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I am submitting herewith a thesis written by Aaron John Kivisto entitled “Attributions and Negative Affect as Moderators of PTSD Symptomatology and Aggression.” I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Psychology.

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Attributions and Negative Affect as Moderators of PTSD Symptomatology and
Aggression

A Thesis
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Master of Arts
Degree
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Aaron John Kivisto
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Abstract

Interpersonal violence has been identified as one of our nation's most important public health concerns and has been empirically linked with Posttraumatic Stress Disorder (PTSD). In order to better understand the relationship between PTSD and violence, we used Chemtob's (1997) Survival Mode model to examine the role of two potentially important proximal factors linking PTSD to violence; cognitive attributions and negative affect. Congruent with theory and previous research, we hypothesized that PTSD would be related to aggression, and that cognitive attributions and negative affect would moderate the relationship between these variables.

Undergraduate students (N = 628) completed an online screening study to assess PTSD symptomatology. Based on screening results, 99 participants were recruited and categorized as having elevated PTSD symptomatology (High-PTSD) or minimal symptomatology (Low-PTSD). Eligible participants completed questionnaires assessing trauma history, psychopathology, attitudes towards violence, and history of violent behavior. Participants then completed the Point Subtraction Aggression Paradigm (PSAP), a lab-based behavioral measure of aggression, in which participants competed against a confederate and had points subtracted on a pre-programmed interval schedule. After completing this task, participants completed measures assessing negative affect and attributions experienced during the PSAP procedure.

Results demonstrate that High-PTSD participants engaged in significantly more aggressive responding on the PSAP than did Low-PTSD participants. Further, High-PTSD participants reported more negative affect than did low-PTSD participants, and a

trend approaching significance suggested that High-PTSD participants tended to make more negative intent attributions regarding confederates' behavior than did low-PTSD participants. Contrary to our hypotheses, findings revealed that negative affect and attributions were unrelated to aggressive responding and did not moderate the relationship between PTSD symptomatology and aggressive responding.

It is possible that the retrospective reporting of negative affect and attributions limited the ability to detect moderation effects in the present study. Implications of the findings are discussed and future research is called for in order to better explicate the complex relationship between PTSD symptomatology and aggression.

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Introduction

Overview of the Project

The Centers for Disease Control have placed a priority on the prevention of violence among those at high risk, acknowledging that injuries resulting from interpersonal violence are one of the most important public health problems facing our country (CDC, 2003). From a public health perspective, the estimated annual cost resulting from interpersonal violence amounts to 3.3% of the Gross Domestic Product of the United States (WHO, 2004). At the individual level, victims of interpersonal violence may suffer any number of deleterious consequences ranging from marital discord in situations of domestic violence to death (WHO, 2004). Based on the ubiquity of interpersonal violence and the consequent burden placed both on the victims and society as a whole, the need to better understand this problem is becoming increasingly evident.

As is true with nearly all behaviors, aggression is complex and multiply determined. While it is beyond the scope of this investigation to examine all the potential factors that may contribute to aggression, the goal of this project is to examine the link between Posttraumatic Stress Disorder (PTSD) symptomatology and aggression. First labeled with terms such as “gross stress reaction” and “transient situational disturbance,” PTSD received its current label in DSM-III based on empirical support obtained from samples of combat veterans (Resick & Calhoun, 2001). Following several decades of research, PTSD is now known to be a debilitating anxiety disorder that follows a terrifying physical or emotional threat to one’s physical integrity (APA, 2000). Among the many deleterious effects of this disorder, in particular, PTSD may increase the risk for

violent behavior. Examining the relationship between PTSD and aggression, both theory and research described below suggest that PTSD may impact thoughts and emotions in potentially threatening situations and increase the likelihood of aggression. Therefore, the specific goal of this research is to examine the meanings that people ascribe to threatening situations and their affective experiences as potential moderators of the relationship between PTSD symptomatology and aggression. In order to achieve the goals of the study, especially assessing the attributions made in response to potentially threatening situations, the proposed project will use a well-established and validated laboratory-based, confederate-involved, competitive aggression paradigm.

