-889.

Cherek, D. R. (1981). Effects of smoking different doses of nicotine on human aggressive behavior. *Psychopharmacology, 74,* 339-345.

Cherek, D. R., Spiga, R., & Egli, M. (1992). Effects of response requirement and alcohol on human aggressive responding. *Journal of the Experimental Analysis of Behavior, 58,* 577-587.

Cherek, D. R., Steinberg, J. L., & Manno, B. R. (1985). Effects of alcohol on human aggressive behavior. *Journal of Studies on Alcohol, 46,* 321-328.

Cherek, D. R., Steinberg, J. L., & Vines, R. V. (1984). Low doses of alcohol affect human aggressive responses. *Biological Psychiatry, 19*, 263-267.

Chermack, S. T., & Giancola, P. R. (1997). The relationship between alcohol and aggression: An integrated biopsychosocial approach. *Clinical Psychology Review, 17,* 621-649.

Dougherty, D. M., Bjork, J. M., Bennett, R. H., & Moeller, F. G. (1999). The effects of a cumulative alcohol dosing procedure on laboratory aggression in women and men. *Journal of Studies on Alcohol, 60,* 322-329.

Dougherty, D. M., Cherek, D. R., & Bennett, R. H. (1996). The effects of alcohol on the aggressive responding of women. *Journal of Studies on Alcohol, 57,* 178-186.

Exum, M. L. (2006). Alcohol and aggression: an integration of findings from experimental studies. *Journal of Criminal Justice, 34*, 131-145.

Foran, H. M., & O'Leary, K. D. (2008). Alcohol and intimate partner violence: A meta-analytic review. *Clinical Psychology Review, 28*, 1222-1234.

Giancola, P. R., & Chermack, S. T. (1998). Construct validity of laboratory aggression paradigms: A response to Tedeschi & Quigley. *Aggression and Violent Behavior, 3,* 237-253.

Giancola, P. R., Levinson, C. A., Corman, M. D., Godlaski, A. J., Morris, D. H., & Phillips, J. P. (2009). Men and women, alcohol and aggression. *Experimental and Clinical Psychopharmacology, 17,* 154-64.

Giancola, P. R., & Zeichner, A. (1997). The biphasic effects of alcohol on human aggression. *Journal of Abnormal Psychology, 106,* 598-607.

Graham, K., & Wells, S. (2001). Aggression among young adults in the social context of the bar. *Addiction Research & Theory, 9,* 193-219.

Graham, K., Bernards, S., Osgood, D. W., & Wells, S. (2006). Bad nights or bad bars? Multi-level analysis of envrionmental predictors of aggression in late-night large capacity bars and clubs. *Addiction, 101,* 1569-1580.

Graham, K., Osgood, D. W., Wells, S., & Stockwell, T. (2006). To what extent is intoxication associated with aggression in bars? A multilevel analysis. *Journal of Studies on Alcohol, 67,* 382-90.

Greenfield, T. K., & Kerr, W. C. (2008). Alcohol measurement methodology in epidemiology: Recent advances and opportunities. *Addiction, 103*(7), 1082-1099.

Hoaken, P. N., & Pihl, R. O. (2000). The effects of alcohol intoxication on aggressive responses in men and women. *Alcohol & Alcoholism, 35,* 471-477.

Hove, M. C., Parkhill, M. R., Neighbors, C., McConchie, J. M., & Fossos, N. (2010). Alcohol consumption and intimate partner violence perpetration among college students: The role of self-determination. *Journal of Studies on Alcohol and Drugs, 71*(1), 78-85.

Ito, T., Miller, N., & Pollock, V. (1996). Alcohol and aggression: A meta-anlaysis of the moderating effects of inhibitory cues, triggering events, and self-focused attention. *Psychological Bulletin, 120,* 60-82.

Jones-Webb, R., McKee, P., Hanan, P., Wall, M., Pham, L., Erickson, D., Wagenaar, A. (2008). Alcohol and malt liquor availability and promotion and homicide in inner-cities. *Substance Use & Misuse, 43,* 159-177.

Kelly, T. H., Cherek, D. R., & Steinberg, J. L. (1989). Concurrent reinforcement and alcohol: Interactive effects on human aggressive behavior. *Journal of Studies on Alcohol, 50,* 399-405.

Kelly, T. H., Cherek, D. R., Steinberg, J. L., & Robinson, D. (1988). Effects of provocation and alcohol on human aggressive behavior. *Drug and Alcohol Dependence, 21,* 105-112.

Landman, A., Ling, P. M., Glantz, S. A. (2002). Tobacco industry youth smoking prevention programs: Protecting the industry and hurting tobacco control. *American Journal of Public Health, 92*, 917-930.

Mann, T., Nolen-Hoeksema, S., Huang, K., Burgard, D., Wright, A., & Hanson, K. (1997). Are two interventions worse than none? Join primary and secondary prevention of eating disorders in college females. *Health Psychology, 16*, 215-225.

Martin, C. S., & Sayette, M. A. (1993). Experimental design in alcohol administration research: Limitations and alternatives in the manipulation of dosage-set. *Journal of Studies on Alcohol, 54,* 750-761.

Miczek, K. A., Barros, H. M., Sakoda, L., & Weerts, E. M. (1998). Alcohol and heightened aggression in individual mice. *Alcoholism, Clinical and Experimental Research*, *22*(8), 1698-1705.

