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ABSTRACT OF THESIS

IRRITABILITY, EXECUTIVE FUNCTIONING, AND THE ALCOHOL- AGGRESSION RELATION

The purpose of this investigation was twofold. First, to test the hypothesis that irritability and executive functioning (EF), two previously established risk factors for alcohol-related aggression, would interact to conjointly confer multiplicative risk for intoxicated violence that is not observed when testing either variable alone. Second, to test the hypothesis that irritability would mediate the relation between EF and alcohol-related aggression. EF was measured using seven well-established neuropsychological tests. Irritability was measured using the *Caprara Irritability Scale-CIS*. Participants were 310 male and female social drinkers between the ages of 21 – 35 years old. After consuming an alcohol or placebo beverage, participants were tested on a laboratory aggression task in which electric shock are given to and received from a fictitious opponent under the guise of a competitive reaction time task. Aggression was operationalized as shock intensities administered to the fictitious opponent. Results indicated that irritability successfully mediated the relation between EF and intoxicated aggression for men only. No support was found to suggest that EF and irritability together confer multiplicative risk for intoxicated aggression. Results are discussed within a cognitive neoassociationistic framework for aggressive behavior.

KEYWORDS: Irritability, Executive Functioning, Alcohol, Aggression, Cognitive neoassociationistic

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June 23, 2008

IRRITABILITY, EXECUTIVE FUNCTIONING, AND THE ALCOHOL-
AGGRESSION RELATION

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THESIS

Aaron John Godlaski

The Graduate School
University of Kentucky

2008

IRRITABILITY, EXECUTIVE FUNCTIONING, AND THE ALCOHOL-
AGGRESSION RELATION

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

By

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2008

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Chapter One Introduction

The research literature has clearly established a relation between acute alcohol consumption and aggressive behavior (Bushman & Cooper, 1990; Chermack & Giancola, 1997; Fishbein, 2003). In fact, a recent study estimated that the economic costs associated with alcohol-related violence exceed \$205 billion, with 85% of those costs attributed to violent crime; with alcohol being responsible for more than double the costs of all other drugs combined (Miller, Levy, Cohen, & Cox, 2006). A consistent and interesting finding is that alcohol increases aggression for some, but not for all individuals. This leads to a complex conceptualization of the relation where individual differences in mood, characteristics of personality, situational factors, and level of intoxication are all part of a multifaceted interaction. Despite vast evidence *describing* the alcohol-aggression relation, questions as to exactly *why* and *how* these factors operate together to engender such behavior continue to be an evolving topic of research.

A large survey study found alcohol consumption to be implicated in 42% of violent crimes (Pernanen, 1991). This number may actually be even higher (closer to 55% to 60%) due to the underreporting or lack of reporting of such behavior (United States Department of Justice, 2005). Among those victims who provided information about their offender's perceived use of alcohol, about 30% reported that the offender had been drinking. With regard to intimate partner or spousal violence, 75% of victims reported having been attacked by an offender who had been drinking (United States Department of Justice, 2005).

Survey studies have also determined that it is alcohol's acute, rather than its chronic effects that have the greatest impact on aggressive behavior (e.g., Fals-Stewart, 2003; Murphy, Winters, O'Farrell, Fals-Stewart, & Murphy, 2005). By design, survey studies do not manipulate alcohol consumption and they typically measure violent behavior in a *post-hoc* fashion either by self-report methods or by accessing archival data. These studies have contributed invaluable information to the alcohol and aggression literature (e.g., Abbey, Zawacki & Buck, 2005; Leonard, Collins & Quigley, 2003; O'Farrell, Fals-Stewart, Murphy & Murphy, 2003; Raskin-White & Spatz-Widom, 2003; Raskin-White, Hansell & Brick, 1993; Testa, 2002; Zhang, Welte & Wiczorek, 1999; Zhang, Welte & Wiczorek, 2002).

Complementing the above survey studies are laboratory investigations that allow for the "live" behavioral assessment of alcohol's effects on aggression. In these studies, aggression has typically been measured using tasks in which participants administer and receive mild electric shocks, tone blasts, or "point subtractions" (redeemable for money) to/from a fictitious opponent under the guise of a competitive interpersonal task (reviewed in Giancola & Chermack, 1998). Such investigations also have the advantage of allowing for the control of alcohol administration, measurement of blood alcohol concentration, and the assessment of personological variables as well as other factors. Over 80 laboratory-based studies report a robust relation between alcohol consumption and aggression showing that persons who receive alcohol are more aggressive than those who receive placebo or a non-alcohol control beverage (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Ito, Miller, & Pollock, 1996; Kelly & Cherek, 1993; Taylor & Chermack, 1993).

As noted above, alcohol-related aggression occurs in some, but not all individuals. This is reflected in the results of several meta-analytic studies demonstrating that alcohol has a “medium” effect size ($d = 0.49$ to 0.61) on aggression (Bushman & Cooper, 1990; Hull & Bond, 1986; Ito, Miller, & Pollock, 1996; Steele & Southwick, 1985). However, it has been hypothesized that this effect size may actually be masking alcohol’s true effect on aggressive behavior because it fails to take into account moderating individual difference variables (Giancola, 2004).

Given this, researchers have focused on identifying risk factor variables that are hypothesized to moderate the alcohol-aggression relation. Research has demonstrated that alcohol is more likely to facilitate aggression for persons with higher levels of dispositional aggressivity (Barnwell, Borders, & Earleywine, 2006; Heyman, O’Leary, & Jouriles, 1995), irritability (Giancola, 2002), trait anger (Parrott & Zeichner, 2002), hostility, permissive beliefs about aggression (Leonard & Senchak, 1993), deviant attitudes (Zhang, Wieczorek, & Welte, 1997), sensation seeking (Cheong & Nagoshi, 1999), as well as lower levels of anger control (Parrott & Giancola, 2004), socialization, self-control (Boyatzis, 1975), dispositional empathy (Giancola, 2003), intelligence (Zhang, Welte & Wieczorek, 1999), and executive functioning (Giancola, 2004). Moreover, alcohol has also been found to potentiate aggression for persons with a difficult temperament (Giancola, 2004a), a desired image of power (Quigley, Corbett, & Tedeschi, 2002), beliefs that alcohol causes aggression (Chermack & Taylor, 1995) as well as high marital conflict (Quigley & Leonard, 1999) and dissatisfaction (Leonard & Senchak, 1993).

The present investigation will focus on two risk factors mentioned above, executive functioning (EF) and irritability. Both have been shown to be significant moderators of the alcohol-aggression relation (Giancola, 2002, 2004a). However, their multiplicative effect on this association has not been examined. Such inquiry is important because, on both empirical and theoretical grounds, EF is purported to regulate affect and thus, by extension, to predict irritability (Moffitt, 1993). Accordingly, a case will be made below that irritability may, in part, mediate the relation between EF and alcohol-related aggression, and that examining the conjoint effects of these variables (i.e., EF and irritability) might show that they confer a multiplicative risk for intoxicated violence that cannot be observed when testing either variable alone.

Executive Functioning

Definition, Theory, and Structure. EF is a higher-order cognitive construct involved in the planning, initiation, and self-regulation of goal-directed behavior (Berger & Posner, 2000; Milner, 1995). Cognitive skills included under the rubric of EF include attentional control, hypothesis generation, previewing, strategic planning, abstract reasoning, temporal response sequencing, cognitive flexibility, set shifting, motor inhibition, and the ability to adaptively manipulate and process information in working memory (Giancola, 2004; Kimberg & Farah, 1993; Stuss & Alexander, 2000).

The structure of the EF construct has been a point of contention with theorists for some time. EF can be understood as a set of distinct, yet interrelated skills. These skills may be measured and conceptualized as distinct component processes (Baddeley & Logie, 1999) or represented and treated as a unified whole (Duncan, Seitz, Kolondny, Bor, Herzog & Ahmed, 2000; Zelazo, Carter, Reznick, & Frye, 1997). This unified structure of EF may be understood from the standpoint of Baddeley’s (1986) model of

working memory. This functional model posits that a “central executive” operates within working memory to allocate resources to different brain functions. The vagueness of the “central executive” stems from the lack of a definitive understanding of how all of these processes work together to exert control over behavior. Baddeley later admitted that the early conceptions of the unified central executive functioned as little more than a “rag-bag” filled with cognitive abilities whose relations were not well understood (Baddeley, 1998). Another issue relating to the conception of a unified EF structure is that it begs the question of the location of a homunculus through which information is filtered and decisions are made. However, because no anatomical structure for such a homunculus has been identified, the structure, and therefore the construct, in all probability does not exist as such (Parkin, 1998). This criticism is sound, but based on the misconception that because the system is referred to as “a whole” that it must function and be visible only as such.

Theoretical opinion varies regarding the unitary versus non-unitary structure of EF. This stems mostly from factor analytic research on the structure of EF. Some analyses have shown EF to be best understood as a unified general construct (Giancola, 2004; Giancola, Mezzich, & Tartar, 1998), while others have fractionated it into separate components (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000; Lehto, Juujarvi, Kooistra, & Pullkinen, 2003). However, the authors of these latter studies did acknowledge that although their data could be represented by a multifactorial structure, the factors were not independent from one another and shared a significant underlying commonality. It is well known that factor analytic results are greatly affected by one’s choice of variables and sample size. Moreover, another reason for the difficulty in solidifying a reliable EF structure is the nature of the tests used to assess its constituent skills. Correlations between these tests naturally tend to be relatively low. This is likely due to the fact that they are assessing what are interrelated, yet ultimately different functions (i.e., abstract reasoning, strategic planning, working memory, etc.). Further amplifying the differences between these separate skills are the varying metrics utilized to assess these functions such as time, accuracy, number of moves, etc. Finally, EF cannot be measured in a neuropsychological or neurological vacuum and as such, scores will be logically related to, and confounded by, other non-EF components. As such, it is our belief that given a large enough test battery, a factor analysis of EF measures will naturally yield a small number of secondary factors that will be overshadowed by a more general superordinate factor. We take the view that neither solution is “more correct” than the other, but simply that they represent different levels of analysis of the same construct. Given the infancy of this area of research, further work examining the structure of EF is clearly warranted. Nevertheless, at this point in time, we adopt the position of leading theorists who assert that although EF is indeed multifaceted, it also possesses a common underlying structure (Miyake et al., 2000; Porecman, 1987; Zelazo et al., 1997).

As such, for the purposes of this investigation, we chose to focus on a wide range of EF processes because our hypotheses center on EF in general; not specific aspects of EF. This decision was motivated by the fact that research on EF as a whole, let alone its subcomponents and how they interact with one another, is still in its early stages (Alexander & Stuss, 2006) and there is very little consensus regarding the specification of the EF concept (see chapters in Miyake & Shah, 2004; Stuss & Alexander, 2005). As

more is understood about the relation between the subcomponents of EF as well as their relation to various outcome variables, we will be in a much better position, both theoretically and empirically, to hypothesize more specific mechanisms. This is in line with the concept of an “executive board of directors” (Baddeley, 1996) of which a number of separate but interrelated skills operate together leading to a common underlying structure.

