

Executive Functioning, Irritability, and Alcohol-Related Aggression

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The purpose of this investigation was to examine (a) whether irritability mediates the relation between executive functioning (EF) and alcohol-related aggression and (b) whether the alcohol-aggression relation is better explained by the interactive effects of EF and irritability above and beyond the effects of either variable alone. EF was measured using seven well-established neuropsychological tests. Irritability was assessed with the *Caprara Irritability Scale*. Participants were 313 male and female social drinkers between 21 and 35 years of age. Following the consumption of an alcohol or a placebo beverage, participants were tested on a laboratory aggression task in which electric shocks were given to and received from a fictitious opponent under the guise of a competitive reaction-time task. Aggression was operationalized as the shock intensities administered to the fictitious opponent. Results indicated that irritability successfully mediated the relation between EF and intoxicated aggression for men only. Despite the fact that irritability and EF both independently moderated the alcohol-aggression relation in previous studies, no significant interaction for their combined effect was detected here. The findings are discussed, in part, within a cognitive neoassociationistic framework for aggressive behavior.

Keywords: alcohol, aggression, irritability, executive functioning

A number of meta-analytic (e.g., Bushman & Cooper, 1990; Hull & Bond, 1986; Ito, Miller, & Pollock, 1996) and narrative (e.g., Begue & Subra, 2007; Chermack & Giancola, 1997) reviews have documented a substantial relation between alcohol intoxication and aggressive behavior. National crime statistics have found alcohol consumption to be implicated in 55% to 60% of violent crimes (U.S. Department of Justice, 2005). Among those victims who provided information about their offender's perceived use of alcohol, approximately 30% reported that the offender had been drinking. With regard to intimate partner violence, 75% of victims reported having been attacked by an offender who was drinking (U.S. Department of Justice, 2005).

A well-accepted finding within this literature is that alcohol increases aggression for some, but not for all, individuals. In other words, some persons are at greater risk for becoming aggressive when intoxicated than others. Two risk factors of interest with regard to this article are executive functioning (EF; Giancola, 2004) and irritability (Giancola, 2002a). Both variables have been found to independently moderate the alcohol–aggression relation. Specifically, alcohol was more likely to increase aggression in men with lower levels of EF (Giancola, 2004) and higher levels of irritability (Giancola, 2002a). However, the combined effect on this association has not been examined. Such inquiry is important for at least two reasons. First, on both empirical and theoretical grounds, EF is purported to regulate affect and thus, by extension,

to predict irritability. Accordingly, a case will be made below that irritability may, in part, mediate the relation between EF and alcohol-related aggression. Second, examining the conjoint effects of these variables (i.e., EF and irritability) might confer a liability for intoxicated aggression that is not observed when testing either variable alone.

Executive Functioning

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

The structure of the EF construct has been a point of contention with theorists for some time. The literature on the nature, structure, and even definition of EF is highly mixed. Regarding the structure of EF, some scholars have advanced a relatively unified view (Duncan et al., 2000; Zelazo, Carter, Reznick, & Frye, 1997), others have proposed a more fractionated component-processes view (Baddeley & Logie, 1999; Lehto, Juujarvi, Kooistra, & Pulkkinen, 2003), and others suggest that both views are defensible (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). These differing viewpoints are primarily attributable to the statistical methods used to derive these EF structures (i.e., factor analytic techniques). Some analyses have found that a broad array of diverse EF tests can be represented as a unitary construct (Giancola, 2004; Giancola, Mezzich, & Tarter, 1998; Giancola, Tarter, Martin, Pelham, & Moss, 1996), while others have found that other EF tests can be fractionated it into separate components (Friedman

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et al., 2007, 2006; Miyake et al., 2000). However, the results of these latter studies yielded inter-factor correlations that were significantly related. Specifically, "inhibition" was significantly respectively correlated with "updating" and "set-shifting" at $r = .62$ and $r = .64$ (Friedman et al., 2006) and at $r = .75$ and $r = .74$ (Friedman et al., 2007). These results are in keeping with those of Miyake et al. (2000), who clearly acknowledged 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.

Mixed findings for the structure of EF tests is not surprising for a number of conceptual and methodological reasons. First, the construct of EF is highly underspecified. Much as the so-called constructs of "impulsivity" and "disinhibition," EF has taken on an almost mystical quality with nebulous boundaries (Perecman, 1987). This is exemplified by the myriad of related yet nonspecific definitions of this construct that abound (reviewed in Giancola, 2000). A logical corollary of this conceptual confusion is that any attempt to operationalize this construct will be a very difficult and arduous process that is unlikely to result in a universally accepted standardized EF test battery. The construct is simply too rich and complex to fit such artificial boundaries (Perecman, 1987). Second, what emerges from a factor analysis is greatly influenced by what goes into one; moreover, factor analysis must also be guided by clear research questions and theory so that the results can be meaningfully interpreted (Bandalos & Boehm-Kaufman, 2008; Mulaik, 1972; Gosuch, 1983).

Giancola's as well as Miyake and Friedman's work reviewed above clearly adhered to such methodological principals. Giancola's work viewed EF as a very broad and highly interrelated construct; understanding that the highly interactive neural circuitry of the brain makes it so that many EF skills are, by definition, intricately tied to one another and to even non-EF skills (Duncan, Duncan, & Hops, 1996; Duncan, Tildesley, Duncan, & Hops, 1995; Miyake et al., 2001; Stuss & Alexander, 2000). This position is borne out by the significant interfactor correlations seen in Miyake and Friedman's research. For these reasons, we selected a broad array of neuropsychological EF tests frequently used in clinical settings. On the other hand, Miyake and Friedman's research group was guided by the theoretical stance that EF can be fractionated into three components (i.e., inhibition, set-shifting, and updating, or in other words, working memory). As such, they selected more specific tests that satisfied their conceptual perspective. Obviously, these two theoretical approaches lead to different operationalizations of the same construct, which inevitably lead to different factor structures.

