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Cognitive and Affective Mediators of Alcohol-Facilitated Intimate Partner Aggression

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Abstract

This multisite study examined whether aggressive cognitions and facial displays of negative affect and anger experienced during provocation mediated the association between alcohol intoxication and intimate partner aggression (IPA). Participants were 249 heavy drinkers (148 men, 101 women) with a recent history of IPA perpetration. Participants were randomly assigned to an Alcohol or No-Alcohol Control beverage condition and completed a shock-based aggression task involving apparent provocation by their intimate partner. During provocation, a hidden camera recorded participants' facial expressions and verbal articulations, which were later coded using the Facial Action Coding System and the Articulated Thoughts in Simulated Situations paradigm. Results indicated that the positive association between alcohol intoxication and partner-directed physical aggression was mediated by participants' aggressive cognitions, but not by negative affect or anger facial expressions. These findings implicate aggressogenic cognitions as a mediating mechanism underlying the association between the acute effects of alcohol and IPA perpetration.

Summary

It is clear that alcohol use interacts with a wide range of individual, dyadic, and contextual factors to increase risk of IPA (Brown & Leonard, 2017). However, the underlying mechanisms of how

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alcohol intoxication facilitates IPA perpetration are not well understood. The present study was undertaken to address this important research gap by specifically assessing how intoxicated individuals reacted, both affectively and cognitively, to sustained provocation by an intimate partner, and whether this reactivity predicted the likelihood of partner-directed aggression. Our findings indicated that a tendency to experience aggressive cognitions fully mediated the association between alcohol intoxication and the likelihood of post-provocation IPA. We did not find sufficient evidence that displays of negative affect and anger expression were significant intervening mechanisms in the link between alcohol and IPA perpetration. In the context of existing theories of aggression, our findings indicate a causal pathway in which alcohol intoxication elicits aggressive cognitions in response to provocation, which is subsequently followed by partner-directed physical aggression.

Keywords

intimate partner aggression; heavy drinking; aggressive cognitions; negative affect; I^3 Model

Intimate partner aggression (IPA) is a serious public health problem that affects the lives of millions of men and women each year and is associated with numerous negative consequences, including mental and physical health problems, increased use of legal and housing services, and financial burden of up to $5.8 billion annually (Black et al., 2011; National Center for Injury Prevention and Control, 2003). It is well-established that heavy alcohol use is a contributing cause of IPA perpetration (Leonard, 2005; Leonard & Quigley, 2017), and myriad risk factors for alcohol-facilitated IPA perpetration have been identified (e.g., Cafferky, Mendez, Anderson, & Stith, 2018; Foran & O'Leary, 2008). Though informative, this literature has not demonstrated empirically that acute alcohol intoxication affects relevant psychosocial mediators to facilitate IPA perpetration. Direct tests of the mechanism(s) by which alcohol facilitates IPA perpetration are critical, as such work can identify critical points of intervention that may inform emerging approaches to offender treatment. As such, the scientific premise of the present study was that alcohol intoxication facilitates IPA perpetration by affecting the regulation of more proximal cognitive and affective responses to relationship conflict. This premise was tested using laboratory-based methods that assessed the effect of acute alcohol intoxication on state cognition and affect that were experienced seconds before a subsequent aggressive response, thereby modeling a temporal and highly proximal association between these theorized mediators and subsequent IPA perpetration.

Alcohol-Facilitated IPA Perpetration

Acute alcohol intoxication and a pattern of heavy alcohol use are among the most robust correlates of IPA perpetration. Survey and cross-sectional data reveal a positive association between IPA perpetration and alcohol-related problems (Caetano, Ramisetty-Mikler, & Field, 2005), alcohol consumption (Abramsky et al., 2011; Graham, Bernards, Wilsnack, & Gmel, 2011; Okuda et al., 2015; Renzetti, Lynch, & DeWall, 2018), and alcohol use disorders (Chermack, Fuller, & Blow, 2000; Friend, Langinrichsen-Rohling, & Eichold, 2011), even after controlling for perpetrator demographics, hostility, and/or relationship

distress (Leonard & Senchak, 1993; Pan, Neidig, & O'Leary, 1994; Renzetti et al., 2018; Smith, Homish, Leonard, & Cornelius, 2012). Longitudinal findings indicate that heavy drinking in the early stages of marriage predicts subsequent IPA (Leonard, 2008; Leonard & Senchak, 1996). Studies of violent couples indicate that IPA perpetration is more likely to occur after alcohol consumption (Testa & Derrick, 2014), with IPA episodes being more frequent, more severe, and are more likely to lead to mutual violence when one partner has been drinking (Murphy, Winters, O'Farrell, Fals-Stewart, & Murphy, 2005; Stuart et al., 2013; Testa, Quigley, & Leonard, 2003). Laboratory studies demonstrate that alcohol intoxication increases (a) negative interaction behaviors among violent couples (Leonard & Roberts, 1998), (b) aggressive verbalizations during simulated relationship conflicts among violent couples (Eckhardt, 2007), and (c) increased white noise blasts towards an intimate partner during a competitive reaction time task (Watkins, DiLillo, & Maldonado, 2015). This cross-method convergence of findings has led to the conclusion that alcohol use is a contributing cause of IPA perpetration (Brown & Leonard, 2017; Leonard & Quigley, 2017).

However, it is also clear that alcohol consumption is neither a necessary nor sufficient criterion for IPA perpetration. The effect size of alcohol on IPA is moderate (for a review see Cafferky et al., 2018), prior laboratory findings involving the acute effects of alcohol on negative interaction behaviors among violent couples did not replicate (Testa et al., 2014), and the effects are dependent on myriad moderating risk factors (Foran & O'Leary, 2008; Stith, Smith, Penn, Ward, & Tritt, 2004). The overall pattern of data suggest that additional laboratory studies are needed to actually demonstrate the acute effects of alcohol intoxication on IPA-related outcomes, given the small number of carefully controlled alcohol administration studies, particularly from samples at high-risk for alcohol-facilitated IPA (Eckhardt, Parrott, & Massa, in press).

The alcohol-aggression relation is most frequently interpreted from the etiologic standpoint of alcohol myopia theory (AMT; Steele & Josephs, 1990). AMT purports that the pharmacological properties of alcohol narrow attentional focus, restrict the internal and external cues individuals perceive, and reduce individuals' capacity to process meaning from information they do perceive. One model of AMT, the attention-allocation model, posits that alcohol impairs attentional capacity, which then restricts the inebriate's ability to perceive and process instigatory and inhibitory cues. As a result, intoxicated individuals allocate their attention such that they perceive and process only the most salient cues of a situation (e.g., a verbal insult from their partner) to the exclusion of less salient inhibitory cues (e.g., legal consequences of IPA).

AMT has garnered extensive empirical support as an explanation for a range of alcohol-related behaviors, including aggression (for a review, see Giancola, Josephs, Parrott, & Duke, 2010). Laboratory data suggest that alcohol use increases or decreases aggression depending upon whether attention is narrowly directed toward cues that promote (e.g., provocation) or inhibit (e.g., non-aggressive norms) aggression, respectively. For instance, distraction from provocative cues reduces physical aggression among intoxicated men (e.g., Giancola & Corman, 2007; Phillips & Giancola, 2008). Meta-analytic reviews evidence smaller effect sizes of alcohol on aggression when participants are distracted (Bushman & Cooper, 1990). Cross-sectional studies suggest that heavy drinking is associated with IPA

primarily among individuals who endorse dispositional tendencies in aggression-related cognitive biases (e.g., high hostility; Lisco, Parrott, & Tharp, 2012; Tharp, Schumacher, Samper, McLeish, & Coffey, 2012) or who are susceptible to alcohol-related shifts in attention toward instigatory cues (e.g., low mindfulness; Gallagher, Hudepohl, & Parrott, 2010). Recent alcohol administration-based research within our lab indicates that individuals with a recent history of IPA and problematic alcohol use were more likely to attend to aggressogenic situational cues while intoxicated but not while sober (Massa, Subramani, Eckhardt, & Parrott, 2019).

While it is clear that AMT is a well-supported explanatory framework for alcohol-facilitated IPA (Parrott & Eckhardt, 2018), the intervening processes by which attentional biases increase (or decrease) the probability of an aggressive response to a provocative situation remain largely unstudied. Indeed, little research has directly examined *mediators* of alcohol-related aggression in high risk samples under controlled laboratory conditions. In the present study, we examined cognitive and affective factors that existing theory (Finkel & Eckhardt, 2013) and research (Giancola et al., 2010) suggest are likely candidate mechanisms that mediate the association between alcohol use and IPA perpetration in a sample of heterosexual couples at high risk for alcohol-facilitated IPA perpetration. These factors are discussed below.

Increased negative affect and anger expression.

According to cognitive-neoassociationistic (CNA) theory (Berkowitz, 1990; 1993), negative affect, anger-related emotions, and hostile cognitions exist within an interconnected associative network. The elicitation of negative affect is posited to activate anger-related emotional, cognitive, and behavioral nodes in the network. In turn, higher-order cognitive processes intensify, suppress, or differentiate the anger experience which, in some cases, results in aggression. The notion that negative affect and anger expression-related processes potentiate aggression has received strong empirical support in the general aggression (Berkowitz, 2012) and IPA literatures (Birkley & Eckhardt, 2015; Norlander & Eckhardt, 2005) and is consistent with other prominent heuristic theories of aggression (e.g., Anderson & Bushman, 2002).

How are these processes affected by alcohol intoxication? Similar to CNA theory, Giancola and colleagues (2010) posited that alcohol-induced attention towards provocation produces a state of general negative affect that may subsequently generate a refined affective state of *anger*. Angry affect may then promote aggression by activating scripts in the associative network that invoke concepts of revenge and retaliation, further focusing the individual on the actions of the instigator and producing a state of excited arousal that impels an approach behavior such as aggression (Carver & Harmon-Jones, 2009). While the literature has clearly established that high levels of trait anger and aggressive personality traits moderate the effects of alcohol on general aggression (Bailey & Taylor, 1991; Bushman & Cooper, 1990; Eckhardt & Crane, 2008; Parrott & Giancola, 2004) as well as IPA (Abbey, Parkhill, Jacques-Tiura, & Saenz, 2009; Crane, Godleski, Przbyla, Schlauch, & Testa, 2016; Leonard, 2008), very little research has examined these factors as potential mediators of the association between alcohol intoxication and IPA perpetration. While prior laboratory

research using an alcohol administration paradigm suggests that IPA perpetration is more likely among individuals with a history of IPA who are intoxicated, provoked, and who experience an increase in state anger during provocation (Eckhardt, 2007), no formal tests of mediation were conducted in this research. No other studies have directly examined anger-related variables as a mechanism that mediates alcohol-facilitated IPA.

Aggressive cognitions.