Relation to Aggression. A number of studies already exist relating EF to aggressive behavior (reviewed in Giancola, 1995; Hawkins & Trobst, 2000; Moffitt, 1993; Moffitt & Henry, 1991; Moffitt, Lyman, & Sylva, 1994; Morgan & Lilienfeld, 2000). That is, deficits in EF are linked to an elevation in aggressive behavior. A meta-analytic study of EF and antisocial behavior found EF’s effect size to be in the “medium” to “large” range (Morgan & Lilienfeld, 2000). Because high EF is related to adaptive planning, initiation, and the self-regulation of goal-directed behavior; low EF is logically related to poor judgment and poor behavioral regulation. Consequently, EF should be viewed as a moderator of aggressive behavior. For example, when an individual is exposed to a provocative situation, s/he is faced with at least one of two options; either to aggress or to inhibit that impulse. If an adequate level of EF is available, the individual will be aware of the inhibitory internal and contextual cues present and appraise the consequences of making either behavioral choice. A person with intact EF, may then decide on the more appropriate non-aggressive action in lieu of the immediate impulse to aggress against the provocateur. Alternatively, an individual with low EF will lack the cognitive abilities to properly process and possibly perceive the information at hand. Confronted with the option to aggress or inhibit in the face of provocation, this individual will be unable to fully appraise the full array of information at hand (e.g., internal and external cues signaling punishment), cognitively generate alternative adaptive behavioral options, and understand the consequences of his/her available choices. As a result s/he will not properly possess the necessary resources to inhibit the immediate impulse to aggress.

Irritability

Caprara (1985) defines irritability as the tendency to act impulsively, conversely, or rudely to the slightest provocation and at the slightest disagreement. Buss and Durkee (1953) describe it in a similar fashion as “a readiness to explode with negative affect at the slightest provocation, including quick temper, grouchiness, exasperation, and rudeness.” (p.344). Both conceptions are descriptive of an individual who, in a provocative situation, is prone to negative cognitions and negative outbursts. To further specify the nature of irritability, it manifests itself as the tendency to take-up an offensive rather than defensive attitude towards a provocative stimulus (Caprara, Renzi, D’Augello, D’Imperio, & Travaglia, 1986).

Several studies support the relation between irritability and aggressive behavior. Men and women high in irritability have been shown to exhibit greater levels of aggression toward a fictitious opponent, in a laboratory setting, compared with their non-irritable counterparts, particularly after being exposed to a frustrating stimulus (Caprara, Renzi, Alcini, D’Imperio, & Travaglia, 1983; Caprara, Cinanni, D’Imperio, Passerini, Renzi, & Travaglia, 1985; Caprara et al., 1986). Parrott and Zeichner (2001) showed that, following nicotine deprivation, individuals with high irritability were more aggressive than those with low irritability. Irritability in children has been shown to be

predictive of difficult and oppositional behaviors later in life (Degangi, Wietlisbach, Goodin, & Scheiner, 1993; Evan, Heriot, & Friedman, 2002; Maziade, Caperaa, Leplante, Thivierge, Boudrault, Cote, & Bouten, 1985). Children who are easily aroused, possess limited self-regulatory behaviors, and have fleeting attentional capacity are also highly prone to being irritable (Evans et al., 2002). These findings are consistent with correlational studies showing irritability to be significantly related to a tolerance for violence (Caprara, Babaranelli, & Zimbardo, 1996), self-reported aggression (Caprara, Barbaranelli, & Comrey, 1992), and dispositional anger (Speilberger et al., 1993). Finally, irritability at age 12 has been shown to predict physical aggression and violence in late adolescence (Caprara, Paciello, Gerbino, & Cugini, 2007).

The Moderating Effects of EF and Irritability on the Alcohol-Aggression Relation

As noted above, both EF and irritability are related to aggression and both have been shown to moderate the alcohol-aggression relation. Two studies have directly examined the moderating effects of EF on alcohol-related aggression (Lau, Pihl, & Peterson, 1995; Giancola, 2004). In the former study, normal males were administered two neuropsychological tests of EF and separated into “high” and “low” functioning groups. They were administered either an alcohol or a non-alcohol beverage and were then tested on a laboratory aggression task. Alcohol and EF both impacted aggression; however, no support for a moderating role of EF was observed (Lau, Pihl, & Peterson, 1995). This negative finding may be due to the fact that only two measures of EF were used; both tests of working memory. As explained above, depending on one’s theoretical framework, working memory is only one part of the larger construct of EF. Viewed in this manner, such an assessment falls short of capturing the larger and more general construct. Furthermore, Lau et al.’s study likely lacked statistical power because only participants falling into the upper and lower score quartiles on the two EF measures were used, thus effectively cutting their sample size in half and lowering the probability to detect a significant interaction effect. Alternatively, Giancola (2004) utilized a larger test battery in an attempt to capture a variety of EF components using much larger sample of both men and women. A confirmatory factor analysis of the EF measures indicated that tests best loaded on a single factor. Results from this study indicated that EF did indeed moderate the alcohol-aggression relation. Specifically, alcohol was most likely to increase aggression for men with low EF.

Regarding irritability, only one study (Giancola, 2002) has assessed its involvement as a moderator of the alcohol-aggression relation. In this investigation, the participant’s level of irritability was assessed using a self-report instrument (Caprara et al., 1985). Participants were grouped into either intoxicated or placebo conditions and then underwent a laboratory aggression task. As with EF, results indicated that irritability moderated the alcohol-aggression relation. Specifically, alcohol was most likely to increase aggression for men with higher, rather than lower, irritability scores.

Does Irritability Mediate the EF-Aggression Relation?

At present, no empirically-tested theoretical framework exists linking EF, irritability, and alcohol-related aggression. Presented below is a heuristic conceptualization of this mediational relation drawing from Berkowitz’s (1993) cognitive neoassociationistic theory of aggressive behavior.

Current conceptualizations of irritability’s relation to aggression suggest that aggression is the result of a combined interplay involving affective and cognitive

behavioral dysregulation (Caprara et al., 2007). When EF functions normally, in the absence of alcohol, an individual is capable of processing information from the environment and properly regulating behavior leading to a capacity to adeptly carry out and realize goal-directed behaviors. However, it is well-established that alcohol disrupts cognitive functioning, particularly EF (Curin & Fairchild, 2003; Finn, Justus, Mazas, & Steinmetz, 1999; Hoaken, Assaad, & Pihl, 1998; Lyvers, & Maltzman, 1991; Peterson, Rothflesch, Zelazo, & Pihl, 1990). With this comes an inability to adequately attend to information from the environment, process that information, and formulate new plans based on that information to achieve one's goals (Steele, & Josephs, 1990). As this process deteriorates and the inability to achieve one's goals continues, one is likely to experience greater levels of negative affect, particularly, irritability. It is through this experience of irritability that the relation between EF and alcohol-related aggression is better explained or, in fact, mediated.

Neuropsychological research indicates that the prefrontal cortex represents the primary cortical substrate that subserves EF (Fuster, 1997; Luria, 1980; Milner & Petrides, 1984; Stuss & Levine, 2002). The prefrontal cortex also plays a role in the regulation and experience of dispositional traits, such as irritability (Alderman, 2003). This neural region also shares connections with the amygdala which is, in part, responsible for attaching affect to incoming information from the environment (Petrides, 1989). It can thus be argued that disruptions in EF lead to an increased experience of irritability and related responses in regard to noxious stimuli and it is this experience that predisposes an individual toward aggressive behavior.

The conception that irritability leads to aggression is consistent with Berkowitz's (1993) cognitive neoassociationistic model. This model posits that aggression is the result of negative affect which includes irritability. The experience of negative affect activates aggression-related memories, emotions, physiological responses, and motor patterns that form an associationistic network that, when activated, predisposes one towards aggression. The effects of the experience of negative affect lead to feelings of anger, frustration, and irritability and their experience can ultimately lead to an aggressive response to provocation. Berkowitz also suggests that the experience of irritability is not free-floating in this case and that the individual experiencing irritability will attribute the source of that irritability to the provoking target and ultimately become openly hostile towards that target (Berkowitz, 1998).

Given the above literature review, both EF and irritability have been established as moderators of the relation between alcohol and aggression. However, their conjoint effects have not been assessed in this capacity. Taken together, they may confer additional information, above and beyond their additive effects, regarding their role as risk factors for alcohol-related aggression. In addition, based on the available data, our hypothetical conceptualization described in the preceding paragraph also makes a case that irritability may also serve as a mediator of the relation between EF and aggressive behavior. Given that alcohol acts as a general disinhibitor of cognitive and affective regulation (Easdon, Izenberg, Armilo, Yu, & Alian, 2005; MacDonald, Fong, Zann, & Martineau, 2000), we hypothesize that such a mediational model would be stronger when participants are intoxicated than when they are sober.

Chapter Two Methodology

Data Source

The data for the study proposed herein was collected between 2000 and 2003 in the laboratory of Dr. Peter Giancola at the University of Kentucky.

Participants

Participants were 310 social drinkers (152 males and 158 females) between the ages of 21 and 35, ($M = 23$ years, $SD = 2.85$ years). The sample consisted of 290 Caucasians, 22 African Americans, and 1 Hispanic participant. Seventy-five percent of participants received a high-school diploma while 24% had an associates, bachelors, or a graduate degree. One percent had not completed a high-school education. Forty-eight percent of participants were financially self-supporting, with a mean annual salary of \$18,294; the remainder were supported by a parent or a spouse. Social drinking was defined as consuming 3 – 4 drinks per occasion on at least two occasions per month. Participants in the study were recruited through advertisements placed in local and university newspapers. They were initially screened over the telephone. Individuals reporting past or present drug or alcohol use problems, serious head injuries, learning disabilities, or serious mental or physical health problems were excluded from participation. Screening for drug, mental, and physical health problems occurred over the telephone using a standardized questionnaire (Giancola, 1999). The Short Michigan Alcohol Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975) was used to screen for alcohol use problems. Individuals scoring an 8 or more (indicative of an alcohol use problem) were excluded from the study. Eligible participants who complete the study were compensated \$50 dollars for their time and effort.

Prelaboratory Procedure

Individuals who successfully completed the telephone screening and met eligibility requirements were scheduled for an appointment at the laboratory. They were asked to refrain from drinking alcohol at least 24 hours prior to their scheduled appointment. They were also asked to refrain from the use of recreational drugs prior to participation and were told that they would undergo a urine drug screening prior to participation to confirm eligibility. Individuals who tested positive on the urine drug screen were given the option to reschedule their appointment for a later date to allow their bodies to detoxify. Individuals were also asked to refrain from eating one hour prior to their beginning the experiment. The standard fast time for studies involving alcohol administration is 4 hours, however, because the screening and test battery given prior to alcohol administration was 3 hours, this standard was observed. Prospective female participants were also required to undergo a urine pregnancy test administered along with the drug test. Women who were pregnant were excluded from participation due to the possible detrimental effects of consuming alcohol during pregnancy. Women within one week of menstruating were also excluded from participation and allowed to reschedule their appointment at least one week prior to or after their menstruation period to minimize the effects of hormonal fluctuation on aggressive responding (Volavka, 1995).