As such, we take the view that neither a unitary or a fractionated EF solution is "more correct" than the other, but that they simply represent different levels of analysis of the same construct. Results from factor analyses must not be blindly accepted as gospel based on arbitrary eigenvector cut-points (i.e., Kaiser rules) or a "selective" choice of ever-growing fit indices. Instead, their interpretation must be guided by an informed choice of statistical indicators and, yet even more importantly, a solid understanding of the theoretical and methodological issues surrounding one's construct of interest (Bandalos & Boehm-Kaufman, 2008; Mulaik, 1972; Gosuch, 1983).

Research on the nature, structure, and even definition of EF is clearly in its infancy and further work is obviously necessary.

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; Perecman, 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, 1999; Stuss & Alexander, 2005). Moreover, to date, there is no empirical or theoretical reason to suspect that any subcomponent of EF will be differentially related to aggression and any attempt to test such a relation would be purely atheoretical and most likely cloud, rather than clarify, the research literature in this area. As more is understood about the relation between the subcomponents of EF as well as their relation to various outcome variables, we may be in a much better position, both theoretically and empirically, to hypothesize more specific mechanisms.

Irritability

Irritability has been defined as the tendency to adopt a hostile attitude and act impulsively, conversely, or rudely to the slightest provocation and at the slightest disagreement (Caprara et al., 1985). Buss and Durkee (1957) described it in a similar fashion as "*a readiness to explode with negative affect at the slightest provocation, including quick temper, grouching, exasperation, and rudeness*" (p. 343). Both conceptions are descriptive of an individual who, in a provocative situation, is prone to negative cognitions and negative outbursts.

Several studies support the relation between irritability and aggressive behavior. Irritable men and women have been shown to exhibit greater levels of aggression, toward a fictitious opponent in a laboratory setting, compared with their nonirritable counterparts, particularly after being exposed to a frustrating stimulus (Caprara et al., 1985; Caprara, Renzi, Alcini, D'Imperio, & Travaglia, 1983; Caprara, Renzi, D'Augello, D'Imperio, & Travaglia, 1986). Following nicotine deprivation, men with high irritability scores were found to be more aggressive in a laboratory setting compared with men with low irritability scores (Parrott & Zeichner, 2001). Finally, irritability at age 12 has been shown to predict physical aggression and violence in late adolescence (Caprara, Paciello, Gerbino, & Cugini, 2007).

EF, Irritability, and Alcohol-Related Aggression

There exist a large number of studies which demonstrate that low EF is related to increased aggression (reviewed in Hawkins & Trobst, 2000; Paschall & Fishbein, 2002; Stevens, Kaplan, & Hesselbrock, 2003). 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 cognitive self-regulation of goal-directed behavior, low EF is logically related to poor judgment and poor behavioral regulation that can predispose toward aggression. Broadly speaking, cognitive and affective dysregulation are generally implicated in the expression of aggressive behavior (Anderson & Bushman,

2002; Berkowitz, 1993). In fact, it has been suggested that one aspect of affective dysregulation that is key in the manifestation of aggression is, irritability (Caprara et al., 2007). However, little research has been directed at attempting to isolate and test specific functional mechanisms within the broad etiological framework of "cognitive and affective dysregulation." However, one could argue that poor cognitive and affective regulation of behavior can lead to the thwarting of one's goals and therefore an increased likelihood of experiencing greater levels of negative affect, particularly, irritability.

Acute alcohol consumption has been shown to disrupt cognitive functioning, particularly EF (Hoaken, Assaad, & Pihl, 1998; Lyvers & Maltzman, 1991; Peterson, Rothfleisch, Zelazo, & Pihl, 1990). Alcohol intoxication leads to an inability to properly perceive and process information from the environment, interpret social cues, and formulate new strategies based on that information in order to cognitively regulate one's behavior and achieve one's goals (Giancola, 2000; Steele & Josephs, 1990). As this process deteriorates and the thwarting of one's goals progresses, there is an increased likelihood of experiencing greater levels of negative affect, particularly, irritability. We would argue that it is through this experience of irritability that the relation between EF and alcohol-related aggression is better explained or, in fact, mediated. The idea 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 (i.e., irritability) activates aggression-related memories, emotions, physiological responses, and motor patterns, which together form an associationistic network that further potentiates one's irritation thus predisposing the individual toward a violent response to provocation. Given that alcohol acts as a general disinhibitor of cognitive and affective regulation (Easdon, Izenberg, Armilio, Yu, & Alian, 2005; MacDonald, Fong, Zanna, & Martineau, 2000), we would also argue that such a mediational model would be stronger when persons are under the influence of alcohol than when they are sober.

In addition to examining whether irritability mediates the relation between EF and aggression, we also tested the interactive effects of EF and irritability on intoxicated aggression. Although both variables have been separately shown to interact with alcohol to facilitate aggressive behavior in men (Giancola, 2002a; 2004), no study has ever examined their conjoint effects. Studied in this manner, they may confer additional information, above and beyond their additive effects, regarding their role as risk factors for alcohol-related aggression.