A variety of models that outline aggression etiology (e.g., Anderson & Bushman, 2002; Berkowitz, 2008; Huesmann, 1988) predict that attentional biases favoring conflict and aggression lead to excessive and biased cognitive processing about the provocation, the transgressor, and the behavioral responses required to resolve the provocative situation (Giancola et al., 2010). This prediction has been supported in conditions involving an insulting provocation delivered by a laboratory confederate (Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005) or after imagining anger-inducing autobiographical memories (Rusting & Nolen-Hoeksema, 1998). In the context of IPA, a variety of theoretical approaches have highlighted the importance of aggressogenic cognitions as treatment-relevant correlates of IPA perpetration. Social information processing (SIP) models of IPA suggest a multi-stage approach to understanding the processing of social information (Holtzworth-Munroe, 1992) involving decoding socially relevant external information, choosing an appropriate response to the incoming information through the use of decision-making skills, and enacting the chosen behavioral response. Applications of SIP theory to the etiology of IPA have been supported by research demonstrating a link between aggressogenic cognitions and both physical and psychological IPA perpetration (Eckhardt & Dye, 2000; Morris, Mrug, & Windle, 2015; Taft, Schumm, Marshall, Panuzio, & Holtzworth-Munroe, 2008). Laboratory-based studies have found that during anger induction, IPA perpetrators show more aggressogenic cognitions such as hostile attribution biases, irrational beliefs, and cognitive biases compared to nonviolent men (Eckhardt, Barbour, & Davison, 1998; Eckhardt & Crane, 2015; Eckhardt, Jamison, & Watts, 2002; Holtzworth-Munroe & Hutchinson, 1993). However, researchers have not yet examined how acute alcohol intoxication impacts the nature and extent of aggressive cognitions as mediating mechanisms, especially among individuals already at risk for IPA perpetration.

The Present Study

Acute alcohol intoxication is a significant contributing cause of IPA perpetration. Extant literature strongly indicates the need to develop clear and testable models of alcohol-facilitated IPA etiology and maintenance. Perhaps the most critical, and underdeveloped, component of this effort is to elucidate the putative mechanisms through which alcohol-facilitated IPA perpetration can be prevented and/or reduced.

To address this need, the present study used alcohol administration research methods and a validated measure of laboratory aggression (the Taylor Aggression Paradigm; Taylor, 1967) to test a mediational model of alcohol-facilitated IPA perpetration. In doing so, this study represents a significant shift in research on alcohol-facilitated IPA perpetration for two key reasons. First, in contrast to most research on alcohol and IPA perpetration, we investigated

how specific affective and cognitive processes may mediate the effect of alcohol on IPA. While myriad laboratory- and field-based studies have examined moderators of the alcohol-aggression link by demonstrating in whom and in which situations alcohol facilitates aggression, we examined affective and cognitive *mediators* of alcohol-facilitated IPA perpetration. This laboratory-based investigation was novel in that we assessed "in-the-moment" affective and cognitive mediators of alcohol-facilitated IPA via the Facial Action Coding System (FACS; Ekman & Friesen, 1975) and Articulated Thoughts in Simulated Situations paradigm (ATSS; Davison et al., 1983), respectively. Unlike self-report measures of state affect, facial coding can be conducted unobtrusively and capture affect in real time (e.g., Ekman, Davidson, & Friesen, 1990). Similarly, the ATSS procedure is a protocol analysis approach that captures automatic, affectively-linked cognition that is more accessible and reportable during simultaneous activation of angry affect in the presence of relevant contextual cues (Zanov & Davison, 2010). Assessing these mediators during the laboratory aggression paradigm allowed us to examine whether the verbalization of aggressive cognitions (cognitive mediator) or facial expressions of negative affect or anger (affective mediator) during a given trial of the aggression task predicted IPA perpetration on the subsequent trial of the aggression task (which occurred seconds later). To our knowledge, this is the first attempt to examine the temporal relation between acute affective and cognitive mediators of alcohol-facilitated IPA perpetration.

Second, we recruited a high-risk sample of men and women with histories of heavy alcohol use and intimate partner conflict from two large urban areas in different geographical regions of the U.S. Despite literature suggesting that heavy drinkers are most at-risk for perpetrating IPA (e.g., Chermack et al., 2000), the majority of laboratory-based alcohol-aggression research recruits social and non-problem drinking samples. Thus, the present research was designed to better understand how alcohol facilitates aggression when at-risk individuals are placed in provoking situations.

Based on the reviewed literature, we made the following predictions:

Hypothesis 1: Compared to sober participants, intoxicated participants will display higher levels of laboratory-based IPA in response to partner provocation.

Hypothesis 2: Compared to sober participants, intoxicated participants will exhibit higher levels of negative affect, anger, and aggressive cognitions in response to partner provocation.

Hypothesis 3: Changes in negative affect, anger, and aggressive cognitions will mediate the association between alcohol intoxication and laboratory-based IPA in response to partner provocation.

Method

Participants

Participants were individuals nested within intimate heterosexual couples recruited from two U.S. cities who responded to print and online advertisements. Interested couples were screened separately by telephone to assess eligibility for the two-session study. To be

eligible, couples had to be dating for at least one month, be at least 21 years of age, and identify English as their native language. Couples were excluded if either partner reported serious head injuries, a medical or psychiatric condition in which alcohol is medically contraindicated, or a desire to seek treatment for alcohol use. Couples in which the female partner tested positive on a urine pregnancy test were also excluded. In addition, at least one partner – termed the index participant – was required to (1) report consumption of an average of at least five (for men) or four (for women) standard drinks per occasion at least twice per month during the past year, and (2) evidence perpetration of at least one act of psychological or non-severe physical IPA in the past year against the partner via self- or partner-report on the Revised Conflict Tactics Scale (Straus, Hamby, Boney-McCoy, & Sugarman, 1996). If both partners within a couple were eligible to serve as the index participant, one individual was selected randomly (i.e., coin flip).

Within one week of completing the telephone screening interview, ineligible couples were contacted by phone and informed that they would not be eligible to participate. Eligible couples were contacted by phone and scheduled for a Session 1 appointment during which eligibility criteria were reassessed. In addition, participants who endorsed a non-heterosexual identity or past-year severe physical IPA during Session 1 were excluded at this time. This process resulted in 289 eligible couples who presented to Session 2. Upon arrival to the laboratory for Session 2, all eligibility criteria were confirmed. Following a manipulation check, 40 participants were excluded due to various reasons, including not reaching the required Breath Alcohol Concentration (BrAC; $n = 7$), not being deceived ($n = 12$), voluntarily withdrawing during Session 2 ($n = 11$), and unpredictable miscellaneous events ($n = 10$) such as not understanding instructions or not being able to finish the alcoholic beverage.

The final sample included 249 participants (148 men, 101 women). Site 1 enrolled 146 of the index participants in the study, with the remaining 103 index participants enrolled at Site 2. See Table 1 for participant demographic information. This study was approved by each university's Institutional Review Board. While this study was not preregistered on an open science website, the sample size as well as the model presented and analyzed in this report closely follow our funded NIH grant proposal from 2012 (1R01AA020578).

Measures

Demographic Form.—This form obtains information such as age, self-identified sexual orientation, race, relationship status and length, years of education, and yearly family income.

Revised Conflict Tactics Scale (CTS-2; Straus et al., 1996).—This 78-item self-report instrument measures a range of events that occur during disagreements within intimate relationships. Participants use a 0 (never) to 6 (more than 20 times) scale to report how many times they have engaged in these behaviors over the past year. In the present study, the minor and severe physical subscale and the minor and severe psychological subscales were used to determine eligibility. Dichotomous scores were derived from each subscale, wherein a score of "1" was assigned to participants who were identified via self- or

partner-report as having perpetrated at least one of the acts on these subscales with the exception of severe physical aggression subscale – those endorsing severe physical IPA were ineligible. Only participants who obtained a score of "1" were deemed eligible for the study.

Drinking patterns questionnaire.—The National Institute on Alcohol Abuse and Alcoholism's (2003) recommended set of alcohol consumption questions were administered to measure participants' past year alcohol use. Heavy drinking was assessed with the question, "During the last 12 months, how many alcoholic drinks did you have on a typical day when you drank alcohol?" A standard drink was defined as one 12-ounce beer, one 5-ounce glass of wine, one mixed drink with 1.5-ounces of 80-proof hard liquor, or a "shot" of hard liquor. Frequency of consumption during the past year was assessed with the question "During the last 12 months, how often did you usually have any kind of drink containing alcohol?" Frequency of binge drinking during the past year was assessed with the question "During the last 12 months, how often did you have 5 or more (males) or 4 or more (females) drinks containing any kind of alcohol within a two hour period?"

Alcohol Use Disorders Identification Test (AUDIT; Babor, Biddle-Higgins, Saunders, & Monteiro, 2001)—This 10-item scale measures problematic drinking and was used to provide personalized alcohol use psychoeducation and treatment referrals prior to discharge. Per AUDIT recommendations and the ethical guidelines of alcohol administration research, all participants received literature focused on the reduction of problems or harms associated with heavy drinking. Further, participants who scored 16-19 also received referrals for brief counseling and continued monitoring; participants who scored 20 or above also received referrals for further diagnostic evaluation for an alcohol use disorder.

Beverage Administration

Upon arrival to the laboratory for Session 2, participants were randomly assigned to an alcohol (n = 122) or no-alcohol control (n = 127) beverage condition. Participants in the alcohol condition received a dose of 0.99 g/kg (men) or 0.90 g/kg (women) body weight of 95% alcohol USP mixed at a 1:5 ratio with Tropicana orange juice. This dose reliably produces BrAC levels between 0.08% and 0.12%. Participants in the no-alcohol control beverage condition received an isovolemic beverage consisting of only orange juice. All participants were allotted 20 minutes to consume their beverage. Participants were explicitly informed whether or not their drink contained alcohol.

Taylor Aggression Paradigm

A modified version (Giancola & Zeichner, 1995) of the Taylor Aggression Paradigm (TAP; Taylor, 1967) was used to assess direct physical aggression. The hardware for the task was developed by Coulbourn Instruments (Allentown, PA) and the computer software was developed by Vibranz Creative Group (Lexington, KY). The TAP was presented as a reaction time competition in which electric shocks are administered to and received from a "fictitious" opponent. Participants were seated at a table in a small room facing a computer screen and keyboard. The numbers *1* through *10* on the computer keyboard were labeled from *low* to *high* to allow participants to determine varying levels of shock to administer.

The keyboard and monitor were connected to a computer located in an adjacent room out of the participant's view. *Physical intimate partner aggression (IPA)* was defined as the average of standardized scores for the intensity and duration of shocks selected (i.e., TAP physical aggression). This scoring procedure was used because previous research has demonstrated that shock intensity and shock duration are highly correlated and part of a more general construct of direct, physical aggression (Carlson, Marcus-Newhall, & Miller, 1990; Hyatt, Chester, Miller, & Zeichner, 2019). The TAP and other similar laboratory paradigms have been repeatedly shown to be safe and valid measures of aggressive behavior (Giancola & Parrott, 2008; Parrott, Miller, & Hudepohl, 2015).