Laboratory Procedure

Once participants completed the required prescreening they were brought into the laboratory to begin the experiment. Demographic data was taken including age, ethnicity, socio-economic status, education level, parental education level, history of alcohol use,

and medical and psychiatric history. Participants then completed a series of neuropsychological test of EF. Participants then complete a series of personality questionnaires, including a measure of irritability. The administration of neuropsychological and personality tests took roughly 3 hours to complete.

Tests of EF

Porteus Maze Test. Participants were required to navigate their way through eight mazes (Porteus, 1965). They were instructed to not lift their pencil from the paper until each maze was completed. The *Impulsive Errors* score (i.e., *Qualitative Score*), was used to index EF (Porteus, 1965). This type of error reflects a lack of foresight, poor judgment, and difficulty learning from experience, as well as poor planning and organizational abilities (Crown, 1952; Porteus & Kepner, 1944).

Go/No-Go Task. Participants completed a computerized version of this task (Newman & Kosson, 1986). Participants were informed that a series of numbers were going to be presented, one at a time, in the center of a computer screen. They were told that they had an opportunity to win money based on their performance on the task. Participants were informed that each time a number appeared on the screen they had to choose whether or not they were going to press the spacebar on the keyboard and that their choice would result in either winning or losing money. They were given no further instructions. Prior to beginning the task, \$5.00 in quarters was placed on the table in front of the participant. The experimenter kept a large stack of quarters on his/her side of the table. Each time the participant won or lost a trial, the experimenter would respectively give or take away a quarter from the participant. Participants did not win or lose money if they made no response at all.

The task had a total of 85 trials. A total of 10 numbers were used. Five numbers were “winners” (37, 96, 78, 53, 29) and five were “losers” (43, 82, 64, 73, 31). The numbers were presented on the computer screen for two seconds with an inter-trial interval of one second. The first five trials were all winning numbers (to establish a dominant response set) and the remainder of the trials were randomly ordered with no consecutive win or lose sequence exceeding three trials. Participants had to learn, by trial and error, when to respond and when to not respond. Trials were presented in eight continuous blocks of 10, excluding the first five. EF was indexed by the total errors of commission (i.e., pressing the spacebar when incorrect) for the last 40 trials of the task. Such errors reflect an inability to inhibit incorrect responding under circumstances involving sustained attention (Newman & Kosson, 1986).

Trails B of the Trail Making Test. Participants were given a sheet of paper randomly arranged with the numbers “1” through “13” and the letters “A” through “L.” They were told that they had to connect the numbers and letters in an alternating sequence (e.g., 1-A-2-B-3-C...) as quickly as possible using a pencil. If an error was made, the experimenter quickly informed the participant so that it could be corrected. Performance on this task was measured as the amount of time taken to complete all of the connections (Reitan, 1992). Success on this task requires good cognitive flexibility and set shifting skills in order to quickly and repeatedly alternate between two different tasks.

Stroop Task. Participants were presented with three stimulus cards. For the first card, they were instructed to read a list of words (red, blue, green, yellow) printed in black ink as quickly as possible. For the second card, they were asked to name the color (red, blue, green, yellow) in which a series of “X”s were printed as quickly as possible.

These first two parts of the task respectively measure verbal and nonverbal perceptual processing speed. For the third card, participants reported the color of the ink in which words were printed as quickly as possible; however, the word names were incongruent with the colors in which they were printed. EF was indexed by the “*interference score*,” derived by subtracting the response time of the second portion of the task (color naming) from the response time of the third portion of the task (incongruent color-word naming) (MacLeod, 1991). Poor performance on this task reflects an inability to inhibit the effects of a distracting stimulus as well as poor attentional skills (MacLeod, 1991; Perret, 1974).

Conditional Associative Learning Test. Seven black squares (1” X 1”) were printed on a laminated 3” X 11” card and placed before the participant. Seven small lights were fixed, in a random arrangement, onto a 10” X 8” metal box which was placed anterior to the card. Participants were asked to learn the manner in which the squares and the lights were associated. The experimenter illuminated the lights in a fixed random order and the participant’s task was to point to the square that s/he believed was associated with the particular illuminated light. Participants learned the associations by trial and error based on feedback from the experimenter. The task ended when 17 consecutive correct responses were achieved or when 210 trials were exhausted. Performance was indexed by the number of errors committed. This test measures the ability to organize and utilize information contained in working memory (Petrides, 1985).

Tower of Hanoi. Participants were presented with a wooden platform mounted with three vertical rods. Five rings (differing in circumference) were stacked on the left-most rod (smaller rings were always stacked on top of a larger ring). Participants were instructed to reproduce the same stacking configuration of rings on the right-most rod by moving the rings according to the following three rules: 1) Only one ring can be moved at a time; 2) a larger ring cannot be placed on a smaller ring; and 3) unless actively being moved, no ring can be removed from a rod. Three trials were conducted. The first involved four rings and the second and third involved all five. Performance was indexed by the number of moves taken to complete the first and third trials (Goel & Grafman, 1995). Scores from the second trial were not used because the test was so difficult that most participants could not solve the problem. This task measures strategic planning and the organization and use of information contained in working memory reflected as the ability to sequentially order a series of responses to achieve a particular goal (Goel & Grafman, 1995).

Wisconsin Card Sorting Test. A computerized version of this task was administered (Heaton, 1993). Participants were presented with four sample “cards” at the top of the screen. The cards depicted between one and four stimulus shapes (i.e., circle, triangle, cross, and square) that were printed in one of four colors (i.e., red, blue, yellow, and green). At the bottom of the screen was a “deck” of 128 cards each printed with different combinations of these shapes and colors. Participants were asked to match each card from the deck to one of the sample cards. The cards could be matched according to their similarity in color, shape, or number of stimuli. However, participants were not informed of the matching principles. Each time 10 consecutive correct matches were achieved, the computer changed the matching principle without notifying the participants. The test proceeded until six sorting categories were completed or until all 128 cards were used. Performance on this task was indexed by the number of errors committed. Success on this task requires the ability to abandon a previous sorting principle and then generate

and test new hypotheses about other solutions thus capitalizing on cognitive flexibility and set shifting skills.

Giancola (2004) conducted a confirmatory factor analysis in which it was demonstrated that the EF tests used in the present study loaded on a single factor. To generate an overall EF variable for data analyses, scores on the above tests were first converted to *z*-scores. These *z*-scores were then summed to obtain an aggregate EF score where higher scores denote worse EF.

Test of Irritability

Once participants completed the neuropsychological test battery they completed a series of personality questionnaires including a measure of irritability. Irritability was assessed using the *Caprara Irritability Scale (CIS; Caprara et al., 1985)*. The CIS consists of 20 items scored on a 6-point Lickert scale. Examples of several items are: “When I am irritated I can’t tolerate discussion”, “Sometimes people bother me by just being around”, and “It takes very little for things to bug me.” All 20 items were summed to produce a total irritability score. Higher scores denote a greater propensity for irritability while lower scores suggest the opposite. The CIS was based on the irritability subscale of the *Buss-Durkee Hostility Inventory (Buss & Durkee, 1957)*. The scale’s design was based on the aforementioned definitions of irritability from Caprara (1985), as well as Buss and Durkee (1953).

The CIS has excellent psychometric properties. Based on a sample of healthy men and women, the scale possesses a Cronbach’s alpha of 0.81, a test-retest coefficient of 0.83, and a split-half coefficient of 0.90 (Caprara et al., 1985). Other studies have demonstrated higher reliability coefficients in both clinical (Tartar et al., 1995) and normal samples (Anderson, 1997). Support for the validity of the CIS has been established by significant positive correlations between CIS scores and self-report measures of state hostility (Anderson, 1997) as well as the administration of mild electric shocks to fictitious opponents on laboratory measures of aggression (Caprara et al., 1986; Parrott & Zeichner, 2001).

Experimental Design

Participants were assigned to one of the following groups: (a) men who received alcohol ($n = 77$), (b) men who received a placebo ($n = 75$), (c) women who received alcohol ($n = 82$), and (d) women who received a placebo ($n = 76$). All participants received both levels of provocation thus making it a repeated measure.

Beverage Administration

Men who received alcohol were administered a dose of 1g/kg of 95% alcohol USP mixed at a 1:5 ratio with Tropicana orange juice. Due to differences in body fat composition, women were given a dose of 0.90g/kg of alcohol. 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. However, during the explanation of the consent form, they were told that they would consume the equivalent

of about 3 to 4 mixed drinks. In order to ensure that participants would be accustomed to the dose of alcohol administered in our study, we excluded anyone that did not consume at least 3-4 drinks per occasion at least twice per month. No participant experienced any adverse effects due to alcohol consumption.

In addition to the two beverage groups 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 have also 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.

Aggression Task

A modified version of the *Taylor Aggression Paradigm* (Taylor, 1967) was used to measure aggression. The hardware for the task was developed by Coulbourne Instruments (Allentown, PA) and the computer software was developed by Vibranz Creative Group (Lexington, KY). 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 selected by the participants. The Taylor task and other similar laboratory paradigms have been repeatedly shown to be safe and valid measures of aggressive behavior for men and women (Anderson & Bushman, 1997; Giancola & Chermack, 1998; Hoaken & Pihl, 2000).

Participants 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.

Measures of Aggression

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

Mean Shock Intensity. This measure comprises the mean shock intensity selection (“1” through “10”) within each provocation condition. 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.

Proportion of “10’s” Selected (“Extreme Aggression”). This measure constitutes the mean proportion of trials that participants selected the highest shock intensity button (i.e., “10”) within each provocation condition. Similar to the shock intensity variable, separate extreme aggression means were calculated for the low and high provocation

conditions, thus yielding two variables. Although issue can be taken with nomenclature, it has been argued that elevated responses on this measure reflect the tendency to exhibit extreme levels of aggression in response to provocation (Chermack and Taylor, 1995).

Procedure

Upon entering the laboratory, subjects were explained the procedures of the study and were asked to sign an informed consent form. The experimenter then assessed their BACs to ensure sobriety. If the BAC test was negative, subjects then underwent a urine drug test and women also underwent a urine pregnancy test. BACs were measured using the Alco-Sensor IV breath analyzer (Intoximeters Inc, St-Louis, MO). Demographic data were then collected and subjects then completed the EF test battery and the CIS.