The following sections will summarize the available research examining the relationship between PTSD and aggression (including the role of cognition and affect in aggression), describe theoretical explanations for the relationship between PTSD symptomatology and aggression, discuss various paradigms for assessing this relationship (with a particular focus on lab-based aggression paradigms), and conclude with a rationale and overview of the current study aiming to examine the potential moderating role of cognitive attributions and negative affect on the association between PTSD symptomatology and aggression.

The PTSD-Aggression Relationship

While various definitions of aggression have been proposed, Baron and Richardson's (1994) definition of aggression as "any form of behavior directed toward the goal of harming or injuring another living being who is motivated to avoid such

treatment” (p.7) captures the salient aspects of behavior commonly regarded as aggression. Examining the prevalence of violence, surveys conducted by the U.S. Department of Justice indicate that assault rates among young adults are second only to adolescents, with 6.7% of 20- to 24-year-old men and 4.3% of 20- to 24-year-old women reporting being the victim of an assault in the past year (Bureau of Justice Statistics, 1998). These rates seem low relative to other large studies, such as those including college populations, which have shown physical victimization rates as high as 28.8% (Leonard, Quigley, & Collins, 2002). Further, males and females recruited from the community reported 37.1% and 22.3% victimization rates, respectively. While the college sample showed significantly lower rates of victimization, they did not differ in their rates of initiating aggression from the community sample (Leonard et al., 2002). Examining gender differences in victimization, the National Crime Victimization Survey (NCVS) shows that while men are twice as likely to be the victims of simple or aggravated assault, women are approximately ten times more likely to be the victims of sexual assault (Bachman & Saltzman, 1995). The prevalence of interpersonal violence is particularly alarming considering the numerous adverse consequences that violence has on its victims, such as PTSD, depression, low self-esteem, lower educational attainment, and a range of somatic health concerns, including chronic pain and gastrointestinal problems (Campbell, 2002; Holtzworth-Munroe, Smutzler, & Sandin, 1997; Koss, & Heslet, 1992; Macmillan, 2001; Tjaden, & Thoennes, 1998).

Akin to the ubiquity of interpersonal violence, PTSD is a prevalent social concern. Within the nosology provided by the DSM IV-TR (APA, 2000), the symptoms

of PTSD follow a physical or emotional threat to one's integrity and fall into three general categories, including 1) re-experiencing symptoms, such as flashbacks, nightmares, and intrusive memories of the traumatic event, 2) avoidance symptoms, including avoiding activities, people, or places that remind the person of the trauma as well as feelings of detachment and a circumscribed range of affect, and 3) arousal symptoms, including irritability, difficulty sleeping and concentrating, hypervigilance, and an exaggerated startle response.

In a nationally representative survey of 5,692 people, Kessler, Chiu, Demler, and Walters (2005) found that 3.5% of individuals met diagnostic criteria for PTSD in the past 12 months. Of those, 36.6% are estimated to be severely impaired, with 33.1% and 30.2% moderately and mildly impaired, respectively. Further, national probability samples estimate lifetime prevalence of PTSD at 6.8% (Kessler, Berglund, Demler, Jin, & Walters, 2005). Breslau and colleagues' (1991) national probability survey suggests that lifetime prevalence rates for PTSD may be higher for women (10.4%) than for men (6%), although men are more likely to be exposed to PTSD-level stressors. While these statistics highlight the prevalence of PTSD, it is also necessary to consider the consequences associated with this disorder, including heightened depression (Merton, & Mohr, 2000; Kilpatrick et al., 2003), alcohol use/abuse (McFarlane, 1998), poorer quality of life (Clark, & Kirisci, 1996), occupational impairments (Brunello et al., 2001), and interpersonal impairments evident in one's family and marital relationships (MacDonald et al., 1999). Further, according to Kessler (2000), individuals with PTSD are six times more likely than demographically matched individuals without PTSD to attempt suicide.

From a societal perspective, Kessler et al.'s (2005) review demonstrated that PTSD is associated with educational failure, teen childbearing and marital instability; all major factors contributing to welfare dependency in Western societies.

