Anger, Hostility, Internalizing Negative Emotions, and Intimate Partner Violence Perpetration: A Meta-Analytic Review

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Abstract

Prior reviews have identified elevated trait anger as a risk factor for intimate partner violence (IPV) perpetration. Given that 10 years have passed since the last comprehensive review of this literature, we provide an updated meta-analytic review examining associations among anger, hostility, internalizing negative emotions, and IPV for male and female perpetrators. One hundred and five effect sizes from 64 independent samples (61 studies) were included for analysis. IPV perpetration was moderately associated with the constructs of anger, hostility, and internalizing negative emotions. This association appeared stronger for those who perpetrated moderate to severe IPV compared to those who perpetrated low to moderate IPV, and did not vary across perpetrator sex, measurement method, relationship type, or perpetrator population. Implications and limitations of findings were reviewed in the context of theoretical models of IPV, and future directions for empirical and clinical endeavors were proposed.

Keywords

Anger; Hostility; Internalizing negative emotions; Intimate partner violence; Meta-analysis

Intimate partner violence (IPV), defined as physical, sexual, or psychological harm perpetrated by a current or former romantic partner (Saltzman et al., 2002), continues to be a pervasive problem of paramount societal, public health, and economic importance. Over a lifetime, more than 1 in 3 women (35.6%) and more than 1 of 4 men (28.5%) will have experienced physical violence, rape, and/or stalking by an intimate partner (Black et al., 2011). The economic burden of the aforementioned acts amounts to more than 8.3 billion dollars annually in the U.S. (National Center for Injury Prevention and Control, 2003; Max et al., 2004). Male-to-female IPV results in more negative health consequences than female-to-male IPV (Archer, 2000; Holtzworth-Munroe, Smutzler, & Sandin, 1997) and female survivors of IPV report more physical and mental health problems than women who do not report partner abuse, including increased use of general health services (Miller, Cohen, &
Rossman, 1993) and greater likelihood for chronic pain, diabetes, depression, substance use, and suicide (Black et al., 2011; Campbell, Sullivan, & Davidson, 1995; Golding, 1996; Kessler et al. 1994). The effect of IPV victimization on mental health consequences in women has been documented in numerous longitudinal studies (Lawrence, Oreno-Aguayo, & Brock, 2012), suggesting that IPV experiences may be important antecedents (rather than consequences) of adverse health outcomes (Ehrensaft, Moffitt, & Caspi, 2006). Male victims of IPV are also at increased risk for mental health symptoms (Hines & Malley-Morrison, 2001) and a subset are victims of severe IPV (Hines & Douglas, 2010). Thus, the ongoing search for reliable risk factors for IPV perpetration is essential to promote: (1) the development of comprehensive etiological models of IPV; (2) improvement of IPV risk assessment methods; and (3) development of empirically supported intervention and treatment programs for perpetrators of IPV.

In the present paper, we will review the available research evidence regarding the status of anger, hostility, and internalizing negative emotions as risk factors for IPV. Prior reviews have suggested that anger and hostility are moderately, if inconsistently, associated with IPV-related outcomes (e.g., Norlander & Eckhardt, 2005), and several theoretical models outlining the etiology of IPV indicate that anger and heightened negative emotionality may serve as risk factors for IPV perpetration (Finkel, 2007; O’Leary, 1988). These findings build on data from the general interpersonal aggression literature that indicate a moderate, positive association between aggressive behavior and anger arousal (Anderson & Bushman, 2002). Moreover, longitudinal studies have also suggested that a pattern of negative affect may prospectively predict IPV in the transition from adolescence to adulthood (e.g., Moffitt, Krueger, Caspi, & Fagan, 2000). Clinically, interventions for partner abusive individuals often include emotion control as a technique to promote nonviolent change (e.g., Murphy & Eckhardt, 2005), and results of several meta-analytic reviews indicate that psychological treatments for anger problems have large effects on the reduction of anger related clinical symptoms (Del Vecchio & O’Leary, 2004). Thus, there are a wide variety of empirically supported reasons to presume that IPV perpetration is significantly associated with anger, hostility, and internalizing negative affect.

However, there are also several factors that complicate the field’s enthusiasm for continued research in this area. First, prior reviews suggest that the aforementioned moderate effect of anger on IPV is inconsistent, and varies widely depending on the specific measurement method used to assess anger-related constructs (Eckhardt et al., 1997). Second, there exists substantial controversy in the IPV field about the inclusion of anger and related affective constructs in etiological models of IPV (Gondolf, 2002). As we will discuss in a later section, some scholars have expressed concern that a focus on anger-related factors may diminish offender accountability and provide the offender with a handy excuse on which to diffuse personal responsibility for their abusive acts (Gondolf & Russell, 1986). Perhaps unsurprisingly, there is very little research on the effectiveness of anger or emotion control interventions for IPV perpetrators; in fact, many state coalitions against domestic violence explicitly prohibit the usage of anger control techniques in their standards for IPV intervention (Rosenbaum & Kunkel, 2009).
Thus, there is a substantial need to provide an empirical answer to the as-yet unresolved question about whether anger-related phenomena are important in understanding and treating IPV. In the present review, we discuss the current state of theory and research regarding anger, internalizing negative emotions, and IPV, and provide an updated meta-analytic review of associations among these constructs. We conclude with a discussion of what is known, as well as what is currently unresolved, in our understanding of the anger-IPV association, and outline the clinical implications of these findings.

Theoretical Overview

There currently exists a substantial, and hotly contested, debate in the IPV field about whether anger has any meaningful relation to IPV (Healey, Smith, & O’Sullivan, 1998; Norlander & Eckhardt, 2005; Dutton, 2010), whether anger-related constructs should be included in assessment for IPV risk (Murphy & Eckhardt, 2005; Holtzworth-Munroe, Beatty, & Anglin, 1995), and whether anger-related variables should be the focus of IPV interventions to any degree (Gondolf & Russell, 1986; McMahon & Pence, 1996; Murphy & Eckhardt, 2005). Much of this debate stems from assumptions based on the earliest, and still currently popular, model of IPV etiology: Power and control theory. This model, which is the predominant perspective in the broader IPV field, focuses exclusively on gender socialization patterns and defines IPV as male-to-female violence deeply rooted in gender-based power dynamics that play out in the romantic context (Bograd, 1988; Pence & Paymar, 1993; Pence & Dasgupta, 2006). Given males’ presumed tacit alignment with patriarchal power structures, men utilize a variety of techniques to coerce and control their female partners in close relationships in order to facillitate overarching goals of male power and privilege, including (but not limited to) physical aggression (Dobash & Dobash, 1979). Thus, adherents to this model place little emphasis on factors internal to the individual (such as anger or other negative emotions) as causes of behavior, preferring instead an analysis of community and contextual-based determinants of power-and-control socialization patterns (e.g., Gondolf, 2012; Gondolf & Russell, 1986; Healey, Smith, & Sullivan, 1998).

The gender-themed power-and-control model continues to be the dominant ideology that wields enormous influence over IPV research, policy, and intervention. However, research findings question many of the core etiological tenets of this approach (for reviews, see Archer, 2000; 2013; Bates, Graham-Kevan, & Archer, 2014; Dutton & Corvo, 2006; Dixon & Graham-Kevan, 2010; Hamel, 2007), and reviews of the effectiveness of interventions for IPV perpetrators report small to nonexistent effects of interventions that are grounded in the power-and-control approach (e.g., the “Duluth Model”; Pence & Paymar, 1993) on IPV-related outcomes (Babcock et al., 2004; Eckhardt et al., 2013; Feder & Wilson, 2006; Smedslund et al., 2012). Nevertheless, of states that currently have standards that outline the structure and content of intervention programs for IPV offenders, the majority include statements of etiology or principles of practice that reference the power-and-control-based patriarchal ideology model (Dutton & Corvo, 2006), with the Duluth Model remaining the “unchallenged treatment of choice for most communities” (Babcock et al., 2004, p. 1026).

This discussion is not intended to suggest that issues relating to power and coercion are unimportant; most, if not all, empirically based models of IPV include the role of gender.
and/or sources of inequality between men and women as distal factors that inform efforts to understand IPV and to intervene with its perpetrators and survivors. However, there is very little empirical support for a strictly gendered analysis of IPV that restricts the understanding of IPV to the behaviors enacted by men towards women, or that organizes IPV risk factors solely around gender-themed attitudes or behaviors, especially as proximal causes of IPV-related outcomes. Rather, the available data suggest a gender-inclusive approach to IPV etiology that considers a wide range of individual, interpersonal, and contextual risk factors that may lead both men and women to act aggressively towards an intimate partner (Dixon & Graham-Kevan, 2011; Felson & Lane, 2010). Of relevance to this report, several theoretical models appear to offer support for anger, hostility, and internalizing negative affect as important risk factors for IPV perpetration. These approaches are discussed below.

Intrapersonal Models

Intrapersonal models of IPV attend to factors internal to the perpetrator, such as heightened anger or negative emotions, that increase the likelihood of IPV perpetration. In contrast to the power-and control model, intrapersonal models allow researchers and clinicians to investigate why different individuals socialized into similar sociocultural circumstances can differ significantly in their tendencies toward IPV perpetration. While there are a wide range of models of IPV that might fall under the classification of an intrapersonal approach, the social learning theory analysis of interpersonal aggression was a noteworthy starting point in understanding how, and in what context, intrapersonal factors influence partner aggression. Social learning theory suggests that people acquire tendencies toward aggression, including IPV perpetration, through basic principles of learning—classical conditioning, operant conditioning, and observational learning—which shape them to act aggressively (Bandura, 1973). Consistent with the social learning model, IPV perpetrators are more likely than non-perpetrators to report witnessing IPV in the family of origin and to have been physically abused as children (e.g., Barnett & Fagan, 1993; Dutton et al., 1996; Howell & Pugliesi, 1988; Kwong, Bartholomew, Henderson, & Trinke, 2003). While reviews of this literature indicate a small-to-moderate effect of family of origin violence on subsequent IPV (Delsol & Margolin, 2004; Stith et al., 2000), the broader implication is that aversive experiences occurring in the family of origin may impact the development of a wide range of internal factors (e.g., aggressogenic attitudes; disrupted emotion regulation; insecure attachments) and relevant behaviors (e.g., verbal and physical aggression), which in turn may reciprocally lead the individual towards similar aversive contexts (e.g., violence-supporting peers; conflict-laden relationships).

The prominence of social learning models of interpersonal aggression was also reinforced by developments in cognitive-behavioral treatments (CBT) for anger and aggression (Beck, 1999; Novaco, 1977). CBT researchers suggested that a variety of cognitive and affective factors place certain individuals at risk for aggressive behavior. For example, social information processing models of aggression posit that individuals engage in a number of mental steps before behaving in social environments, including encoding of sensation and perceptual cues (subject to attentional biases); integration of cues with memory/past experience and forming interpretations (subject to hostile attribution biases); generation of response styles (e.g. anger control, anger expression, aggression); and evaluation of
consequences and selection of response (Crick & Dodge, 1994). Individuals at risk for interpersonal aggression may selectively encode certain aspects of instigating situations and interpret them in ways that increase hostile cognitions, promote angry affect, and endorse aggressive response styles (Dodge & Pettit, 2003).

Anger was also implicated as a risk factor for IPV based on associative network and script models of aggressive behavior. These theories indicated that contextual stimuli serve to automatically initiate not only biased social information processes, but also activate other components of one’s cognitive-affective-memory network that may be associatively linked with these contextual cues (Berkowitz, 1989; Huesmann, 1988). For example, the cognitive neoassociation model (Berkowitz, 1990; 1993; 2012) suggests that exposure to particular external or internal contextual cues (e.g., tone of voice; spillover of work stress) may initiate an experience of unpleasant negative affect, which then activates related thoughts, memories, images, attributions, etc., associated with the social context. These largely automatic and implicit factors serve to further refine the individual’s initial undifferentiated negative affect and guide behavioral outputs; for some, these processes may intensify the emotional experience into maladaptive levels of anger or related negative emotions, which in turn may lead to a higher probability of aggressive problem-solving responses. Reviews of the literature support this model (Berkowitz, 2008), as activation of physiological responses in hostile contexts and angry appraisals of the aggressive target are predictive of aggressive responding following exposure to instigating situational cues.