Subjects were then escorted into the testing room where they received their beverages. Twenty minutes were allotted for beverage consumption. In order to allow the alcohol to be sufficiently absorbed into the bloodstream, persons receiving alcohol had their pain thresholds tested (described below) 15 minutes after they finished their drinks. In order to maximize the placebo manipulation, individuals in the placebo group had their pain thresholds tested 2 minutes after they finished their drinks. It has been shown that placebo manipulations are only effective shortly after beverage consumption (Bradlyn and Young, 1983; Martin, Finn, & Young, 1990; Martin & Sayette, 1993). As such, testing pain thresholds 2 minutes after beverage consumption ensured that aggression was assessed while the placebo manipulation was most effective (Martin, et al., 1990; Martin & Sayette, 1993). BACs were measured following the pain threshold testing. The placebo group began the aggression task immediately after the pain threshold testing.

Given that the aggression-potentiating effects of alcohol are more likely to occur on the ascending limb of the BAC curve (Giancola & Zeichner, 1997) and because previous studies have found a BAC of at least 0.08% to be effective in eliciting aggressive behavior in some individuals (Giancola & Zeichner, 1997; Gustafson, 1992; Pihl, Smith, & Farrell, 1984), the alcohol group began the task after they reached an ascending BAC of at least 0.09%. One could argue that the time duration between the end of beverage consumption and beginning the aggression task should have been standardized for both beverage groups. This was not done because it would have reduced the effectiveness of the placebo manipulation (noted above) and would have produced undesirable large individual differences in BACs during the aggression task. Pertinently, following a standardized 30 minute wait after beverage consumption, Giancola and Zeichner (1995) found a BAC range between .06 and .14 using the same alcohol dose administered in the present investigation. More importantly, they also found a significant positive relation between this BAC range and aggression. Interestingly, these researchers additionally determined that even a long time delay (approximately 3 hours) between the end of alcohol consumption and the measurement of aggression does not affect responses on the Taylor task (Giancola & Zeichner, 1997). As such, it is very unlikely that the extra 15-20 minutes alcohol subjects had to wait before beginning the aggression task adversely affected their results. Given these data and arguments, it is clear that more experimental control is gained by standardizing BACs at the time aggression is assessed rather than simply waiting a pre-determined time following beverage consumption. Immediately before beginning the aggression task, subjects 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.”

Subjects’ 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 (1 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 fingertip electrodes attached to the index and middle fingers of the non-dominant hand using Velcro straps. Subjects 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, subjects 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 subject 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 subject through a hidden video camera.

Following the pain threshold testing, subjects were once again explained the instructions for the aggression task. They were informed that shortly after the words “Get Ready” appeared on the screen, the words “Press the Spacebar” would appear at which time they 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 subjects to deliver a shock to their opponent and a losing trial resulted in receiving a shock from this individual. Following a winning trial and pressing a shock button, subjects could 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 corresponded with the shock level they selected. These images were used to reinforce subjects’ beliefs that they were actually administering shocks. Upon losing a trial, subjects 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.

Subjects 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 subjects 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 procedure consisted of two successive blocks of trials. During the first block, subjects received shock intensities between “1” and “4” (mean intensity = 2.5) after they lost a trial. This denoted the low provocation condition. During the next block, they received shock intensities between “7” and “10” (mean intensity = 8.5) after they lost a trial. This denoted the high provocation condition. Each block consisted of 16 trials (8 wins and 8 losses). There were two “transition trials” between the blocks. Subjects lost both of the 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. Thus, there were a total of 34 trials. Having the high provocation condition always follow the low provocation condition is an intentional aspect of this task. Taylor and Chermack (1993) have argued that using the low-high sequence adds an increased degree of external validity to the task because this ordering best reflects how an escalation in interpersonal provocation leads to increased violence in “real-life” situations.

All shocks delivered to the subjects were of a 1 second duration. In actuality, reaction-times were not measured; the competitive task was used to lead subjects to believe that they were engaging in an adversarial interaction with another individual. The win/lose sequence was predetermined and controlled by the computer program that executed the task. The sequence was presented in a randomized fashion; however, each subject received the same sequence. The trials were interspersed by 5 second intervals. The initiation of trials, administration of shocks to the subjects, and the recording of the subjects’ responses were controlled by a computer. The experimenters, other electronic equipment, and the computer that controlled the task were located in an adjacent control room out of the subjects’ view. The experimenter secretly monitored the subject through a hidden video camera and microphone throughout the procedure.

Immediately following the testing procedure, BACs were measured and subjects 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.” Subjects 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 experimental manipulation (see below). Subjects were then compensated. All individuals who received alcohol were required to remain in the laboratory until their BAC dropped to 0.019%.

Deception Manipulation

In order to disguise the task as a measure of aggression, subjects were given a fictitious cover story. They were informed that the study was aimed at understanding how a person’s “thinking-style” and personality influence alcohol’s effects on reaction-time in a competitive situation. In order to convince subjects 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 subject into the testing room, s/he identified the confederate (same gender as the subject) as the “opponent.” No opportunity for an interaction between the subject and the confederate was allowed. Furthermore, immediately prior to testing their pain thresholds, subjects were informed that their competitor would undergo the same threshold testing procedure first. They were also informed that they would be able to hear his/her responses 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 his/her pain thresholds in accordance with a list of predetermined responses. All subjects heard the same experimenter-confederate verbal exchange. Of course, in actuality, there was no real opponent.

In previous research conducted in our laboratory, we found that subjects who were debriefed sometimes informed prospective participants about the procedures of the

study, thus invalidating the aggression data. Consequently, we no longer debrief subjects about the actual purpose of our research and its deception manipulations. Previous research has shown that our deceptions have no short- or long-term adverse effects (Pihl, Zacchia, & Zeichner, 1981). This investigation was approved by the University of Kentucky Institutional Review Board.

Proposed Analyses

Two main analyses are proposed for this investigation. The first involves testing a multiple regression model to assess a 4-way interaction between beverage type, gender, irritability, and EF on physical aggression as measured by the TAP. The second will be a test of irritability as a mediator between EF and alcohol-related aggression as measured by the TAP. The method and guidelines for detecting mediation were those put forth by Baron and Kenny (1986). To test the significance of the mediation analyses a bootstrap resampling method in addition to Sobel's test of significance will be used according to Preacher and Hayes (2004).

Power Analysis

Given that our largest hypothesized interaction consists of a 4-way effect, it is important to determine the number of subjects needed to detect such an interaction while holding all other variables constant. Parameters for the power analysis were $\alpha = .05$ and power = .80. Based on previous research in our laboratory (Giancola, 2002b, 2002d), we estimated that the full moderation model (i.e., EF X Irritability X Gender X Beverage) would account for approximately 20% of the total variance [i.e., "medium" effect size ($f^2 = .15$)]. This is a conservative estimate in that many of our models accounted for around 30% of the variance (Giancola, 2002a, 2002b). The next estimate, also based on past studies from our laboratory (Giancola, 2002a, 2002b), was that the b value for the 4-way interaction term would be around .10 ("small" effect size). Based on these data, we conservatively chose a b value of .10. On the basis of these parameters it was determined that approximately 300 subjects would provide adequate power to detect these effects (Cohen, 1992). In order to avoid confusion, it should be noted that our power calculation was based on the ability to detect a significant "medium" effect for the full model and a "small" effect for the 4-way interaction. Given that this is the first systematic program of research aimed at isolating risk factors for intoxicated aggression, we conservatively estimated a "small" effect for the 4-way interaction to ensure that we would not fail to detect a meaningful effect.

Chapter Three Results

Manipulation Checks

Aggression Task Checks. In order to verify the success of the 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. The deception manipulation appeared successful. Typical descriptions from participants about their opponents included profane derogatory remarks as well as statements that the person was “okay,” “a good competitor,” and “fast.” Some participants also indicated that they had “no feelings for this person either way.” The majority of participants stated that they did well on the task and thought that their opponent tried hard to win. All felt that the task was a good measure of reaction-time. Five individuals [2 women (1 placebo and 1 alcohol) and 3 men (2 placebo and 1 alcohol)] reported that they did not believe that they were competing against another person and were thus removed from the analyses. Otherwise, no suspicion was raised about the credibility of the task.

Placebo Checks. All participants in the placebo group indicated that they believed that they drank alcohol. In response to the question regarding how drunk they felt, persons in the alcohol group reported average pre- and post-task ratings of 4.5 and 5.0 (scale range: 0 to 11). The placebo group reported average ratings of 1.7 and 2.0, respectively, [pre-task ratings: $t(308) = -15.6, p < .05$; post-task ratings: $t(308) = -14.2, p < .05$]. In response to the question about whether the alcohol they drank caused any impairment, persons in the alcohol group reported an average rating of 5.8 and those in the placebo group reported an average rating of 2.0, $t(308) = -16.3, p < .01$, (scale range: 0 to 10). There were no significant Gender or Gender X Beverage group effects for any of the placebo check measures.

BAC Levels. All participants tested in this study had BACs of 0% upon entering the laboratory. Individuals in the alcohol group had a mean BAC of 0.098% ($SD = 0.016$) just before beginning the aggression task and a mean BAC of 0.104% ($SD = 0.019$) immediately after the task. Persons given the placebo had a mean BAC of 0.01% ($SD = 0.008$) just before the task and a mean BAC of 0.01% ($SD = 0.016$) immediately after the task. There were no significant Gender or Gender X Beverage group effects for any of the BAC measures. Persons in the alcohol group had a mean BAC of 0.11% approximately 10 minutes after the last BAC assessment indicating that they were on the ascending limb of the BAC curve during the aggression task. BACs peaked around 0.11% to 0.12%.

Demographic Data

In order to test for unexpected group differences, all demographic variables were analyzed using 2 (beverage) X 2 (gender) between-groups design analyses of variance (ANOVA). There were no such differences. However, compared with women, men began drinking regularly at an earlier age, $F(1, 308) = 5.91, p < .05$, were first intoxicated at an earlier age, $F(1, 308) = 5.43, p < .05$, consumed more drinks per occasion, $F(1, 308) = 67.08, p < .05$, had more drinking occasions per week, $F(1, 308) = 18.94, p < .05$, consumed more alcoholic drinks per week, $F(1, 308) = 60.14, p < .05$, had higher SMAST scores, $F(1, 309) = 37.68, p < .05$. However, men and women did not differ with regard to age, years of education, salary, age at first drink, and EF. These data are

presented in Table 1.

Mediation Analyses

One purpose of this investigation was to determine whether irritability would mediate the relation between EF and alcohol-related aggression. According to Baron and Kenny (1986) mediation is tested by regressing the proposed mediator (irritability) onto the independent variable (EF), the dependent variable (aggression) onto the independent variable, and finally the dependent variable onto both the independent variable and the proposed mediator. If the explanatory value of the independent variable is reduced with the inclusion of the mediator in the model then mediation has occurred. To assess if this mediation was conditional upon beverage group and/or gender the analysis was carried out separately for the four experimental groups (male/alcohol, male/placebo, female/alcohol, female/placebo). Since the TAP yields three measures of aggression (first trial shock intensity, mean shock intensity, and extreme aggression) the mediational analyses were carried out separately for each measure of aggression.