In addition to the prevalence and societal costs of aggression and PTSD, the co-occurrence of these phenomena has intrigued researchers (i.e., Chemtob et al., 1994). While the role of PTSD symptomatology in aggression has been explored across a wide range of traumas in the current literature, this research began exclusively with male combat veterans in the 1980's. Based in part on the Congressional mandate to assess the social readjustment of Vietnam veterans following their deployment that resulted in the *National Vietnam Veterans Readjustment Study* (NVVRS), there has been a considerable amount of research devoted to the link between combat-related PTSD and interpersonal violence perpetration. McFall et al. (1999) demonstrated that Vietnam combat veterans receiving inpatient psychiatric treatment for PTSD exhibited more violent behavior, such as property destruction, threats with and without a weapon, and physical fighting, than inpatients without PTSD. This finding is congruent with other findings from the NVVRS, which found that 46% of veterans with PTSD had committed at least one violent act in the past year, with 37% committing six or more such acts (Kulka et al., 1988). By demonstrating that veterans with PTSD receiving inpatient psychiatric treatment exhibit more aggression than veterans with PTSD not undergoing inpatient treatment, McFall et al. (1999) demonstrate that PTSD symptom severity should be considered in assessing the potential for aggression, with greater symptom severity suggesting a greater risk of

aggression. In sum, these studies using male combat veterans consistently point to a strong association between PTSD and aggression.

Social learning theory (Bandura, 1986), which emphasizes learning by observing others and developing subsequent expectations regarding the consequences of behaviors, may help to explain the relationship between combat experience and aggression. Such an explanation, however, is not sufficient because it fails to address differential outcomes among those with similar learning histories. Because these studies have compared veterans with veterans, these findings raise an interesting question. Specifically, why is it that those veterans who go on to develop PTSD show higher rates of aggression than veterans who develop other psychiatric disorders? Based on the aforementioned research, it appears that PTSD symptomatology may make a unique contribution to aggressive behavior. Further, this heightened aggression remains even after controlling for combat exposure among veterans (Castillo et al., 2002; Taft et al., 2007) and impulsivity (Chemtob et al., 1994). While these findings consistently indicate a positive correlation between PTSD symptomatology and interpersonal violence perpetration, many questions remain unanswered. While the NVVRS (Kulka et al., 1988) brought the PTSD-violence link to our attention, the fact that each of these studies focused exclusively on male Vietnam combat veterans reduces the generalizability of these findings.

In light of the necessity of addressing this societal problem, researchers have examined numerous correlates and predictors of violence. Of particular importance, past research has noted an association between PTSD and aggression (e.g., Cunningham, Connor, Miller, & Melloni, 2003). More specifically, Castillo and colleagues (2002)

found that a group of individuals with PTSD demonstrated consistently higher rates of interpersonal violence perpetration than a group comprised of individuals with other psychiatric diagnoses, including depression, substance abuse, adjustment disorder, anxiety disorders other than PTSD, and personality disorders. In addition, individuals with PTSD have been shown to evidence higher levels of anger than those without PTSD (Chemtob et al., 1994), suggesting that PTSD may be a risk-factor for aggression that necessitates further study in an effort to deter interpersonal violence. Both as a social concern and a frequently encountered clinical problem, PTSD-related violence perpetration has only recently received empirical attention.

Moving beyond this circumscribed population of male combat veterans with PTSD, other forms of trauma leading to PTSD have also been associated with heightened aggression. For example, Moe, King, and Bailly (2004) found heightened aggressive responding among male and female college students reporting childhood parental physical abuse. Utilizing a lab-based aggression paradigm, these authors found that nearly half of those with a history of abuse engaged in high levels of aggressive responding (≥ 400 B-button responses) that were similar to the average previously found among violent parolees (Cherek, Moeller, Schnapp, & Dougherty, 1997). This figure is in contrast to none of the non-abused group engaging in this high level of aggressive responding. This research offers further support for the association between trauma and violence perpetration in a controlled laboratory setting.

Examining adolescents who have witnessed parental domestic violence, Moretti et al. (1996) found that adolescents witnessing domestic violence to be at increased risk for

perpetrating aggressive acts towards friends and romantic partners. Further, nearly one-third of this sample met diagnostic criteria for PTSD, suggesting that witnessing domestic violence in one's family of origin serves as a risk factor for this psychiatric diagnosis. Taken together, Moretti et al. (1996) found that individuals witnessing domestic violence who subsequently met diagnostic criteria for PTSD were particularly likely to be aggressive as adolescents.