Method

Data and Participants

The data presented in this article were drawn from a large project aimed at isolating risk factors for alcohol-related aggression. As noted earlier, two studies from this project demonstrated that EF (Giancola, 2004) and irritability (Giancola, 2002a) moderated the alcohol-aggression relation. However, those studies do not contain any tests regarding the mediational models contained in the present article, nor do they contain an analysis of the conjoint moderating effects of EF and irritability. In fact, the

current article is based on a separate theoretical foundation and proposes to test fundamentally different questions compared with our previous reports.

Participants were 313 (156 men and 157 women) healthy social drinkers between 21 and 35 years of age ($M = 23.07$; $SD = 2.85$). Social drinking was defined as consuming at least 3 to 4 drinks per occasion at least twice per month. Participants were recruited through advertisements placed in various newspapers in Lexington, Kentucky. Respondents were initially screened by telephone. Individuals reporting any past or present drug- or alcohol-related problems, serious head injuries, learning disabilities, or serious psychiatric symptomatology were excluded from participation. Individuals reporting abstinence from alcohol use or a condition in which alcohol consumption is medically contraindicated were also excluded. Respondents were screened for alcohol use problems using the *Short Michigan Alcoholism Screening Test* (SMAST; Selzer, Vinokur, & van Rooijen, 1975). Any person scoring an "8" or more on the SMAST was excluded from participation. Anyone with a positive breath alcohol concentration (BrAC) reading or a positive urine pregnancy or drug test (i.e., cocaine, marijuana, morphine, amphetamines, benzodiazepines, and barbiturates) result was also excluded. All pregnancy tests were negative. Two men had a positive BrAC, and one woman had a positive drug test. These individuals were not tested.

The sample consisted of 290 Caucasians, 22 African Americans, and 1 Hispanic. Regarding marital status, 89.1% of the participants were never married, 7.1% were married, 3.5% were divorced, and 0.3% were separated. The sample had a mean of 16.2 years of education ($SD = 2.07$). Forty-eight percent of the sample supported themselves financially and earned approximately \$18,500 per year; the remainder were supported by a parent or by a spouse. This study was approved by the University of Kentucky's Institutional Review Board.

Prelaboratory Procedures

Following the telephone screening interview, individuals eligible for participation were scheduled for an appointment to come to the laboratory. They were told to refrain from drinking alcohol 24 hr prior to testing, to refrain from using recreational drugs from the time of the telephone interview, and to refrain from eating 1 hr prior to testing (given that participants did not begin drinking until 3 hr into the experiment, the standard 4-hr fast used in most alcohol studies was observed). Because of hormonal variations associated with menstruation that may affect aggressive responding (Volavka, 1995), women were not tested between 1 week prior to and the beginning of menstruation. Participants were told that they would receive \$50 at the completion of the study as compensation.

Neuropsychological Test Selection

Selection of the EF test battery was guided by functional and neuroanatomical considerations according to guidelines put forth by Diamond (1991). Specifically, from a functional perspective, tests of EF were chosen to reflect a wide variety of skills encompassed by this construct such as attentional control, previewing ability, strategic goal planning, abstract reasoning, cognitive flexibility (set shifting), hypothesis generation, inhibition, and the

ability to organize and adaptively utilize information contained in working memory. From a neuroanatomical perspective, tests of EF were selected on the basis of being generally accepted as measures of functions that are subserved primarily by the prefrontal cortex. The prefrontal cortex and its subcortical circuits are thought to be the primary neurological substrates that subservise EF (Fuster, 1997; Luria, 1980; Stuss & Alexander, 2000). There are extensive neuroimaging data from normal individuals and ample neuropsychological evidence from patients with acquired brain lesions demonstrating that the EF tests selected for this investigation measure primarily prefrontal cortical functions (e.g., Casey et al., 1997; Demakis, 2003; Goel & Grafman, 1995; Karnath, Walleesch, & Zimmermann, 1991; Petrides, Alvisatos, Evans, & Meyer, 1993; Rezai et al., 1993; Sasaki, Gemba, Nambu, & Matsuzaki, 1993; Stuss, Floden, Alexander, & Katz, 2001). Tests were also selected on the basis of being well-established and psychometrically sound measures that have proven sensitivity and specificity to assess a broad array of EF domains. As was alluded to earlier, the literature on the structure and definition of EF is mixed. Consequently, we chose to focus on a broad array of EF processes because our hypotheses center on EF in general, not specific aspects of EF.

Tests of EF

The following seven tests of EF were administered to all participants: *Porteus Mazes* (Porteus, 1965), *Go/No-Go Task* (Newman & Kosson, 1986), *Trails B of the Trail Making Test* (Reitan, 1992), *Stroop Test* (MacLeod, 1991), *Conditional Associative Learning Test* (Petrides, 1985), *Tower of Hanoi* (Goel & Grafman, 1995), and the *Wisconsin Card Sorting Test* (Heaton, 1993). All tests were administered according to standard procedures. To conserve space, interested readers are directed to Giancola (2004) for a full description of these tests, their administration, and their psychometric properties. Giancola (2004) conducted a confirmatory factor analysis comparing three different models, which 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 from the neuropsychological tests were first converted to *z*-scores and then summed to obtain an aggregate EF score where higher scores denote increased errors on the EF tests.

Irritability

Irritability was assessed using the *Caprara Irritability Scale* (CIS; Caprara et al., 1985). The CIS is a measure of dispositional irritability that consists of 20 items scored on a 6-point Likert 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. The development of the CIS was based on, and is very similar to, the *Irritability Scale* from the *Buss-Durkee Hostility Inventory* (Buss & Durkee, 1957).