The above conditions for mediation were satisfied for male/alcohol participants only for mean shock intensity and extreme aggression. According to Baron and Kenny (1986) full mediation occurred for the model utilizing mean shock intensity as the dependent variable; while partial mediation occurred for the model with extreme aggression as the dependent variable. As can be seen in Table 2, the β estimates for the relations between the EF and the dependent variables were significant for each model. To test the potential mediating role of irritability, these models were then re-calculated with the addition of the irritability variable (Step 2). As hypothesized, the final β estimates for the relations between EF and the dependent variables were no longer significant when irritability was included in the model, the β estimates for the relations between irritability and the dependent variables were statistically significant, therefore demonstrating a mediating role for irritability. Results indicated a 40% reduction in the relation between EF and mean shock intensity. However, in the model utilizing extreme aggression as the dependent variable, the relation between EF and extreme aggression was reduced slightly, but not to non-significance indicating that irritability partially mediated the relation between EF and extreme aggression. Results indicated a 25% reduction in the relation between EF and extreme aggression when including irritability in the model.

A drop in significance, or the lack of, does not always guarantee that mediation has occurred. It was therefore important to utilize a *post-hoc* test of the significance for both of the above mediational analyses (Holmbeck, 2002).

Several tests exist to assess the significance of mediation effects such as the normal theory approach (Sobel, 1986), structural equation modeling (Holmbeck, 1997), and bootstrapping (Preacher & Hayes, 2004). Sobel's test is a commonly used and powerful means of testing mediation, however, it relies on the assumption of a normally distributed large data set (Fritz & MacKinnon, 2007). Percentile bootstrapping was chosen for this analysis for its simplicity and power in detecting a significant effect by generating a confidence interval around a series of resamplings of the dataset. If the confidence interval does not contain zero then the hypothesis that mediation has occurred gains support (Preacher & Hayes, 2004). Sobel's test as well as percentile bootstrapping were employed to assess the significance of the mediational effect. For the test utilizing mean shock intensity as the dependent variable, the results for Sobel's test were

significant, with 95% confidence intervals between 0.0108 - 0.1525. Using 1,000 bootstrap resamples of the data the results were also significant, with 95% confidence intervals between 0.0174 – 0.1484. As noted above, inasmuch as these confidence intervals do not contain zero, the mediation effect is considered to be significant. For the test utilizing extreme aggression as the dependent variable, the results for Sobel’s test was nearly significant, with 95% confidence intervals between -0.0008 - 0.0128. Using 1,000 bootstrap resamples of the data the results were significant, with 95% confidence intervals between 0.0001 – 0.0131. Because the results are based on a large number of resamplings of the dataset confidence interval and critical values are subject to slight change (within 10,000ths of a point) between calculations of the test, therefore results should be viewed with slight caution.

Moderation Analyses

The second purpose of the analyses was to test if EF and irritability had conjoint effects above and beyond one another for predicting intoxicated aggression. First, values for the EF and irritability variables were converted to *z*-scores. The categorical variables of gender and beverage were dummy coded and all possible interaction terms amongst all of the variables were created. Moderation analyses were carried out according to the methods put forth by Aiken and West (1991). For each of the models all variables were entered hierarchically with main effects in the first step, 2-way effects in the second step, 3-way effects in the third step, and the 4-way interaction in the fourth step.

Because the dependent variable in this study was a repeated measure (low and high provocation conditions), the use of standard regression techniques was not possible. To allow for testing interactions involving provocation, the sum/difference regression technique, which allows for the ability to test interaction terms involving repeated-measure variables was used (Hope, 1975; Judd, Kenny, McClelland, 2001). This method is the regression equivalent of a between-within, or mixed model ANOVA. To complete this analysis, two new dependent variables were created. The first, DV1, was the sum of low and high provocation responses (DV1 = low provocation + high provocation), and the second, DV2, was the difference between the two response conditions (DV2 = low provocation – high provocation). The regression model was then computed for both DVs. Coefficients for DV1 represent the “between” effects, and coefficients for DV2 represent all the “within” effects for provocation (Stephen West, personal communication, 2002).

The above analyses were carried out for the following DVs: 1st trial shock intensity, mean shock intensity, and extreme aggression. None of the results yielded significant interaction effects. Significant main effects were found for the following dependent variables: for (1st trial shock intensity), irritability ($\beta = .292, p < .01$), gender ($\beta = .244, p < .001$); for (mean shock intensity), EF ($\beta = .149, p < .01$), irritability ($\beta = .408, p < .001$), beverage ($\beta = .134, p < .05$), gender ($\beta = .189, p < .001$); and for (extreme aggression), EF ($\beta = .181, p < .01$), irritability ($\beta = .329, p < .001$), beverage ($\beta = .192, p < .001$), and gender ($\beta = .349, p < .01$). Despite the previous findings that EF (Giancola, 2004) and irritability (Giancola, 2002) moderated the alcohol-aggression relation on their own; no support was found to suggest that they confer an additional interactive risk above and beyond their interactive effects with beverage.

Ancillary Analyses

It was suggested that the Vocabulary subtest of the WAIS-III is general proxy for overall IQ and thus a possible predictor of intoxicated aggression, similar to EF. We went

about testing this possibility in two ways. First, we re-computed the above moderation model, substituting the vocabulary score for EF. Results indicated that only the following main effects were significant for (mean shock intensity): Beverage ($\beta = .159, p < .001$), Gender ($\beta = .211, p < .001$), Irritability ($\beta = .299, p < .001$), and Vocabulary ($\beta = -.245, p < .001$); (1st trial shock intensity): Beverage ($\beta = .114, p < .05$), Gender ($\beta = .297, p < .001$), Irritability ($\beta = .168, p < .05$), and Vocabulary ($\beta = -.234, p < .001$); and (extreme aggression): Beverage ($\beta = .225, p < .001$), Gender ($\beta = .351, p < .001$), Irritability ($\beta = .206, p < .001$), and Vocabulary ($\beta = -.181, p < .001$). Our second analysis consisted of a two-step regression model in which EF was entered in the first step ($\beta = .092, p < .001$). We then added Vocabulary in the 2nd step and found that both variables were significant in the final model [(EF; $\beta = .061, p < .05$) (Vocabulary; $\beta = -.310, p < .05$)]. The above analyses were significant for average shock intensity but not for 1st trial or extreme aggression.

It was also suggested that it would be useful to assess the correlations between 1st trial shock intensity (unprovoked aggression) and overall aggression (shock intensity) under low and high provocation conditions. Results indicated that the correlations between unprovoked aggression and shock intensity under low and high provocation were correlated at $r = 0.77$ and $r = .44$, respectively (p 's $< .05$). Furthermore the relation between aggression under low and high provocation was correlated at $r = 0.63, p < .05$. See Table 3 for the correlation matrix.

Table 3.1. Demographic Data.

| Measure | Men | | Women | |
|---------------------------------|----------|-----------|----------|-----------|
| | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |
| Age | 23.34 | 3.07 | 22.72 | 2.58 |
| Years of Education | 15.92 | 2.02 | 16.31 | 2.00 |
| Salary | \$18.86K | \$12.72K | \$17.80K | \$9.80K |
| Executive Functioning† | 0.00 | 1.00 | -0.01 | 1.00 |
| SMAST | 1.93 | 2.85 | 0.36 | 1.46* |
| Age at first drink | 15.22 | 2.61 | 15.70 | 2.50 |
| Age when first drunk | 15.90 | 2.45 | 16.57 | 2.51* |
| Age when regular drinking began | 18.48 | 2.32 | 19.11 | 2.17* |
| Drinking occasions per week | 2.36 | 1.37 | 1.73 | 1.18* |
| Drinks per occasion | 6.30 | 3.48 | 3.75 | 1.67* |
| Drinks per week | 15.75 | 12.91 | 6.78 | 6.35* |

Note. *SMAST = Short Michigan Alcoholism Screening Test*

K = \$1,000

† data represented as *z*-scores.

* $p < .05$.

Table 3.2. Regression Equations Assessing the Mediating Role of Irritability for the Relation Between EF and Aggression in Intoxicated Males.

| Step and Measure | β | p-value |
|--|---------------------------|----------------|
| Mean Shock Intensity | | |
| Step 1: EF | .320 | .007 |
| Step 2: EF | .175 | .ns |
| Irritability | .382 | .001 |
| $F(2,75) = 10.957, p = .000; R^2 = 0.219$ | | |
| Extreme Aggression | | |
| Step 1: EF | .319 | .004 |
| Step 2: EF | .241 | .031 |
| Irritability | .246 | .028 |
| $F(2,75) = 7.233, p = .001; R^2 = 0.156$ | | |
| Note: EF = Executive Cognitive Functioning | | |

Table 3.3. Pearson-Product Moment Correlations Between the TAP Aggression Variables and Provocation.

| Measure | 1 | 2 | 3 | 4 |
|--|----------|----------|----------|----------|
| 1) 1 st trial Shock Intensity | | 0.768 | 0.444 | 0.606 |
| 2) Low Provocation | | | 0.631 | 0.863 |
| 3) High Provocation | | | | 0.934 |
| 4) Mean Shock Intensity | | | | |

Note: All correlations significant at the $p < .01$ level.

Chapter Four Discussion

The results of this investigation partially supported the hypotheses. Specifically, irritability mediated the relation between EF and aggression, but only for intoxicated males. Specifically, the introduction of irritability into the relation between EF and intoxicated aggression reduced the association between EF and aggression by 40%. As expected, this mediation effect did not occur in sober men because they likely did not experience the cognitive dysregulation and subsequent increase in negative affect associated with intoxication. Moreover, the mediation effect did not occur in either sober or intoxicated women. It has been suggested that women do not exhibit physical aggression to the same degree as men due to the possibility that they might have a greater liability threshold to exhibit such behavior. In other words, compared with males, females may require a greater degree of biological and/or environmental vulnerability in order to display aggression (Cloninger, Reich, & Guze, 1975; Cloninger, Christiansen, Reich, & Gottesman, 1978). Clearly, further examination of the ways in which females experience and engage in aggressive behavior is warranted.

With regard to the moderation analyses, previous research has demonstrated that both irritability and EF independently moderate the alcohol-aggression relation (Giancola, 2002; 2004). However, the hypothesis that EF and irritability would have an interactive effect above and beyond their constituent interactions was not supported. This may be due to the possibility that their relation is best described as a mediational one in which alcohol-induced higher-order cognitive dysregulation gives rise to irritability and ultimately aggressive behavior rather than the two variables interacting in a conjoint fashion.