Regarding IPV, Holtzworth-Munroe (1992) developed a social information processing model specific to IPV, in which errors in decoding, decision making, and enactment were hypothesized to increase the likelihood of intense emotional experiences, such as anger, and to facilitate aggressive responding. Relative to their nonviolent counterparts, IPV perpetrators exhibit (a) decoding, interpretation, and hostile attribution biases; (b) less competent decision making (i.e., greater generation of aggressive response options); and (c) more positive and less negative evaluations of violence in close relationships (for reviews, see Eckhardt & Dye, 2000; Holtzworth-Munroe, 2000; Murphy & Eckhardt, 2005; Stith et al., 2004). There is also evidence to support specific predictions from associative models of aggression regarding the automatic nature of cued associations that may relate to IPV. Specifically, IPV perpetrators show more biased implicit attitudes favoring aggressive stimuli relative to nonviolent individuals (Eckhardt, Samper, Suhr, & Holtzworth-Munroe, 2012), with biased attitudes also relating to the frequency of prior year IPV and responsiveness to IPV interventions (Eckhardt et al., 2012; Eckhardt & Crane, 2014). Together, these cognitively-oriented, intrapersonal models indicate that as a result of these cognitive biases and distortions, IPV perpetrators experience higher levels of anger, contempt, disgust, and other forms of intense negative affect during relationship conflict (Eckhardt, 2007; Eckhardt, Barbour, & Stuart, 1997; Gottman et al., 1995; Jacobson et al., 1994; Norlander & Eckhardt, 2005; O’Leary, 1988).

Lastly, in contrast to social information processing models, some intrapersonally focused scholars propose that IPV may stem from a pattern of individual differences that reflect long-standing disturbances to personality dynamics, attachment styles, and psychopathological symptoms. For example, one of the more consistent risk factors for IPV...
is the early and stable presence of antisocial behaviors and oppositional/conduct disorder symptoms (for a comprehensive review of this literature, see Capaldi et al., 2012). Importantly, anger, hostility, and anxiety are highly correlated with this antisocial trait and are predictive of both IPV-related and non-IPV related violent crimes in longitudinal studies (Moffitt, Krueger, Caspi, & Fagan, 2000; White & Widom, 2003). Researchers have demonstrated that individuals with high levels of negative emotionality (anger, hostility, anxiety) and neuroticism perpetrate higher rates of IPV than those lower in these traits (Hellmuth & McNulty, 2008). The traumatic origin of this affective instability was discussed by Dutton (2010), who proposed a developmental model of IPV that highlights the role of attachment insecurity in developing a specific and pervasive pattern of affective instability similar to the borderline personality organization construct. As noted by attachment theorists (e.g., Bowlby, 1973), early traumatic experiences with highly rejecting and/or abusive caregivers establishes a lingering fear of being abandoned in close relationships combined with heightened trauma-related anger or rage (“intimacy anger”). During subsequent romantic conflicts, this fearful attachment style is activated and the individual may emit angry displays, aggressive actions, and other coercive behaviors in efforts to prevent abandonment and exert control (albeit ineffective) over their partner and their own unstable sense of self (Dutton, 2010). This pattern leads to a tendency for the perpetrator to blame the partner as the primary cause of relationship conflicts, and to produce hypervigilance and rumination about the next potential rupture in the relationship. Research has generally supported these core assumptions. Intimacy anger, borderline personality organization, preoccupation with partner blame, and fearful-angry attachment predict IPV perpetration (Dutton, 1995; 2010; Dutton, Starzomski, Saunders, & Bartholomew, 1994), although the proximal nature of how these factors impact acute IPV and the exact developmental trajectory of this pattern remain to be articulated.

**Interpersonal Models**

In contrast to power-and-control and intrapersonal perspectives, researchers investigating the nature of close relationships and couples’ therapy scholars have noted that (1) relationship conflict emerges from partners’ frequently interdependent exchange of behaviors and negative affective expressions (e.g., Gottman, 1998; Gottman et al., 1976; Gurman & Jacobson, 2002), and (2) relationship conflict and relationship distress are consistent predictors of IPV (Jacobson, 1994; Murphy & Eckhardt, 2005; Schumacher et al., 2001; Stith et al., 2004). A large body of research indicates that this negative reciprocity sequence is in part characterized by repeated exchanges of anger, hostility, contempt, and belligerence (Cordova et al., 1993; Gottman, 1979; Hellmuth & McNulty, 2008). This pattern has also been found to differentiate couples with a history of IPV from couples who are distressed but not violent (Burman, Margolin, & John, 1993), such that when one spouse displayed angry/contemptuous behavior it was much more likely to trigger an angry/hostile behavioral response from the other spouse. Furthermore, higher levels of negative reciprocity were found for both spouses in violent couples but not within non-violent satisfied or non-violent discordant couples (Cordova et al., 1993). Given evidence suggesting that IPV victimization may the single strongest predictor of IPV perpetration (O’Leary, Slep, & O’Leary, 2007), it is therefore critical to understand the affective
dynamics that characterize the ways in which couples with at least one violent partner interact, and to incorporate this information into effective clinical interventions.

Meta-Theoretical Approaches

There have been relatively few attempts to construct broad multifactorial models of IPV perpetration, and even fewer that extend these factors to a predictive, process-level of model of IPV risk. Current theories attempting to integrate findings across the widely varying IPV literature essentially provide a topographic overview of the variables, including those devoted to negative affect and personality, that are associated with IPV perpetration (e.g., Bell & Naugle, 2008; Dutton, 1995; Holtzworth-Munroe & Stuart, 1994; O’Leary et al., 2007). However, these approaches emphasize levels of analysis or classes of variables, rather than the mechanisms through which particular risk factors interact to increase the probability of IPV perpetration. Another comprehensive model of IPV, Capaldi et al.’s (Capaldi, Kim, & Shortt, 2004; Capaldi, Shortt, & Kim, 2005) dynamic developmental systems perspective (DDS), offers a marked improvement over traditional topographic multicomponent models. The DDS model is an heuristic that examines the interaction of three factors in the prediction of IPV: (1) contextual characteristics (e.g., background factors); (2) developmental characteristics, and (3) relationship influences. These risk factors can be examined as relational dynamics or as individual-level risk factors, and can be used to model dynamic cascade effects in which the presence of a risk factor may trigger a cascade of consequences and alter developmental trajectories. This model would appear to have particular relevance to researchers interested in an interpersonal / developmental theoretical framework.

In contrast to other models of IPV, we present I$^3$ Theory (“I-Cubed”) as a comprehensive, multifactorial, and process level model to understand the role of anger and negative emotions in IPV (Finkel, 2007). This model not only outlines the classes of variables related to abusive behavior but also the fundamental processes (and their interaction) that are necessary and sufficient for IPV to occur (Finkel & Eckhardt, 2013). This framework can be considered a “meta-theoretical” approach to understanding IPV risk. Like other meta-theoretical approaches, such as the General Aggression Model (Anderson & Bushman, 2002), I$^3$ Theory does not restrict the prediction of IPV to one decisive risk factor (or set of factors) or to one particular theoretical level of analysis (e.g., intrapersonal vs. interpersonal). Rather, I$^3$ Theory suggests that scholars can predict whether a given interaction between intimate partners will be violent versus nonviolent if they can discern the strength of instigation (i.e., context), degree of impellance (e.g., aggression-facilitating traits), and presence of inhibitory factors. Despite the limitations of the monothetical approaches reviewed above, each can contribute to an I$^3$ Theory analysis of IPV perpetration as long as data emerging from these approaches can be shown to represent a process or set of processes through which their hypothesized risk factors (e.g., power and control, intimacy anger) promote IPV perpetration (Finkel, in press).

Specifically, I$^3$ Theory suggests that instances of IPV can be predicted by three general process-level factors: Instigation (situational factors that normatively potentiate an urge to aggress; i.e., provocation); Impellance (dispositional or situational factors that promote a
strong urge to aggress; i.e., anger, hostility, negative emotions); and Inhibition (factors that increase the likelihood that the urge to aggress will be over-ridden; e.g., self-control) (Finkel & Eckhardt, 2013). While any particular process-level factor may be associated with IPV, the advantage of this model is that it allows researchers to model interactions among these processes. Theoretically, the greatest likelihood for IPV would occur when instigation and impellance processes are strong and inhibitory processes are weak, previously described as “perfect storm” conditions (Finkel et al., 2012; Finkel & Eckhardt, 2013). For example, IPV is especially likely (a) during a particularly volatile verbal argument over perceived infidelity (strong instigation) involving a partner (b) with dispositional tendencies towards high anger arousal during conflict and a trait tendency to interpret the behaviors of others as hostile (strong impellors), and (c) who possesses unusually poor self-control due to high work stress and current alcohol intoxication (weak inhibition). Prior research has supported the use of the Instigation x Impellance x Inhibition perfect storm interaction to predict interpersonal aggression and IPV (Eckhardt, 2007; Finkel, in press; Finkel et al., 2012; Sinclair, Ladny, & Lyndon, 2011; Slotter et al., 2012).

Summary—There are a multitude of theoretical models that support the notion that high levels of anger, hostility, and internalizing negative emotions may be linked to the perpetration of IPV. Whether the route from anger to IPV is via social information processing, personality dynamics, and/or as a moderated effect in combination with other factors, a variety of frameworks support this connection. As summarized by Anderson and Bushman (2002), there are at least three ways that our understanding of violence risk may be improved by considering the perpetrator’s manner of experiencing and expressing anger and negative emotions. First, the experience of anger may justify the use of aggression; individuals who chronically experience anger and strong negative emotions are more likely to excuse such behaviors as context-appropriate. Second, each angry interaction serves to keep the person focused on aggressive motivations and immersed in a script revolving around themes central to anger and aggressive revenge. To the extent that individuals report more frequent and intense anger experiences, the more likely these aggressive scripts are reinforced by their hostile interactions with the social environment. Third, anger is an approach-related emotion (Carver & Harmon-Jones, 2009) that is associated with increased arousal states. These states of excitation may be transferred to a romantic partner and increase risk of aggressive problem solving styles.

Thus, there are persuasive theoretical reasons to presume that anger is at least associated with IPV perpetration. With the goal of providing an evidence-based review of this association, we next discuss prior reviews of this literature and conclude with clear definitions of the core constructs involved in this meta-analysis.

Prior Reviews and Meta-Analyses

While several early qualitative reviews of the literature on risk factors for IPV suggested the importance of anger and hostility (Holtzworth-Munroe et al., 1997; Tolman & Bennett, 1990), the first targeted review of anger, hostility, and IPV (Eckhardt, Barbour, & Stuart, 1997) concluded that there was an association between these constructs that depended on the specific construct (i.e., anger vs. hostility) and the method in which these constructs were
assessed (self-report vs observational). Subsequent meta-analyses of IPV risk factors confirmed a modest association between anger, hostility, and IPV (Schumacher et al., 2001; Stith et al., 2004). A meta-analysis by Norlander and Eckhardt (2005) reported moderate effect sizes for anger/hostility and IPV ($d = .51$), with the effect size for hostility ($d = .58$) higher than the effect for anger ($d = .47$) in differentiating between men with and without an IPV history.