The CIS has excellent psychometric properties. The Cronbach alpha coefficient for the present investigation was 0.89. Furthermore, based on a sample of healthy men and women, Caprara et al. (1985) found that the scale possesses a Cronbach alpha coefficient of 0.81, a test-retest coefficient of 0.83, and a split-half coefficient

of 0.90. Other studies have demonstrated higher reliability coefficients in both clinical (Tarter, Alterman, & Edwards, 1985) and normal samples (Anderson, 1997). Moreover, 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 ($n = 81$), (b) men who received a placebo ($n = 75$), (c) women who received alcohol ($n = 82$), and (d) women who received a placebo ($n = 75$).

Beverage Administration

Men who received alcohol were administered a dose of 1 g/kg of 95% alcohol United States Pharmacopeia mixed at a 1:5 ratio with Tropicana orange juice. Because of differences in body fat composition, women were given a dose of 0.90 g/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 cc's of alcohol were added to each placebo beverage and 3 cc'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. 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 to 4 drinks per occasion at least twice per month. No participant experienced any adverse effects because of 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. 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 from one another (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997). In recognition of this latter finding, we only employed an alcohol and a placebo group.

Aggression Task

A modified version of the *Taylor Aggression Paradigm* (TAP; Taylor, 1967) was used to measure aggression. The hardware for the TAP was developed by Coulbourne Instruments (Allentown, PA), and the computer software was developed by Vibranz Creative Group (Lexington, KY). The TAP 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 TAP 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.

Procedure

Upon entering the laboratory, participants were explained the procedures of the study and were asked to sign an informed consent form. The experimenter then assessed their BrACs to ensure sobriety. If the BrAC test was negative, participants then underwent a urine drug test and women also underwent a urine pregnancy test. BrACs 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.

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

Given that the aggression-potentiating effects of alcohol are more likely to occur on the ascending limb of the BrAC curve (Giancola & Zeichner, 1997) and because a BrAC of at least 0.08% is effective in eliciting robust levels of aggression (Giancola & Zeichner, 1997; Gustafson, 1992; Pihl, Smith, & Farrell, 1984), the alcohol group began the TAP shortly after they reached an ascending BrAC of at least 0.09% (two men and one woman never achieved this BrAC and were thus removed from the study). This methodology indicates that we decided to standardize BrAC rather than the time latency following beverage consumption. One could argue that the time duration between the end of beverage consumption and beginning the TAP 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 undesirably large individual differences in BrACs during the aggression task. Finally, imme-

diately before beginning the TAP, participants provided subjective ratings of their level of intoxication. This was done using a specially constructed scale ranging from 0 to 11 on which "0" was labeled "not drunk at all," "8" was labeled "drunk as I have ever been," and "11" was labeled "more drunk than I have ever been."

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

Following the pain threshold/tolerance testing, participants were once again explained the TAP. 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" appeared at which time they had to lift their fingers off of the spacebar as quickly as possible. A "win" was signaled by the words "You Won. You Get to Give a Shock" and a "loss" was signaled by the words "You Lost. You Get a Shock." A winning trial allowed participants to deliver a shock to their opponent and a losing trial resulted in receiving a shock from this individual. Participants could not elect to not shock their opponent. That is, following a winning trial, the task would pause until a shock was selected. However, participants were told that shock button "#1" delivers a very low intensity shock that is best characterized as "very mild" and "definitely not painful." Following a winning trial and pressing a shock button, participants 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 participants' beliefs that they were actually administering shocks. Upon losing a trial, participants received a shock and were given feedback regarding the level of that shock in the form of a signal on the volt meter and the illumination of one of the 10 "shock lights" on the computer screen.

Participants were told that they had a choice of 10 different shock intensities to administer at the end of each winning trial. Regardless of beverage group assignment, all participants were informed that their opponents were 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 34 trials (17 wins and 17 losses). Participants received increasingly higher intensity shocks follow-

ing losing trials. Taylor and Chermack (1993) argued that increasing provocation as the task progresses adds an increased degree of external validity to the TAP because this ordering best reflects how an escalation in interpersonal provocation leads to increased violence in "real-life" situations. Aggression was operationalized as the mean shock intensity selection ("1" through "10") across all trials, which represents a measure of retaliatory aggression.

All shocks delivered to the participants were of a one second duration. In actuality, reaction times were not measured; the competitive task was used to lead participants to believe that they were 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 fixed-random order with no more than three consecutive wins or losses. The trials were interspersed by 5-s intervals. A computer controlled the initiation of trials, administration of shocks to the participants, and the recording of their responses. The experimenter secretly viewed and heard the participant through a hidden video camera and microphone throughout the procedure.

Immediately following the testing procedure, BrACs were measured and participants were again asked to rate their subjective state of intoxication. In addition to this, they were asked whether the alcohol they drank caused them any impairment on a scale ranging from 0 to 10 on which "0" was labeled "no impairment," "5" was labeled "moderate impairment," and "10" was labeled "strong impairment." Participants were then asked a yes/no question regarding whether they believed that they had consumed alcohol. They were also asked a variety of questions to indirectly assess the credibility of the experimental manipulation (see below). Participants were then debriefed and compensated. All individuals who received alcohol were required to remain in the laboratory until their BAC dropped to 0.04%.

Deception Manipulation

To disguise the TAP as a measure of aggression, participants were given a fictitious cover story. They were informed that the purpose of the study was to determine how a person's "thinking-style" and personality would influence alcohol's effects on reaction time in a competitive situation. To convince participants that they were actually competing against another person, a confederate was seated in a room adjacent to the testing room. As the experimenter led the participant into the testing room, s/he identified the confederate (same gender as the participant) as the "opponent." No opportunity for an interaction between the participant and the confederate was allowed. Furthermore, immediately before assessing their pain thresholds, participants were informed that their opponent would undergo the threshold assessment procedure first. Participants were also informed that they would be able to hear their opponent's responses to the procedure over an intercom that ostensibly served the two testing rooms and the control room. In actuality, the confederate acted as the fictitious opponent and answered the experimenter's questions regarding the testing of his/her pain threshold/tolerance in accordance with a list of predetermined responses. All participants heard the same experimenter-confederate verbal exchange. Of course, in reality, there was no actual opponent.