The possibility that the Vocabulary subtest of the WAIS-III could be a proxy for general cognitive ability was also tested. We wanted to determine whether the vocabulary subtest score would predict aggressive behavior as well as EF. The rationale behind this analysis was to determine whether a simpler single test would predict aggression better or to the same degree as the larger EF variable. As can be seen in the results section, in order to carry this out, the moderation analysis was recalculated substituting the vocabulary score for the EF variable. A main effect for vocabulary was found, however there were no significant higher-order interactions with beverage as was detected in another study using EF (Giancola, 2004). We then conducted another analysis in which a two-step regression model was tested with EF predicting aggression in the first step and EF and vocabulary predicting aggression in the second step. Results indicated that EF was a significant predictor in the 1st step ($\beta = .09$). The addition of vocabulary in the 2nd step ($\beta = -.31$) reduced the magnitude of EF from $\beta = .09$ to $\beta = .06$, however both variables (EF & Vocabulary) were significant in the final model. These results indicate that vocabulary and EF contribute unique and significant portions of variance in explaining aggression, however the impact of vocabulary is clearly stronger. Other studies that have controlled for vocabulary when assessing the relation between EF and anti-social behavior have found that both variables predict significant portions of variance as well (Lynam, Moffitt, & Stouthamer-Loeber, 1993; Seguin, Assaad, Negin, & Tremblay, 2004; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999). However, an examination of the pertinent literature shows that very few studies have assessed the simultaneous effects of EF and vocabulary. No study other than this investigation has

actually compared the magnitude of both variables simultaneously. The few studies that have measured EF and vocabulary (Lynam, Moffitt, & Stouthamer-Loeber, 1993; Seguin, Assaad, Negin, & Tremblay, 2004; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999) have shown that both variables explain significant and unique variance in antisocial behavior; however, an examination of their statistical procedures does not allow for a comparison of the magnitude of these effects. Nigg (2001) notes that controlling for IQ and its components is notoriously absent from studies where it should be considered. It is therefore particularly important to control for IQ related variables such as vocabulary because such tests most likely measure non-executive components that are necessary for executive functioning (Pennington et al., 1996).

Despite some of the negative findings noted above, the results of the present investigation are in keeping with the theoretical formulations described in the Introduction section where it was postulated that the cognitive skills subsumed under the EF rubric are involved in regulating irritability (Luria, 1961; 1980; Moffitt, 1993; Tarter et al., 1985). Specifically, when EF fails to function normally, and alcohol is introduced into the system, an individual's capacity for processing information from the environment and properly regulating behavior becomes impaired. This leads to deficits in the capacity to adeptly carry out and realize goal-directed behaviors. It is a well-established finding that alcohol disrupts cognitive functioning, particularly EF (Curin & Fairchild, 2003; Finn, Justus, Mazas, & Steinmetz, 1999; Hoaken, Assaad, & Pihl, 1998; Lyvers, & Maltzman, 1991; Peterson, Rothfliesch, Zelazo, & Pihl, 1990). With this comes an inability to adequately attend to information from the environment, process that information, and formulate new plans based on that information to achieve one's goals (Steele, & Josephs, 1990). As this process deteriorates and the inability to achieve one's goals continues, one is likely to experience greater and greater levels of negative affect, particularly, irritability (Berkowitz, 1993). It is through this experience of irritability that the relation between EF and alcohol-related aggression is mediated.

In addition to this, our findings also bolster several theoretical formulations of the relation between EF, negative affect, and aggression. Moffitt's (1993) theory states that neuropsychological deficits, particularly EF deficits, underlie a dysregulation of temperament (which includes irritability) that can predispose toward aggressive behavior. Similarly, Luria (1980) concluded that the cognitive regulation of affect and behavior is governed predominantly by the prefrontal cortex; the primary neural substrate for EF. Parenthetically, others have demonstrated empirically that EF plays a major role in the regulation and expression of temperament (which again, includes irritability) on intoxicated aggression (Giancola, Parrott, & Roth, 2006).

Following Luria, Tarter and colleagues (Tarter et al., 1985, Tarter, 1988) later put forth a similar theoretical stance to explain the neurobehavioral underpinnings of alcoholism. Specifically, Tarter's model implicated childhood difficult temperament, which may manifest as irritability, as a risk factor for alcoholism as well as antisocial and aggressive behavior (Giancola & Tarter, 1999). These theoretical formulations, as well as the results of the present investigation, are consistent with studies showing that patients with acquired lesions to the prefrontal cortex often show a symptom complex consisting of cognitive and behavioral inflexibility, impulsivity, irritability, emotional dysregulation, as well as heightened aggression (McAllister, 1992; Tateno, Jorge, & Robinson, 2003).

Acute alcohol intoxication acts as a proxy for symptoms similar to those seen in

persons with mild damage to the prefrontal cortex such as poor inhibition, attention, planning, and affect regulation. The pharmacological disinhibition model of alcohol-related aggression posits that alcohol affects brain regions responsible for the inhibitory control of behavior and that this allows for the expression of aggressive behaviors that are typically inhibited in persons with intact cognitive functioning (Chermack & Giancola, 1997). Within this model, alcohol leads to aggression by disrupting cognitive processes causing individuals to be less able to attend to situational cues and less aware of the consequences of their actions (Steele & Josephs, 1990; Zeichner & Pihl, 1979). Intoxicated individuals are also less capable of regulating their emotional responses and tend to react to the most salient and noxious cues of provocation (Steele & Josephs, 1990). The cognitive dysregulation brought on by impaired EF, alcohol intoxication, or both together can lead to a heightened experience of negative affect; specifically, irritability. Consequently, aggression results from irritability directed at the source of provocation and an inability, due to cognitive dysregulation, to adequately deal with that affective response and thus properly regulate behavior.

This conception is consistent with Berkowitz's cognitive neoassociationistic model. According to Berkowitz (1993), aggression is the result of negative affect. The experience of negative affect is brought on by disruptions in cognitive functioning when an individual is unable to adequately attend to information from the environment, process that information, and formulate new plans based on that information to achieve one's goals (Steele & Josephs, 1990). As this process deteriorates and the inability to achieve one's goals continues, one is likely to experience greater levels of negative affect, particularly, irritability. This activates aggression-related memories, emotions, physiological responses, and motor patterns that form an associationistic network that predisposes one towards aggression. It is the experience of negative affect that leads to feelings of anger, frustration, and irritability; and this experience can ultimately lead to an aggressive response to provocation. Within Berkowitz's (1998) model, it is also possible for an individual to react with fear-related escape responses to provocation. However, when taking into account the effects of alcohol intoxication and lowered EF, individuals may be more strongly cued towards more salient and noxious irritable responses towards provocation because of their inability to adequately attend to, perceive, and cognitively process other peripheral inhibitory cues in the situation (see also Steele & Josephs, 1990). Berkowitz also suggests that the experience of irritability is not "free-floating" in this case, and that the individual experiencing irritability will attribute the source of that irritability to the provoking target and ultimately become openly hostile towards that target more so than less irritable individuals (Berkowitz, 1998).

Future Directions in Research

Given the mixed findings of this investigation and the complexity of alcohol-related aggression as understood from Berkowitz's (1993) cognitive neoassociationistic framework, several future directions for research warrant exploration. The most immediate, for explanatory purposes, is exploring the possibility that other forms of negative affect might mediate the relation between EF and alcohol-related aggression in a fashion similar to irritability. This would bolster support for a cognitive neoassociationistic model of aggression as well as support the idea that aggression is the result of a combined interplay involving affective and cognitive behavioral dysregulation (Caprara et al., 2007).

Berkowitz (1993) speaks a great deal on the importance of negative affect in relation to aggressive behavior. However, not all negative affective states are associated with aggressive behavior (i.e. depressed mood). It would be very telling to fractionate negative affect into a number of constituent parts and explore those that are most readily related to aggressive behavioral outcomes in an effort to better understand how varied negative experiences might lead to aggression. Further exploration of a cognitive neoassociationistic framework should also involve looking at “reactive” aggressors (see Crick & Dodge, 1996; Fontaine & Dodge, 2006). “Reactive” aggressors are those who experience heightened levels of irritability, have been shown to be particularly hostile towards a targeted provocateur, and exhibit limited inhibitory control over their behavior (Raine et al., 2006). According to Berkowitz (1993), a provocateur, such as one used in the TAP, is very proximal to the source of negative affect for the participant and therefore becomes the target of aggression. For a “reactive” aggressor, this association between targeted provocateur and negative affect is a powerful one, to the point that it may short-circuit any of the limited inhibitory control of the aggressor (Berkowitz, 2008). Characteristics of the aggressor, such as personality risk factors, cognitive regulatory capacities, mood states, and/or level of reactivity, have been of primary concern for aggression researchers for some time. However, it may prove useful to begin to understand the characteristics and qualities of targets, namely what causes some individuals to be more vulnerable to attack than others. Manipulation of target qualities may shed new light on aggression understood from a cognitive neoassociationistic framework.

References

- Abbey, A., Zawacki, T., & Buck, P. (2005). The effects of past assault perpetration and alcohol consumption on men's reactions to women's mixed signals. *Journal of Social and Clinical Psychology, 24*, 129-155.
- Aiken, L., & West, S. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park: Sage Publications
- Alderman, N. (2003). Contemporary approaches to the management of irritability and aggression following traumatic brain injury. *Neuropsychological Rehabilitation, 13*, 211-240.
- Alexander, M. & Stuss, D. (2006). Frontal injury: Impairment of fundamental processes lead to functional consequences. *Journal of the International Neuropsychological Society, 12*, 192-193.
- Anderson, C. (1997). Effects of violent movies and trait hostility on hostile feelings and aggressive thoughts. *Aggressive Behavior, 23*, 161-178.
- Baddeley, A.D. (1998). The central executive: A concept and some misconceptions. *Journal of the International Neuropsychological Society, 4*, 523-526.
- Baddeley, A.D. (1986) *Working Memory*. Oxford: Oxford University Press.
- Barnwell, S., Borders, A., & Earleywine, M. (2006). Alcohol-aggression expectancies and dispositional aggression moderate the relationship between alcohol consumption and alcohol-related violence. *Aggressive Behavior, 32*, 517-525.
- Berger, A. & Posner, M. (2000). Pathologies of brain attentional networks. *Neuroscience and Biobehavioral Reviews, 24*, 3-5.
- Berkowitz, L. (2008). On the consideration of automatic as well as controlled psychological processes in aggression. *Aggressive Behavior, 34*, 117-129.
- Berkowitz, L. (1998). Affective aggression: The role of stress, pain, and negative affect. In E. Donnerstein & R. Geen (Ed.) *Human aggression: Theories, research, and implications for social policy*. (pp. 49-72). San Diego: Academic Press. 309 pp.
- Berkowitz, L. (1993). More thoughts about the social cognitive and neoassociationistic approaches: Similarities and differences. In T. Srull & R. Wyer (Eds.) *Perspectives on anger and emotion*. (pp. 179-197). Hillsdale: Lawrence Erlbaum. 206 pp.
- Boyatzis, R. (1975). The predisposition toward alcohol-related interpersonal aggression in men. *Journal of Studies on Alcohol, 36*, 1196-1207.
- Bradlyn, A. & Young, L. (1983). Parameters influencing the effectiveness of the balanced placebo design in alcohol research. In: Pohorecky, L., Brick, J., (Eds.), *Stress and alcohol use*. Elsevier, New York, NY, pp. 87-103.
- Bushman, B., & Cooper, H. (1990). Effects of alcohol on human aggression: an integrative research review. *Psychological Bulletin, 107*, 341-354.
- Buss, A., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting and Clinical Psychology, 21*, 343-349.
- Caprara, G., Paciello, M., Gerbino, M., & Cugini, C. (2007). Individual differences conducive to aggression and violence: Trajectories and correlates of irritability and hostile rumination through adolescence. *Aggressive Behavior, 33*, 359-374.

