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Mechanisms of Alcohol-Facilitated Intimate Partner Violence

Christopher I. Eckhardt[1], **Dominic J. Parrott**[2], and **Joel G. Sprunger**[1]

[1]Purdue University, West Lafayette, IN, USA

[2]Georgia State University, Atlanta, USA

Abstract

Intimate partner violence (IPV) is a critical public health problem that requires clear and testable etiological models that may translate into effective interventions. While alcohol intoxication and a pattern of heavy alcohol consumption are robust correlates of IPV perpetration, there has been limited research that examines the mediating mechanisms of *how* alcohol potentiates IPV. We provide a theoretical and methodological framework for researchers to conceptualize how alcohol intoxication causes IPV, and propose innovative laboratory methods that directly test mediational mechanisms. We conclude by discussing how these innovations may lead to the development of interventions to prevent or reduce alcohol-related IPV.

Keywords

intimate partner violence; alcohol intoxication; alcohol myopia; I^3 theory; treatment

Surveys of adults conducted in the United States and abroad indicate that intimate partner violence (IPV) occurs at alarmingly high rates across a multitude of age groups, across both sexes, and among individuals of all ethnic, racial, and cultural backgrounds (Black et al., 2011; Jose & O'Leary, 2009). Acute alcohol intoxication and a pattern of heavy alcohol consumption are among the most robust correlates of IPV perpetration (see Leonard, 2008), even after controlling for perpetrator demographics, hostility, and relationship distress (Leonard & Senchak, 1993; Pan, Neidig, & O'Leary, 1994). Cross-sectional research suggests that alcoholic males evidence higher rates of IPV relative to non-alcoholic males (Chermack, Fuller, & Blow, 2000). Longitudinal findings indicate that heavy drinking in the early stages of marriage predicts subsequent IPV (Leonard & Senchak, 1996). Studies of violent couples indicate that when one partner has been drinking, IPV episodes are more frequent, severe, and more likely to lead to mutual violence (Murphy, Winters, O'Farrell, Fals-Stewart, & Murphy, 2005; Testa, Quigley, & Leonard, 2003). Laboratory studies demonstrate that alcohol intoxication increases negative interaction behaviors among violent couples (Leonard & Roberts, 1998) and aggressive verbalizations during simulated relationship conflicts, especially among violent men prone to anger (Eckhardt, 2007). This

Corresponding Author: Christopher I. Eckhardt, Department of Psychological Sciences, Purdue University, 703 3rd St., West Lafayette, IN 47907, USA. eckhardt@purdue.edu.

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cross-method convergence of findings has led to the conclusion that alcohol use is a contributing cause of IPV (Leonard, 2005).

However, accumulated findings in this area also reveal that while we have made progress in understanding whether alcohol is associated with IPV, we have a poor understanding of how alcohol use actually functions to increase risk for IPV. Indeed, most of our current understanding of the alcohol–IPV association concerns moderators of this relationship. In line with the data on alcohol and general aggression (Ito, Miller, & Pollock, 1996; Parrott & Giancola, 2004), reviews of the relatively scant literature on alcohol–IPV moderators indicate that men who are most likely to exhibit partner violence when intoxicated are those with preexisting aggressive propensities, including high state and trait anger (Eckhardt, 2007; Leonard & Blane, 1992), antisocial traits (Jacob, Leonard, & Haber, 2001), and a verbally aggressive conflict style (Quigley & Leonard, 1999). To date, however, there have been *no* studies that have directly examined the actual mediating mechanisms of *how* alcohol intoxication, and the variables with which it interacts, potentiates alcohol-related aggression. This is a significant deficit in the current literature and is surprising, given the abundance of rich theoretical explanations of these putative mechanisms. The perspective we have adopted in our work, and that we will champion in this article, seeks to move beyond simply identifying moderating risk factors and attempting to uncover theoretically based mediating processes that underlie reported associations among risk factors and IPV. As such, the aim of the present article is to provide the theoretical and methodological framework for researchers to take a first step toward understanding how acute alcohol intoxication causes IPV. Specifically, we will (a) discuss new theoretical developments that provide a solid framework upon which to study the role of alcohol and IPV, and (b) propose innovative laboratory-based methods derived from this theoretical framework that will directly test these putative mechanisms. See Figure 1 for an overview. We envision that data generated by the proposed approach can provide the necessary foundation for subsequent research on methods to reduce alcohol-facilitated IPV.