Results

Manipulation Checks

TAP checks. To verify the success of the TAP, 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 posttask ratings of 4.5 and 5.0 (scale range = 0 to 11). The placebo group reported average ratings of 1.7 and 2.1, respectively, pretask ratings: $t(311) = -15.7, p < .05$; posttask ratings: $t(311) = -14.1, p < .05$. 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, $t(311) = -16.4, p < .01$ (scale range = 0 to 10). There were no significant Gender or Gender \times Beverage group effects for any of the placebo check measures.

BrAC levels. All participants tested in this study had BrACs of 0% upon entering the laboratory. Individuals in the alcohol group had a mean BrAC of 0.097% ($SD = 0.014$) just before beginning the aggression task and a mean BrAC of 0.105% ($SD = 0.018$) immediately after the task. Persons given the placebo had a mean BrAC of 0.01% ($SD = 0.009$) just before the task and a mean BrAC of 0.008% ($SD = 0.007$) immediately after the task. There were no significant Gender or Gender \times Beverage group effects for any of the BAC measures. Persons in the alcohol group had a mean BrAC of 0.11% approximately 10 min after the last BrAC assessment indicating that they were on the ascending limb of the BAC curve during the aggression task. BrACs peaked around 0.11% to 0.12%.

Demographic Data

To test for unexpected group differences, all demographic variables were analyzed using 2 (beverage) \times 2 (gender) between-groups design analyses of variance (ANOVA). There were no such differences. However, compared with women, men began drinking at an earlier age, $F(1, 311) = 5.94, p < .05$; were first intoxicated at an earlier age, $F(1, 311) = 5.28, p < .05$; consumed more drinks per occasion, $F(1, 311) = 51.86, p < .001$; had more drinking occasions per week, $F(1, 311) = 18.89, p < .001$; consumed more alcoholic drinks per week, $F(1, 311) = 41.73, p < .001$; and had higher SMAST scores, $F(1, 311) = 37.21, p < .001$. However,

men and women did not differ with regard to age, years of education, salary, age at first drink, as well as EF and CIS scores. These data are presented in Table 1.

Correlations

Table 2 provides Pearson Product-Moment correlations between the CIS (irritability scale), the EF variable, as well as the individual tests that comprised the EF variable for both men and women. Recall, all of these measures were assessed while participants were sober.

Mediation Analyses

The first aim of this study was to examine whether irritability would mediate the relation between EF and aggression. According to Baron and Kenny (1986), conditions for mediation were only met for the alcohol/male group. A two-step hierarchical multiple regression procedure was used. The first step involved testing the relation between EF and aggression and the second step involved adding the effects of irritability to the previous model to assess the change in the original relation between EF and aggression. As can be seen in Table 3 and in Figure 1, the standardized β estimate for the relation between EF and aggression was statistically significant in the first step of the model. In Step 2, the addition of the irritability variable reduced the relation between EF and aggression by 42% thus rendering it no longer statistically significant (indicating full mediation). The β estimate for the relation between irritability and aggression was statistically significant. To provide a more complete picture of the mediation analyses, they were also calculated for the other experimental groups (placebo/male; alcohol/female; and placebo/female) that did not meet Baron and Kenny's (1986) specified conditions to carry out mediation analyses. As can be seen in Table 3, the results indicated no consistent pattern of significant findings.

Despite its widespread use, Baron and Kenny's (1986) approach suffers from issues with low statistical power, controlling Type-I error rates, and efficiency in parameter estimation (James, Mulaik,

& Brett, 2006; LeBreton, Wu, & Bing, 2008; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). It was therefore important to conduct post hoc tests of the significance of the above mediation analyses (Holmbeck, 2002). Several tests exist to assess the significance of mediation effects such as normal theory approach (Sobel, 1986), structural equation modeling (Holmbeck, 1997), and percentile bootstrapping resampling (Preacher & Hayes, 2004). Sobel's test is a commonly used and powerful means of testing mediation; however, it relies on the assumption of a very large normally distributed data set (Fritz & MacKinnon, 2007).

Bootstrap resampling is becoming an increasingly popular means of testing indirect effects such as those found in mediation analyses (Preacher & Hayes, 2004). This is because of its increased accuracy over other methodologies at computing confidence limits around indirect effects inasmuch as bootstrap resampling does not assume that indirect effects follow a normal distribution (MacKinnon, Lockwood, & Williams, 2004; MacKinnon, Fritz, Williams, & Lockwood, 2007; Preacher & Hayes, 2004). Bootstrapping involves multiple resamplings of a given dataset; from that, a series of independent and identical distributions are generated whereby a given test may be repeated. This process yields a confidence interval that provides parameters rather than a single estimate of an effect. This method has been argued to be superior to a normal-theory based approach because of its simplicity and power in detecting a significant effect by generating a confidence interval around a series of resamplings of an actual dataset (MacKinnon et al., 2002; Preacher & Hayes, 2004). As noted earlier, we began by assessing mediation using the method put forth by Baron and Kenny (1986). However, because the indirect mediation effect was visible only within the alcohol/male group, we chose to separately recalculate our analyses to test for the presence of possible significant indirect (mediation) effects within all four of our groups using the more stringent percentile bootstrap re-sampling approach (Preacher & Hayes, 2004).