Facial Expression Coding

Facial expressions were coded using the Facial Action Coding System (FACS; Ekman, Friesen, & Hagar, 2002). The FACS is a comprehensive, anatomically-based system that classifies all observable facial activity into 44 unique action units (AUs). In the FACS, coders do not interpret emotions displayed. Instead, they describe all visible facial movements in terms of discrete AUs and combinations of AUs. Facial coding can be conducted unobtrusively and can capture discrete emotions or general affect in real time (Ekman et al., 1990; Rosenberg & Ekman, 1994; Sayette & Parrott, 1999; Wallace & Taylor, 2009). The FACS provides accurate and specific information across a range of emotional experiences (Ekman, Friesen, & O'Sullivan, 1988; Ekman & Rosenberg, 1997; Rosenberg, Ekman, & Blumenthal, 1998) and has successfully assessed distinct emotions in laboratory aggression research (Parrott, Gallagher, & Zeichner, 2012; Parrott, Zeichner, & Stephens, 2003; Wallace & Taylor, 2009).

Definition of coding intervals.—During each trial in the competitive task, participants' facial expressions were digitally and continuously recorded for subsequent coding with a high definition button camera that was hidden in a small box underneath the computer monitor. Only a specific time interval (10-sec) during each trial was coded. This time interval began 1-sec after the participant was informed as to whether s/he won or lost a trial and ended after a 6-sec "window" during which the participant administered or received an electric shock. Thus, facial expressions were coded during select periods of time when the adversarial interaction was most likely to be emotionally evocative. Facial coding was conducted using the Observer XT 11.0 software package (Noldus Information Technology, Leesburg, VA, USA).

Interpretation of facial codes.—Following prior work (Ekman & Friesen, 1975; Parrott et al., 2003; Rosenberg et al., 1998), core actions of anger include lowering and bringing together of the eyebrows (AU 4), raising of the eyelids (AU 5), and tightening and/ or compressing of lips (AU 23 and AU 24). All three facial areas (i.e., eyebrows, eyes, and lips) must be active to unambiguously infer the presence of anger. If facial areas are not all active, ambiguity can be reduced by considering the context of the expression (Ekman & Friesen, 1975). Given that the current investigation involved a context in which anger was likely to be elicited (i.e., an adversarial interaction in which provoking shocks are administered to the participant), activation of at least two facial areas was required in order to interpret the AUs as an expression of anger. However, of the three anger-related facial areas, only AU 23 and

AU 24 are associated solely with the expression of anger. Thus, the activation of either of these AUs in the absence of AU 4 or AU 5 was also identified as anger. This method of interpretation is considered conservative enough to exclude false positives, yet flexible enough to capture anger-related affect. In addition, facial expressions that included any combination of these core actions in addition to core actions of other emotions were labeled as anger. It was deemed appropriate to include the latter facial expressions due to the broad emotion-disinhibiting effects of alcohol and the fact that a given emotion is not necessarily expressed without some concurrent features of other emotions (Ekman, 1992; Ekman & Friesen, 1975). State negative affect was operationalized by summing the number of facial expressions containing an AU or AU combination associated with basic negative emotions, which included anger (as described above), fear (e.g., AU1 [inner brow raiser] + AU2 [outer brow raiser] + AU4 [brow lowerer]), sadness (e.g., AU 15 [lip corner depressor] or AU1+ AU4), disgust (e.g., AU9 [nose wrinkler] or AU10 [upper lip raiser]), and contempt (e.g., unilateral AU10 or unilateral AU14). Using these parameters, state anger and state negative affect were operationalized by summing the number of anger and negative affect facial expressions, respectively, displayed during each trial of the competitive task. Coders showed excellent interrater agreement (κ = .77).

Articulated Thoughts in Simulated Situations

A modified version of the ATSS paradigm (Davison et al., 1983) was used to collect and code verbalizations of aggressive cognitions while index participants completed the TAP. Before participants began the TAP, the experimenter provided them with the following instructions: "*Remember, we want to know what you are thinking as the task goes on. So, we encourage you to 'talk out loud' and let us know your thoughts. You can say whatever you want about your performance, your partner, or what you are thinking about at that moment. You won't be able to hear each other – the intercom has been turned off between your rooms*."

The duration and content of each ATSS verbalization was coded using the Observer XT 11.0 software package. Two trained raters transcribed and coded each articulation reflecting the verbalization of aggressive cognitions throughout the duration of the TAP. ATSS statements reflecting aggressive cognitions were defined in a manner that was consistent with prior usage of this outcome (Eckhardt, 2007; Eckhardt & Crane, 2015) and referred to any stated intention to cause physical harm to the partner (e.g., "I'm going to shock you with a ten for that"), any insulting or emotionally harmful statement about the partner (e.g., "You are such an asshole"), and any statement intended to instigate a confrontation with the partner (e.g., "Why don't you try saying that to my face?"). Coders showed excellent interrater agreement (κ = .95).

Procedure

Participants presented to the lab on two separate days. To minimize potential coercion, informed consent was obtained separately (i.e., in separate rooms) for the index participant and his/her partner upon arrival to each session. Session 1 involved reassessment of all eligibility criteria as well as administration of a questionnaire battery. For Session 2, participants were met in a room separate from the aggression laboratory. In order to disguise

the task as a measure of aggression, participants were given a fictitious cover story. They were informed that the purpose of the study was to examine the relation between alcohol and reaction time under competitive conditions. As such, they would consume an alcoholic or nonalcoholic beverage prior to engaging in a competitive reaction time task against their partner. At this time, participants were also weighed and their BrAC was assessed to confirm sobriety. Participants with a BrAC above 0% were rescheduled on a subsequent day.

Both members of the couple were then escorted to the index participant's testing room. Participants received instructions regarding the reaction time competition. For each trial, participants were informed that shortly after the words "*Get Ready*" appeared on the screen, the words "*Press the Spacebar*" would appear, at which time they would press and hold down the spacebar. Following this instruction, the words "*Release the Spacebar*" would appear, at which time they would 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*." A "loss" was signaled by the words "*You Lost. You Get a Shock*." A winning trial required participants to deliver a shock to their partner, and a losing trial resulted in receiving a shock from their partner. Participants were told that they had a choice of 10 different shock intensities to administer at the end of each winning trial for a duration of their choosing. Participants were informed that, while they could not elect to not shock their partner, the shock button "1" would deliver a low-intensity shock that is best characterized as "very mild" and "definitely not painful."

Next, the partner was escorted to a separate testing room to ostensibly consume his or her beverage. In actuality, this participant received a full debriefing of the study and was compensated and discharged. This procedure was done as a safety precaution so that the partner could provide consent for the deception protocol to continue with the index participant. Upon obtaining this consent, index participants were administered an alcoholic or non-alcoholic beverage. After consuming their beverages, index participants' pain thresholds were assessed to determine the intensity parameters for the shocks they would receive. The pain threshold assessment was accomplished via the administration of one-second duration shocks presented in an incremental stepwise intensity method from the lowest available shock setting, which is imperceptible, until the shocks reached a reportedly "painful" level. All shocks were administered through two electrodes that were attached to the index and middle fingers of the nondominant hand. Participants were asked to state when the shocks were "first detectable" and then when they reached a "painful" level. This procedure was conducted while index participants were seated in the testing room and the experimenter was in an adjacent control room. They communicated through an intercom.

After the pain thresholds were determined and participants reached an ascending BrAC of 0.075%, participants completed six "practice" TAP trials, ostensibly against their intimate partner, so that they could become familiar with the procedure. In actuality, the "practice" trials were rigged so that all participants received physical and verbal provocations from their partner in order to create an adversarial interpersonal interaction. Participants "lost" four out of six trials and received moderate intensity shocks (i.e., 4's and 5's) on each of these losing trials. Next, participants received scripted written negative feedback on a form they believed to be from their partner. Most relevant to this feedback was the partner's

statement that s/he intended to deliver the highest possible level shock in the subsequent reaction time trials.

Upon reaching a BrAC of 0.08%, the full aggression task commenced and consisted of 20 trials (10 wins and 10 losses). On losing trials, participants received shocks from their "partner" that were one second in duration and ranged from 95% (a "9") to 100% (a "10") of their highest tolerated shock intensity. A specially designed "volt meter" and the illumination of one of the 10 "shock lights" (ranging from 1 [low] to 10 [high]) on the computer screen signaled to the participant the shock that s/he or the partner selected. A randomly generated win/loss sequence was predetermined and incorporated into the computer program that executed the task. All participants received the same sequence. A computer controlled initiation of trials, administration of shocks to participants, and recordings of their responses.

Immediately upon completion of the TAP, BrACs were measured. Participants were then asked a variety of questions to assess indirectly the credibility of the experimental manipulation (see below), debriefed, and compensated. All individuals who received alcohol remained in the laboratory until their BrAC fell to .03%, at which time they were escorted to prearranged transportation by laboratory staff.

Data Analytic Plan

Descriptive statistics and mean comparisons were computed using SPSS v.25, and all primary analysis models were estimated using Mplus v.8. The primary analysis models were conducted within a multilevel modeling framework, with trials nested within individuals, to account for person-level dependence. Mediator variables (i.e., facial expressions of negative affect and anger, aggressive cognitions) were scored in two different ways for analysis: (1) within person (trial-by-trial) scores were computed to represent the lagged time intervals leading up to each shock administration, respectively; and (2) between-person (across trial) scores were computed as each participant's total number of negative affect facial expressions, anger facial expressions, or aggressive cognitions across all trials, respectively. This scoring system and trial-within-person framework allowed for both distinct and compositional interpretations of trial-by-trial and across-trial effects. It was not necessary to additionally nest within dyad, as only one member of the couple completed the TAP. All primary models controlled for data-collection site and participant gender. All multilevel models were fit using weighted least squares mean and variance adjusted estimation, which is robust to non-normally distributed data (Brown, 2006). Multi-level mediation was tested within a multilevel structural equation modeling framework, with average trial-by-trial effects of anger expressions and aggressive verbalizations estimated as latent slopes in the model at the between-subjects level, (Preacher, Zyphur, & Zhang, 2010); thus residual variances were estimated at the between-subjects level for anger expressions and aggressive verbalizations in addition to the TAP physical aggression outcome and at the within-subjects level for anger expressions and aggressive verbalizations. This approach allowed us to compute indirect effects that combined the effect of alcohol on relevant mediators across participants (i.e., between-subjects effects) and the effects of those mediators on IPA perpetration across participants (i.e., between-subjects effects) and on a trial-by-trial basis

(i.e., within-subjects effects). Any predictors and paths that did not significantly contribute to the model were trimmed throughout the process to test and present a parsimonious final model. While the present report is part of a larger parent project that included other measures, all measures, data analytic plans, and power considerations included in the present report were proposed a priori in our funded NIH grant proposal from 2012 (1R01AA020578).