- Caprara, G., Barbaranelli, C., & Zimbardo, P. (1996). Understanding the complexity of human aggression: Affective, cognitive, and social dimensions of individual differences in propensity toward aggression. *European Journal of Personality, 10*, 133-155.
- Caprara, G., Barbaranelli, C., & Comrey, A. (1992). A personological approach to the study of aggression. *Personality and Individual Differences, 13*, 77-84.
- Caprara, G., Renzi, P., D'Augello, G., D'Imperio, G., & Travaglia, G. (1986). Interpolating physical exercise between instigation to aggress and aggression: The role of irritability and emotional susceptibility. *Aggressive Behavior, 12*, 83-91.
- Caprara, G., Cinanni, V., D'Imperio, G., Passerini, S., Renzi, P., & Travaglia, G. (1985). Indicators of impulsive aggression: present status of research on irritability and emotional susceptibility scales. *Personality and Individual Differences, 6*, 665-674.
- Caprara, G., Renzi, P., Alcini, P., D'Imperio, G., & Travaglia, G. (1983). Instigation to aggress and escalation of aggression examined from a personological perspective: The role of irritability and emotional susceptibility. *Aggressive Behavior, 9*, 345-351.
- Cheong, J. & Nagoshi, C. (1999). Effects of sensation seeking, instruction set, and alcohol/placebo administration on aggressive behavior. *Alcohol, 17*, 81-86.
- Chermack, S. & Giancola, P.R. (1997). The relation between alcohol and aggression: An integrated biopsychosocial conceptualization. *Clinical Psychology Review, 17*, 621-649.
- Chermack, S., & Taylor, S. (1995). Alcohol and human physical aggression: Pharmacological versus expectancy effects. *Journal of Studies on Alcohol, 56*, 449-456.
- Crick, N., & Dodge, K. (1996). Social information processing mechanisms on reactive and proactive aggression. *Child Development, 67*, 993-1002.
- Crown, S. (1952). An experimental study of psychological changes following prefrontal lobotomy. *Journal of General Psychology, 47*, 3-41.
- Curtin, J. & Fairchild, B. (2003). Alcohol and cognitive control: Implications for regulation of behavior in response conflict. *Journal of Abnormal Psychology, 112*, 424-436.
- Degangi, G., Wietlisbach, S., Goodin, M., & Scheiner, N. (1993). A comparison of structured sensorimotor therapy and child-centered activity in the treatment of preschool children with sensorimotor problems. *American Journal of Occupational Therapy, 47*, 777-786.
- Duncan, J., Seitz, R., Kolodny, J., Bor, D., Herzog, H., Ahmed, A. (2007). A neural basis for general intelligence. *Science, 289*, 457-460.
- Easdon, C., Izenberg, A., Armilio, M., Yu, H., & ALian, C. (2005). Alcohol consumption impairs stimulus and error-related processing during a Go/No-Go task. *Cognitive Brain Research, 25*, 873-883.
- Evans, I., Heriot, S., & Friedman, A. (2002). A behavioral pattern of irritability, hostility, and inhibited empathy in children. *Clinical Child Psychology and Psychiatry, 7*, 211-224.

- Engle, R.W., Tuholski, S.W., Laughlin, J.E., & Conway, A.R. (1999). Working memory, short-term memory and general fluid intelligence: A latent variable approach. *Journal of Experimental Psychology*, *128*, 309-331.
- Fals-Stewart, (2003). The occurrence of partner physical aggression on days of alcohol consumption: A longitudinal diary study. *Journal of Consulting and Clinical Psychology*, *71*, 41-52.
- Faul, F. & Erdfelder, E. (1992). G-Power: A priori, post-hoc, and compromise power analyses for MS-DOS [computer program]. Bonn, FRG: Bonn University.
- Finn, P., Justus, A., Mazas, C., & Steinmetz, J. (1999). Working memory, executive processes and the effects of alcohol on Go/No-Go learning: Testing a model of behavioral regulation and impulsivity. *Psychopharmacology*, *16*, 465-472.
- Fontaine, R. & Dodge, K. (2006). Real-Time Decision Making and Aggressive Behavior in Youth: A Heuristic Model of Response Evaluation and Decision (RED). *Aggressive Behavior*, *32*, 604-624.
- Fritz, M. & MacKinnon, D. (2007). Required sample size to detect a mediated effect. *Psychological Science*, *18*, 233-239.
- Fuster, J. (1997). Network Memory. *Trends in Neuroscience*, *20*, 451-459.
- Giancola, P. R. (2004). Executive functioning and alcohol-related aggression. *Journal of Abnormal Psychology*, *113*(4), 541-555.
- Giancola, P.R. (2004a). Difficult temperament, acute alcohol intoxication, and aggressive behavior. *Drug and Alcohol Dependence*, *74*, 135-145.
- Giancola, P.R. (2003). The moderating effects of dispositional empathy on alcohol-related aggression in men and women. *Journal of Abnormal Psychology*, *11*, 275-281.
- Giancola, P.R. (2002). Alcohol-related aggression during the college years: Theories, risk factors, and policy implications. *Journal of Studies on Alcohol*, *suppl. 14*, 129-139.
- Giancola, P.R. (2002a). Irritability, acute alcohol consumption and aggressive behavior in men and women. *Drug and Alcohol Dependence*, *68*, 263-274.
- Giancola, P.R. (2002b). Alcohol-related aggression in men & women: The influence of dispositional aggressivity. *Journal of Studies on Alcohol*, *63*, 969-708.
- Giancola, P. R. (2000). Executive functioning: A conceptual framework for alcohol-related aggression. *Experimental and Clinical Psychopharmacology*, *8*, 576-597.
- Giancola, P.R. (1999). University of Kentucky Alcohol Research Laboratory Participant Screening Questionnaire. (unpublished manuscript).
- Giancola, P. R. (1995). Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behavior. *Aggressive Behavior*, *21*, 431-450.
- Giancola, P. & Chermack, S. (1998). Construct validity of laboratory aggression paradigms: a response to Tedeschi and Quigley 1996. *Aggression and Violent Behavior*, *3*, 237-253.
- Giancola, P. & Tarter, R. (1999). Executive cognitive functioning and risk for substance abuse. *Psychological Science*, *10*, 203-205.
- Giancola, P. & Zeichner, A., (1997). The biphasic effects of alcohol on human aggression. *J. Abnormal Psychology*, *106*, 598-607.

- Giancola, P. & Zeichner, A. (1995). Alcohol-related aggression in males and females: Effects of blood alcohol concentration, subjective intoxication, personality, and provocation. *Alcohol: Clinical and Experimental Research*, *19*, 130-134.
- Giancola, P.R., Mezzich, A., & Tartar, R. (1998). Executive cognitive functioning, temperament, and antisocial behavior in conduct disordered adolescent females. *Journal of Abnormal Psychology*, *107*, 629-641.
- Giancola, P., Parrott, D., & Roth, R. (2006). The influence of difficult temperament on alcohol-related aggression: Better accounted for by executive functioning. *Addictive Behavior*, *31*, 2169-2187.
- Goel, V., & Grafman, J. (1995). Are the frontal lobes implicated in “planning” functions? Interpreting data from the Tower of Hanoi. *Neuropsychologica*, *33*, 623-642.
- Gustafson, R., 1992. Alcohol and Aggression: A replication study controlling for potential confounding variables. *Aggressive Behavior*, *18*, 21-28.
- Hawkins, K. & Trobst, K. (2000). Frontal lobe dysfunction and aggression: conceptual issues and research findings. *Aggression and Violent Behavior: A Review Journal*, *5*, 147–157.
- Heaton, R. (1993). *Wisconsin Card Sorting Test: Computer version 2*. Odessa: Psychological Assessment Resources.
- Heyman, R., O’Leary, D., & Jouriles, E. (1995). Alcohol and aggressive personality styles: Potentiators of serious physical aggression against wives? *Journal of Family Psychology*, *9*, 44-57.
- Hoaken, P., Assaad, J., & Pihl, R. (1998). Cognitive functioning and the inhibition of alcohol-induced aggression. *Journal of Studies on Alcohol*, *59*, 599-607.
- Holmbeck, G. (2002). Post-hoc probing of significant moderational and mediation effects in studies of pediatric populations. *Journal of Pediatric Psychology*, *27*, 87-96.
- Holmbeck, G. (1997). Towards terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from child clinical and pediatric psychological literatures. *Journal of Consulting and Clinical Psychology*, *65*, 599-610.
- Hope, K., 1975. Models of status inconsistency and social mobility effects. *American Sociological Review*, *40*, 322-343.
- Hull, J., & Bond, C. (1986). Social and behavioral consequences of alcohol consumption and expectancy: A meta-analysis. *Psychological Bulletin*, *99*, 347-360.
- Ito, T., Miller, N., & Pollock, V. (1996). Alcohol and aggression: a meta-analysis of the moderating effects of inhibitory cues, triggering events, and self-focused attention. *Psychological Bulletin*, *120*, 60-82.
- Judd, C., Kenny, D., & McClelland, H. (2001). Estimating and testing mediation and moderation in within-subject designs. *Psychological Methods*, *6*, 115-134.
- Kelley, T., & Cherek, D. (1993). The effects of alcohol on free operant aggressive behavior. *Journal of Studies on Alcohol Supplement*, *11*, 40-52.
- Kimberg, D., & Farah, M. (1993). A unified account of cognitive impairments following frontal lobe damage: The role of working memory in complex, organized behavior. *Journal of Experimental Psychology: General*, *122*, 411-428.