Previous quantitative and qualitative reviews have also examined whether there is an association between various internalizing negative emotions (e.g., depression, anxiety) and IPV. Relative to nonviolent comparisons, perpetrators of IPV have higher rates of depressive disorders (Kessler et al., 2001), and perpetrators of severe IPV report higher rates of a broad range of psychopathology, especially anxiety-related or nonaffective psychotic disorders (Danielson et al., 1998; Kessler et al., 2001). Prospective studies suggest that the presence of major depressive episodes at age 18 for men and women predicts greater odds of being involved at age 26 in an abusive relationship involving injury or requiring intervention by the criminal justice system (Ehrensaft, Moffitt, & Caspi, 2006). A meta-analytic review by Stith et al. (2004) reported a moderate effect size between depression and IPV ($d = .48$, $k = 14$), but only among male IPV perpetrators. The importance of depression as an indicator of risk for IPV was recently explored in a qualitative review of the literature by Dutton and Karakanta (2013). The authors concluded that depressive affect may increase angry rumination and impulsivity, which in turn may increase risk of aggressive responding via an “affective swamping” effect, whereby perpetrators have difficulty differentiating between internal and external attributions for negative affect and target their internal dysphoria towards their partner.

Thus, while previous reviews and meta-analyses of the constructs of interest have provided an important foundation for understanding whether IPV is associated with anger, hostility, and internalizing negative emotions, there are compelling reasons for an updated review of this literature. First, it has been almost a decade since the last targeted review of anger/hostility and IPV, and an updated synthesis of the literature is needed. Second, it is important to evaluate the comparative effects of anger/hostility on IPV relative to internalizing negative emotions (e.g., depression, anxiety). Third, existing meta-analyses have not evaluated the burgeoning literature on female perpetrators of IPV as it relates to affective risk factors (Bair-Merritt et al., 2010; Straus, 2005; Stuart et al., 2006). Specifically, the current review will provide a quantitative investigation of the association between internalizing negative emotions (e.g., depression, anxiety, and other negative affect) and IPV; an updated synthesis of the literature examining the association between anger, hostility, and IPV that includes not only the most recent literature but that also used novel observational methods to assess these constructs; and the inclusion of a wider range of potential moderators of the anger/hostility/internalizing negative emotions and IPV association, such as sex of perpetrator, perpetrator population, and type of relationship.

**Definitions of Constructs**

As noted in prior reviews of this literature (e.g., Norlander & Eckhardt, 2005), researchers have traditionally ignored important distinctions among many of the constructs involved in
this review. In particular, there has been a long-standing tendency to combine measures of anger and hostility into a single construct, or to use these terms interchangeably when reporting results. While this practice is less commonly observed in the current literature relative to 10–15 years ago, there is still a critical need to consider separately the constructs of anger and hostility. Similarly, researchers do not routinely specify how anger-related constructs are linked with other negative affective experiences. Indeed, personality and psychopathology researchers (e.g., Markon, Krueger, & Watson, 2005; Wright et al., 2012) have suggested that while there is empirical support for differentiating among anger-related traits (“antagonism”) relative to anxious traits (“negative affect”) and depression-related traits (“detachment”), these factors are nested within a broader hierarchy of Internalizing and Externalizing dimensions. Additional research is needed to examine broader commonalities existing between the affective risk factors investigated in IPV research.

While there is substantial difficulty in defining any particular emotion (cf. Izard, 2010), scholars have emphasized their adaptive nature and the multiple components that are variously activated but simultaneously experienced as a particular emotion (Nesse & Ellsworth, 2009). With this multicomponent model in mind, we define anger as a multidimensional construct comprised of physiological (sympathetic nervous system arousal), cognitive (appraisals about the meaning, causes, and significance of an event), phenomenological (labeling of angry feelings, self-awareness), and behavioral variables (approach-related motivations; verbal and behavioral expressions) (Berkowitz, 1993; Eckhardt et al., 1997; Eckhardt & Deffenbacher, 1995). There is insufficient evidence to suggest that any one of these components is more central to understanding the emotion of anger relative to another (Barrett, 2013), and most measures of anger emphasize a limited range of factors related to these multiple components.

Hostility, in contrast to anger, has been conceptualized traditionally as an attitudinal disposition characterized by a tendency toward negative judgment and dislike of others (Buss, 1961). The most recent meta-analysis on anger, hostility and IPV (Norlander & Eckhardt, 2005) used Miller and colleagues’ (1996) definition of hostility as a variable characterized by cynicism (believing the behaviors of others are motivated by selfish interests), mistrust (overarching belief that others are intentionally hurtful and provoking), and denigration (viewing others as mean and dishonest). The cognitive components of hostility are conceptualized as a predisposition to anger experience and expression that may motivate aggression or create aversive contexts that increase its likelihood (Spielberger, 1988). While there are theoretical and functional areas on which anger and hostility overlap, it remains important to distinguish between these constructs as a means to estimate the construct validity of how these factors affect IPV risk (Eckhardt et al., 2007; Norlander & Eckhardt, 2005).

Internalizing negative emotions—Several internalizing negative emotions appear to serve as both risk factors (Dutton & Karakanta, 2013; Julian & McKenry, 1993; Mauiro et al., 1998; Pan, Neidig, & O’Leary, 1994) and outcomes (Swan & Snow, 2005) of IPV perpetration. In order to establish whether internalizing constructs have a unique association with IPV separate from the effects of anger and hostility, we included a variety of variables related to internalizing negative emotion (i.e., anxiety, depression, negative affect, negative
emotionality) in this meta-analytic review. Negative affect refers to the experience of a range of negative emotions, while negative emotionality implies a trait-like tendency/susceptibility toward the experience of negative affect, including anger, hostility, mistrust of others, anger and anxiety, ineffective reactions to stress, and thoughts of revenge (Moffit, Caspi, Rutter, & Silva 2001). Longitudinal findings by Capaldi and colleagues (Capaldi & Stoolmiller, 1999; Kim & Capaldi, 2004; Kim, Laurent, Capaldi, & Feingold, 2008) indicate that depression may indirectly increase risk for male-to-female IPV primarily through its destructive effects on relationship quality and increase in conflict/arguments, especially for women’s depression symptoms (see also Dutton & Karakanta, 2013). Anxiety and depression are heterogeneous in definition across the studies reviewed in this meta-analysis. These constructs were assessed within a subset of studies as indicators of specific psychopathology that contained symptoms of internalizing negative affect alongside behavioral and cognitive indicators (e.g., loss of sleep, excessive rumination), while observational assessments defined depression and anxiety as purely affect-based (e.g., sadness) or a mix of physiological and affective experiences (e.g., tension/fear).

Moderators of the Associations Among Anger, Hostility, Internalizing Negative Emotions and IPV

Based on prior reviews of the literature, we examined the following variables as potential moderators of the association among anger, hostility, internalizing negative emotions and IPV: sex/gender of perpetrator, relationship status, population, and measurement type.

Sex of perpetrator—Recent reviews of IPV have found comparable rates of perpetration for males and females (Archer, 2000; 2002; 2006), and in some circumstances rates of female-to-male IPV in nationally representative community samples have exceeded those of male-to-female IPV (Caetano, McGrath, Ramisetty-Mikler, & Field, 2005). As an increasing number of studies have included or exclusively examined female-to-male IPV perpetration (Kim & Capaldi, 2004; Swan et al., 2005), it is necessary to investigate sex/gender differences on the associations of interest in the current meta-analysis.

Relationship status—Traditionally, the literature examining male-to-female IPV has focused almost exclusively on married couples or couples who were living “as if married” (Barbour et al., 1998; Dutton & Browning, 1988; Margolin, 1988), perhaps reflecting relatively stable or longer-term relationships. More recent investigations of IPV have included a wider range of relationship types (e.g., dating or cohabitating) that could account for some of the variability in the constructs of interest. In the present review, married, dating-cohabiting, and “living as if married” samples will be compared to dating, non-cohabitating samples to test the assertion by Stith and colleagues (2004) that married/cohabitation IPV is a distinctly different phenomenon from IPV perpetration outside of a cohabitating relationship. An additional exploratory moderation analysis was conducted in order to compare married to dating (cohabitating, non-cohabitating, and mixed by cohabitation) relationship samples.

Population—it is unclear whether heterogeneity in the relationships between anger, hostility, negative emotions, and IPV may be accounted for by differences in the perpetrator...
populations sampled across studies (e.g., clinical vs. community). It is important to test for population-based differences as results could have the potential to inform assessment and intervention efforts.

**Measurement type**—While self-report measures may provide the perpetrator’s account of their anger experiences or negative affective tendencies, it is difficult to generalize from responses to generally worded questionnaires to specific relationship situations and IPV-related contexts. Relative to self-report endorsement measures, observational assessment approaches provide a more externally valid avenue for discerning acute anger and negative affect in the context of imagining relationship-specific scenarios (e.g., the articulated thoughts in simulated situations task; Davison, Robins, & Johnson, 1983; Eckhardt, 2007) or during acute conflict discussions with one’s partner (e.g., the specific affect coding system, or SPAFF; Gottman & Krokoff, 1989). No predictions were made for measurement type, as the effects of this variable on the association between anger, hostility, and IPV have been inconclusive across reviews (Eckhardt et al., 1997; Norlander & Eckhardt, 2005).

**Current Meta-Analysis**

Prior literature reviews, as well as recent theory and research, suggest that the field’s understanding of IPV etiology and intervention may be improved by accounting for perpetrators’ tendencies towards anger, hostility, and internalizing negative emotions. In the current review, we provide an updated synthesis of this research literature in light of recent definitions of the constructs of interest and given the potential influence of several moderators previously outlined. Specifically, we predicted: (1) a moderate, positive association among anger, hostility, and IPV; (2) a positive association between internalizing negative emotions and IPV, albeit smaller than the anger/hostility IPV correlation given the less extensive etiological support for this association; and (3) sex/gender would not moderate the associations among anger, hostility, internalizing negative emotions, and IPV given evidence of symmetry across sex for IPV perpetration (Archer 2000, 2002) and consistency in IPV motivations between males and females (Straus, 2005; Stuart et al., 2006). Given inconsistencies in the literature, we did not make specific predictions about the moderating roles of IPV perpetrator population (i.e., community, clinical), relationship type (e.g., dating, married, cohabitating), or measurement method (e.g., self-report, observational).

**Method**

**Procedure**

First, all studies evaluated in the previous meta-analysis on anger and hostility among male perpetrators of IPV (Norlander & Eckhardt, 2005) were gathered and evaluated for inclusion. Next, computerized searches of the PsycInfo, Medline, Educational Resources (ERIC), and National Criminal Justice Reference Service Abstracts (NCJRS) databases were conducted using the following key words: partner violence, partner abuse, wife abuse, husband abuse, perpetrator, and batterer. These words were paired separately with the following keywords: anger*, hostil*, negative affect*, negative emotion*, anxiety, and depression. Database search parameters were as follows: peer reviewed articles only, studies

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published the English language, and exclusion of child* keyword and dissertations. Empirical studies were additionally identified through review of the references of collected articles and book chapters obtained using the previously stated approaches. Finally, authors in the field were contacted by email via a targeted special interest group to identify any relevant articles that were currently in press. These search methods yielded a total of 2,644 abstracts of which 113 studies (available from authors by request) were identified for inclusionary review.

Criteria for Inclusion

The following selection criteria were used to determine eligibility for inclusion in this meta-. Each study included must have: (1) been published in a peer-reviewed journal, reported in English; (2) reported data from measures of anger, hostility and/or negative emotions (not aggression); (3) included samples of male and/or female perpetrators of IPV; (4) reported participant data independently by sex versus using couple/dyad scores; (5) reported data from independent samples for each publication unless different measures/constructs of interest were discussed; (6) specified that partner assault occurred within heterosexual married, co-habiting, dating couples (or a mix of one of these and other relationship types); and (7) included North American samples only. Of note, physical IPV (defined as the intentional use of physical force with the potential for causing a range of harm, e.g., pushing, choking, use of weapon; Saltzman et al., 2002) must have been examined, and was the primary and exclusive form of IPV investigated in the majority of included studies (although several studies used a composite measure of different types of IPV, e.g., physical and verbal). Upon application of these criteria, 52 studies were excluded and 61 studies comprised the final sample for this meta-analysis. Studies were excluded for the following reasons: 24 studies were excluded because they did not assess physical IPV and/or any of the constructs of interest; six studies included the same sample as studies selected for the meta-analysis (in these cases, article content was compared to the purpose of the meta-analysis to select the most appropriate article to include); five studies reported couples data only; five studies were comprised of groups based on substance use and substance use disorders were the dependent variable; four studies reported on groups that were mixed by sex; three studies included samples outside North America; three studies only reported effect sizes for comparisons between groups based on sex of perpetrator; two studies reported physical health outcomes as the primary outcome measure; and one study included a non-heterosexual sample.