The bootstrapping results confirmed those yielded using Baron and Kenny's (1986) methods. Using 1,000 bootstrap resamples of the data, the results were significant, within 95% confidence

Table 1
Demographic Data

Measure	Men		Women	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	23.41	3.07	22.73	2.59
Years of education	16.03	2.13	16.31	2.01
Yearly salary	\$18.54K	\$12.69K	\$17.93K	\$9.83K
SMAST	1.91	2.82	0.36	1.47*
Age at first drink	15.21	2.62	15.68	2.47
Age when first drunk	15.90	2.42	16.56	2.52*
Age when regular drinking began	18.48	2.30	19.10	2.18*
Drinking occasions per week	2.34	1.37	1.72	1.17*
Drinks per occasion	6.32	3.45	3.89	2.38*
Drinks per week	15.67	12.86	7.31	9.76*
Executive functioning	-0.21	4.02	0.02	3.72
Caprara Irritability Scale	55.15	13.79	53.76	15.10

Note. SMAST = Short Michigan Alcoholism Screening Test; K = \$1,000; Executive functioning data is represented as z-scores.

* $p < .05$.

Table 2
Pearson Product-Moment Correlations Between Executive Functioning and Irritability for Men and Women

Scales	1	2	3	4	5	6	7	8	9
EF	—	.152	.481**	.528**	.538**	.581**	.525**	.702**	.584**
CIS	.055	—	-.033	.024	.093	.044	.016	.167*	.246**
G/NG	.462**	.026	—	.077	.092	.261**	.137	.296**	.122
Trails	.617**	-.096	.183*	—	.124	.235**	.163*	.339**	.118
TOH	.432**	.031	.005	.169*	—	.106	.178*	.267**	.310**
Stroop	.581**	.103	.031	.413**	.217**	—	.264**	.267**	.196*
Porteus	.578**	.030	.081	.150	.104	.135	—	.276**	.153
CALT	.578**	.026	.204*	.297**	.026	.199*	.325**	—	.289**
WCST	.610**	.094	.227**	.181*	.176*	.245**	.371**	.217**	—

Note. EF = executive functioning variable; CIS = Caprara Irritability Scale; G/NG = Go/ No-Go; Trails = Trail Making Test; TOH = Tower of Hanoi; Stroop = Stroop Test; Porteus = Porteus Mazes; CALT = Conditioned Associative Learning Test; WCST = Wisconsin Card Sorting Test. Values above diagonal represent men and those below represent women.
* $p < .05$. ** $p < .01$.

intervals (CIs), denoted by the noninclusion of a zero value, for the male/alcohol group only (CIs = 0.02 to 0.15). Nonsignificant findings, denoted by the inclusion of a zero value within the confidence intervals, were found for the male/placebo group (CIs = -0.03 to 0.04), the female/alcohol group (CIs = -0.05 to 0.01), and the female/placebo group (CIs = -0.01 to 0.04).

Moderation Analyses

The second aim of this study was to examine the interactive effects of EF and irritability on intoxicated aggression. Although both EF and irritability have been separately shown to interact with

alcohol to facilitate aggressive behavior in men (Giancola, 2002a, 2004), no study has ever examined their conjoint effects. Taken together, they may confer additional information, above and beyond their additive effects, regarding their role as risk factors for alcohol-related aggression.

The relations between the independent variables (i.e., EF, irritability, gender, and beverage) and aggression were assessed using multiple regression. Beverage and gender groups were dummy coded according to procedures outlined in Aiken and West (1991). Interaction terms were calculated by obtaining the cross-products of standardized first-order variables. Standardizing the first-order variables also automatically centers the values (i.e., deviation scores with a mean of zero), which reduces multicollinearity between interaction terms and their constituent lower-order terms (Aiken & West, 1991). All main effects were entered into the models on the first step, followed by 2-way effects in the second step, 3-way effects in the third step, and the 4-way interaction in the fourth step. This resulted in a full model comprised of 15 variables.

To not reproduce previously published data, we confine our reporting of results to only the 4-way interaction term between EF, irritability, gender, and beverage on aggression. The 4-way interaction was not significant. There were significant interactions between beverage and EF and between beverage and irritability. However, as noted earlier, these findings were reported in previous studies (Giancola, 2002a, 2004). As such, the interactive effects of EF and irritability did not confer additional explanatory variance

Table 3
Regression Equations Assessing the Mediating Role of Irritability for the Relation Between Executive Functioning (EF) and Aggression

Step and measure	β	p
Intoxicated males		
Step 1: EF	.300	.007
Step 2		
EF	.175	ns
Irritability	.382	.001
$F(2, 78) = 11.0, p < .001; R^2 = .22$		
Sober males		
Step 1: EF	.123	ns
Step 2		
EF	.116	ns
Irritability	.254	.028
$F(2, 72) = 3.11, p = .050; R^2 = .08$		
Intoxicated females		
Step 1: EF	.048	ns
Step 2		
EF	.073	ns
Irritability	.264	.017
$F(2, 79) = 3.05, p = .053; R^2 = .07$		
Sober females		
Step 1: EF	.228	.049
Step 2		
EF	.201	ns
Irritability	.152	ns
$F(2, 72) = 2.90, p = .062; R^2 = .07$		

Note. ns = nonsignificant.