- Lau, M., Pihl, R., & Peterson, J. (1995). Provocation, acute alcohol intoxication, cognitive performance, and aggression. *Journal of Abnormal Psychology, 104*, 150-155.
- Lehto, J., Juujarvi, P., Kooistra, L., & Pulkkinen, L. (2003). Dimensions of executive functioning: Evidence from children. *British Journal of Developmental Psychology, 21*, 59-80.
- Leonard, K. & Senchak, M. (1993). Alcohol and premarital aggression among newlywed couples. *Journal of Studies on Alcohol. suppl. 11*, 96-108.
- Leonard, K., Collins, R., & Quigley, B. (2003). Alcohol consumption and the occurrence and severity of aggression: An event based analysis of male to male barroom violence. *Aggressive Behavior, 29*, 346-365.
- Luria, A. (1980). *Higher Cortical Functions in Man*. New York: Basic Books.
- Luria, A. (1961). *The role of speech in the regulation of normal and abnormal behavior*. New York: Basic Books.
- Lynam, D., Moffitt, T., & Stouthamer-Loeber, M. (1993). Explaining the relation between IQ and delinquency: Class, race, test motivation, school failure, or self-control? *Journal of Abnormal Psychology, 102*, 187-196.
- Lyvers, M., & Maltzman, I. (1991). Selective effects of alcohol on Wisconsin Card Sorting Test performance. *British Journal of Addiction, 86*, 399-407.
- MacDonald, T., Fong, G., Zanna, M., & Martineau, A. (2000). Alcohol myopia and condom use: Can alcohol intoxication be associated with more prudent behavior? *Journal of Personality and Social Psychology, 78*, 605-619.
- MacLeod, C. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin, 109*, 163-203.
- Martin, C., Earleywine, M., Finn, P., & Young, R., (1990). Some boundary conditions for effective use of alcohol placebos. *Journal of Studies Alcohol, 51*, 500-505.
- Martin, C. & Sayette, M. (1993). Experimental design in alcohol administration research: Limitations and alternatives in the manipulation of dosage-set. *Journal of Studies on Alcohol 54*, 750-761.
- Maziade, M., Caperaa, P., Leplante, B., Thivierge, J., Boudreault, M., Cote, R., & Bouten, P. (1985). Value of difficult temperament among 7-year-olds in the general population for predicting psychiatric diagnosis at age 12. *American Journal of Psychiatry, 142*, 943-946.
- McAllister, T. (1992). Neuropsychiatric sequelae of head injuries. *Psychiatric Clinics of North America, 15*, 395-413.
- Miller, T., Levy, D., Cohen, M., & Cox, K. (2006). Costs of alcohol and drug-involved crime. *Prevention Science, 7*, 333-342.
- Milner, B. (1995). Aspects of frontal lobe function. In H. Jasper, S. Riggio, & P. Goldman-Rakic (Eds.), *Epilepsy and the functional anatomy of the frontal lobe* (pp. 67-84). New York: Raven Press.
- Milner, B., & Petrides, M. (1984). Behavioral effects of frontal lobe lesions in man. *Trends in Neuroscience, 7*, 403-407.
- Miyake, A., & Shah, P. (1999). *Models of Working Memory: Mechanisms of active maintenance and executive control*. Cambridge, UK: Cambridge University Press.

- Miyake, A., Friedman, N., Emerson, M., Witzki, A., & Howerter, A. (2000). The unity and diversity of executive functions and their contribution to “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*, *41*, 49-100.
- Moffitt, T. E. (1993). The neuropsychology of conduct disorder. *Development and Psychopathology*, *5*, 133–151.
- Moffitt, T., & Henry, B. (1989). Neuropsychological assessment of executive functions in self-reported delinquents. *Development and Psychopathology*, *1*, 105–118.
- Moffitt, T. E., Lyman, D. R., & Sylva, P. A. (1994). Neuropsychological tests predicting persistent male delinquency. *Criminology*, *2*, 277–300.
- Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, *20*, 113–136.
- Moscovitch, M. & Umiltà, C. (1990). Modularity and neuropsychology: Modules and central processes in attention and memory. In M. Schwartz (Ed.) *Modular deficits in Alzheimer-type dementia* (pp. 1-59). Cambridge: MIT Press.
- Murphy, C.M., Winters, J., O’Farrell, T.J., Fals-Stewart, W., & Murphy, M. (2005). Alcohol consumption and intimate partner violence by alcoholic men: Comparing violent and non-violent conflicts. *Psychology of Addictive Behavior*, *19*, 35-42.
- Newman, J., & Kosson, D. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology*, *95*, 252-256.
- Nigg, J. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin*, *127*, 571-598.
- O’Farrell, T., Fals-Stewart, W., Murphy, M., & Murphy, C. (2003). Partner violence before and after individually based treatment for male alcoholic patients. *Journal of Consulting and Clinical Psychology*, *72*, 92-102.
- Parkin, A. (1998). The central executive does not exist. *Journal of the International Neuropsychological Society*, *4*, 518-522.
- Parrott, D. & Giancola, P.R. (2004). A further examination of the relation between trait anger and alcohol-related aggression: The role of anger control. *Alcoholism: Clinical and Experimental Research*, *28*, 855-864.
- Parrott, D. & Zeichner, A. (2002). Effects of alcohol and trait anger on physical aggression in men. *Journal of Studies on Alcohol*, *63*, 196-204.
- Paschall, M., & Fishbein, D. (2002). Executive functioning and aggression: a public health perspective. *Aggression and Violent Behavior*, *7*, 215-235.
- Pennington, B. F., Bennetto, L., McAleer, O., & Roberts, R. J. (1996). Executive functions and working memory: Theoretical and measurement issues. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 327–348). Baltimore: Brookes.
- Perecman, E. (1987). Consciousness and the meta-functions of the frontal lobes: Setting the stage. IN E. Parnanen (Ed.) *The frontal lobes revisited* (pp. 1- 10). Hillsdale: Earlbaum.
- Parnanen, K. (1991). *Alcohol in Human Violence*. New York: Guilford Press.
- Perret, E. (1974). The left frontal lobe of man and the suppression of habitual responses in verbal categorical behavior. *Neuropsychologica*, *12*, 323-330.
- Peterson, J., Rothfleisch, J., Zelazo, P., & Pihl, R. (1990). Acute alcohol intoxication and cognitive functioning. *Journal of Studies on Alcohol*, *51*, 114-122.

- Petrides, M. (1985). Deficits on conditional associative learning tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologica*, *23*, 601-614.
- Pihl, R., Smith, M., & Farrell, B. (1984). Alcohol and aggression in men: A comparison of brewed and distilled beverages. *Journal of Studies Alcohol*, *45*, 278-282.
- Pihl, R., Zacchia, C., & Zeichner, A. (1981). Follow-up analysis of the use of deception and aversive contingencies in psychological research. *Psychological Reports*, *48*, 927-930.
- Porteus, S. (1965). *Porteus Maze Test: Fifty year's application*. Paolo Alto: Pacific Books.
- Porteus, S., & Kepner, R. (1944). Mental changes after bilateral prefrontal lobotomy. *Genetic Psychological Monographs*, *29*, 3-115.
- Preacher, K. & Hayes, A. (2004). SPSS and SAS procedures for assessing indirect effects in simple mediation models. *Behavioral Research Methods, Instruments, and Computers*, *36*, 717-731.
- Quigley, B. & Leonard, K. (1999). Husband alcohol expectancies, drinking, and marital-conflict styles as predictors of severe marital violence among newlywed couples. *Psychology of Addictive Behaviors*, *13*, 49-59.
- Quigley, B., Corbett, A., & Tedeschi, J. (2002). Desired image of power, alcohol expectancies and alcohol-related aggression. *Psychology of Addictive Behavior*, *16*, 318-324.
- Raine, A., Dodge, K., Loeber, R., Gatzke-Kopp, L., Lynam, D., Reynolds, C., Stouthamer-Loeber, M., Liu, J. (2006). The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*, *32*, 159-171.
- Raskin-White, H., Hansell, S., & Brick, J. (1993). Alcohol use and aggression among youth. *Alcohol Health and Research World*, *17*, 144-150.
- Raskin-White, H. & Spatz-Widom, C. (2003). Intimate partner violence among abused and neglected children in young adulthood: The mediating effects of early aggression, antisocial personality, hostility, and alcohol problems. *Aggressive Behavior*, *29*, 332-345.
- Reitan, R. (1992). *Trail Making Test: Manual for administration and scoring*. Tuscon: Reitan Neuropsychological Laboratory.
- Seguin, J., Assaad, J., Nagin, D., Tremblay, R. (2004). Cognitive-neuropsychological function in chronic physical aggression and hyperactivity. *Journal of Abnormal Psychology*, *112*, 603-613.
- Seguin, J., Boulerice, B., Harden, P., Tremblay, R., & Pihl, R. (1999). Executive function and physical aggression after controlling for Attention Deficit Hyperactivity Disorder, general memory, and IQ. *Journal of Child Psychology and Psychiatry*, *40*, 1197-1208.
- Selzer, M., Vinokur, A., & van Rooijen, L. (1975). A self-administered Short Michigan Alcoholism Screening Test (SMAST). *Journal of Studies on Alcohol*, *36*, 117-126.
- Sobel, M. (1986). Some new results on indirect effects and their standard errors in covariance structure models. In N. Tuma (Ed.), *Sociological Methodology 1986* (pp. 159-186). Washington, DC: American Sociological Association.

- Steele, C., & Josephs, R. (1990). Alcohol myopia: Its prized and dangerous effects. *American Psychologist, 45*, 921-933.
- Steele, C., & Southwick, L. (1985). Alcohol and social behavior I: The psychology of drunken excess. *Journal of Personality and Social Psychology, 48*, 18-34.
- Stuss, D., & Alexander, M. (2000). Executive functions and the frontal lobes: A conceptual framework. *Psychological Research, 63*, 289-298.
- Stuss, D., & Levine, B. (2002). Adult clinical neuropsychology: Lessons from studies of the frontal lobe. *Annual Review of Psychology, 53*, 401-433.
- Tarter, R. (1988). Are there inherited behavioral traits that predispose to substance abuse. *Journal of Consulting and Clinical Psychology, 56*, 189-196.
- Tarter, R., Alterman, A., & Edwards, K. (1985). Vulnerability to alcoholism in men: A behavior-genetic perspective. *Journal of Studies on Alcohol, 46*, 329-356.
- Tateno, A. Jorge, R., Robinson, R. (2003). Clinical correlates of aggressive behavior after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neuroscience, 15*, 155-160.
- Taylor, S. (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression.
- Taylor, S., & Chermack, S. (1993). Alcohol, drugs, and human physical aggression. *Journal of Studies on Alcohol Supplement, 11*, 78-88.
- Testa, M. (2002). The impact of men's alcohol consumption on perpetration of sexual aggression. *Clinical Psychology Review, 22*, 1239-1263.
- United States Department of Justice (2005). *Bureau of Justice Statistics: Crime Characteristics*. Retrieved June 26, 2007, from http://www.ojp.usdoj.gov/bjs/cvict_c.htm#alcohol
- Volavka, J. (1995). *Neurobiology of violence*. Washington, D.C.: American Psychiatry Press.
- Zelazo, P. Carter, A., Reznick, S., & Frye, D. (1997). Early development of executive function: A problem-solving framework. *Review of General Psychology, 1*, 198-226.
- Zhang, L. Welte, J., & Wiczorek, W. (2002). The role of aggression-related alcohol expectancies in explaining the link between alcohol and violent behavior. *Substance Use and Misuse, 37*, 457-471.
- Zhang, L. Welte, J., & Wiczorek, W. (1999). The influence of parental drinking and closeness on adolescent drinking. *Journal of Studies on Alcohol, 60*, 245-251.
- Zhang, L, Wiczorek, W., & Welte, J. (1997). The nexus between alcohol and violent crime. *Alcoholism: Clinical and Experimental Research, 21*(7), 1264-1271.

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