McDowell, M. A., Fryar, C. D., Hirsch, R., & Ogden, C. L. (2005). *Anthropometric Reference Data for Children and Adults: U.S. Population, 1999-2002*. Hyattsville, MD: National Center for Health Statistics.

Miller, T. R. (2006). Costs of alcohol and drug-involved crime. *Prevention Science, 7,* 333-342.

Neal, D. J., & Fromme, K. (2007). Event-level covariation of alcohol intoxication and behavioral risks during the first year of college. *Journal of Consulting and Clinical Psychology, 75,* 294-306.

NIAAA. (2000). *Tenth Special Report to the U.S. Congress on Alcohol and Health*. National Institutes of Health, Washington, DC (NIH Publication No. 00-1583).

Oosterbaan, R. J. (1994). Frequency and regression analysis of hydrologic data. In H.P.Ritzema (Ed.), *Drainage principles and applications.* Wageningen, The Netherlands: International Institute for Land Reclamation and Improvement.

Parrott, D. J., & Zeichner, A. (2001). Effects of nicotine deprivation and irritability on physical aggression in male smokers. *Psychology of Addictive Behaviors, 15,* 133–139.

Patel, T. (1996). 'Wrong needs' do more harm than good. *New Scientist, 149*, 6.

Phillips, S., Matusko, J., & Tomasovic, E. (2007). Reconsidering the relationship between alcohol and lethal violence. *Journal of Interpersonal Violence, 22,* 66-84.

Pridemore, W. A. (2002). Vodka and violence: Alcohol consumption and homicide rates in Russia. *American Journal of Public Health, 92*, 1921-1930.

Quigley, B. M. & Leonard, K. E. (2006). Alcohol expectancies and intoxicated aggression. *Aggression and Violent Behavior, 11,* 484-496.

Rolfe, A., Dalton, S., Krishnan, M., Orford, J., Mehdikhani, M., Cawley, J., & Ferrins-Brown, M. (2006). Alcohol, gender, aggression and violence: Findings from the Birmingham untreated heavy drinkers project. *Journal of Substance Use*, *11*, 343-358.

Ross, D. F., & Pihl, R. O. (1989). Modification of the balanced-placebo design for use at high blood-alcohol levels. *Addictive Behaviors, 14,* 91-97.

Selzer, M. L., Vinokur, A., & Rooijen, L. V. (1975). A Self-Administered Short Michigan Alcoholism Screening Test (SMAST). *Journal of Studies on Alcohol, 36,* 117-126.

Taylor, S. P. (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. *Journal of Personality, 35***,** 297-310.

Taylor, S. P., & Chermack, S. T. (1993). Alcohol, drugs and human physical aggression. *Journal of Studies on Alcohol, Supplement No. 11,* 78-88.

Taylor, S. P., & Gammon, C. B. (1975). Effects of type and dose of alcohol on human physical aggression. *Journal of Personality and Social Psychology, 32,* 169-175.

Taylor, S.P., Vardaris, R.M., Rawtich, A.B., Gammon, C.B., Cranston, J.W., & Lubetkin, A.I. (1976). The effects of alcohol and delta-9-tetrahydrocannabinol on human physical aggression. *Aggressive Behavior, 2,* 153-161.

Tremblay, P. F., Graham, K., & Wells, S. (2008). Severity of physical aggression reported by university students: A test of the interaction between trait aggression and alcohol consumption. *Personality and Individual Differences, 45,* 3-9.

Turner, C. (1990). How much alcohol is in a standard drink - an analysis of 125 studies. *British Journal of Addiction, 85*, 1171-1175.

U.S. Department of Justice, Bureau of Justice Statistics. (2008). *Criminal Victimizations in the United States, 2006 Statistical Tables.* Retrieved April 6, 2009, from Crime and Victim Statistics: http://www.ojp.usdoj.gov/bjs/pub/pdf/cvus06.pdf

Volavka, J. (1995). *Neurobiology of violence.* DC: American Psychiatric Press.

Volavka, J., & Nolan, K. A. (2008). Methodological structure for aggression research. *Psychiatry Quarterly, 79,* 293-300.

Ward, A., Mann, T., Westling, E. H., Creswell, J. D., Ebert, J. P., & Wallaert, M. (2008). Stepping up the pressure: Arousal can be associated with a reduction in male aggression. *Aggressive Behavior, 34,* 584-92.

Watson, P. E., Watson, I. D., & Batt, R. D. (1981). Prediction of blood alcohol concentrations in human subjects. *Journal of Studies on Alcohol, 42,* 547-556.

Wells, S., & Graham, K. (2003). Aggression involving alcohol: Relationship to drinking patterns and social context. *Addiction, 98,* 33-42.

Wells, S., Graham, K., & West, P. (2000). Alcohol-related aggression in the general population. *Journal of Studies on Alcohol, 61*, 626-632.

Wells, S., Graham, K., Speechley, M., & Koval, J. (2006). Do predisposing and family background characteristics modify or confound the relationship between drinking frequency and alcohol-related aggression? A study of late adolescent and young adult drinkers. *Addictive Behaviors, 31*, 661-675.

Wells, S., Mihic, L., Tremblay, P. F., Graham, K., & Demers, A. (2008). Where, with whom, and how much alcohol is consumed on drinking events involving aggression? Event-level associations in a Canadian National Survey of university students. *Alcoholism, Clinical and Experimental Research, 32,* 522-533.

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