Our theoretical review will first discuss a unifying framework of risk for alcohol-related IPV (I^3 Theory) and then integrate this framework with a well-accepted theory (alcohol myopia theory [AMT]) of *how* alcohol plays a role in translating risk into actual IPV perpetration. Next, we review methodologies that allow for direct assessment of mechanisms of alcohol-facilitated IPV assessed in the laboratory. This focus on laboratory methods is intentional and purposeful. Laboratory experiments afford researchers' control over study variables—importantly, the amount of alcohol ingested by participants. In addition, only the laboratory experiment can establish causal relationships as well as directly and precisely examine the pharmacological effects of alcohol on IPV. Unfortunately, there exist few laboratory studies directly addressing the role of alcohol in IPV. Thus, advancements here are necessary to complement the exciting and innovative field methods being used to examine the day-to-day, and even hour-to-hour, link between alcohol use and IPV (e.g., Testa & Derrick, 2014).

Theoretical Considerations

Overview

Research in the area of IPV has largely been conducted via several prominent monotheoretical models that conceptualize IPV as (a) a male-to-female act performed for purposes of power and control that is tolerated or even encouraged within a patriarchal society (i.e., sociocultural models); (b) the result of social learning or modeled violence within the family of origin, with more proximal factors related to maladaptive cognitions and affect regulation deficits, and disturbed personality characteristics or psychopathology (i.e., intrapersonal models); or (c) the result of a cascade of dissonant dyadic factors that emerge within the context of the communication styles of a specific couple and that increase the likelihood of partner aggression (i.e., interpersonal models). This reliance on unitary monoperspective models has been useful in terms of framing research questions that arise from specific tenets of these theories (e.g., do particular patterns of personality traits characterize IPV perpetrators?) and in generating lists of potential risk factors for IPV. But many of the variables that appear on IPV risk factor lists are static or distal predictors (e.g., age, psychopathology, sociopolitical values) that do not directly inform our understanding of process-level causal relationships. In addition, adherence to a singular theoretical perspective becomes problematic when data are irreconcilable with the primary tenets of that theory. For example, data indicating that males and females show nearly equal rates of IPV perpetration make the singular reliance on the sociocultural/patriarchal socialization perspective problematic (Straus, 2010). Strict adherence to *any* single theoretical approach may arbitrarily constrict the range of relevant variables and interactions among these variables that one is able to investigate.

I^3 Theory

Thus, while there is no shortage of risk factor lists and monotheoretical models of IPV, we believe that it is time to move toward a unifying theoretical framework that can allow for the framing and exploring of variables relevant to understanding the causes of IPV that is not bound by the usual theoretical limitations and that may provide a clearer understanding of potential mediators of the alcohol–IPV association. One such "metatheory" is known as I^3 Theory (pronounced "I-cubed"), which is a process-oriented framework that offers a means of understanding predictors of IPV (and other behaviors) according to three process categories—*I*nstigation, *I*mpellance, and *I*nhibition (hence, "I^3")—that are considered necessary and sufficient for predicting the likelihood of IPV in a given situation (Finkel & Eckhardt, 2013). A key advantage to using I^3 theory in understanding alcohol-induced IPV rests in its interactional framework. The model suggests that we may be able to predict, with greater accuracy, whether a given interchange between intimate partners will be violent versus non-violent if we can discern the strength and patterning of instigation, impellance, and inhibition factors. These factors are reviewed in more detail below.

Instigating factors are situational or contextual experiences—such as interpersonal provocation—that may trigger an urge to become aggressive for the average person under typical circumstances. These factors essentially provide the initial momentum toward an aggressive action that represents the availability of an aggressive response (e.g., an argument

with an intimate partner). *Impelling* factors are dispositional and/or situational factors that psychologically prepare an individual to experience a strong urge to aggress when encountering instigation in a particular context, and include many of the traditional static individual difference risk factors for IPV (e.g., high anger arousal, antisocial traits). Instigating and impelling factors interact with each other and share a relationship akin to a match and gasoline; the gasoline alone will not ignite, but the introduction of a flame produces a reaction of exaggerated magnitude. Thus, a proclivity toward aggression will persist unless there is an equally strong inhibitory surge to override it (Finkel, 2014). *Inhibitory* factors are those that increase the likelihood that a person will be able to resist an urge to behave aggressively at a specific point in time. In the context of IPV, inhibitory factors essentially set the threshold beyond which aggressive urges would result in partner-directed aggression. The integrity of these inhibitory capabilities may be acted upon by *disinhibiting* influences, which decrease the effectiveness of inhibitory efforts and, therefore, decrease the likelihood that a person will be able to resist an urge to behave in an aggressive manner at a specific point in time. Examples of disinhibiting influences include cognitive resource depletion, physical pain, and—of relevance to this report—alcohol intoxication (Berkowitz, 1993a; Finkel, DeWall, Slotter, Oaten, & Foshee, 2009; Giancola, Josephs, Parrott, & Duke, 2010).