Intoxicated Men

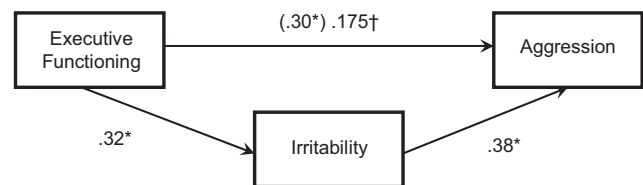


Figure 1. Regression model representing the mediating effect of irritability on the relation between executive functioning and aggression in intoxicated men.

regarding the alcohol-aggression relation. In addition to examining the 4-way interaction, we also conducted a multiple regression analysis of the four independent variables entered simultaneously in the absence of their higher-order effects. The omnibus model was significant, $F(4, 308) = 13.72, p < .001; R^2 = .15$. All linear effects within the model were also significant: EF ($\beta = .159, p < .01$), irritability ($\beta = .257, p < .001$), beverage ($\beta = .127, p < .05$), and gender ($\beta = .182, p < .001$). In summary, despite the previous findings that EF (Giancola, 2004) and irritability (Giancola, 2002a) independently moderated the alcohol-aggression relation; no support was found to suggest that they conferred an interactive liability.

Stepwise Regression Analyses

Given that the four independent variables were significant in the model described earlier, we conducted a stepwise multiple regression analysis to determine the relative importance of each variable in predicting aggressive behavior. Results indicated that irritability entered the model first accounting for 7.8% of the variance ($F\Delta = 26.24, p < .001$), followed by gender, which accounted for an additional 3.1% ($F\Delta = 10.80, p < .005$), followed by EF, which accounted for an additional 2.6% ($F\Delta = 9.39, p < .005$), followed by beverage, which accounted for an additional 1.6% ($F\Delta = 5.83, p < .01$).

Discussion

The results of this investigation partially supported the hypotheses. Specifically, irritability mediated the relation between EF and aggression, but only for intoxicated men. As expected, this effect was not likely to occur in sober men because we would speculate that they presumably did not experience the cognitive dysregulation, and subsequent increase in irritability in response to provocation, associated with alcohol intoxication. Moreover, the mediation effect did not occur in sober or intoxicated women either. Men and women differ on their experience of aggression when placed in a negative affective state, with men being significantly more prone to aggressive behavior than women (Verona & Curtin, 2006). It has been suggested that women do not manifest physical aggression to the same degree as men because of the possibility that they might have a greater liability threshold to exhibit such behavior. In other words, compared with men, women may require a greater degree of biological and/or environmental vulnerability in order to display aggression (Cloninger, Christiansen, Reich, & Gottesman, 1978; Cloninger, Reich, & Guze, 1975). This is illustrated in a study showing that acute alcohol intoxication only increased aggression in women who reported excessively high levels of dispositional physical aggressivity (Giancola, 2002b).

The secondary hypothesis that EF and irritability would have an interactive effect above and beyond their constituent effects was not supported. This may be because of the possibility that their relation is best described as a mediational one in which alcohol-induced cognitive dysregulation gives rise to irritability and ultimately aggressive behavior, rather than the two variables interacting in a conjoint fashion. However, it should be kept in mind that previous studies have demonstrated that both EF (Giancola, 2004) and irritability (Giancola, 2002a) independently increased the risk for intoxicated aggression. Moreover, the simultaneous and step-

wise entry regression models presented in the current study were consistent with one another indicating that all four independent variables (EF, irritability, gender, and beverage) were significant predictors of aggressive behavior. The best predictors, in order of decreasing importance were irritability, gender, EF, and beverage.

Our findings are in keeping with a number of theoretical formulations suggesting that the cognitive skills subsumed under the EF rubric are involved in regulating affect (i.e., irritability). One of the first accounts of this idea was put forth by Luria (1961, 1980) who suggested that the cognitive regulation of affect and behavior is governed predominantly by the prefrontal cortex; the primary neural substrate for EF. Following Luria, Tarter, and colleagues (Tarter, 1988; Tarter et al., 1985) advanced a similar theoretical stance to explain the neurobehavioral underpinnings of alcoholism. Specifically, Tarter's model implicated childhood difficult temperament, which, in part, manifests as irritability, as a risk factor for alcoholism, as well as antisocial and aggressive behavior (Giancola & Tarter, 1999). Lastly, Moffitt (1993) suggested that neuropsychological deficits, particularly EF deficits, predispose toward a dysregulation of temperament that can display as irritability and poor impulse control that can lead to violence. In keeping with these theoretical accounts, a recent study by Giancola, Roth, and Parrott (2006) demonstrated that EF did indeed play a regulatory role over temperament. These theoretical formulations and empirical findings, as well as the results of the present investigation, are consistent with studies showing that patients with acquired lesions to the prefrontal cortex often present with a symptom complex consisting of cognitive and behavioral inflexibility, impulsivity, and emotional dysregulation which manifests as irritability that, in turn, can predispose toward violent behavior (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. In fact, acute alcohol intoxication is well known to disrupt cognitive functioning, especially EF (Hoaken et al., 1998; Lyvers & Maltzman, 1991; Peterson et al., 1990). The so called "disinhibition model" of alcohol-related aggression contends that alcohol acts as a general disinhibitor; in a sense, anesthetizing brain regions or loosening constraints important in maintaining inhibitory control over behavior (Collins, 1988; Graham, 1980). More contemporary models suggest that alcohol leads to aggression by disrupting cognitive processes involved in perceiving and processing peripheral inhibitory cues, interpreting social cues, formulating flexible plans to achieve goals, as well as considering the consequences of engaging in maladaptive behaviors (Collins, 1988; Giancola, 2000; Graham, 1980; Steele & Josephs, 1990; Zeichner & Pihl, 1979). As such, the cognitive dysregulation brought on by impaired EF, alcohol intoxication, or both, in conjunction with provocation, can lead to a heightened experience of negative affect; specifically irritability, that can then lead to an aggressive reaction. We maintain that it is through this experience of irritability that the relation between EF and alcohol-related aggression is mediated.