Results

Manipulation Check

Aggression task checks.—Participants were interviewed to confirm their belief that they were competing against their partner on a "reaction time" task and that this task was not a measure of aggression. First, participants were asked whether or not they thought the task was a good measure of reaction time. Second, they were asked how they thought their partner performed on the task. The main criteria for exclusion were participants' beliefs that they were not actually competing against their partner or that the task was a measure of aggression. As noted above, of the 289 participants, 12 (4%) indicated that the task was not a measure of reaction time and/or that they were not actually competing against their partner.

BrAC levels.—All participants tested in this study had BrACs of 0.00% upon entering the laboratory. A paired samples *t*-test indicated that participants' BrACs in the alcohol group were significantly higher post-TAP ($M = .106\%$, $SD = .016$) than pre-TAP ($M = .093\%$, $SD = .014$), $t(120) = -9.55$, $p < .001$. This finding, and inspection of these data at the individual level, confirmed that all intoxicated participants were on the ascending limb of the BrAC curve during the experimental procedures. Participants in the no-alcohol control condition had a mean BrAC of 0.00% before and after the experimental procedures.

Primary analyses.—Table 1 contains descriptive statistics related to both alcohol use and previous IPA perpetration and victimization. IPA scores reflect the highest report (self or partner) of an individual's behavior. Aggressive cognitions did not significantly covary with anger expressions at the within-subjects (trial-by-trial; $r = -.005$, $SE = .004$, $p = .93$) or between-subjects level ($r = .006$, $SE = .009$, $p = .52$). IPA significantly covaried with aggressive verbalizations at both the within-subjects ($r = .006$, $SE = .003$, $p = .03$) and between-subjects levels ($r = .019$, $SE = .005$, $p = .001$). IPA did not significantly covary with anger expressions at either the within-subjects ($r = .005$, $SE = .03$, $p = .85$) and between-subjects levels ($r = .04$, $SE = .05$, $p = .32$). To test Hypothesis 1, we simply regressed the TAP physical aggression score on beverage condition (Alcohol vs. No-Alcohol control) at the between-person level. As expected, participants who consumed alcohol displayed significantly higher levels of IPA on the TAP than those who did not consume alcohol ($B = .68$, $SE = .30$, $p = .023$). To test Hypothesis 2, we fit a multivariate regression model at the individual level with facial expressions of negative affect, facial expressions of anger, and aggressive cognitions regressed on beverage condition. Our hypotheses were partially supported, as facial expressions of anger ($B = -.36$, $SE = 12$, $p < .001$) and aggressive cognitions ($B = .07$, $SE = .02$, $p < .001$) significantly differed as a function of beverage condition. Interestingly, participants who consumed alcohol evidenced significantly more

aggressive cognitions but significantly fewer anger expressions than those who consumed the no-alcohol control beverage. Facial expressions of negative affect did not significantly differ across beverage conditions ($B = -.145$, $SE = .12$, $p = .23$). Thus, this variable was removed from subsequent models.

We then fit a multiple mediator model to test Hypothesis 3. Results from fitting the multiple mediator model supported the aggressive cognitions pathway (indirect effect: $B = .07$, $SE = .03$, $p = .025$), but not the anger facial expressions pathway (indirect effect: $B = -.10$, $SE = .10$, $p = .311$), from alcohol to IPA perpetration (see Figure 1, top panel). These results also supported a significant and positive within-person effect of aggressive cognitions, but not anger facial expressions, on IPA perpetration during the subsequent opportunity trial (see Figure 1, bottom panel).[1] That is, participants who verbalized aggressive cognitions during a given trial administered significantly higher shocks to their partner on the next trial in which they could administer a shock.

Thus, a final model was fit to isolate the aggressive cognitions mediational pathway (see Figure 2, top panel). Trial-by-trial means for aggressive cognitions ranged from .14 to .29 in the Alcohol group and .05 to .16 in the No-Alcohol Control group. Trial-by-trial means for IPA pepetration ranged from .03 to .17 in the Alcohol group and −0.11 to .07 in the No-Alcohol Control group. Results of this model supported significant between-person effects of alcohol on aggressive cognitions, and aggressive cognitions on IPA perpetration. These results also supported a significant overall within-person effect of aggressive cognitions on IPA perpetration during the subsequent opportunity trial (see Figure 2, bottom panel). Thus, as in the prior analysis, participants who verbalized aggressive cognitions during a given trial administered significantly higher shocks to their partner on the next trial in which they could administer a shock. Collectively, these within and between-person effects contributed to a significant indirect effect of beverage condition on IPA perpetration via aggressive cognitions ($B = .25$, $SE = .11$, $p = .02$). The direct, between-person effect of beverage condition on IPA perpetration was rendered non-significant after the aggressive cognitions pathway was estimated, which supports a full mediation interpretation.

Discussion

This multi-site study examined the association between acute alcohol intoxication and IPA perpetration using validated methods of alcohol administration and laboratory-assessed aggression in a high-risk sample of heavy-drinking, abusive couples. Hypothesis 1 posited that individuals with a history of IPA and heavy drinking would be more aggressive towards a putative partner on the TAP after consuming alcohol relative to sober participants. This hypothesis was supported; intoxicated subjects directed higher levels of aggression on the laboratory task towards an intimate partner than sober participants. Hypothesis 2 posited that in response to provocation from an intimate partner, intoxicated participants would exhibit higher levels of negative affect, anger, and aggressive cognitions relative to sober participants. This hypothesis was partially supported, as alcohol intoxication led to an

[1]We tested an additional model with anger expressions as the only mediator, to examine its effect unconditioned on aggressive verbalizations. The effect of anger expressions on IPA in this model was similar to what is presented in Figure 1.

increase in aggressive cognitions but not in anger or negative affect. Hypothesis 3 predicted that the association between alcohol intoxication and TAP partner aggression would be mediated by FACS-assessed negative affect and anger as well as ATSS-assessed aggressive cognitions. This hypothesis was partially supported. Analysis of indirect effects of alcohol on laboratory-based IPA perpetration which combined both between- and within-subject effects indicated that the positive association between alcohol use and partner-directed physical aggression was fully mediated by participants' aggressive cognitions, but not by expressions of negative affect or anger. In the sections below, we review the implications of these results in more detail and consider limitations that may impact how these findings are interpreted.

The finding that acute alcohol intoxication is associated with increased partner-directed aggression is consistent with broader research on the association between alcohol misuse and IPA (for reviews, see Cafferky et al., 2018; Eckhardt, Parrott, & Massa, in press). Our tests of both between- and within-subjects effects support articulated aggressive cognitions as a significant mediator of alcohol-facilitated IPA perpetration. These findings are consistent with previous research, including alcohol administration studies using the ATSS paradigm (Eckhardt, 2007), suggesting that heavy drinking is associated with IPA primarily among individuals who endorse dispositional tendencies in aggression-related cognitive biases (e.g., Tharp et al., 2012) and who are susceptible to alcohol-related shifts in attention toward provocative cues (Gallagher et al., 2010; Massa, Subramani, Eckhardt, & Parrott, 2019).

Cognitive factors have been theorized to mediate the alcohol-aggression association for quite some time (e.g., Giancola, 2000; Heinz, Beck, Meyer-Lindenberg, Sterzer, & Heinz, 2011; Hoaken, Giancola, & Pihl, 1998), but there has been very little direct evidence in support of these models, especially in the context of IPA perpetration. The present findings provide important evidence about the proximal effects of alcohol on cognitive processing mechanisms and extend prior work in this area by assessing participants' spontaneously verbalized aggressogenic cognitions, experienced during a highly provocative conflict with their intimate partners, as an intervening mechanism that fully mediates the effects of alcohol intoxication on partner-directed aggression. The present findings may align with a recent study of the neurocognitive correlates of alcohol-facilitated aggression (using the TAP), which demonstrated that the neural mechanisms underlying intoxicated TAP aggression were found in areas associated with the processing of threatening stimuli and inhibitory control (Denson, Blundell, Schofield, Schira, & Kramer, 2018). Future research that merges neural imaging approaches with concurrent assessment of participants' cognitive experiences during alcohol intoxication would provide important data regarding the broader neuropsychological mechanisms that contribute to provoked aggression towards an intimate partner.

Our analyses failed to find a significant indirect effect of facial displays of general negative affect or specific expressions of anger. In fact, participants who consumed alcohol evidenced significantly fewer anger facial expressions than those who consumed the no-alcohol control beverage. Because post-hoc explanations of why a significant negative, as opposed to positive, association was found would be speculative at best, we do not do so here. That

stated, it is important to note that these results are consistent with prior studies suggesting that heavy alcohol use did not predict subsequent increases in anger towards an intimate partner (Crane, Testa, Derrick, & Leonard, 2014; Eckhardt, 2007). Such findings stand in contrast to prior reviews of the literature suggesting that negative affect, especially externalizing-related affective experiences such as dispositional anger, is an important risk factor for IPA perpetration (e.g., Birkley & Eckhardt, 2015) and may be an important moderator of alcohol-involved IPA (Eckhardt, 2007; Shorey, McNulty, Moore, & Stuart, 2017). However, it is important to note that prior studies in this area have neither specifically conceptualized nor directly examined negative affective displays, recorded unobtrusively via hidden camera during provocation, as mediating mechanisms of alcohol-facilitated IPA (for additional discussion see Eckhardt, Parrott, & Sprunger, 2015). Thus, our specific methodology of assessing negative affective experiences may have allowed for a more fine-grained analysis of the mediating mechanisms involved in discrete instances of IPA and therefore may stand in contrast to prior research in this area.

The broader pattern of results reinforces the importance of conceptualizing the factors involved in alcohol-facilitated IPA according to the relative balance between variables that *instigate* and *impel* aggression versus those that *inhibit* aggression (Leonard & Quigley, 2017; Taylor & Leonard, 1983). As described by the Instigating-Impelling-Inhibiting ("I^3") Model (Finkel, 2007; 2014), the greatest likelihood for IPA occurs when instigation (e.g., provocation) and impellance processes (e.g., aggressogenic traits) are strong, inhibitory processes (e.g., emotion regulation) are weak, and disinhibitory processes (e.g., alcohol intoxication) are present (e.g., Watkins et al., 2015). From the standpoint of the I^3 Model (Finkel, 2014), our results suggest a more mechanistic understanding of the pathway towards alcohol-facilitated IPA. Specifically, alcohol intoxication exacerbated participants' verbalizations of aggressive cognitions following instigation (being shocked by an intimate partner), which in turn caused an increase in partner-directed aggression. Within I^3, aggressive cognitions would be conceptualized as both an impelling factor as well as a *behavioral proclivity* mediator, which must temporally follow the instigator and temporally precede the behavioral outcome; when present, the behavioral proclivity mediator results in the enactment of a behavior unless it is overridden by inhibitory processes (Finkel, 2014; p. 13). In the present study, aggressive cognitions are therefore an impelling behavioral proclivity factor that mediates the association between provocation and IPA, which is further moderated by the disinhibiting effects of alcohol intoxication (see Figure 3). Future research that applies this type of multidimensional approach to understanding the mechanisms of alcohol-facilitated IPA will better inform the development of effective intervention and prevention programs, as discussed in the next section.