A common pattern of the I^3 approach has become known as the *perfect storm theory* (Finkel, 2007), which predicts that an individual is most likely to enact a given behavior in a specific situation when instigation and impellance are strong and inhibition is weak. This perfect storm theory is especially relevant in the case of alcohol-induced IPV, as research suggests that such behavior is especially likely under interactive contexts involving partner provocation (high instigation), among individuals with a history of aggression and a proneness toward anger arousal (high impellance), and during alcohol intoxication (low inhibition). Thus, knowledge of these three processes, and of the interplay among them, may be both *necessary and sufficient* for predicting IPV perpetration and understanding process-level mediators of specific abusive acts.

I^3 Theory and Alcohol Myopia

A singular advantage to a unifying model such as I^3 theory is its theoretical inclusiveness, which allows researchers to incorporate the most empirically supported theories available as a means of establishing empirically based, multifactorial conceptualizations of risk. Specific theories can then be brought to bear to examine how hypotheses related to risk can be translated into process-oriented mediation models. While research has clearly established that alcohol-induced aggression is more likely when the perpetrator (a) is provoked (e.g., Taylor, Schmutte, Leonard, & Cranston, 1979; that is, the role of *Instigation*) and (b) possesses particular aggressogenic traits (Parrott & Giancola, 2004; that is, the role of *Impellors*), evidence is converging that the pathway from alcohol intoxication to aggressive behavior primarily involves factors specifically related to *Inhibition* (Giancola et al., 2010). Thus, alcohol does not appear to unitarily impel acts of aggression; rather, alcohol intoxication produces key neuropsychological changes that alter executive functioning and impede self-regulatory capacities.

The most prominent and empirically supported model for understanding *how* alcohol affects inhibitory processes related to human behavior is 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 (AAM), 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 one's partner) to the exclusion of less salient inhibitory cues (e.g., legal consequences of IPV).

AMT has garnered extensive empirical support as an explanation for a range of alcohol-related behaviors, including aggression (for a review, see Giancola et al., 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., Gallagher & Parrott, 2011; Giancola & Corman, 2007). 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 IPV primarily among individuals who endorse dispositional tendencies in aggression-related cognitive biases (e.g., high hostility; Leonard & Blane, 1992) or who are susceptible to alcohol-related shifts in attention toward instigatory cues (e.g., low mindfulness; Gallagher, Hudepohl, & Parrott, 2010). To this end, prior research has demonstrated that individuals at risk for aggression show attentional biases toward aggression-relevant contextual stimuli (Eckhardt & Cohen, 1997; Smith & Waterman, 2004). *Thus, it follows from AMT that alcohol use should potentiate IPV by narrowing attention onto salient, provocative cues—particularly in already high-risk persons.* While prior research has examined the moderating effects of information-processing biases (e.g., executive functioning deficits; Giancola, 2004), the mediational attention allocation hypothesis assumed to underlie the alcohol–aggression association has been studied only once in an experimental context (Gallagher & Parrott, 2011), and has yet to be examined among individuals with a history of IPV.

The AAM has largely been used to explain how alcohol increases aggressive behavior. However, as indicated above, this model also allows for the counterintuitive prediction that alcohol intoxication *can actually decrease* aggression, even below that of sober individuals (Giancola & Corman, 2007). Specifically, in a situation where non-provocative cues are most salient, the narrowed attentional capacity of the inebriate will be focused on those cues, leaving little space in working memory to focus on less salient provocative cues. In contrast, sober persons faced with the same situation possess enough working memory to allot attention to provocative and non-provocative cues, thus increasing their risk of aggression above that of intoxicated persons. This counterintuitive prediction has compelling implications for interventions designed to prevent or reduce alcohol-related aggression (Giancola, Josephs, DeWall, & Gunn, 2009).

While it is clear that AMT is a well-supported model that fleshes out the inhibitory process dimension of the I^3 metatheory, the intervening processes by which attentional biases increase (or decrease) the probability of an aggressive response to a provocative situation remain largely unstudied (and are *completely* unstudied in the IPV literature). To address this important gap in the literature, Giancola and colleagues (2010) sought to explain the mechanisms that mediate the association between attention allocation and interpersonal aggression. These proposed mechanisms of AMT are discussed below.

Increased negative affect and anger—In his cognitive-neoassociationistic theory, Berkowitz (1990, 1993a) argued that negative affect and anger-related emotions and cognitions exist within an 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. Therefore, anger-related cognitions at this latter stage are likely to result in a higher risk of aggression. This model has received strong empirical support (Berkowitz, 1993b) and is consistent with other prominent heuristic theories of aggression (e.g., Anderson & Bushman, 2002).