This conceptualization is consistent with Berkowitz's cognitive neoassociationistic model. According to Berkowitz (1993), aggression is the result of negative affect, which includes irritability. As noted earlier, the experience of negative affect is a result of one's goals being thwarted by disruptions in cognitive functioning when

one is unable to properly appraise and process environmental cues and devise adaptive solutions based on changing environmental demands (Berkowitz, 1993). As this deteriorative process continually thwarts the ability to achieve one's goals, one is likely to experience even greater levels of negative affect and irritability. According to Berkowitz (1993), this will activate aggression-related memories, emotions, physiological responses, and motor patterns that form an associationistic network that predisposes toward aggression. It is noteworthy that 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 are more likely to focus on, and thus be influenced by, more salient, noxious, and provocative cues that would lead to an irritable response to provocation. Given these parameters (i.e., alcohol intoxication and low EF), it is less plausible that one would alternatively focus on more peripheral fear-potentiating cues that might signal escape from the potential altercation (see also Steele & Josephs, 1990).

As noted earlier, within the context of Berkowitz's (1993) model, we argued that the relation between low EF and increased aggression is mediated by negative affect, specifically irritability. However, there exist alternative viewpoints regarding the relation between alcohol intoxication and negative affect. Although findings are generally mixed, it has been postulated that alcohol can attenuate, rather than increase, the experience of negative affect (reviewed in Greely & Oei, 1999; Sayette, 1993; Sher, 1987). However, there are important conceptual and methodological differences between these studies and our results. Alcohol has been shown to reduce negative affect in the form of anxiety, (Josephs & Steele, 1990; Steele & Josephs, 1988) and anticipatory fear responses (Curtin, Lang, Patrick, & Stritzke, 1998; Curtin, Patrick, Lang, Cacioppo, & Birbaumer, 2001) to a stressor, but only when one's attentional focus is distracted away from the impending stressor. In fact, some studies have shown that if attention is actually focused upon a given stressor, persons given alcohol will actually experience increased negative affect (i.e., anxiety) compared with those given no alcohol (Josephs & Steele, 1990; Steele & Josephs, 1988). In our study, participants' attention was directly focused on the highly salient stressor of provocation in the form of receiving electric shocks from their fictitious opponent. Consistent with contemporary theoretical accounts (Berkowitz, 1993; Steele & Josephs, 1990), this highly salient, provocative, and aggressive cue; coupled with the disinhibiting effects of alcohol, lead to an activation of negative affect, specifically, the experience of irritability, agitation, and hostility. This activation of hostility and subsequent aggression seems to be particularly prevalent in males with a propensity for experiencing negative affect in response to stress (Verona & Kilmer, 2007; Verona, Reed, Curtin, & Pole, 2007). As such, we contend that our results are in keeping with others demonstrating that when attention is focused upon cues with a hostile negative affective valence (i.e., being provoked with electric shocks), alcohol will be more likely to increase negative affect, specifically irritability, and therefore aggression.

Limitations and Other Considerations

It is important to highlight the fact that our data are largely correlational in nature and collected within the context of a cross-

sectional design which places limitations on drawing distinct causal assertions related to mediation. EF and irritability were measured in the sober state as dispositional variables prior to testing aggression. As such, this investigation was not designed to test the acute effects of alcohol on EF or on irritability. Furthermore, the study was also not designed to assess how intoxicated versus sober EF differentially impact state irritability and how such a state variable would affect aggression. Thus, we would like to note that we are not testing *causal* mediation. Obviously, these are all important questions that are clearly worthy of study and necessary to obtain a better understanding of the relations between the variables being examined in the present study. Nevertheless, our research question was aimed at determining whether the dispositional trait of irritability mediates and moderates the relation between the dispositional trait of EF and aggressive behavior measured under alcohol and placebo. Research reviewed above indicates that alcohol can have an anxiogenic effect (Josephs & Steele, 1990; Steele & Josephs, 1988) that can theoretically lead to an experience of irritability and subsequent aggression, however credence should also be given to other existing literatures showing that alcohol has the capacity to disrupt attention and thus reduce anxiety (Sayette, 1993; Sher, Bartholow, Peuser, Erikson, & Wood, 2007). It is possible that our mediation effect was specific to intoxicated males because of differential processing of provocation from the TAP by the intoxicated and sober groups. Despite these theoretical claims, the design of this study did not allow for the inclusion of measures to assess focus of, or alcohol's effect on, attention. Given the mixed findings regarding alcohol's anxiogenic versus anxiolytic effects (see Sher et al., 2007), future research into attentional processes while under the influence of alcohol and subsequent aggressive behavior is warranted.

In conclusion, given our data and the complexity of the alcohol-aggression relation as understood from Berkowitz's (1993) cognitive neoassociationistic framework, one key avenue for future research involves 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 serve to elaborate upon the cognitive neoassociationistic model by providing a better understanding of which forms of negative affect and their associated cognitions and behavioral patterns lead to aggression. 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 such behavior. It would be very telling to fractionate negative affect into a number of constituent parts and explore how such components are related to aggression as well as alcohol-related aggression. Along similar lines, given the current debate about the structure of EF, it would also be interesting to examine whether different aspects of EF are differentially related to aggressive behavior in comparison to a more unitary variable. A reasonable starting point would be to examine Miyake et al.'s (2000) tripartite model as well as EF conceptualizations put forth by others (Zelazo et al., 1997).

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