Coding of Studies

The following variables were coded independently by the author and three research assistants: publication characteristics (sample size), type of relationship (marital/cohabitating or dating), demographics (e.g., sex of perpetrator, geographic location, ethnicity, age), participant selection and recruitment (community, criminal justice, clinical), constructs of interest (e.g., anger, hostility, negative emotions), and measures employed. The first author met with each coder separately for three, 2-hour meetings to complete training and review coder questions, and met with each coder on an as-needed basis to address any study-specific questions. Discrepancies between coders were documented and evaluated by the first author, after which no differences in coding remained.
Sample Description

Overall descriptive information for the studies included in this meta-analysis is as follows: the majority of the pooled sample were in their early thirties ($M = 31.92$, $SD = 5.69$), had completed high school ($M$ years of education = 12.86, $SD = 1.42$), and earned on average around $30K annually ($M= 31,959$, $SD = 10,658$). In 44% of studies, the majority of the sample was Caucasian, in 5% of studies African Americans comprised the majority of the sample, in 15% of studies ethnicity was mixed with none greater than 60% of the sample (36% of studies did not report ethnicity). Of note, many studies provided limited to no descriptive information; as a result, these demographic variables should be interpreted with caution.

Effect Size Estimates

Standard mean difference ($d$) effect sizes and 95% confidence intervals were calculated using the Practical Meta-Analysis Effect Size Calculator (Lipsey & Wilson, 2001). Cohen’s $d$ is an effect size estimate derived from the standardized difference between two group means divided by their pooled standard deviations (Cohen, 1977). When means and standard deviations were used for subgroups (e.g., more than one IPV or NV group), between-subgroup variance was added to the pooled standard deviation (Lipsey & Wilson, 2001). The standardized mean difference statistic was the chosen effect size estimate for this meta-analysis as a main goal of this analysis was to analyze differences between violent and nonviolent partners (at varying levels of IPV severity) on the constructs of interest. That being said, it is important to note that several effect sizes were correlation coefficients between IPV and the constructs, particularly among female samples and on the relationship between IPV and depression. Effect sizes were placed in a common metric through standardization in order to compare the impact of constructs of interest across included studies (Lipsey & Wilson, 2001; odds ratio-to-$d$ effect size conversions were necessary for four studies and were calculated and verified by hand). A small effect size is $\leq .20$, a medium effect size is $= .50$, and a large effect size is $\geq .80$ (Cohen 1977, 1988). Standardized mean difference ($d$) effect sizes were calculated from the following (listed in order of frequency): means and standard deviations, $F$-scores, $t$-scores, correlation coefficients, odds ratios, $X^2$, and a $p$-value (when no other data were available). Each effect size was weighted by the inverse of its variance in order account for the weight of each variable by study sample size (Hedges & Olkin, 1985; Lipsey & Wilson, 2001).

Effect sizes for the relationships among IPV and anger, hostility, and internalizing negative emotions were averaged separately across measures of each construct within each individual study. On several occasions, data for subscales rather than the total score were provided; for these cases, effect sizes were calculated for each subscale and then averaged across subscales to produce an effect size for the measure. Effect sizes across studies were then averaged to obtain an accumulated, overall effect for each construct (Lipsey & Wilson, 2001). Effect sizes across all three constructs, and an anger/hostility composite, were calculated by summing effect sizes across studies in order to arrive at grand effect sizes. A fixed effects model was employed in analyses of moderation as some or all of the variability in effect sizes for the constructs of interest across studies was assumed to be systematic based on hypothesized categories/moderating variables. The $Q$ statistic was calculated to
determine whether there was significant heterogeneity within \((Q_w)\) and between \((Q_b)\) effect size groups (Lipsey & Wilson, 2001). The \((Q_b)\) is an indicator of the variability between groups/categories for the construct of interest, while the \((Q_w)\) is an indicator of within-group variability. If the \(Q\) statistic was significant, the null hypothesis of homogeneity between or within groups was rejected since the groups differed by more than what would be expected due to sampling error. If the null hypothesis was not rejected (e.g., \(p\)-value for \(Q\) test was non-significant) then there were no significant differences between or within groups (Lipsey & Wilson, 2001).

**Measure Descriptions by Type**

Self-report measures were used in 87% of studies. The most commonly used self-report measures by construct were the: State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988), State-Trait Anger Expression Inventory 2 (STAXI-2; Spielberger, 1999), and Multidimensional Anger Inventory (MAI; Siegel, 1985, 1986) for anger; the Buss–Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) for hostility; and the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff 1977), Beck Depression Inventory (BDI; Beck 1978), and Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943) for negative emotions. Observational measures were employed in 18% of studies; the most commonly employed observational measures included the Articulated Thoughts in Simulated Situations (ATSS; Davison, Robins, & Johnson, 1983) and the Specific Affect Coding System (SPAFF; Gottman & Krokoff, 1989).

The reported reliability estimates for all self-report and observational measures included were within an acceptable range (.74 – .94; \(M = .93, SD = .28\)).

**Results**

**Composite Effect Size for Anger, Hostility, and Negative Emotion**

A grand effect size was calculated for a total of 105 effect sizes from 64 independent samples (from 61 studies) and the fixed effects meta-analysis suggested that IPV was moderately associated with anger, hostility, and internalizing negative emotions \((d = .51, k = 61, 95\% CI = .48 – .54, p < .001; \text{see Table 1 for effect sizes for each study and construct})\). Tests of homogeneity revealed that the grand effect size across all three constructs was not representative of the individual effect sizes across studies \((Q_w(103) = 1251.72, p < .001)\). Following the elimination of the most extreme outlier (Chambers & Wilson, 2007; \(d = 2.45\)) heterogeneity was \((Q_w(102) = 1194.35, p < .001)\) reduced. After elimination of the second most extreme outlier, (Tweed & Dutton, 1998; \(d = -1.31\)) heterogeneity was again reduced \((Q_w(101) = 1147.70, p < .001)\). The third most extreme outliers (Mauro et al., 1998; Swan & Snow, 2003; \(d\) for both studies = -1.26) were then eliminated and heterogeneity was reduced further \((Q_w(99) = 1005.62, p < .001)\). Although heterogeneity was reduced with the elimination of the four most extreme outliers, all effect sizes were included in the present analysis in order to account for the full range of effects.

In order to address the “file-drawer” problem of publication bias in which non-significant results are less likely to be both submitted and accepted for publication (Rosenthal, 1991), a fail-safe \(N\) was calculated at 69. This means that 69 unpublished studies with null results
would be needed to reduce the overall effect size across studies below the critical $d$ value of .20 for a small effect (Lipsey & Wilson, 2001; Orwin, 1983).

**Effect Sizes for Each Construct**

Effect sizes were calculated for each construct across studies and these analysis suggested moderate effects for the relationships between anger and IPV ($d = .48, k = 37, 95\% \text{ CI} = .27 – .68, p < .001$) and hostility and IPV ($d = .56, k = 17, 95\% \text{ CI} = .27 – .85, p < .001$), and a small effect for the relation between internalizing negative emotions and IPV ($d = .33, k = 47, 95\% \text{ CI} = .16 – .51, p < .001$; see Table 2 for all moderator effect sizes, confidence intervals, and $Q$ tests of homogeneity). An effect size was also calculated for anger/hostility composite scores (measures that represented a composite of these two constructs of interest) that suggested a moderate relationship between anger/hostility and IPV ($d = .64, k = 3, 95\% \text{ CI} = -.04 – 1.32, p = .064$). Homogeneity tests failed to reject the null hypothesis of homogeneity among constructs (including the anger/hostility composite as a separate construct), as the between-category test was not significant ($Q_b (3) = 2.51, p = .473$). Marginally non-significant variability was observed within the construct category of internalizing negative emotions ($Q_w (46) = 61.46, p < .063$), but not within anger ($Q_w (36) = 21.33, p = .975$), hostility ($Q_w (16) = 3.20, p=.999$), or the anger/hostility composite ($Q_w (2) = .03, p = .983$). The construct of internalizing negative emotions was explored further by examining the following categories: depression-related (e.g. CES-D depression, SPAFF sadness; $d = .42, k = 26, 95\% \text{ CI} = .12 – .72, p < .01$), anxiety-related (e.g. PANAS anxiety, SPAFF tension/fear; $d = .36, k = 13, 95\% \text{ CI} = -.07 – .79, p = .099$), and other negative emotions ($d = .15, k = 4, 95\% \text{ CI} = -.63 – .93, p = .70$). Between-group tests of homogeneity for internalizing negative emotion categories were not significant ($Q_b (2) = .39, p = .821$) and within-group tests of homogeneity were also not significant ($Q_w (40) = 32.00, p = .812$).

As there was no between-group heterogeneity among the four constructs of interest (anger, hostility, anger/hostility composite, and internalizing negative emotions) and IPV, moderation analyses were conducted on the entire sample.

**Analyses of Moderators**

**Sex of perpetrator**—Effect sizes were estimated for the composite of all four constructs of interest for the 93 effect sizes for males ($d = .43, k = 45, 95\% \text{ CI} = .32 – .55, p < .001$) and the 30 effect sizes for females ($d = .37, k = 18, 95\% \text{ CI} = .17 – .57, p < .001$). Homogeneity tests revealed that there were no significant group differences ($Q_b (1) = .29, p = .59$). Thus, the moderate relationship ($d = .51$) between anger, hostility, anger/hostility, internalizing negative emotion and IPV was consistent between males and females.

**IPV perpetrator population**—Composite effect sizes were calculated for the 45 effect sizes among community samples ($d = .40, 95\% \text{ CI} = .22 – .57, p < .001$), 38 clinical samples ($d = .50, 95\% \text{ CI} = .31 – .69$), and 21 mixed samples ($d = .37, 95\% \text{ CI} = .11 – .63, p < .01$; e.g., mixed community/clinical, clinical/incarcerated). Homogeneity tests revealed no significant differences between groups ($Q_b (2) = .88, p = .645$). The moderate effect size
observed between the constructs of interest and IPV was therefore consistent across IPV perpetrator population.

**Type of relationship**—Analyses were conducted using two different coding techniques. First, married and dating cohabitating samples were compared to dating non-cohabitating, dating mixed samples (cohabitating and non-cohabitating), and mixed relationships to see if cohabitation served as a moderator. Effects sizes across the entire sample were estimated for the 58 effect sizes for IPV perpetrators who were married or dating-cohabitating ($d = .41$, 95% CI = .25 – .56, $p < .001$), two effect sizes for dating non-cohabitating perpetrators ($d = .37$, 95% CI = -.56 – 1.30, $p = .434$), 29 relationship samples that were mixed ($d = .59$, 95% CI = .36 – .81, $p < .001$; e.g., married, single, divorced, dating), and 6 mixed dating samples ($d = .46$, 95% CI = -.02 – .93, $p = .061$; e.g., cohabitating and non-cohabitating). Of note, nine studies did not report relationship type. There were no significant differences among groups ($Q_{ba}(3) = 1.72$, $p = .633$).