Examining maritally violent men, Holtzworth-Munroe and colleagues (1997) found that physically aggressive men were more likely than nonviolent men to have experienced the trauma of witnessing domestic violence in their family of origin. Although previous research has yet to examine the association between other forms of trauma and aggression, the existing body of research consistently points to a correlation between aggression across the types of trauma that have been studied. Therefore, it appears likely that many forms of trauma and trauma symptomatology may put people at risk for the perpetration of interpersonal violence.

While a majority of the current literature indicates a correlation between PTSD and violence and seems to suggest that PTSD predicts violence over time, the correlational nature of the work prohibits such conclusions. Acknowledging that correlations alone offer little evidence of predictive value, Wolfe and colleagues (2004) conducted a one-year longitudinal study examining the extent to which child maltreatment and trauma predicted violence perpetration in adolescent dating relationships. To our knowledge, this study appears to be the only longitudinal examination of the trauma-violence link over time. Using a large sample ($N = 1,317$) of

students from 10 high schools, these authors assessed levels of partner violence perpetration at Time-1, along with three potential mediators of the relationship between a history of childhood abuse and partner violence perpetration: 1) current trauma-related symptoms, 2) attitudes justifying dating violence, and 3) empathy and self-efficacy in dating relationships. Using structural equation modeling techniques, results indicated that current trauma-related symptoms alone were predictive of increases in partner violence perpetration between Time-1 and Time-2 (one year later) and were the only variable to mediate the relationship between childhood abuse and partner violence perpetration. In sum, this research suggests that childhood maltreatment is a significant distal correlate of violence, a finding corroborated by previous researchers (Moe, King, & Bailly, 2004), while highlighting the importance of current trauma symptoms in understanding the relationship between childhood maltreatment and partner violence over time.

Taken together, these studies have begun to elucidate the PTSD-violence relationship. As Haapasalo and Pokela (1999) point out, however, our current understanding lacks “information about the variables that mediate and moderate the relationship between trauma and antisocial outcomes” (p. 123). Put another way, traumatic experiences represent a distal correlate of aggression; future research requires an examination of more proximal variables in order to glean a better understanding of this relationship. This study intends to address this limitation by examining the role of cognitive attributions and negative affect in the PTSD symptomatology-violence relationship. The following sections discuss the potential role of cognitive processes and negative affect in increasing risk for violence among individuals with PTSD.

Cognition and Aggression

One variable that might influence the relationship between PTSD symptomatology and aggression is cognitive attributions. A substantial amount of research has suggested that cognitive attributions, or the meanings that people ascribe to events and behavior, might serve to influence aggressive behavior (Berkowitz, 1983; Chemtob et al., 1997; Dodge, Pettit, Bates, & Valente, 1995; Lochman & Dodge, 1994; McNiel, Eisner, & Binder, 2003; Nader, Coles, & Foa, 2002). For example, McNiel et al. (2003) discuss the theoretical construct of the hostile attributional style, characterized by the perception that others are driven by negative dispositional traits to cause one harm. Put more graphically, Dill and colleagues (1997) describe individuals characterized by a hostile attributional style as tending “to view the world through blood-red tinted glasses” (p. 275). Examining this predilection with self-report measures among 110 psychiatric inpatients, McNeil et al. (2003) found that patients who made more hostile attributions reported a greater number of aggressive acts in the past two months, providing information about their risk for violence above and beyond that of demographic and diagnostic variables alone. These results suggest that biased social information processing might translate into overt aggressive behaviors.

Whereas the work of McNeil and colleagues (2003) offers preliminary information regarding a potentially useful construct for aggressive adults, there exists a broader literature examining hostile attributional styles among aggressive adolescents. Examining the attributional styles of severely aggressive adolescents in a maximum-

security prison for adolescent offenders, Dodge, Price, Bachorowski, and Newman (1990) showed 16 video-recorded vignettes to participants. In addition to assessing attributional styles based on these vignettes, prison staff rated each individual's proactive (e.g., bullying) and reactive (e.g., defensive) aggression. Results suggested that hostile attributional biases were positively correlated with offenders' levels of conduct disorder, reactive and proactive aggression, and violent crimes. These associations held even after controlling for the effects of race, socioeconomic status, and intelligence.