Clinical Implications

Individuals with a history of heavy drinking and IPA, such as those in the present study, have few effective choices for interventions that reduce these problematic behaviors. Standard intervention programs for IPA perpetrators are not typically structured to address co-occurring substance use problems (Murphy & Ting, 2010), resulting in separate referrals to substance treatment programs, often held in different locations by a different set of treatment providers with little evidence of effectiveness (Klostermann, 2006; Klostermann, Kelley,

Mignone, Pusateri, & Fals-Stewart, 2010; Kraanen, Vedel, Scholing, & Emmelkamp, 2014). While there are promising findings regarding the IPA-reducing effect of substance-focused interventions (Easton & Crane, 2016; Murphy & Ting, 2010; O'Farrell, Murphy, Stephan, Fals-Stewart, & Murphy, 2004), this effect is limited in that gains in IPA reduction are lost when the individual reengages in alcohol misuse (Easton & Crane, 2016; Mignone, Klostermann, & Chen, 2009). In the context of the present study, additional interventions are needed that could target aggressogenic cognitive processes *that could be applied during episodes of acute intoxication* (Gallagher & Parrott, 2011; Giancola, Duke, & Ritz, 2011), such as recently developed "just-in-time" adaptive interventions delivered through smartphone technology (e.g., Goldstein et al., 2017; Hardeman et al., 2019).

The results of the present study offer some potential areas for further inquiry. As described earlier, the attention-allocation model of AMT (Steele & Josephs, 1990) allows for the counterintuitive hypothesis that intoxicated persons will behave less aggressively than sober individuals if their attention is redirected away from cues of instigation by more salient cues of inhibition. Proposed interventions grounded in this model call for the use of highly salient and easy-to-process inhibitory cues designed to capture the inebriate's attention. With a focus on such cues, the likelihood of IPA should be reduced. Basic research demonstrates the efficacy of AMT-informed laboratory-based interventions (e.g., Gallagher & Parrott, 2011; 2016; Giancola & Corman, 2007). Thus, there is an evidence base to support the development of interventions that could be disseminated at the individual or societal level (e.g., for a review, see Giancola et al., 2010; Parrott & Eckhardt, 2017). The present findings suggest that such efforts should specifically target inhibitory cues that would reduce the likelihood of alcohol-induced aggressive cognitions, as these cognitions are a more proximal predictor of IPA perpetration.

Limitations

The findings of the present study should be interpreted in light of several important limitations. First, there is no doubt that our laboratory-aggression task did not perfectly model situations encountered by couples who are in conflict. For example, when physical IPA occurs, partners are face-to-face with a complex progression and escalation of affect-laced conflict that is not captured in the standardized provocation utilized in the present study. This limitation reflects more general concerns raised about laboratory measures of aggression, such as the TAP, where the balance of affective and cognitive factors may be unique (Ferguson & Rueda, 2009; Tedeschi & Quigley, 1996). That stated, there is extensive literature that addresses concerns of ecological validity in laboratory-based research (e.g., Berkowitz & Donnerstein, 1982; Mook, 1982) and the internal and external validity of the TAP specifically (e.g., Chester & Lasko, 2019; Giancola & Parrott, 2008; King & Russell, 2019; Miller, Wilson, Hyatt, & Zeichner, 2015). Importantly, despite these criticisms, our results demonstrate that even under controlled circumstances, our predictions were still supported. Second, the present findings are limited to a situation in which participants' experience of provocation was timed to a relatively high peak blood alcohol level. In naturalistic settings, the timing and dose of alcohol consumption relative to the experience of provocation is far more variable. To address this limitation, ambulatory studies which can simultaneously track blood alcohol levels and IPA in real-time are needed. Third, the present

sample was unique in that all participants were considered as high-risk for IPA perpetration given their history of heavy drinking and previous perpetration of IPA. Thus, it is not clear whether the present findings generalize to lower-risk individuals, or those who perpetrate severe forms of IPA. Fourth, while ample evidence supports the use of the ATSS paradigm to assess "in-the-moment" cognitive processes (Zanov & Davison, 2010), it is possible that asking participants to report on their thinking might have increased attentional focus and potentially altered participants' naturalistic cognitive processes, especially among individuals with inhibitory control deficits. Finally, the generalizability of the findings is limited to cisgender heterosexual individuals. Although some risk factors predict IPA perpetration across sexual and gender identities, future research should examine the role of risk factors specific to sexual and gender minorities, such as sexual minority stress (Edwards, Sylaska, & Neal, 2015).

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References

Abbey A, Parkhill MR, Jacques-Tiura AJ, & Saenz C (2009). Alcohol's role in men's use of coercion to obtain unprotected sex. Substance Use & Misuse, 44(9-10), 1329–1348. doi:10.1080/10826080902961419 [PubMed: 19938921]

Abramsky T, Watts CH, Garcia-Moreno C, Devries K, Kiss L, Ellsberg M, … Heise L (2011). What factors are associated with recent intimate partner violence? Findings from the WHO multi-country study on women's health and domestic violence. BMC Public Health, 11, 109–127. [PubMed: 21324186]

Anderson CA, & Bushman BJ (2002). Human aggression. Annual Review of Psychology, 53, 27–51. doi:10.1146/annurev.psych.53.100901.135231

Attwood S, Parke H, Larsen J, & Morton KL (2017). Using a mobile health application to reduce alcohol consumption: A mixed-methods evaluation of the drinkaware track & calculate units application. BMC Public Health, 17, 394–417. [PubMed: 28511698]

Babor TF, Biddle-Higgins JC, Saunders JB, Monteiro MG (2001). AUDIT: The Alcohol Use Disorders Identification Test: Guidelines for use in primary health care. Geneva, Switzerland: World Health Organization.

Bailey DS, & Taylor SP (1991). Effects of alcohol and aggressive disposition on human physical aggression. Journal of Research in Personality, 25(3), 334–342. doi:10.1016/0092-6566(91)90024-K

Berkowitz L (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. American Psychologist, 45(4), 494–503. doi:10.1037/0003-066X.45.4.494

Berkowitz L (1993). Towards a general theory of anger and emotional aggression: Implications of the cognitive-neoassociationistic perspective for the analysis of anger and other emotions. In Wyer RS Jr. & Srull TK (Eds.), Perspectives on anger and emotion (pp. 1–46). Hillsdale, NJ: Lawrence Erlbaum Associates.

Berkowitz L (2008). On the consideration of automatic as well as controlled psychological processes in aggression. Aggressive Behavior, 34(2), 117–129. doi:10.1002/ab.20244 [PubMed: 18183563]

Berkowitz L (2012). A different view of anger: The cognitive-neoassociationistic conception of the relation of anger to aggression. Aggressive Behavior, 38(4), 322–333. doi:10.1002/ab.21432 [PubMed: 25363699]

Berkowitz L, & Donnerstein E (1982). External validity is more than skin deep: Some answers to criticisms of laboratory experiments. American Psychologist, 37, 245–257. doi:10.1037/0003-066X.37.3.245

Birkley EL, & Eckhardt CI (2015). Anger, hostility, internalizing negative emotions, and intimate partner violence perpetration: A meta-analytic review. Clinical Psychology Review, 37, 40–56. doi:10.1016/j.cpr.2015.01.002 [PubMed: 25752947]

Black MC, Basile KC, Breiding MJ, Walters ML, Merrick MT, Chen J, & Stevens MR (2011). The National Intimate Partner and Sexual Violence Survey (NISVS): 2010 summary report. Atlanta, GA.

Brown T (2006). Confirmatory factor analysis for applied research. New York: Guilford.

Brown W, & Leonard KE (2017). Does alcohol cause violence and aggression? In Sturmey P (Ed.), The Wiley handbook of violence and aggression. Hoboken, NJ: John Wiley & Sons. doi:10.1002/9781119057574.whbva029

Bushman BJ, & Cooper HM (1990). Effects of alcohol on human aggression: An integrative research review. Psychological Bulletin, 107(3), 341–354. doi:10.1037/0033-2909.107.3.341 [PubMed: 2140902]

Bushman BJ, Bonacci AM, Pedersen WC, Vasquez EA, & Miller N (2005). Chewing on it can chew you up: Effects of rumination on triggered displaced aggression. Journal of Personality and Social Psychology, 88(6), 969–983. doi:10.1037/0022-3514.88.6.969 [PubMed: 15982116]

Caetano R, Ramisetty-Mikler S, & Field CA (2005). Unidirectional and bidirectional intimate partner violence among White, Black, and Hispanic couples in the United States. Violence and Victims, 20(4), 393–406. doi:10.1891/088667005780927584 [PubMed: 16250407]

Cafferky BM, Mendez M, Anderson JR, & Stith SM (2018). Substance use and intimate partner violence: A meta-analytic review. Psychology of Violence, 8, 110–131. doi:10.1037/vio0000074

Carlson M, Marcus-Newhall A, & Miller N (1990). Effects of situational aggression cues: A quantitative review. Journal of Personality and Social Psychology, 58(4), 622–633. doi:10.1037/0022-3514.58.4.622 [PubMed: 14570078]

Carver CS, & Harmon-Jones E (2009). Anger is an approach-related affect: Evidence and implications. Psychological Bulletin, 135(2), 183–204. doi:10.1037/a0013965 [PubMed: 19254075]

Chermack ST, Fuller BE, & Blow FC (2000). Predictors of expressed partner and non-partner violence among patients in substance abuse treatment. Drug and Alcohol Dependence, 58, 43–54. doi:10.1016/S0376-8716(99)00067-8 [PubMed: 10669054]

Chester DS, & Lasko EN (2019). Validating a standardized approach to the Taylor Aggression Paradigm. Social Psychological and Personality Science, 10, 620–631. doi:10.1177/1948550618775408

Crane CA, & Easton CJ (2017). Integrated treatment options for male perpetrators of intimate partner violence. Drug and Alcohol Review, 36, 24–33. doi:10.1111/dar.12496 [PubMed: 28109172]

Crane CA, Godleski SA, Przbyla SM, Schlauch RC, & Testa M (2016). The proximal effects of acute alcohol consumption on male-to-female aggression: A meta-analytic review of the experimental literature. Trauma, Violence, & Abuse, 17(5), 520–531. doi:10.1177/1524838015584374

Crane CA, Testa M, Derrick JL, & Leonard KE (2014). Daily associations among self-control, heavy episodic drinking, and relationship functioning: An examination of actor and partner effects. Aggressive Behavior, 40, 440–450. doi:10.1002/ab.21533 [PubMed: 24700558]

Davison GC, Robins C, & Johnson MK (1983). Articulated thoughts during simulated situations: A paradigm for studying cognition in emotion and behavior. Cognitive Therapy and Research, 7, 17–39. doi:10.1007/BF01173421