Second, relationship type was examined as a moderator using the following re-coded categories: married, dating (cohabitating, non-cohabitating or a mix of these two types), and mixed (e.g., married, dating, divorced, separated, single). Composite effect sizes were estimated for 50 effect sizes for married samples ($d = .38$, 95% CI = .21 – .54, $p < .001$), seven effect sizes for dating samples ($d = .39$, 95% CI = -.06 – .84, $p = .086$), and 37 effect sizes for mixed relationship samples ($d = .59$, 95% CI = .39 – .78, $p < .001$). Tests of homogeneity revealed no significant differences among groups for relationship type, $Q_b(2) = 2.64$, $p = .267$. Specifically, there appeared to be significant within-group heterogeneity for mixed relationship samples only ($Q_{w}(36) = 53.76$, $p = .03$). Thus, the moderate effect sizes for anger, hostility, anger/hostility, negative emotion, and IPV appeared consistent across type of relationship.

**Method of assessment**—Composite effect sizes were computed for 83 effect sizes for self-report measures ($d = .48$, 95% CI = .35 – .60, $p < .001$) and 23 effect sizes for observational measures ($d = .25$, 95% CI = .00 – .49, $p < .05$) in order to test whether assessment method affected associations between the constructs and IPV. Homogeneity tests revealed no significant differences between different types of assessment methods ($Q_{b}(1) = 2.68$, $p = .101$).

**IPV severity**—Effect sizes from 12 independent samples (collapsed across construct) were analyzed for mean differences across severity of IPV perpetrated within these samples. Men and women who perpetrated moderate to severe IPV reported higher levels of anger, hostility, and negative emotions than those who reported low to moderate IPV ($d = 1.00$, 95% CI = .92 – 1.08, $p < .05$).

**Discussion**

The results of the present meta-analysis indicated that IPV was moderately associated with anger, hostility, internalizing negative emotions ($d = .51$), which is consistent with prior quantitative reviews (Norlander & Eckhardt, 2005). Surprisingly, there were no significant differences in effect sizes among the constructs and their respective associations with IPV.
The hypothesis that anger and hostility would have moderate associations with IPV was partially supported ($d = .48$, $d = .56$, respectively). While the association between internalizing negative emotions and IPV ($d = .33$) was smaller than that of anger or hostility, there were no significant differences across the affective constructs in their association with IPV, which was not predicted. In contrast to earlier reviews, no significant differences were observed for the associations among anger, hostility, internalizing negative affect, and IPV across the hypothesized moderating variables of sex, relationship type, type of assessment, or perpetrator population. However, the anger-IPV association does appear to be moderated by IPV severity, with stronger effects observed between anger-related constructs and IPV with increasing IPV severity.

**Implications**

Consistent with recent etiological models of IPV, the present results confirm that anger, hostility, and internalizing negative emotions are moderately associated with IPV, both in terms of differentiating among those with a history of such aggression as well as covarying with acute instances of partner violence. These data come from a wide range of research methodologies, assessment approaches, and populations, and have been compiled into diverse models of IPV perpetration. These theories have posited that dysregulation of negative emotions, particularly anger, increases risk of IPV perpetration through a variety of interacting mechanisms (Finkel & Eckhardt, 2013). Heightened negative affective states increase the availability of aggression-relevant beliefs, images, and memories (Anderson & Bushman, 2002; Berkowitz, 2012), and provide aggression-prone individuals with an immediately accessible script to manage the provocation (Huesmann, 1988). When these scripts contain cognitive distortions that increase (a) the blameworthiness of the target, (b) the unfairness of the provoking situation, and (c) the perception that aggressive action is justified, IPV risk is therefore heightened (e.g., Beck, 1999). But it is also clear that IPV-prone individuals are not aggressive with everyone or in all situations. As predicted by interpersonal models of IPV, the proximal context of IPV is the specific relationship in which the abusive behavior is embedded (Margolin et al., 1988; Neidig & Friedman, 1984). Patterns of affective expression, and dyadic reactions to that expression, are shaped by reciprocally determined reinforcement processes and soon become automatically initiated by the presence of a specific contextual cue (e.g., a long sigh; a particular facial expression; the “silent treatment”). Individuals with a history of childhood trauma or disrupted attachment may be particularly prone to experience dysregulated emotions during relationship conflict, with the aforementioned cognitive, affective, and relational processes maintaining these patterns over time (Dutton, 2010). Additional factors, such as alcohol intoxication (Eckhardt, 2007), work stress spillover (Doumas, Margolin, & John, 2008), or weak relationship commitment (Slotter et al., 2012) may further weaken the normal inhibitory processes that would otherwise curb aggressive inclinations when individuals high in anger or negative emotions experience are involved in a relationship conflict (Finkel & Eckhardt, 2013).

Despite the significant effects shown to exist between anger, internalizing negative emotions, and IPV, and the consistency of these results with those of prior reviews, it is quite likely that a section of the IPV field will continue to regard any links between anger /
emotion dysregulation and IPV with a high level of suspicion (e.g., Gondolf, 2012; Healey et al., 1998). In some ways, such caution may be warranted; this review suggests that anger and internalizing negative emotion-related variables have only a moderate association with IPV perpetration outcomes. However, it is worth highlighting the fact that there does not appear to be (and perhaps never has been) theoretical support for the proposition that problematic negative emotions, in isolation, are a univariate cause of IPV. Few, if any, aggression or IPV scholars have ever actually proposed such a simple anger-to-IPV causal analysis, yet it is perhaps this overly simplified causal connection that appears to be at the root of power-and-control adherents’ concerns about the need to investigate anger and internalizing negative emotions in the context of IPV, or to examine the importance of including emotion regulation components in treatment programming for IPV perpetrators. The results of this review suggest that researchers, practitioners, criminal justice personnel, and agencies that provide services for both survivors and perpetrators of IPV should seriously consider anger and internalizing negative emotions as important factors that should inform both their models of IPV risk as well as their methods of intervention and prevention. To blithely dismiss the role of anger and emotions, as do most state standards for IPV intervention programs (Maiuro & Eberle, 2008), or to suggest that an acknowledgement of the importance of negative emotions distracts from the “real” causes of violence, only serves to keep existing ideologies in their traditional positions of power and to covertly stand in the way of progress in intervention and prevention.

Based on the present results, we would advance the conclusion that for some partner abusive individuals, perhaps as many as 50% (Eckhardt et al., 2008; Murphy et al., 2007), anger-related problems are meaningfully associated with IPV perpetration and should be taken into account when designing programming for IPV offenders, regardless of whether the perpetrator is male or female. These findings are also informed by decades-old findings documenting interdependence between males and females’ displays of negative conflict behaviors during arguments (Burman et al., 1993; Jacobson et al., 1994; Margolin, John, & Gleberman, 1988), as well as recent research demonstrating that males and females do not differ in their use of controlling behaviors in close relationships (Bates et al., 2014; Graham-Kevan & Archer, 2009), countering claims from some researchers that women’s use of IPV is defensive and noncoercive in nature (Gondolf, 2007). Furthermore, recent research using daily reporting methods to assess anger and IPV-related behaviors supports the notion that increases in angry affect are associated with increases in IPV risk for both men and women (Crane & Eckhardt, 2013a; Elkins, Moore, McNulty, Kivisto, & Handsel, 2013), with recent research indicating that female anger may be a stronger predictor of both female-to-male and male-to-female IPV than male anger (Crane & Testa, 2014). While there are several empirically supported treatments for individuals with general anger problems (Del Vecchio & O’Leary, 2004; Kassinove & Tafrate, 2002), there is severely limited information regarding whether anger-control treatments or anger-focused components of broader CBT interventions are effective in reducing assault rates among IPV perpetrators (for a review, see Eckhardt et al., 2013). The prevailing conclusion in the peer-reviewed literature appears to be that intervention programs that contain an emotion regulation component are as effective as those that do not (e.g., Dunford, 2000). Thus, there are viable programs that
indeed target how individuals attempt to manage the experience and expression of negative emotions.

The link between anger and IPV may be partially accounted for by processes related to self-regulation, emotion dysregulation, and/or other externalizing behaviors (e.g., impulsivity; Krueger et al., 2002; Murphy et al., 2007; Magdol, Moffitt, Caspi, & Silva, 1998). Specifically, male perpetrators of IPV evidence internalizing and externalizing emotion regulation difficulties (Babcock, Johnson, Gottman, & Yerington, 2000; Holtzworth-Munroe & Stuart, 1994) and would likely benefit from practice and refinement of adaptive emotion expression skills (Murphy & Eckhardt, 2005). Within CBT-based IPV intervention programs, higher levels of anger, impulsivity, anxiety/depression, and difficulties with emotion regulation may impede CBT skill based work, particularly in the areas of communication and relationship-building (Murphy et al., 2007). CBT-adapted programs that target emotional expression skills, active listening, and negotiation/respectful assertion skills have greater empirical support than the utilization of a “one size fits all” anger management program (Costa & Babcock, 2008; Murphy & Eckhardt, 2005).

The problem, however, is that the few published randomized trials of IPV intervention programs typically fail to show a difference in effectiveness between an active treatment and a relevant control group, such as a probation-only condition (e.g., Labriola et al., 2008). While it may be the case that negative emotion-focused treatments may be no more effective than other interventions, it is important to consider the context of these findings. Researchers have noted that traditional interventions for IPV perpetrators were rapidly and broadly implemented in communities well in advance of systematic efforts to evaluate their effectiveness (Stuart, Temple, & Moore, 2007). The clinical need for an intervention that could be widely implemented (e.g., the Duluth Model) outweighed the need to ask questions about whether such interventions were indeed empirically supported. More recently, however, there has been a shift in this approach that has favored the development of “alternative” treatments for IPV perpetrators (for a review see Eckhardt et al., 2013) that focus on applying methods of behavior change validated in non-IPV populations, such as motivational interviewing (Alexander et al., 2010; Crane & Eckhardt, 2013b), couples therapy (Stith, Rosen, McCollum, & Thomsen, 2004), and combined substance use-IPV treatments (Easton et al., 2007). These treatment methods are prime examples of how the IPV field can develop and implement empirically supported treatments (or treatment components) with clear evidence of effectiveness (Eckhardt et al., 2013), despite opinions from IPV traditionalists that such interventions are unnecessary (Gondolf, 2011). This approach needs to be applied to the constructs of interest to this review; there are simply no targeted studies of an anger / emotion control treatment package, and no systematic study of an emotion regulation focused component of a broader treatment program for IPV offenders. A treatment dismantling design would therefore be of substantial benefit to examining ways of improving treatment effectiveness in this area, as well as providing some much-needed empirical evidence to the long-standing debate in state standards for IPV intervention programs regarding the value of anger control treatments.
Limitations

There were several limitations to the current meta-analytic review. Few studies ($N = 12$) reported IPV severity; therefore, the strength of the mean difference ($d = 1.00$) between moderate-to-severe and low-to-moderate IPV perpetrators on anger, hostility, and depression should be interpreted with some caution. There was also limited geographic and ethnic diversity within the present sample as North American populations were exclusively examined and a large proportion of perpetrators were Caucasian. In addition, relatively few studies examined anger and internalizing negative affect arousal as a potential proximal cause of IPV-related behaviors, thus restricting our ability to specify the extent to which episodes of anger or emotional arousal are associated with acute incidents of IPV.

In conclusion, anger, hostility, and internalizing negative emotions are moderately associated with IPV perpetration, and these associations are consistent across perpetrator sex, measurement format, and perpetrator population. These negative emotions exhibit a heightened association with partner abuse with increasing IPV severity. In order to continue to move forward in the pursuit of a deeper understanding of the factors and mechanisms that inform IPV risk, it is essential to put aside controversies that explicitly limit or prohibit continued exploration of factors shown to empirically relate to IPV. A singular advantage of an approach informed by data, as opposed to ideology, is an openness towards novel findings, to new and ‘controversial’ constructs such as anger and internalizing negative emotions, and to new collaborations that will undoubtedly inform our understanding of IPV etiology and intervention/prevention. “Given our awareness of the limitations of current approaches …, it is our obligation to apply what we know about the complexity of partner abuse to improve the programs intended to end it” (Stuart, 2005, p. 262.). With this in mind, it follows that our ability to prevent violence against intimate partners and to achieve the goal of zero-recidivism programs for IPV perpetrators can best be achieved through open collaboration between researchers, clinicians, and policy makers and creative development of more empirically-based IPV prevention and intervention programs.