In line with this model, Giancola and colleagues (2010) posited that alcohol-induced attention toward 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. In the context of IPV, prior research suggests the importance of understanding how the interaction between trait (as well as state) anger and alcohol intoxication increases risk for IPV (Eckhardt, 2007). In this study, IPV perpetrators and a control sample of non-violent males were randomly assigned to one of three alcohol administration conditions in the laboratory (alcohol, active placebo, control). All men were induced to experience anger arousal and, while imagining these scenarios, they reported their emotions and conflict behaviors, which were subsequently coded by trained research assistants. Results indicated that while alcohol intoxication alone did not exert a direct effect on angry and aggressive responding to the imagined conflicts, alcohol interacted with IPV history and change in anger arousal, such that IPV perpetrators given alcohol who also had increases in state anger responded with the highest level of aggressive responding (Eckhardt, 2007). However, no formal tests of mediation were conducted in this research, and no other studies have directly examined state anger as a mediator of alcohol-facilitated IPV. Likewise, no studies have examined whether redirecting the inebriate's attention away from state anger while in the midst of an altercation with an intimate partner will also reduce aggression. Future research is needed to directly test anger as a mediator of alcohol-facilitated IPV as well as to develop methods to reduce alcohol-facilitated IPV via the redirection of attention away from negative affect and anger.

Hostile cognitive rumination—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 rumination 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). These findings are replicated in work with partner violent males. Eckhardt and colleagues (Eckhardt, Barbour, & Davison, 1998; Eckhardt, Jamison, & Watts, 2002) had partner violent and non-violent males imagine scenarios involving a neutral relationship interaction or an anger-inducing marital conflict paradigm, and then instructed participants to articulate their thoughts, affective reactions, and prepotent behaviors when prompted. Partner violent males articulated significantly higher levels of irrational beliefs, cognitive distortions, hostile attributions, and aggressive verbalizations during anger arousal (see also Holtzworth-Munroe & Hutchinson, 1993).

Together, these findings suggest that one important functional pathway through which alcohol-induced attentional biases may increase aggression risk is by concentrating subsequent cognitive processes on the hostile transgression. Borders and Giancola (2011) examined this hypothesis in a sample of individuals randomly assigned to alcohol or placebo groups who participated in a competitive reaction-time laboratory task to assess aggressive behavior. Hostile rumination was assessed via questionnaire prior to (trait) and immediately after (state) the laboratory aggression task. Findings revealed that aggressive behavior was highest among those with high trait and state hostile rumination. While the researchers' methodology did not allow for an analysis of state hostile rumination as a mediator of the alcohol–aggression relationship, it is likely that these cognitive distortions and interpretive biases served as critical information cues that prolonged angry affect and guided the transgressed toward an aggressive resolution of the conflict. While there have been few investigations of hostile rumination as a risk factor for IPV (see Sotelo & Babcock, 2013), there are currently no published studies that have examined hostile rumination in the context of alcohol-induced IPV. Thus, a second factor that should mediate the relationship between alcohol intoxication and IPV is hostile rumination.

Self-awareness—Distraction is posited to reduce intoxicated aggression by redirecting attention away from provocative cues. However, the aggression-attenuating effect of distraction may be more effective if the inebriate's attention is redirected toward cues that increase self-awareness. Self-awareness theory posits that when attention is focused on the self, automatic comparisons between self and social standards of appropriate behavior are initiated (Silvia & Duval, 2001). Pertinently, alcohol intoxication reduces self-awareness by disrupting the encoding of self-relevant information that could be used to modulate behavior in accordance with non-aggressive social norms (Hull, 1981; Hull, Levenson, Young, & Sher, 1983). Indeed, a seminal meta-analysis by Ito and colleagues (1996) found smaller effect sizes of alcohol on aggression when social drinking participants' attention was focused on self-relevant information. Indeed, research suggests that interventions designed to increase self-awareness (e.g., the addition of mirrors, emphasis of one's behavior in relation to non-aggressive norms) reduce alcohol-related aggression toward oneself

(Berman, Bradley, Fanning, & McCloskey, 2009) and others (Bailey, Leonard, Cranston, & Taylor, 1983; Jeavons & Taylor, 1985) in social drinking samples. Thus, by increasing self-awareness, the inebriate is distracted from provocation and able to process cues of inhibition.

Laboratory Methods to Assess Mechanisms of Alcohol-Facilitated IPV

The preceding review illustrates the rich theoretical explanations for putative mechanisms of alcohol-facilitated IPV. As noted earlier in this review, an abundance of research has focused on moderators of the alcohol–IPV link while devoting much less empirical study to the specific affective and cognitive mediators of this relation. This limitation prevents research from developing and testing theoretically based interventions designed to reduce alcohol-facilitated IPV. Below, we propose methodologically innovative approaches to address this limitation.