Denson TF, Blundell KA, Schofield TP, Schira MM, & Krämer UM (2018). The neural correlates of alcohol-related aggression. Cognitive, Affective, & Behavioral Neuroscience, 18(2), 203–215. doi:10.3758/s13415-017-0558-0

Easton CJ, & Crane CA (2016). Interventions to reduce intimate partner violence perpetration among people with substance use disorders. International Review of Psychiatry, 28(5), 533–543. doi:10.1080/09540261.2016.1227307 [PubMed: 27696955]

Eckhardt CI (2007). Effects of alcohol intoxication on anger experience and expression among partner assaultive men. Journal of Consulting and Clinical Psychology, 75, 61–71. doi:10.1037/0022-006X.75.1.61 [PubMed: 17295564]

Eckhardt CI, Barbour KA, & Davison GC (1998). Articulated thoughts of martially violent and nonviolent men during anger arousal. Journal of Consulting and Clinical Psychology, 66(2), 259–269. doi:10.1037/0022-006X.66.2.259 [PubMed: 9583329]

Eckhardt CI, & Crane C (2008). Effects of alcohol intoxication and aggressivity on aggressive verbalizations during anger arousal. Aggressive Behavior, 34, 428–436. [PubMed: 18307248]

Eckhardt CI, & Crane CA (2015). Cognitive and aggressive reactions of male dating violence perpetrators to anger arousal. Journal of Interpersonal Violence, 30(8), 1348–1368. doi:10.1177/0886260514540330 [PubMed: 25023727]

Eckhardt CI, & Dye ML (2000). The cognitive characteristics of maritally violent men: Theory and evidence. Cognitive Therapy and Research, 24(2), 139–158. doi:10.1023/A:1005441924292

Eckhardt CI, Jamison TR, & Watts K (2002). Anger experience and expression among male dating violence perpetrators during anger arousal. Journal of Interpersonal Violence, 17(10), 1102–114. doi:10.1177/088626002236662

Eckhardt CI, Parrott DJ, & Massa AA (In press). Substance abuse and intimate partner violence perpetration. In Handbook of interpersonal violence across the lifespan. Springer.

Eckhardt CI, Parrott DJ, & Sprunger JG (2015). Mechanisms of alcohol-facilitated intimate partner violence. Violence Against Women, 21(8), 939–957. doi:10.1177/1077801215589376 [PubMed: 26059921]

Edwards KM, Sylaska KM, & Neal AM (2015). Intimate partner violence among sexual minority populations: A critical review of the literature and agenda for future research. Psychology of Violence, 5, 112–121. doi:10.1177/0886260514535260

Ekman P (1992). Telling lies: Clues to deceit in the marketplace, politics, and marriage. New York, NY: Norton.

Ekman P, Davidson RJ, & Friesen WV (1990) The Duchenne smile: Emotional expression and brain physiology II. Journal of Personality and Social Psychology, 58, 342–353. [PubMed: 2319446]

Ekman P, & Friesen WV (1975). Unmasking the face: A guide to recognizing emotions from facial cues. Englewood Cliffs, NJ: Prentice Hall.

Ekman P, Friesen WV, & Hagar JC (2002). Facial Action Coding System on CD-ROM. Salt Lake City, UT: Network Information Research.

Ekman P, Friesen WV, & O'Sullivan M (1988). Smiles when lying. Journal of Personality and Social Psychology, 54, 414–420. [PubMed: 3361418]

Ekman P, & Rosenberg EL (Eds.) (1997). What the face reveals: Basic and applied studies of spontaneous expression using the Facial Action Coding System (FACS). New York, NY: Oxford University Press.

Ferguson CJ, & Rueda SM (2009). Examining the validity of the modified Taylor competitive reaction time test of aggression. Journal of Experimental Criminology, 5, 121–137. doi:10.1007/s11292-009-9069-5

Finkel EJ (2007). Impelling and inhibiting factors in the perpetration of intimate partner violence. Review of General Psychology, 11(2), 193–207. doi:10.1037/1089-2680.11.2.193

Finkel EJ (2014). The I-cubed model: Metatheory, theory, and evidence. In Olson JM & Zanna MP (Eds.), Advances in experimental social psychology (Vol. 49, pp. 1–104). San Diego, CA: Academic Press.

Finkel EJ, & Eckhardt CI (2013). Intimate partner violence. In Simpson JA & Campbell L (Eds.), The Oxford handbook of close relationships (pp. 452–474). New York, NY: Oxford University Press.

Foran HM, & O'Leary KD (2008). Alcohol and intimate partner violence: A meta-analytic review. Clinical Psychology Review, 28, 1222–1234. doi:10.1016/j.cpr.2008.05.001 [PubMed: 18550239]

Friend J, Langhinrichsen-Rohling J, & Eichold BH (2011). Same-day substance use in men and women charged with felony domestic violence offenses. Criminal Justice and Behavior, 38(6), 619–633. doi:10.1177/0093854811

Gallagher KE, Hudepohl AD, & Parrott DJ (2010). Power of being present: The role of mindfulness on the relation between men's alcohol use and sexual aggression toward intimate partners. Aggressive Behavior, 36(6), 405–413. doi:10.1002/ab.20351 [PubMed: 20623578]

Gallagher KE, & Parrott DJ (2011). Does distraction reduce the alcohol-aggression relation? A cognitive and behavioral test of the attention-allocation model. Journal of Consulting and Clinical Psychology, 79(3), 319–329. doi:10.1037/a0023065 [PubMed: 21500889]

Gallagher KE, & Parrott DJ (2016). A self-awareness intervention manipulation for heavy-drinking men's alcohol-related aggression toward women. Journal of Consulting and Clinical Psychology, 84(9), 813. doi:10.1037/ccp0000118 [PubMed: 27176660]

Giancola PR (2000). Executive functioning: A conceptual framework for alcohol-related aggression. Experimental and Clinical Psychopharmacology, 8(4), 576–597. doi:10.1037/1064-1297.8.4.576 [PubMed: 11127429]

Giancola PR, & Corman MD (2007). Alcohol and aggression: A test of the attention-allocation model. Psychological Science, 18(7), 649–655. doi:10.1111/j.1467-9280.2007.01953.x [PubMed: 17614875]

Giancola PR, Duke AA, & Ritz KZ (2011). Alcohol, violence, and the alcohol myopia model: Preliminary findings and implications for prevention. Addictive Behaviors, 36(10), 1019–1022. doi:10.1016/j.addbeh.2011.05.006 [PubMed: 21665371]

Giancola PR, Josephs RA, DeWall CN, & Gunn RL (2009). Applying the attention-allocation model to the explanation of alcohol-related aggression: Implications for prevention. Substance Use & Misuse, 44(9-10), 1263–1279. doi:10.1080/10826080902960049 [PubMed: 19938917]

Giancola PR, Josephs RA, Parrott DJ, & Duke AA (2010). Alcohol myopia revisited: Clarifying aggression and other acts of disinhibition through a distorted lens. Perspectives on Psychological Science, 5(3), 265–278. doi:10.1177/1745691610369467 [PubMed: 26162159]

Giancola PR, & Parrott DJ (2008). Further evidence for the validity of the Taylor Aggression Paradigm. Aggressive Behavior, 34, 214–229. doi: 10.1002/ab.20235 [PubMed: 17894385]

Giancola PR & Zeichner A (1995). An investigation of gender differences in alcohol-related aggression. Journal of Studies on Alcohol, 56, 573–579. [PubMed: 7475039]

Goldstein SP, Evans BC, Flack D, Juarascio A, Manasse S, Zhang F, & Forman EM (2017). Return of the JITAI: applying a just-in-time adaptive intervention framework to the development of m-health solutions for addictive behaviors. International Journal of Behavioral Medicine, 24(5), 673–682. 10.1007/s12529-016-9627-y [PubMed: 28083725]

Graham K, Bernards S, Wilsnack SC, Gmel G (2011). Alcohol may not cause partner violence but it seems to make it worse: A cross national comparison of the relationship between alcohol and severity of partner violence. Journal of Interpersonal Violence, 26(8), 1503–1523. doi:10.1177/0886260510370596 [PubMed: 20522883]

Hardeman W, Houghton J, Lane K, & Naughton F (2019). A systematic review of just-in-time adaptive interventions (JITAIs) to promote physical activity. International Journal of Behavioral Nutrition and Physical Activity, 16, 31–53. 10.1186/s12966-019-0792-7

Heinz AJ, Beck A, Meyer-Lindenberg A, Sterzer P, & Heinz A (2011). Cognitive and neurobiological mechanisms of alcohol-related aggression. Nature Reviews Neuroscience, 12(7), 400–413. doi:10.1038/nrn3042 [PubMed: 21633380]

Hoaken PN, Giancola PR, & Pihl RO (1998). Executive cognitive functions as mediators of alcohol-related aggression. Alcohol and Alcoholism, 33(1), 47–54. doi:10.1093/oxfordjournals.alcalc.a008347 [PubMed: 9539177]

Holtzworth-Munroe A (1992). Social skill deficits in maritally violent men: Interpreting the data using a social information processing model. Clinical Psychology Review, 12, 605–617. doi:10.1016/0272-7358(92)90134-T

Holtzworth-Munroe A, & Hutchinson G (1993). Attributing negative intent to wife behavior: The attributions of maritally violent versus nonviolent men. Journal of Abnormal Psychology, 102(2), 206–211. doi:10.1037/0021-843X.102.2.206 [PubMed: 8315133]

Huesmann LR (1988). The role of social information processing and cognitive schema in the acquisition and maintenance of habitual aggressive behavior. In Green RG & Donnerstein E (Eds.),

Human aggression: Theories, research, and implications for social policy (pp. 73–109). San Diego, CA: Academic Press. doi:10.1016/B978-012278805-5/50005-5

Hyatt CS, Chester DS, Zeichner A, & Miller JD (2019). Analytic flexibility in laboratory aggression paradigms: Relations with personality traits vary (slightly) by operationalization of aggression. Aggressive Behavior, 45, 377–388.10.1002/ab.21830 [PubMed: 30852848]

King AR, & Russell TD (2019). Lifetime Acts of Violence Assessment (LAVA) predictors of laboratory aggression. Aggressive Behavior. Online first publication. doi:10.1002/ab.21835

Klostermann KC (2006). Substance abuse and intimate partner violence: Treatment considerations. Substance Abuse Treatment, Prevention, and Policy, 1(1), 24. doi:10.1186/1747-597X-1-24

Klostermann K, Kelley ML, Mignone T, Pusateri L, & Fals-Stewart W (2010). Partner violence and substance abuse: Treatment interventions. Aggression and Violent Behavior, 15, 162–166. doi:10.1016/j.avb.2009.10.002

Kraanen FL, Vedel E, Scholing A, & Emmelkamp PMG (2014). Prediction of intimate partner violence by type of substance use disorder. Journal of Substance Abuse Treatment, 46(4), 532–539. doi:10.1016/j.jsat.2013.10.010 [PubMed: 24365101]

Leonard KE (1984). Alcohol consumption and escalatory aggression in intoxicated and sober dyads. Journal of Studies on Alcohol, 45(1), 75–80. doi:10.15288/jsa.1984.45.75 [PubMed: 6700223]

Leonard KE (2005). Alcohol and intimate partner violence: When can we say that heavy drinking is a contributing cause of violence? Addiction, 100, 422–425. doi:10.1111/j.1360-0443.2005.00994.x [PubMed: 15784050]

Leonard KE (2008). The role of drinking patterns and acute intoxication in violent interpersonal behaviors. Alcohol and Violence: Exploring Patterns and Responses (Monograph), 29–55.