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Highlights

- Meta-analysis on associations of anger, hostility, internalizing negative emotions and IPV.
- Moderate associations consistent for perpetrator sex and measurement/relationship.
- Association between internalizing negative emotions and IPV was moderated by IPV severity.
- Results inform etiological factors, mechanisms of risk, and intervention for IPV.
- Openness to collaboration on empirical exploration of IPV risk is advocated.
### Table 1

Descriptive information, measures, and effect sizes of studies included in the meta-analysis.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sex</th>
<th>Population Characteristics</th>
<th>N and Sample Size by Group</th>
<th>Measures &amp; Description of Findings</th>
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<td>Anderson (2002)</td>
<td>M &amp; F</td>
<td>Married, Dating Cohabitating; Community</td>
<td>N=7,395; Males: CMIPV (3,132); Females: CMIPV (3,726)</td>
<td>CES-D; IPV significantly increases the odds of depression for males (OR=1.02, p=.001, d=.01) and females (OR=1.03, p=.001, d=.01).</td>
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<tr>
<td>Babcock et al. (2005)</td>
<td>M</td>
<td>Married, Dating Cohabitating; Community</td>
<td>N=102; SNV (30), LMIPV (37), MSIPV (35)</td>
<td>STAXI trait anger scales, SPAFF; IPV higher trait anger than SNV (d=.67, CI = .24 – 1.11). IPV higher observed contempt and belligerence than SNV (d=.58, CI = .14 – 1.01).</td>
</tr>
<tr>
<td>Barbour et al. (1998)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=88; SNV (34); DNV (23), CMIPV (31)</td>
<td>STAXI, ATSS; CMIPV higher trait anger (d=.87, CI = .41 – 1.33), state anger (d=.52, CI = .08 – .97), and anger out scores (d=.97, CI = .51 – 1.43) than DNV and SNV. DNV and SNV had higher anger control than CMIPV (d= −.91, CI = −1.37 – −.45). No differences between groups on ATSS anger/rage (d=.01, CI = ns), annoyance (d= −.30, CI = ns), or negative emotions (d= −.33, ns).</td>
</tr>
<tr>
<td>Barnett, Fagan, &amp; Booker (1991)</td>
<td>M</td>
<td>Married; Community, Probation</td>
<td>N=228; SNV (50), DNV (43), CLIPV (93) – counseled/uncounseled, GV (42)</td>
<td>BDHI; CLIPV and GV higher overall BDHI scores than SNV and DNV (d=−.34, CI = .07 – .60).</td>
</tr>
<tr>
<td>Beasley &amp; Stoltenberg (1992)</td>
<td>M</td>
<td>Married, Cohabitating, Divorced, Separated; Clinical</td>
<td>N=84; NV (35), CLIPV (49)</td>
<td>STAS; IPV had higher scores than NV for state (d=.97, CI = .51 – 1.43) and trait (d=.59, CI = .15 – 1.04) anger.</td>
</tr>
<tr>
<td>Boyle &amp; Vivian (1996)</td>
<td>M</td>
<td>Married; Clinical/ Community</td>
<td>N=312; MSIPV (100), LMIPV (69), DNV (94), SNV (49)</td>
<td>BDI, MAI-H, SS-Ang, STAS-T; IPV had higher scores than NV on anger on the STAS-T (d=.44, CI = .20 – .67) and spouse-specific anger (d=.50, CI = .26 – .73) and on depression (d=.69, CI = .46 – .92). IPV as not different from NV on hostility (d=.07, CI = ns) and MSIPV did not differ from LMIPV on any of these variables.</td>
</tr>
<tr>
<td>Burman, Margolin, &amp; John (1993)</td>
<td>M &amp; F</td>
<td>Married; Community</td>
<td>N=65; Couples; SNV (15), MSIPV (17), VA (15), WI (18)</td>
<td>SPAFF; No differences between groups on anger/contempt for husbands (d=.49, n.s.) or wives (d=.59, n.s.) or on non-hostile negative affect for husbands (d= −.26, n.s.) or wives (d= −.12, n.s.).</td>
</tr>
<tr>
<td>Cadsky &amp; Crawford (1988)</td>
<td>M</td>
<td>Married and other not specified; Clinical</td>
<td>N=172; MSIPV (66), LMIPV (106)</td>
<td>BDHI; MSIPV scored higher than LMIPV on BDHI (d=.40, CI = .10 – .71). *Not enough info. provided to calculate effect size for depression.</td>
</tr>
<tr>
<td>Caetano &amp; Cunradi (2003)</td>
<td>M &amp; F</td>
<td>Married or Dating Co-habitating; Community</td>
<td>N=1635; Couples</td>
<td>CES-D; Female, but not male, IPV perpetrators had significantly greater odds of depression (d=1.91, CI = 1.80 – 2.02; d=.12, n.s.).</td>
</tr>
<tr>
<td>Chambers &amp; Wilson (2007)</td>
<td>M</td>
<td>Married, Separated, Divorced, Single, Widowed; Clinical</td>
<td>N=93; LMIPV (51), MSIPV (32), DB (10)</td>
<td>PAI; DB cluster and MSIPV moderately distressed personality cluster had higher depression scores (d=2.05; CI = 1.55 – 2.55;DEP scale) and higher anxiety scores than the LMIPV non-elevated personality cluster on the ANX and ARD scales, respectively (d=1.37, CI = .92–1.83 ; d= 1.71; CI = 1.23 – 2.18).</td>
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<tr>
<td>Clements &amp; Holtzworth-Munroe (2009)</td>
<td>M &amp; F</td>
<td>Married; Community</td>
<td>N=66; Couples; SNV (14), DNV (14), IPV (38)</td>
<td>Emotion Checklist; After conflict discussions with spouse there were no differences between IPV and NV for males and females, respectively on reported tense/anxious/tear-related (d=−.16, CI = n.s.; d= .30, CI = n.s.), anger/contempt/disgust (d=.02, CI = n.s.; d= −.12, CI = n.s.), or sad/discouraged (d=−1.14, CI = n.s.; d= .14, CI = n.s.) emotions.</td>
</tr>
<tr>
<td>Cogan &amp; Fennell (2007)</td>
<td>M &amp; F</td>
<td>Dating; Community</td>
<td>N=396; Males: NV (44), MSIPV (9), VO (75), GV (65); Females: NV (64), MSIPV (26), VO (27), GV (86)</td>
<td>CES-D, TAS; IPV groups reported greater depression than NV among males (d=.41, CI = .07 – .75), and females (d=.28, CI = .02–.56). Greater trait anger reported among IPV groups than NV for males (d=.84, CI = .47–1.21) but not females (d=.35, n.s.).</td>
</tr>
<tr>
<td>Costa &amp; Babcock (2008)</td>
<td>M</td>
<td>Married or dating cohabitating “as if married”; Community</td>
<td>N=178; SNV (21), DNV (27), MSIPV (130)</td>
<td>ATSS; MSIPV articulated more anger than SNV and DNV across ATSS scenarios (d = .39, CI = .06 – .72), and reported more anger than SNV, specifically (d = .61, CI = .15 – 1.08).</td>
</tr>
<tr>
<td>Date &amp; Ronan (2000)</td>
<td>M</td>
<td>Married, Dating non-cohabitating, other non-specified; Incarcerated</td>
<td>N=59; NV (20), IPV (20), VO (20)</td>
<td>TAS; Violent to non-partner only scored higher than NV (d=1.14, CI = .47 – 1.82).</td>
</tr>
<tr>
<td>Dewhurst, Moore, &amp; Alfano (2008)</td>
<td>M</td>
<td>Relationship type not reported; Clinical, Incarcerated, CCG</td>
<td>N=73; NV (22), IPV (22), GV (10), SO (19)</td>
<td>BPI; Greater depression reported among IPV groups than NV for males (d=.84, CI = .47–1.21) but not females (d=.35, n.s.).</td>
</tr>
<tr>
<td>Dutton &amp; Browning (1988)</td>
<td>M</td>
<td>Married; CCG, Clinical</td>
<td>N=60; NV (18) – some levels of IPV, VA (18), CLIPV (24)</td>
<td>Using anger self ratings, CLIPV scored higher on anger during the abandonment scene than VA or NV (d=.68, CI = .15 – 1.21).</td>
</tr>
<tr>
<td>Dutton &amp; Starzomski (1993)</td>
<td>M</td>
<td>Married*; Clinical, Incarcerated, CCG *Wife reports of IPV</td>
<td>N=75; NV* (17), *IPV in control group, CLIPV (32), INCIPV (26)</td>
<td>MAI; Across all groups, medium correlation between total anger and severe IPV (d=.49 CI = .04 – .95) and no correlation between total anger and CTS total (d=.14, CI = n.s.).</td>
</tr>
<tr>
<td>Dutton et al. (1994)</td>
<td>M</td>
<td>Married, Other relationships, unknown; Community, Clinical</td>
<td>N=160; NV* (40) *IPV in control group, MSIPV (120)</td>
<td>MAI; MSIPV higher anger than NV (d=.31, CI = .0004 – .62).</td>
</tr>
<tr>
<td>Dutton, Starzomski, &amp; Ryan (1996)</td>
<td>M</td>
<td>Married; Clinical, CCG</td>
<td>N=185; NV* (45) *IPV in control group, CLIPV (140)</td>
<td>MAI; CLIPV reported more anger than NV (d=.41, CI = .07 – .75).</td>
</tr>
<tr>
<td>Dye &amp; Eckhardt (2000)</td>
<td>M &amp; F</td>
<td>Dating cohabitating and non-cohabitating; Clinical, CCG</td>
<td>N=257; Males: SNV (71), MSIPV (24); Females: SNV (109), MSIPV (43)</td>
<td>STAXI; For males, SNV reported greater anger control than MSIPV (d=−.49, CI = −.96 – −.02). For females, MSIPV had higher anger out (d=.51, CI = .15 – .87) and anger expression scores (d=.45, CI = .09 – .80).</td>
</tr>
<tr>
<td>Eckhardt &amp; Kassinove (1998)</td>
<td>M</td>
<td>Married; Community/Community mixed IPV group, CCG</td>
<td>N=40; SNV (20), IPV (20)</td>
<td>ATSS; SNV had more anger control strategies across ATSS scenarios than CLIPV (d=−.77, CI = −1.41 – −.13).</td>
</tr>
<tr>
<td>Eckhardt, Barbour, &amp; Davidson (1998)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=88; SNV (34), DNV (23), IPV (31)</td>
<td>ATSS; IPV articulated more hostile attribution biases than DNV (d=.84, CI = .28 – 1.40) and SNV (d=1.04, CI = .52 – 1.56). During anger scenario, SNV scored higher on anger control statements than IPV (d=−.98, CI = −1.49 – −.46), and there were no differences between IPV and DNV (d=−.19, CI = ns).</td>
</tr>
<tr>
<td>Eckhardt (2007)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=102; NV (56), IPV (46)</td>
<td>STAXI-2; IPV higher scores than NV on trait anger (d=.80, CI = .39 – 1.20)</td>
</tr>
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<tr>
<td>Eckhardt et al., (2008)</td>
<td>M</td>
<td>Married, Dating cohabitating, Single, Separated, Divorced, Clinical</td>
<td>N=199; LMIPV: FO (61); MSIPV (138) comprised of: LLA (86), DB (40), GV (12)</td>
<td>HAT, TAS; MSIPV scored higher on trait anger than LMIPV (d=1.11, CI = .79 – 1.44); MSIPV also scored higher than LMIPV on the HAT (d=63, CI = .32 – .94).</td>
</tr>
<tr>
<td>Eckhardt, Jamison, &amp; Watts (2002)</td>
<td>M</td>
<td>Dating non-cohabitating; Community</td>
<td>N=33; NV (16), LMIPV (17)</td>
<td>STAXI, ATSS; LMIPV higher overall score on STAXI trait anger (d=1.73 CI = .93 – 2.53), anger in (d=.78, CI = .07 – 1.48), anger out (d=2.12, CI = 1.26 – 2.97) than NV except for anger control (d=−1.80, CI = −2.61 – −.99). No difference between groups on ATSS anger expression variables (d=−.09, CI = ns), or other negative emotions (d=−.43, ns).</td>
</tr>
<tr>
<td>Else et al. (1993)</td>
<td>M</td>
<td>Married, Divorced, Separated, Never Married; Clinical, CCG</td>
<td>N=42; NV (21), CLIPV (21)</td>
<td>HDHQ, MMPI, BDI; No difference between CLIPV and NV on hostility (d=.45, CI = ns); CLIPV did not differ from NV on reported depression across two measures (d=.19, CI = ns).</td>
</tr>
<tr>
<td>Feldbau-Kohn, Heyman, &amp; O'Leary (1998)</td>
<td>M</td>
<td>Married; Clinical</td>
<td>N=89; MSIPV</td>
<td>State-Trait Anger Scale, BDI; Medium sized correlations between IPV and state-trait anger (d=.58, CI = .16 – 1.01) and IPV and depression (d=.43, CI = .01 – .85).</td>
</tr>
<tr>
<td>Follingstad et al. (1992)</td>
<td>M</td>
<td>Dating cohabitating or non-cohabitating, Married; Community</td>
<td>N=74; LMIPV (37) – termed “non-abusive” but IPV reported, MSIPV (37)</td>
<td>BDHI; MSIPV had higher BDHI assault scores than LMIPV (d=.63, CI = .17 – 1.10). No differences between groups on BDHI verbal (d=−.14, CI = ns) or irritable scores (d=−.12, CI = ns).</td>
</tr>
<tr>
<td>Gavazzi, Julian, &amp; McKeny (1996)</td>
<td>M</td>
<td>Married, Dating cohabitating; Clinical/Community mixed, CCG</td>
<td>N=152; NV (100), IPV (52)</td>
<td>BSI; IPV had higher scores than NV on depression (d=.46, CI = .12 – .80), anxiety (d=.45, CI = .12 – .79), and hostility (d=.67, CI = .33 – 1.02).</td>
</tr>
<tr>
<td>Gordis, Margolin, &amp; Vickerman (2003)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=90; NV (38), IPV over 1 year ago (23), recent IPV (29)</td>
<td>Marital Coding System (observational); Greater hostility observed for IPV groups compared to NV (d=.66, CI = .18 – 1.03); IPV within past year reported greater hostility than IPV perpetrated over 1 year ago (d=1.07, CI = .49 – 1.66) and NV (d=1.17, CI = .65–1.70).</td>
</tr>
<tr>
<td>Graham et al. (2012)</td>
<td>M &amp; F</td>
<td>Rel. type not reported; Community</td>