Measuring IPV in the Laboratory

There exist several well-established and validated laboratory paradigms to assess aggressive behavior (for a review, see Anderson & Bushman, 1997; Giancola & Parrott, 2008). The most widely used is the TAP (Taylor, 1967), in which participants are told that they will compete against an opponent (seated in an adjacent room) on a reaction-time task. Depending on whether participants win or lose a reaction-time trial, they will either administer or receive an electric shock to, or from, their opponent. Of course, no opponent actually exists, and the sequence of wins and loses as well as the intensity of the shocks set by the “opponent” is predetermined by the experimenter. The bogus competition merely exists to deceive individuals into believing that they are engaged in a competitive and adversarial relationship with another person. Aggression is operationalized by the intensity and duration of shock administered to the fictitious opponent. While the specific procedures of other approaches will not be reviewed here, a critical element of these paradigms is that they afford a participant the ostensible opportunity to inflict harm upon another participant via some noxious stimulus (e.g., electric shock, adverse noise, hot sauce, negative evaluation).

Critics of the TAP have suggested that the task may assess constructs superfluous to aggression (e.g., competitiveness) and has limited external validity (Tedeschi & Quigley, 1996). While researchers have countered these claims (e.g., Anderson & Bushman, 1997; Giancola & Chermack, 1998), others have attempted to measure aggression-related outcomes in the context of actual dyadic conflicts, albeit in a structured laboratory context. Studies using these methods randomly assign couples to an alcohol consumption condition and then implement a conflict resolution paradigm in which couples discuss real-life disagreements (e.g., Haber & Jacob, 1997; Leonard & Roberts, 1998). While these studies provide an important relational context to examine relevant dyadic conflicts, there are obvious ethical limitations surrounding the extent to which researchers can measure aggressive acts designed to hurt their partner. However, with two recent exceptions (Watkins, DiLillo, Hoffman, & Templin, 2015; Watkins, DiLillo, & Maldonado, 2015), published studies to date have not used a laboratory aggression paradigm—wherein each member of the couple is afforded the ostensible opportunity to inflict harm upon their

partner—to examine IPV. To overcome this limitation, both members of the couple must be recruited into a study that employs the TAP or another validated laboratory aggression paradigm (for a review, see Parrott & Giancola, 2007).

Assessment of In Vivo Attention, Cognition, and Affect

Establishing mediation is based on several criteria (for a review, see Kazdin & Nock, 2003), one of which is the demonstration of a *temporal relation* between the mediator(s) and the outcome. In laboratory experiments, this criterion is achieved only by assessing proposed mediating variables in vivo and prior to the display of aggressive behavior. Unfortunately, as outlined by Lindsay and Anderson (2000), laboratory-based studies are not ideal for examining the mediational effects of state variables (e.g., anger) on aggressive behavior. Specifically, tests of mediation also require that the assessment of the mediating variable (e.g., state anger) not interfere with the assessment of the criterion variable (e.g., aggression). In laboratory studies, it may take several minutes for participants to complete self-report measures of anger prior to engaging in an aggression task. As a result, high levels of state anger that presumably mediate alcohol-related aggression may dissipate over time. Alternatively, assessment of aggression-related mediators in the laboratory may inadvertently reveal the true purpose of the aggression task. Collectively, these limitations call for relatively unobtrusive, real-time measures of proposed mediators. Below, we propose several well-established methods that overcome these limitations.

Attention—The concurrent assessment of attention allocation and aggressive behavior is clearly a challenge, and likely explains why the fundamental tenant of the AAM was not directly tested until recently (Gallagher & Parrott, 2011). Indeed, traditional laboratory measures of attention are logistically difficult, if not impossible, to integrate with traditional laboratory measures of aggression. We offer two potential solutions to this challenge.

First, reaction-time-based attentional measures, such as the dot probe task (MacLeod, Mathews, & Tata, 1986), could be integrated into an early stage of the laboratory aggression task. For instance, in a study by Gallagher and Parrott (2011), participants were provoked via reception of electric shocks and a verbal insult from a fictitious male opponent in an ostensible series of practice trials of the TAP. Then, participants' attention allocation to aggression words was assessed via a dot probe task. Immediately thereafter, the TAP resumed and physical aggression was measured. A strength of this approach is that it allows researchers to demonstrate the presence of alcohol-related attentional biases toward provocative stimuli immediately prior to the assessment of aggression. Of course, a limitation of this method lies in the presumption that attentional focus remains constant throughout the subsequent assessment of aggression and that participants possess a sufficient level of verbal fluency to quickly read and understand the aggressive words in short order.