Contrasting severely violent with moderately aggressive and non-aggressive adolescent boys, Lochman and Dodge (1994) suggest that the attributional biases of severely violent adolescents represent a form of cognitive misperception that impedes socially competent behavior. Using 12 video-recorded vignettes depicting conflictual situations to assess attributions, these authors found that severely violent adolescents differed both in their recall of social cues and in their attributional biases from moderately aggressive and non-aggressive adolescents. The deficiencies among severely violent adolescents in attending to and recalling relevant interpersonal cues suggest an impediment to accurate social information processing, possibly contributing to their qualitatively different attributional style.

In a review of the literature on social information processing, Crick and Dodge (1994) concluded that the positive relationship between hostile attributional biases and aggression held not only in hypothetical situations, such as those found in studies utilizing vignettes, but also in actual lab-based situations as well. Based on their review, there is consistent evidence that hostile attributional biases are frequently present in

children or adolescents with externalizing or aggressive behavior. While a majority of this correlational research operates, at least implicitly, under the assumption that these hostile attributional biases may cause the aggression, Dodge et al. (1995) attempted to delineate a causal relationship through a mediational model. Within this model, early abuse served as the independent variable, teacher-rated externalizing problems served as the dependent variable, and social information processing variables served as potential mediators. Results indicated that abuse was associated with social information processing deficits, and these deficits (including a hostile attributional bias) predicted later externalizing behaviors. Relevant to the trauma-violence association discussed above, Dodge et al. (1995) found that nearly half of the effect of early physical harm on externalizing problems can be accounted for by deficient social information processing. Not only do these findings suggest that social information processing influences aggression, but they also suggest the need to consider social information processing variables in future research examining the trauma-violence association.

In addition to demonstrating that cognitive attributions are linked to aggression, research has also shown that attributions are associated with PTSD symptomatology. To better understand the deficits in social information processing among adults with PTSD, the homograph paradigm has been implemented to examine the tendency of those with PTSD to interpret ambiguous information as threatening (Nader, Coles, & Foa, 2002). By presenting words with two meanings (e.g. mug), one of which may have a threatening connotation, these authors compared the performance of those with PTSD to those with a trauma history without PTSD. Results indicate that individuals with PTSD not only

interpret greater threat in these ambiguous words, but they show higher levels of difficulty in inhibiting the activation of this interpretation due to an inability to generate alternative explanations. These findings support the view that individuals with PTSD show general deficits in social information processing that often results in interpreting ambiguous stimuli as threatening while failing to recognize alternative explanations. Therefore, it is possible that those with PTSD will frequently demonstrate similar deficits in social information processing that might partially account for the relationship between PTSD and violence.

Comparing the social information processing of those with PTSD to those with Panic Disorder (PD) with the emotional Stroop task, Buckley, Blanchard, and Hickling (2002) found that those with PD showed greater interference with words specific to their clinical condition compared to those with PTSD. In contrast, those with PTSD showed a much more generalized interference with words of negative valence, not only those specific to their disorder, compared to those with PD. These findings further suggest that individuals with PTSD tend to exhibit a generalized deficiency in social information processing whereby they selectively focus on negative stimuli that, in turn, interfere with cognitive processing.

Based on prior research examining the attributional style of aggressive individuals in general (i.e., Crick & Dodge, 1994), it is the intention of the present investigation to examine the cognitive attributions made by those with PTSD symptomatology within the context of a laboratory-based aggression paradigm. By obtaining a retrospective account of participants' histories of interpersonal violence, trauma histories, and trauma

symptomatology, in conjunction with a laboratory-based aggression procedure, it will be possible to examine several aspects of the relationship between PTSD symptomatology and aggression, especially the role of cognitive attributions as a moderator of the relationship between PTSD symptomatology and aggression. While a majority of the previous research explicating the link between PTSD and aggression has assessed aggression through retrospective self-report methods, the utilization of a laboratory-based aggression procedure facilitates the study of the cognitive attributions that may moderate the PTSD symptomatology-aggression relationship immediately following provocation.