<td>N=577; Males: BDIPV (128), LMIPV (56); Females BDIPV (157), LMIPV (236)</td>
<td>Composite International Diagnostic Interview; Odds ratios between IPV and depression for males (d=.29, OR = 3.37, p = .001) and females (d = .35, OR = 4.35, p = .001).</td>
</tr>
<tr>
<td>Greene, Coles, &amp; Johnson (1994)</td>
<td>M</td>
<td>Dating Cohabiting, Married, Divorced, Single; Clinical</td>
<td>N=40; 4 IPV groups based on personality clusters; Histrionic (9), Depressed (11), Normal (11), Disturbed (9)</td>
<td>Histrionic and Depressed IPV clusters had lower STAXI anger expression scores than Normal and Disturbed IPV clusters (d=1.38, CI = .69 – 2.06); Histrionic cluster had highest Hysteria scale score (d=.77, CI = .01 – 1.53); Normal cluster had the lowest (d=.69, CI = .004 – 1.37). No difference between Normal and all other clusters on depression (d=.55, CI = ns).</td>
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<tr>
<td>Hamberger et al. (1996)</td>
<td>M</td>
<td>Married; Clinical</td>
<td>N=833; MSIPV (148), LLA (212), LMIPV (335)</td>
<td>NAS; BDI; Non-pathological MSIPV and LLA clusters scored higher on the NAS than the LMIPV passive-aggressive dependent cluster (d=.64; CI:.49-.79); MSIPV higher depression scores than LLA and LMIPV (d=.90; CI:.72-.109)</td>
</tr>
<tr>
<td>Hastings &amp; Hamberger (1988)</td>
<td>M</td>
<td>Married, Single, Divorced, Separated; Clinical, CCG</td>
<td>N=168; DNV (43), IPV alcohol problems (47), IPV no-alcohol problems (78)</td>
<td>NAS, MCMI; BDI; DNV had higher anger scores than both IPV groups (d=-.48, CI=-.88-.08); IPV with and without alcohol problems reported more depression than DNV, respectively (d=.85, CI=.42-1.28; d=.66, CI=.28-1.04) on the BDI. IPV reported more depression (d=.76, CI=.41-1.12) and anxiety (d=.68, CI=.33-1.04) than DNV on MCMI.</td>
</tr>
<tr>
<td>Hershorn &amp; Rosenbaum (1991)</td>
<td>M</td>
<td>Dating cohabitating and non-cohabitating, Married, Separated, Single; Clinical</td>
<td>N=41; MSIPV (17 overcontrolled /24 undercontrolled)</td>
<td>BDHI; Overcontrolled hostile more severe IPV (d=.87, CI=.22-.52), but less frequent IPV (d=.73, CI=.09-1.37) than undercontrolled hostile.</td>
</tr>
<tr>
<td>Holtzworth-Munroe et al. (2000)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=164; SNV (39), DNV (23), FO (37), DB (15), LLA (34), MSIPV (16)</td>
<td>SPAFF; IPV groups rated higher on anger/hostility composite than SNV and DNV (d=.66, CI=.34-.98). SNV group had the lowest score on anger/hostility (d=.79, CI=.42-.16). No differences between groups on rated sadness (d=&lt;.06, n.s.).</td>
</tr>
<tr>
<td>Holtzworth-Munroe &amp; Smulzier (1996)</td>
<td>M</td>
<td>Married; Community/ Clinical mixed IPV sample; CCG</td>
<td>N=97; SNV (28), DNV (23), CLIPV (25), CMIPV (21)</td>
<td>Biglan rating system; There were no significant difference between IPV and NV groups on anger (d=.21, CI=ns) or hostility (d=.30, CI=ns) in response to written and videotaped stimuli.</td>
</tr>
<tr>
<td>Jacobson et al. (1994)</td>
<td>M</td>
<td>Married; Community/Clinical mixed sample</td>
<td>N=92; DNV (32), MSIPV (60)</td>
<td>SPAFF; MSIPV scored higher on SPAFF anger/contempt composite (d=.48, CI=.03-.93), and belligerence (d=.50, CI=.07-.93) than DNV. There were no differences between groups on sadness (d=&lt;.04, n.s.) or tension/fear (d=.17, n.s.).</td>
</tr>
<tr>
<td>Julian &amp; McKenry (1993)</td>
<td>M</td>
<td>Married, Separated, Divorced, Single; Community/ Clinical mixed sample</td>
<td>N=94; NV (50), IPV (42)</td>
<td>CES-D; NV scored higher on depression than IPV (d=−.55, CI=−.94−.13).</td>
</tr>
<tr>
<td>Kendra, Bell, &amp; Guimond (2012)</td>
<td>F</td>
<td>Dating cohabitating &amp; non-cohabitating; Community</td>
<td>N=496; LMIPV</td>
<td>MAI; Medium sized correlation between anger arousal and IPV (d=.47, CI=.29-.65)</td>
</tr>
<tr>
<td>Kim &amp; Capaldi (2004)</td>
<td>M &amp; F</td>
<td>Dating cohabitating/ non-cohabitating, Married; Community</td>
<td>N=158; Couples; IPV Wave 2 (158), IPV Wave 3 (148) *Waves were 3 years apart, same sample with attrition</td>
<td>CES-D; Depression and IPV are correlated at wave 2 for females (d=.77, CI=.45-.1.10) but not males (d=.16, n.s.). Wave 2 depression is correlated with wave 3 IPV for females (d=.70, CI=.37-.1.03) and males (d=.47, CI=.15-.80)</td>
</tr>
<tr>
<td>Leonard &amp; Senchak (1993)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=607; IPV</td>
<td>STAXI Trait Anger, BDHI, and permissiveness for aggression composite; Moderate correlation between anger/hostility composite and IPV (d=.61, CI=.44-.77).</td>
</tr>
<tr>
<td>Maiuro, Cahn, &amp; Vitaliano (1986)</td>
<td>M</td>
<td>Married, Dating Cohabiting;</td>
<td>N=107, NV (29), IPV (78)</td>
<td>BDHI; IPV had higher BDHI scores than NV (d=.86, CI=.42-.130).</td>
</tr>
<tr>
<td>Maiuro, Vitaliano, &amp; Cahn (1987)</td>
<td>M</td>
<td>Never married, Separated/Divorced, Married; Clinical, CCG</td>
<td>N=119, NV (26), LMIPV (30), VO (26), MSIPV (37)</td>
<td>BAAQ; IPV scored higher than NV (d=1.38, CI=.91-.1.85). No difference between LMIPV and MSIPV on BAAQ (d=-.08, CI=.01).</td>
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<tr>
<td>Maiuro et al. (1988)</td>
<td>M</td>
<td>Cohabitating/ never married, Separated/ divorced, Married; Clinical, CCG</td>
<td>N=129; NV (29), LMIPV (39), VO (29), MSIPV (38)</td>
<td>BDHI, HDHQ, BDI, IPV scored higher on BDHI (d=.85, CI = .42 – 1.27) and HDHQ (d=.86, CI= .44 – 1.29) than NV. LMIPV scored higher than NV, VO, and MSIPV on depression (d = −1.26, CI = −1.63 – −.86).</td>
</tr>
<tr>
<td>Margolin (1988)</td>
<td>M &amp; F</td>
<td>Married; Community</td>
<td>N=45; Couples; IPV (25) – PA and VA; NV (20) – includes WI type with some IPV</td>
<td>NAI, MMPI; No differences between IPV and NV for husbands and wives, respectively, on anger (d=55, CI = ns; d= −.02, CI = ns), depression (d=.59, CI = ns; d=.29, CI = ns), or hysteria (d=.42, CI = ns; d=.16, CI = ns).</td>
</tr>
<tr>
<td>Margolin, John, &amp; Foo (1998)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=171; NV (140); Emot. Abusive (69), Non Abusive (71); IPV (31): Phys. &amp; Emot. Abusive</td>
<td>BDHI, IPV Phys. and Emot. Abusive scored higher than both NV groups (d= .46, CI = .07 – .85). Emot. Abusive NV scored higher on BDHI than Non Abusive (d= .40, CI = .07 – .74).</td>
</tr>
<tr>
<td>Margolin, John, &amp; Gleberman (1988)</td>
<td>M &amp; F</td>
<td>Married; Community</td>
<td>N=78; SNV (19), IPV groups: IPV (19), VA (18), WI (22)</td>
<td>Self-reported affected state after conflict discussion with spouse; IPV groups scored higher than NV for husbands and wives, respectively, on anger (d=1.16, CI = .60 – 1.72; d=1.35, CI = .78 – 1.92), anxiety (d=.93, CI = .39 – 1.48; d=1.08, CI = .53 – 1.63), and sadness (d=.94, CI = .39 – 1.49; d=.85, CI = .30 – 1.39).</td>
</tr>
<tr>
<td>McNulty &amp; Hellmuth (2008)</td>
<td>M &amp; F</td>
<td>Married; Community</td>
<td>N=123; Males: LMIPV (61); Females MSIPV (62)</td>
<td>PANAS; IPV was not significantly correlated with negative affect for husbands (d=1.2, CI = n.s.) or wives (d=.39, CI = ns).</td>
</tr>
<tr>
<td>Murphy, Taft, &amp; Eckhardt (2007)</td>
<td>M</td>
<td>Dating cohabitating and non-cohabitating, Separated; Clinical</td>
<td>N=139; IPV: Pathological Anger (26), Low Anger Control (40), Normal Anger (70)</td>
<td>Pathological anger group reported more IPV than low anger control and normal anger groups at pre tx (d=.79, CI = .35 – 1.22), post tx (d=.59, CI = .15 –1.02), and at 6 month follow up (d=.61, CI = .18 – 1.04).</td>
</tr>
<tr>
<td>Pan, Neidig, &amp; O’Leary (1994)</td>
<td>M</td>
<td>Married and other non-specified; Army population</td>
<td>N=11,870; LMIPV</td>
<td>Modified BDI, 15 items; Odds ratios between depression and mild (d=1.04) and severe IPV (d=.86).</td>
</tr>
<tr>
<td>Saunders (1992)</td>
<td>M</td>
<td>Married; Clinical</td>
<td>N=165; LMIPV (86), MSIPV; GV (48), EV (31)</td>
<td>NAS, BDI; MSIPV higher in anger (d=.69, CI = .37 – 1.00) and depression (d = .79, CI = .47 – 1.11) than LMIPV.</td>
</tr>
<tr>
<td>Schumacher et al. (2008)</td>
<td>M &amp; F</td>
<td>Married; Community</td>
<td>N=634; Couples; 4 Longitudinal Waves</td>
<td>10 item hostility measure; Medium correlations between hostility and IPV averaged across time of marriage and 1, 2, and 4 year anniversaries for males (d=.54, CI = .38 – .70) and females (d=.57, CI = .41 – .73).</td>
</tr>
<tr>
<td>Shorey et al. (2010)</td>
<td>F</td>
<td>Married, Dating Cohabitating; Clinical</td>
<td>N=80; MSIPV</td>
<td>STAXI Trait Anger Scales; Large sized correlation between physical IPV and trait anger (d=1.07, CI = .60 –1.53.</td>
</tr>
<tr>
<td>Shorey et al. (2012)</td>
<td>M</td>
<td>Married, Dating cohabitating and non-cohabitating, single, divorced, separated; Clinical</td>
<td>N=308; CLIPV</td>
<td>PDSQ; Small to medium correlations for IPV and depression (d=.41, CI=.18 – .63), PTSD (d=.54, CI = .31 – .77), GAD and social phobia (d=.54, CI = .31 – .77, respectively), and panic disorder (d=.39, CI = .16 – .61).</td>
</tr>
<tr>
<td>Sullivan et al. (2013)</td>
<td>F</td>
<td>Relationship type not reported; Community</td>
<td>N=369; CMIPV</td>
<td>CES-D; STAI: State-Anxiety subscale; Small correlation between IPV and depression (d=.24, CI = .04 – .44). No correlation between IPV and anxiety (d=.04, ns).</td>
</tr>
<tr>
<td>Swan et al. (2005)</td>
<td>F</td>
<td>Relationship type not reported; Community/ Clinical mixed sample</td>
<td>N=108; IPV</td>
<td>CES-D, Anger Expression Scale; Medium sized correlations between</td>
</tr>
<tr>
<td>Author</td>
<td>Sex</td>
<td>Population Characteristics</td>
<td>N and Sample Size by Group</td>
<td>Measures &amp; Description of Findings</td>
</tr>
<tr>
<td>------------------------</td>
<td>-----</td>
<td>-----------------------------</td>
<td>---------------------------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Swan &amp; Snow (2003)</td>
<td>F</td>
<td>Relationship type not reported; Clinical</td>
<td>N=95; IPV groups varied on coercion by male or female and female as sole aggressor; VNV (32)</td>
<td>IPV and anger in (d=.63, CI = .24–1.02) and anger control (d=−.49, CI = −.88 − −.11). Large sized correlations between IPV and depression (d=.70, CI = −.31 to 1.09) and IPV anger out (d=.98, CI = .58–1.38). Anger Expression Scale, State Trait Anxiety Inventory, CES-D; VNV, interestingly, scored higher on anger in than IPV groups (d=−.44, CI = −.87 − −.01). No differences between groups on anger out (d=.20, n.s.) or anger control (d=.29, n.s.). VNV reported greater anxiety (d= −.73, CI = −1.17 − −.30) and depression (d= −.126, CI = −1.72 − −.80) than IPV groups.</td>
</tr>
<tr>
<td>Tharp et al. (2012)</td>
<td>M</td>
<td>Dating Cohabitating/Married/other; Community</td>
<td>N=121; LMIPV</td>
<td>BDHII; Large sized correlation between IPV and hostility (d=.93, CI = .55 − 1.30).</td>
</tr>
<tr>
<td>Tweed &amp; Dutton (1998)</td>
<td>M</td>
<td>Rel. type not reported; Clinical</td>
<td>N=114; NV (44), LMIPV (70) – 2 groups based on personality types</td>
<td>MCMI-II, MAI; LMIPV greater anger than NV (d=.47, CI = .09 − .85). Impulsive LMIPV reported greater anger than NV (d=.63, CI = .18 − 1.07). Impulsive LMIPV had greater depression (d = −1.37, CI = −1.89 − −.85) and anxiety (d = −1.31, CI = −1.83 − −.79) scores than Instrumental LMIPV.</td>
</tr>
<tr>
<td>Waltz, Babcock, &amp; Gottman (2000)</td>
<td>M</td>
<td>Married; Community</td>
<td>N=103; DNV (32), GV (17), Pathological (16), Family Only (38)</td>
<td>SPAFF; No difference between IPV groups and DNV on SPAFF anger (d=.21, CI = n.s.), tension/fear (d=.13, CI = n.s.), or sadness (d=.28, CI = n.s.). IPV groups scored higher on SPAFF contempt than DNV (d=.46, CI = .03−.88).</td>
</tr>
</tbody>
</table>