Eye tracking technology represents a second solution to in vivo measurement of attention allocation. Eye tracking technology allows for relatively direct and continuous measurement of overt visual attention and is able to address the limitations of reaction-time measures (e.g., the dot probe). This methodology reduces the need for inference regarding attentional focus on specific external cues and provides an important complement to traditional

measures of attention. The small number of studies that examined selective attention toward aggression-relevant stimuli using eye tracking methods suggests that individuals with high dispositional anger may immediately bias their attention toward hostile situational cues (e.g., Horsley, de Castro, & van der Schoot, 2010; Wilkowski, Robinson, Gordon, & Troop-Gordon, 2007). However, these attentional processes have not been examined in the context of alcohol use and/or IPV. The added level of fidelity gained by the online measurement of human visual attention during aggression-relevant tasks in the context of alcohol would contribute greatly toward observing the mechanism by which alcohol influences attention to environmental cues present during relationship conflict and subsequent aggressive responding.

Cognition—Real-time sampling of cognitive reactions during aggression assessment can be achieved via thought sampling methods, such as the articulated thoughts in simulated situations (ATSS) paradigm (Davison, Robins, & Johnson, 1983; Eckhardt, 2007). The ATSS paradigm is a general method for assessing cognitive activity and affective experiences simultaneous to the participant taking part in an emotionally arousing situation. In traditional applications of this procedure, participants are asked to visualize an emotionally stimulating situation (e.g., intimate partner conflict). When prompted by a tone, they verbally report their thoughts and affective reactions to the current situation. These verbal reports are recorded and later coded for social information-processing distortions. We propose an integration of the ATSS with laboratory aggression assessment, such that participants are instructed to verbally report their thoughts during the laboratory aggression task. This innovation will capture verbal articulations that precede or are concurrent with the display of aggressive behavior.

Affect—Observational methods allow researchers to assess affect in vivo while overcoming the well-documented limitations of self-report. These methods typically involve the recording of subject behavior and later coding for content. However, the presence of a video camera aimed at a participant can enhance self-awareness and thus complicate interpretation of alcohol's effect on IPV. To circumvent this problem, we propose the use of a high-resolution hidden camera(s) embedded in the testing room.

Among the available observational methods for coding real-time affect, the facial action coding system (FACS; Ekman & Friesen, 1978a) is the most comprehensive. The FACS is a comprehensive, anatomically based system that classifies all observable facial activity into 44 unique action units (AUs). Coders do not interpret emotions displayed. Instead, they describe all visible facial movements in terms of discrete AUs and combinations of AUs. These descriptive data are then analyzed to identify and generate frequency counts of discrete emotional expressions. 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). FACS has twice been utilized to examine state emotion during the TAP (Parrott, Zeichner, & Stephens, 2003; Wallace & Taylor, 2009), thus speaking to the feasibility of integrating this procedure into laboratory-based IPV research.

Sampling Considerations

We propose that studies of alcohol-facilitated IPV recruit high-risk samples of heavy drinking men and women with a recent history of IPV. Despite the literature suggesting that heavy drinkers are most at risk for perpetrating alcohol-related physical aggression (Parrott & Giancola, 2006) and IPV (e.g., Chermack et al., 2000), the majority of laboratory-based alcohol–aggression research involves non-problem drinking samples, which may indeed be appropriate if the goal is to generalize to a moderate or moderate-to-heavy drinking population. However, if one wishes to apply findings to the population at highest risk for IPV perpetration, recruitment of high-risk samples (e.g., defined by common measures of problem drinking or presence of an alcohol-use disorder) will yield results with greater generalizability to populations of clinical interest than currently exists in the extant literature. It is important to highlight that this type of proposed research, which involves the administration of alcohol to heavy drinkers at high risk for physical aggression, requires attention to several key ethical considerations in accordance with National Institute on Alcohol Abuse and Alcoholism (NIAAA) guidelines.[1] Of particular note, participants' treatment seeking status must be considered as it relates to eligibility determination. In the context of IPV, it is important to rule out those with recent episodes of severe partner-directed physical aggression to limit risks of impulsive aggression to intimate partners as well as to staff. Despite these and many other obstacles inherent to alcohol administration research involving high-risk participants, the inclusion of psychoeducational materials, referral lists, and brief motivational interventions is viewed as direct benefits to participants. Other procedures may be used to maximize participant safety. These include researchers' assessment of current relationship conflict to determine readiness for the experimental protocol, use of structured protocols which assess for both partners' perception of safety in the relationship and conducting of follow-up phone interviews.