Leonard KE, & Quigley BM (2017). Thirty years of research show alcohol to be a cause of intimate partner violence: future research needs to identify who to treat and how to treat them. Drug and Alcohol Review, 36(1), 7–9. doi:10.1111/dar.12434 [PubMed: 27305859]

Leonard KE, & Roberts LJ (1998). The effects of alcohol on the marital interactions of aggressive and nonaggressive husbands and their wives. Journal of Abnormal Psychology, 107(4), 602–615. doi:10.1037/0021-843X.107.4.602 [PubMed: 9830248]

Leonard KE, & Senchak M (1993). Alcohol and premarital aggression among newlywed couples. Journal of Studies on Alcohol, Supplement, (11), 96–108. doi:10.15288/jsas.1993.s11.96 [PubMed: 8410969]

Leonard KE, & Senchack M (1996). Prospective prediction of husband marital aggression within newlywed couples. Journal of Abnormal Psychology, 105(3), 369–380. 10.1037/0021-843X.105.3.369 [PubMed: 8772007]

Lisco CG, Parrott DJ, & Tharp AT (2012). The role of heavy episodic drinking and hostile sexism in men's sexual aggression toward female intimate partners. Addictive Behaviors, 37(11), 1264–1270. [PubMed: 22749405]

Massa AA, Subramani OS, Eckhardt CI, & Parrott DJ (2019). Problematic Alcohol Use and Acute Intoxication Predict Anger-Related Attentional Biases: A Test of the Alcohol Myopia Theory. Psychology of Addictive Behaviors, 33(2), 139–143. doi:10.1037/adb0000426 [PubMed: 30451511]

Mignone T, Klostermann K, & Chen R (2009). The relationship between relapse to alcohol and relapse to violence. Journal of Family Violence, 24(7), 497–505. doi:10.1007/s10896-009-9248-1

Miller JD, Wilson LF, Hyatt CS, & Zeichner A (2015). Psychopathic traits and aggression: Which trait components predict aggressive responding in a laboratory task?. Personality and Individual Differences, 87, 180–184. doi:10.1016/j.paid.2015.08.008

Mook DG (1983). In defense of external invalidity. American Psychologist, 38, 379–387.

Morris AM, Mrug S, & Windle M (2015). From family violence to dating violence: Testing a dual pathway model. Journal of Youth and Adolescence, 44(9), 1819–1835. doi:10.1007/s10964-015-0328-7 [PubMed: 26208831]

Murphy CM, & Ting L (2010). The effects of treatment for substance use problems on intimate partner violence: A review of empirical data. Aggression and Violent Behavior, 15, 325–333. doi:10.1016/j.avb.2010.01.006

Murphy CM, Winters J, O'Farrell TJ, Fals-Stewart W, & Murphy M (2005). Alcohol consumption and intimate partner violence by alcoholic men: Comparing violent and nonviolent conflicts. Psychology of Addictive Behaviors, 19(1), 35–42. doi:10.1037/0893-164X.19.1.35 [PubMed: 15783276]

O'Farrell TJ, Murphy CM, Stephan SH, Fals-Stewart W, & Murphy M (2004). Partner violence before and after couples-based alcoholism treatment for male alcoholic patients: the role of treatment involvement and abstinence. Journal of Consulting and Clinical Psychology, 72(2), 202–217. [PubMed: 15065955]

National Center for Injury Prevention and Control. (2003). Costs of intimate partner violence against women in the United States. Atlanta (GA): Centers for Disease Control and Prevention.

National Institute on Alcohol Abuse and Alcoholism. (2003). National Council on Alcohol Abuse and Alcoholism recommended sets of alcohol consumption questions. Retrieved September 4, 2018, from https://www.niaaa.nih.gov/research/guidelines-and-resources/recommended-alcohol-questions.

Norlander B, & Eckhardt CI (2005). Anger, hostility, and male perpetrators of intimate partner violence: A meta-analytic review. Clinical Psychology Review, 25, 119–152. doi:10.1016/j.cpr.2004.10.001 [PubMed: 15642644]

Okuda M, Olfson M, Wang S, Rubio JM, Xu Y, & Blanco C (2015). Correlates of intimate partner violence perpetration: Results from a national epidemiologic survey. Journal of Traumatic Stress, 28, 49–56. doi:10.1002/jts.21986 [PubMed: 25624189]

Pan HS, Neidig PH, & O'Leary KD (1994). Predicting mild and severe husband-to-wife physical aggression. Journal of Consulting and Clinical Psychology, 62(5), 975. doi:10.1037/0022-006X.62.5.975 [PubMed: 7806729]

Parrott DJ, & Eckhardt CI (2017). Effects of alcohol and other drugs on human aggression. In Bushman BJ (Ed.), Aggression and violence: A social psychological perspective (pp. 199–222). New York, NY: Routledge/Taylor & Francis Group.

Parrott DJ, & Eckhardt CI (2018). Effects of alcohol on human aggression. Current Opinion in Psychology, 19, 1–5. doi:10.1016/j.copsyc.2017.03.023 [PubMed: 28649583]

Parrott DJ, Gallagher KE, & Zeichner A (2012). Liquid courage or liquid fear: Alcohol intoxication and anxiety facilitate physical aggression. Substance Use & Misuse, 47, 774–786. [PubMed: 22452787]

Parrott DJ, & Giancola PR (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(6), 855–864. doi:10.1097/01.ALC.0000128226.92708.21

Parrott DJ, Miller CA, & Hudepohl AD (2015). Immediate and short-term reactions to participation in laboratory aggression research. Psychology of Violence, 5, 209–216. doi: 10.1037/a0035922

Parrott DJ, Zeichner A, & Stephens D (2003). Effects of alcohol, personality, and provocation on the expression of anger in men: A facial coding analysis. Alcoholism: Clinical and Experimental Research, 27, 937–945.

Phillips JP, & Giancola PR (2008). Experimentally induced anxiety attenuates alcohol-related aggression in men. Experimental and Clinical Psychopharmacology, 16(1), 43–56. doi:10.1037/1064-1297.16.1.43 [PubMed: 18266551]

Preacher KJ, Zyphur MJ, & Zhang Z (2010). A general multilevel SEM framework for assessing multilevel mediation. Psychological Methods, 15(3), 209–233. doi:10.1037/a0020141 [PubMed: 20822249]

Renzetti CM, Lynch KR, & DeWall CN (2018). Ambivalent sexism, alcohol use, and intimate partner violence perpetration. Journal of Interpersonal Violence, 33(2), 183–210. doi:10.1177/0886260515604412 [PubMed: 26354500]

Rosenberg EL, & Ekman P (1994). Coherence between expressive and experiential systems in emotion. Cognition and Emotion, 8, 201–229.

Rosenberg EL, Ekman P, & Blumenthal JA (1998). Facial expression and the affective component of cynical hostility in male coronary heart disease patients. Health Psychology, 17, 376–380. [PubMed: 9697948]

Rusting CL, & Nolen-Hoeksema S (1998). Regulating responses to anger: Effects of rumination and distraction on angry mood. Journal of Personality and Social Psychology, 74(3), 790–803. doi:10.1037/0022-3514.74.3.790 [PubMed: 9523420]

Sayette MA, & Parrott DJ (1999). Effects of olfactory stimuli on urge reduction in smokers. Experimental and Clinical Psychopharmacology, 7(2), 151–159. doi:10.1037/1064-1297.7.2.151 [PubMed: 10340155]

Smith PH, Homish GG, Leonard KE, & Cornelius JR (2012). Intimate partner violence and specific substance use disorders: Findings from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychology of Addictive Behaviors, 26(2), 236–245. doi:10.1037/a0024855 [PubMed: 21823768]

Steele CM, & Josephs RA (1990). Alcohol myopia: Its prized and dangerous effects. American Psychologist, 45(8), 921–933. doi:10.1037/0003-066X.45.8.921

Stith SM, Smith DB, Penn CE, Ward DB, & Tritt D (2004). Intimate partner physical abuse perpetration and victimization risk factors: A meta-analytic review. Aggression and Violent Behavior, 10, 65–98. doi:10.1016/j.avb.2003.09.001

Straus M, Hamby S, Boney-McCoy S, & Sugarman D (1996). The revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. Journal of Family Issues, 17, 283–316. doi:10.1177/019251396017003001

Stuart GL, Moore TM, Elkins SR, O'Farrell TJ, Temple JR, Ramsey SE, & Shorey RC (2013). The temporal association between substance use and intimate partner violence among women arrested for domestic violence. Journal of Consulting and Clinical Psychology, 81(4), 681–690. Doi: 10.1037/a0032876 [PubMed: 23647284]

Taft CT, Schumm JA, Marshall AD, Panuzio J, & Holtzworth-Munroe A (2008). Family-of-origin maltreatment, posttraumatic stress disorder symptoms, social information processing deficits, and relationship abuse perpetration. Journal of Abnormal Psychology, 117(3), 637–646. doi:10.1037/0021-843X.117.3.637 [PubMed: 18729615]

Taylor SP (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. Journal of Personality, 35, 297–310. doi: 10.1111/j.1467-6494.1967.tb01430.x [PubMed: 6059850]

Taylor SP, & Leonard KE (1983). Alcohol and human physical aggression. Aggression: Theoretical and Empirical Reviews, 2, 77–101.