aSex = sex of IPV perpetrator; Construct = Anger (A), Hostility (H), and Negative Emotion (NE); ( ) = N for each subgroup; CI = 95% confidence interval; IPV = intimate partner violence (severity unspecified); LMIPV = low/moderate IPV; MSIPV = moderate/severe IPV; NV = non-violent; DNV = distressed/dissatisfied non-violent; SNV = satisfied non-violent; VO = violent outside intimate relationship; GV = generally violent within and outside of intimate relationship; SO = sex offenders; DB = dysphoric/borderline; EV= emotionally volatile; FO = family only; LLA = low-level antisocial; VNV = nonviolent victim of IPV; VA = verbally abusive; WI = nonviolent withdrawing; CLIPV = clinical IPV; CMIPV = community IPV; BDIPV = bidirectional IPV; INCIPV = incarcerated IPV.
Table 2

Mean effect sizes and homogeneity tests for moderators of the relationship between anger, hostility, anger/hostility, negative emotion and IPV.

<table>
<thead>
<tr>
<th>Moderator Variables</th>
<th># of effects</th>
<th>Mean d</th>
<th>95% CI</th>
<th>Q between</th>
<th>Q within</th>
</tr>
</thead>
<tbody>
<tr>
<td>Construct</td>
<td>2.51</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Anger</td>
<td>37</td>
<td>.48***</td>
<td>.27 – .68</td>
<td>21.33</td>
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<tr>
<td>Hostility</td>
<td>17</td>
<td>.56***</td>
<td>.27 – .85</td>
<td>3.20</td>
<td></td>
</tr>
<tr>
<td>Anger/Hostility Composite</td>
<td>3</td>
<td>.64**</td>
<td>-.04 – 1.32</td>
<td>.03</td>
<td></td>
</tr>
<tr>
<td>Negative Emotions</td>
<td>47</td>
<td>.33***</td>
<td>.16 – .51</td>
<td>61.46</td>
<td></td>
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<tr>
<td>Depression</td>
<td>26</td>
<td>.42**</td>
<td>.12 – .72</td>
<td>24.16</td>
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<tr>
<td>Anxiety-related</td>
<td>13</td>
<td>.36*</td>
<td>-.07 – .79</td>
<td>7.30</td>
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<tr>
<td>Other</td>
<td>4</td>
<td>.15</td>
<td>-.63 – .93</td>
<td>.53</td>
<td></td>
</tr>
<tr>
<td>Sex of Perpetrator</td>
<td>.29</td>
<td></td>
<td></td>
<td>117.70</td>
<td>32.00</td>
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<tr>
<td>Male</td>
<td>93</td>
<td>.44***</td>
<td>.32 – .55</td>
<td>84.85</td>
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<tr>
<td>Female</td>
<td>30</td>
<td>.37***</td>
<td>.17 – .57</td>
<td>32.84</td>
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<tr>
<td>IPV Perpetrator Population</td>
<td>.88</td>
<td></td>
<td></td>
<td>91.11</td>
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<tr>
<td>Community</td>
<td>45</td>
<td>.40***</td>
<td>.22 – .57</td>
<td>25.10</td>
<td></td>
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<tr>
<td>Clinical</td>
<td>38</td>
<td>.50***</td>
<td>.31 – .69</td>
<td>55.99*</td>
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<tr>
<td>Mixed Sample</td>
<td>21</td>
<td>.37***</td>
<td>.11 – .63</td>
<td>10.02</td>
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<tr>
<td>Relationship Type</td>
<td>1.72</td>
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<td></td>
<td>1.72</td>
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<tr>
<td>Married or Dating Cohabitating</td>
<td>58</td>
<td>.41***</td>
<td>.25 – .56</td>
<td>24.53</td>
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<tr>
<td>Dating, Non-Cohabitating</td>
<td>2</td>
<td>.47</td>
<td>-.56 – 1.30</td>
<td>.02</td>
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</tr>
<tr>
<td>Mixed</td>
<td>29</td>
<td>.59***</td>
<td>.36 – .81</td>
<td>51.68**</td>
<td></td>
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<tr>
<td>Dating Mixed</td>
<td>6</td>
<td>.46</td>
<td>-.02 – .93</td>
<td>2.64</td>
<td>.71</td>
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<tr>
<td>Married (only)</td>
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<td>.38***</td>
<td>.21 – .54</td>
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<td>Dating</td>
<td>7</td>
<td>.39</td>
<td>-.06 – .84</td>
<td>.41</td>
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<tr>
<td>Mixed</td>
<td>37</td>
<td>.59***</td>
<td>.39 – .78</td>
<td>53.76*</td>
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<tr>
<td>Method of Assessment</td>
<td>.268</td>
<td></td>
<td></td>
<td>2.68</td>
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<tr>
<td>Self-Report</td>
<td>83</td>
<td>.48***</td>
<td>.35 – .60</td>
<td>82.47</td>
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</tr>
</tbody>
</table>
### Moderator Variables

<table>
<thead>
<tr>
<th># of effects</th>
<th>Mean</th>
<th>95% CI</th>
<th>Q between</th>
<th>Q within</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observational</td>
<td>23</td>
<td>0.25&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.00 – 0.49</td>
<td>11.98</td>
</tr>
</tbody>
</table>

<sup>a</sup> Full list of self-report measures available upon request.

<sup>b</sup> Three effect sizes from anger/hostility composite measures were included in this analysis.

<sup>c</sup> There were moderate effect sizes for anger, hostility, and anger/hostility composite scores and IPV (d = 0.51, k = 56, 95% CI = 0.46 – 0.56, p < 0.001), an anger and anger/hostility composite and IPV (d = 0.52, k = 40, 95% CI = 0.46 – 0.57, p < 0.001), and hostility and anger/hostility composite scores and IPV (d = 0.53, k = 19, 95% CI = 0.46 – 0.60, p < 0.001). These effect sizes were calculated for comparison purposes to the findings of Norlander and Eckhardt (2005).

<sup>+</sup> = 0.07,

* = p = 0.05;

** = p = 0.01;

*** = p = 0.001;

CI = confidence interval