Intervention Implications

Should the use of the proposed methods yield empirical data in support of these hypothesized mechanisms, the field will be ready for the development of AAM-informed intervention strategies designed to reduce IPV following alcohol consumption. The critical ingredient of these strategies will be their ability to direct the inebriate's attention away from cues that instigate IPV and toward cues that inhibit IPV. Such strategies can then be tested within the laboratory environment and in the field to demonstrate that they reduce alcohol-facilitated aggression via these mechanisms. Examples of AAM-informed strategies are reviewed elsewhere (see Giancola et al., 2010). However, given that the most likely location for alcohol-related IPV is in the home (Leonard, Quigley, & Collins, 2002), application of such strategies—which include potentially discreet physical cues such as non-violence chips or wristbands—to domestic settings merits attention here. Importantly, the implementation of AAM-informed strategies in domestic settings must be tailored to the individual. AMT posits that cue salience is the critical predictor of attentional focus; however, individuals certainly differ in what they perceive to be salient as well as in their dominant response to a given salient cue (K. Leonard, personal communication, August 13, 2013). For these

[1]Key National Institute on Alcohol Abuse and Alcoholism (NIAAA) guidelines may be found at http://www.niaaa.nih.gov/Resources/ResearchResources/job22.htm#populations

reasons, the clinician must work closely with an individual to ensure that the selected intervention cue(s) is likely to be salient to the individual and facilitate the desired inhibitory (or non-aggressive) response.

Conclusion

We propose a new line of research that has yet to be undertaken in the field of IPV: a direct test of the theoretically based mechanisms for alcohol-facilitated IPV. The "perfect storm" hypothesis stemming from I^3 theory is especially relevant here, highlighting the risk for IPV when instigation is high, when provoked individuals possess aggression-eliciting impellors, and when factors are present that lower inhibitory mechanisms. Interactions among these factors can then be tested in more detail using empirically informed models, such as AMT, that can establish mediational mechanisms to estimate causal relationships among the variables of interest. Of course, these efforts require specific research methods to establish these types of causal inferences, and we have outlined a series of design and measurement innovations to assess the mediational roles played by attention, cognition, and affect.

These innovations have important implications for the field, which at present has limited evidenced-based options for the reduction of alcohol-facilitated IPV. Extant literature indicates that treatment for an alcohol-use disorder exerts a small-to-moderate effect on the reduction of IPV (Murphy & Ting, 2010). Continued development of these interventions is necessary, as reducing or eliminating alcohol use will clearly reduce alcohol-related IPV. However, even if treatment for an alcohol-use disorder was deemed a first-line intervention for IPV, the reality is that many patients do not achieve sustained abstinence, and the long-term effects of these interventions are unknown. Thus, it is critical that we also develop intervention strategies that reduce IPV for individuals who have *already consumed alcohol*. The long-term aim of the proposed work is to address this need. In addition, the field may also consider approaches that harness the power of bystanders. Decades of research have examined predictors of bystander behavior in response to emergency and non-emergency situations (for a review, see Fischer et al., 2011). However, there exists virtually no research on the factors that facilitate bystanders' decision to intervene in an alcohol-related episode of IPV. Collectively, these three intervention strategies could form a robust and comprehensive approach to the reduction of alcohol-facilitated IPV. However, to realize the potential of these approaches, rigorous and innovative research is needed to provide the necessary evidence base for their implementation.

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Biographies

Christopher I. Eckhardt, PhD, is an associate professor of psychological sciences at Purdue University in West Lafayette, Indiana. His research examines the cognitive and affective correlates of intimate partner violence, the effects of alcohol on these processes, and how risk factor research may inform interventions for IPV offenders. His research has resulted in more than 50 publications and has been supported by awards from the National Institute on Alcohol Abuse and Alcoholism, National Institute of Mental Health, National Institute of Justice, the Centers for Disease Control and Prevention, and the H.F. Guggenheim Foundation.

Dominic J. Parrott, PhD, is a clinical psychologist and an associate professor at Georgia State University. His research uses laboratory and survey methods to examine risk factors and mechanisms for aggression, with a particular emphasis on the effects of alcohol on IPV, aggression toward sexual minorities, and sexual aggression. An end goal of his research program is to inform directly the development of interventions that prevent or reduce alcohol-related violence. His work has resulted in over 70 scientific articles and been funded by the National Institute on Alcohol Abuse and Alcoholism and the Centers for Disease Control and Prevention.

Joel G. Sprunger, MS, is a doctoral student in the clinical psychology program at Purdue University. He received his BA in psychology from Purdue University at the Indiana University—Purdue University Fort Wayne campus and his MS in psychology from Purdue University in West Lafayette, Indiana. His research interests involve the examination of process models for IPV risk factors, emphasizing the simultaneous consideration of aggression-promoting (e.g., alcohol intoxication, affective, and social information-processing biases) and aggression-inhibiting (e.g., relationship investment, antiviolence attitudes) factors to inform estimates of future partner-directed aggressive events and treatment/prevention efforts.