Testa M, Crane CA, Quigley BM, Levitt A, & Leonard KE (2014). Effects of administered alcohol on intimate partner interactions in a conflict resolution paradigm. Journal of Studies on Alcohol and Drugs, 75(2), 249–258. 10.15288/jsad.2014.75.249 [PubMed: 24650819]

Testa M, & Derrick JL (2014). A daily process examination of the temporal association between alcohol use and verbal and physical aggression in community couples. Psychology of Addictive Behaviors, 28(1), 127–138. 10.1037/a0032988 [PubMed: 24341618]

Testa M, Quigley BM, & Leonard KE (2003). Does alcohol make a difference? Within-participants comparison of incidents of partner violence. Journal of Interpersonal Violence, 18(7), 735–743. doi:10.1177/0886260503253232 [PubMed: 14675506]

Tharp AT, Schumacher JA, Samper RE, McLeish AC, & Coffey SF (2012). Relative importance of emotion dysregulation, hostility, and impulsiveness in predicting intimate partner violence perpetrated by men in alcohol treatment. Psychology of Women Quarterly, 37, 51–60. [PubMed: 23990693]

Wallace P, & Taylor S (2009). Reduction of appeasement-related affect as a concomitant of diazepam-induced aggression: Evidence for a link between aggression and the expression of self-conscious emotions. Aggressive Behavior, 35, 203–212. [PubMed: 19090555]

Watkins LE, DiLillo D, & Maldonado RC (2015). The interactive effects of emotion regulation and alcohol intoxication on lab-based intimate partner aggression. Psychology of Addictive Behaviors, 29(3) , 653–663. 10.1037/adb0000074 [PubMed: 25844831]

Zanov MV, & Davison GC (2010). A conceptual and empirical review of 25 years of cognitive assessment using the Articulated Thoughts in Simulated Situations (ATSS) think-aloud paradigm. Cognitive Therapy and Research, 34(3), 282–291. doi:10.1007/s10608-009-9271-9

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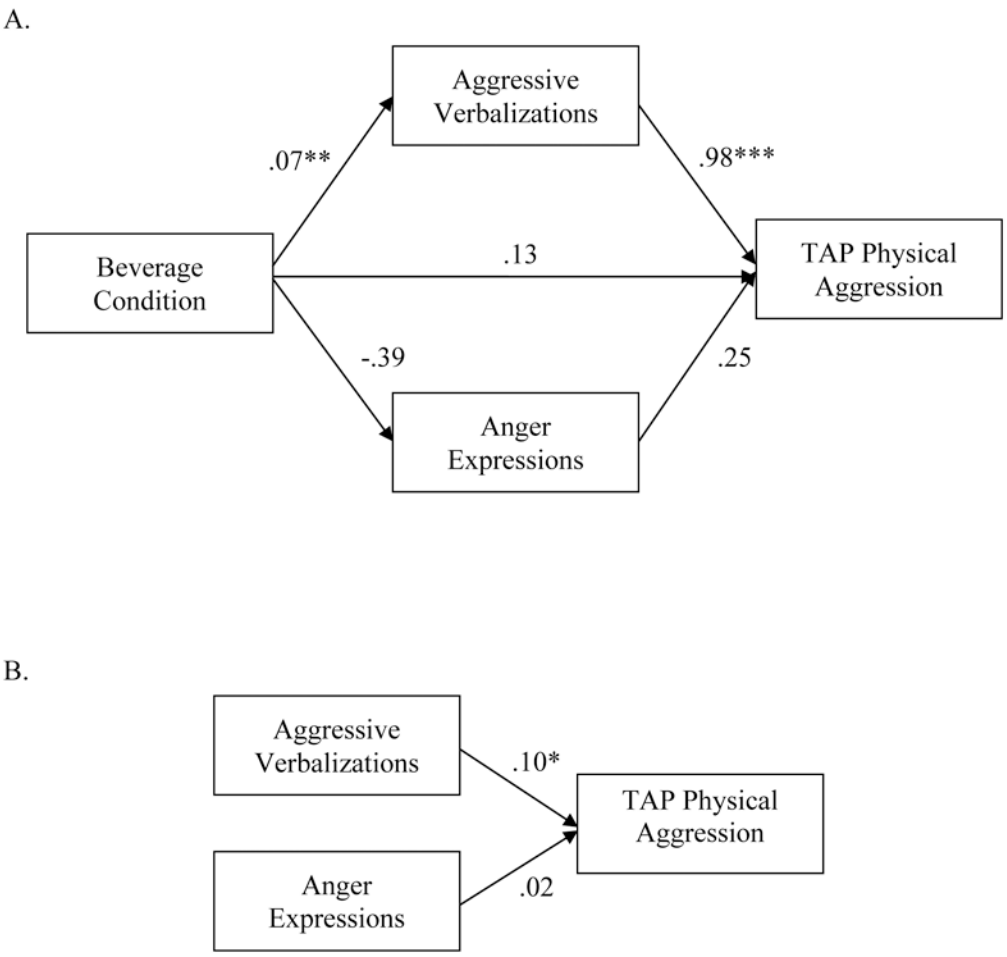

Figure 1.

Multiple-mediator model. (A) Across Trial Effects (Between Person); (B) Trial-by-Trial Effects (Within Person). *$p < .05$, **$p < .01$, ***$p < .001$

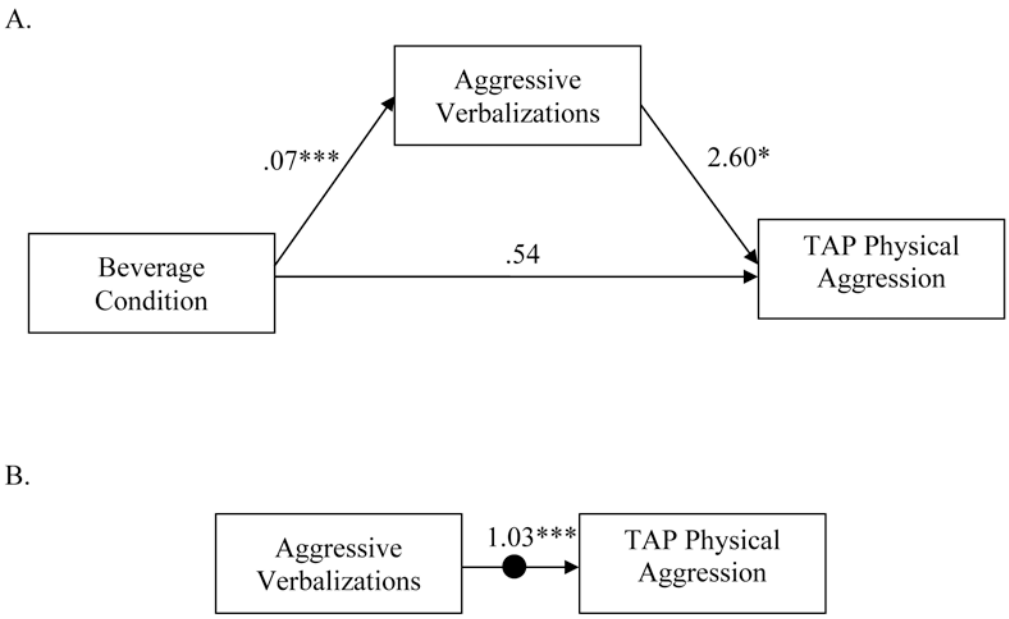

Figure 2.

Single-mediator model. (A) Across Trial Effects (Between Person); (B) Trial-by-Trial Effects (Within Person). *$p < .05$, **$p < .01$, ***$p < .001$

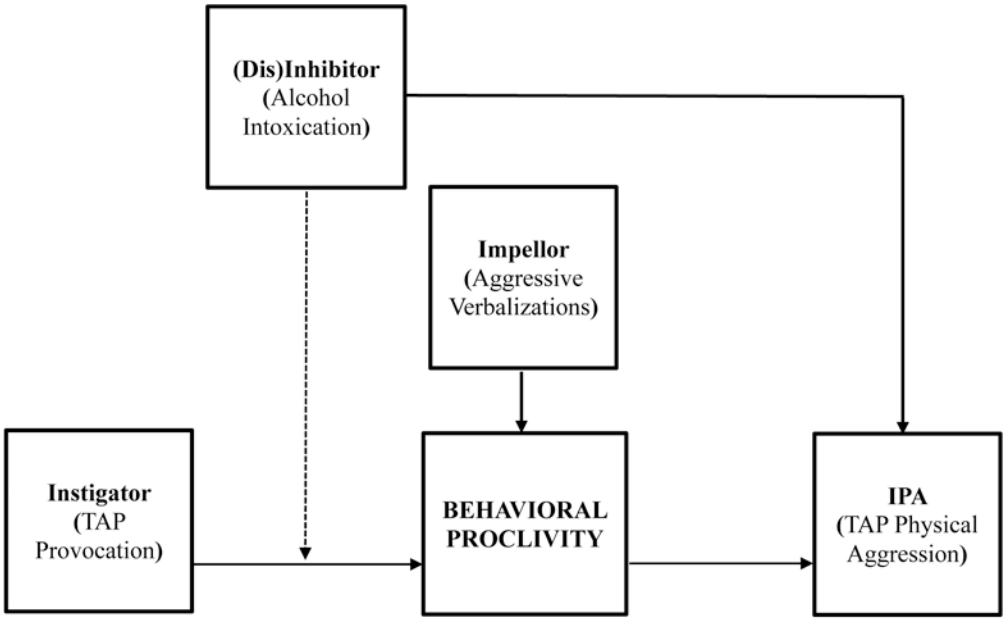

Figure 3.

Provocation, Aggressive Verbalizations, and Alcohol Intoxication as facilitators of IPA: A "Perfect Storm" Representation (adapted from Finkel, 2014, p. 15). Solid lines represent mediation effects, and dashed lines represent moderating effects.

Table 1

Sample Demographics and Prior Year Alcohol Use and Intimate Partner Aggression (by Gender)

Gender (%)	
Male	59.4
Female	40.6
Marital status (%)	
Single	41.4
Married	13.0
Unmarried, living with partner	37.2
Divorced	6.3
Separated	2.1
Age (*M, SD*)	32.62 (10.44)
Years education (*M, SD*)	13.68 (2.79)
Relationship length, months (*M, SD*)	54.26 (57.46)
Race/ethnicity (%)	
White/Euro American	25.6
Black/African American	65.1
Asian American	1.3
Multiracial	8.0
Latino/Hispanic	4.3
Annual income (%)	
< 10k	36.1
$10-20k	23.5
$20-30k	18.1
$30-40k	11.8
> $40k	10.5

	Male (*n* = 148)	**Female (*n* = 101)**	**Total (*n* = 249)**
Alcohol Use			
Total AUDIT score	10.74 (5.56)[a]	8.31 (4.38)[b]	9.75 (5.24)
Drinking days per week	3.04 (1.89)[a]	2.65 (1.78)[a]	2.88 (1.85)
Drinks per drinking day	7.03 (3.74)[a]	5.39 (2.80)[b]	6.36 (3.48)
Binge drinking days per week	1.44 (1.71)[a]	1.27 (1.72)[a]	1.37 (1.75)
Intimate Partner Aggression			
Physical Perpetration	8.74 (11.01)[a]	7.63 (9.52)[a]	8.29 (10.43)
Psychological Perpetration	20.71 (20.36)[a]	16.38 (18.64)[a]	18.95 (19.76)
Physical Victimization	5.10 (11.32)[a]	1.82 (3.76)[b]	3.78 (9.18)
Psychological Victimization	26.98 (26.15)[a]	18.47 (22.62)[b]	23.53 (25.08)
Mediator Variables			
Anger Expressions	2.49 (5.77)[a]	2.94 (6.54)[a]	2.67 (6.09)
Aggressive Verbalizations	.49 (.84)[a]	.88 (1.60)[a]	.68 (1.28)
Negative Affect	15.28 (18.08)[a]	20.00 (18.53)[b]	17.20 (18.37)

Note: Note: $n = 249$. Means in same row with different superscripts differ, $p < .001$.