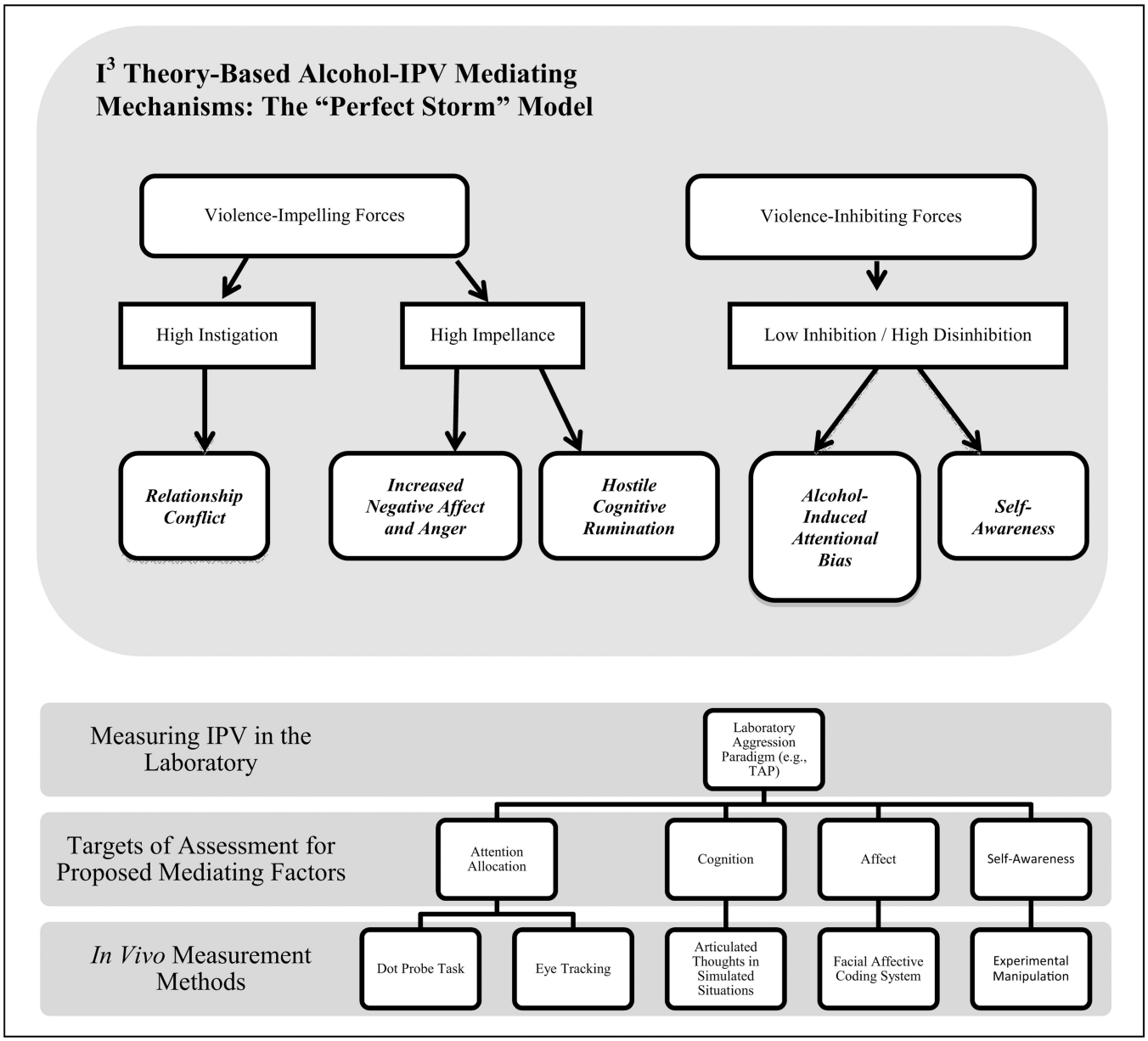

Figure 1.

I^3 theory-based alcohol–IPV mediating mechanisms: The "perfect storm" model.

Note. IPV = intimate partner violence.

This chart represents the flow of discussion for measuring IPV in the laboratory. IPV-inducing factors, including potential mediating mechanisms, are proposed via I^3 theory (Finkel & Eckhardt, 2013). Methods for assessing IPV in the laboratory are presented as a means to assess mediating mechanisms in the context of an IPV-relevant provocation (e.g., the Taylor Aggression Paradigm [TAP]; Taylor, 1967). Next, target constructs of proposed mediators are presented along with specific laboratory methods for their measurement or manipulation.