Negative Affect and Aggression

To this point this review has summarized the literature linking PTSD to aggression and reviewed the relationship between cognitive attributions and aggression. Before examining the theoretical explanations linking PTSD symptomatology and aggression, it is first necessary to discuss the role of negative affect as another potential moderator variable between PTSD symptomatology and aggression.

From a theoretical standpoint, Berkowitz (1990) proposed a cognitive neoassociationistic model of aggression. Fundamental to this model is the notion that a wide variety of unpleasant feelings can give rise to anger and aggression. Further, it is presumed that an associative network links specific feelings with particular thoughts, memories, motor movements, and physiological reactions. By activating any part of this network, it is thought that all associated parts will be subsequently activated. While thought is not considered to be an essential ingredient in this process, Berkowitz posits

that certain thoughts or attributions might serve to enhance or minimize aggressive behavior.

Congruent with Berkowitz's (1990) proposition that negative affect of any kind will activate anger-related feelings, thoughts, memories, and actions, more recent research has found support for the role of negative affect on aggression, even when this negative affect cannot be attributed to the misdeeds of another individual. For example, in recent years there has been a heated debate in the social psychological literature regarding the effects of unpleasantly hot weather on interpersonal violence. According to Berkowitz (2000), unpleasantly high temperatures result in negative affect, which in turn leads to aggressive inclinations through the activation of an associative network. Lending support to Berkowitz's theory, Rotton and Cohn (2000) replicated previous research showing that violence is a curvilinear function of weather, suggesting that as individuals become uncomfortably warm they become more aggressive, although this increase shows a sharp inflection point when the temperatures enter the 95-100 degree range. Similarly, researchers have found that unpleasantly cold temperatures lead to heightened aggression (Anderson, 1989). While this line of research is plagued by the possibility of a third variable playing an unacknowledged role in contributing to heightened aggression, it serves to bridge the gap between theory and empirical examination. Fortunately, more rigorous experimental control has been implemented in other examinations linking negative affect to aggression and this body of evidence consistently points to the relationship between these two variables.

Examining the role of negative affect on aggressive responding using an air-blast aggression paradigm, Verona, Patrick, and Lang (2002) provide evidence of increased aggression following an experimental induction of negative affect. Within this procedure, aggression was operationalized as an amalgam of three components: 1) shock latency, 2) shock intensity, and 3) shock duration. Results indicate that increases in negative affect, induced with a blast of air to the participant's throat, appeared to potentiate aggressive responding by decreasing shock latency, whereas shock intensity and duration did not increase significantly.

While Verona and colleagues (2002) found no evidence of negative affect contributing to greater shock intensity and duration, correlational analyses revealed that shock intensity was related to the motives participants attributed to their aggression. Further, these authors suggest "shock intensity (and duration) may not be influenced directly by emotional reactions to a specific stressor but may instead be influenced by cognitive biases, rumination, and perceptions of the situational context" (p. 256). While these findings regarding the cognitive processes are presented as auxiliary to those supporting the negative affect-aggression relationship, they serve to highlight the roles of negative affect and attributions potentially working in combination to influence aggressive responding.

Bjork, Dougherty, and Moeller (1997) demonstrated a relationship between depressive symptoms and aggression among females using a lab-based aggression paradigm, although this finding was not found among male participants. Further demonstrating the relationship between negative affect and aggression using a sample of

combat veterans from the NVVRS, Taft et al. (2007) examined the role of dysphoric negative affect in the PTSD-aggression relationship using the Beck Depression Inventory. Based on findings derived from structural equation modeling, these authors found that symptoms of PTSD were directly related to aggression and also indirectly related to aggression through dysphoric symptoms. Similarly, previous research has shown that comorbid depression is a significant risk factor for intimate partner violence among veterans with PTSD, further corroborating the role of negative affect in the PTSD-violence relationship (Taft et al., 2005). Taken together, previous research has consistently supported the relationship between negative affect, including anger and depression, and aggression.

Negative Affect and Cognition

It is important to note that this relationship between negative affect and cognition appears to be bidirectional and has been approached as such by various researchers (i.e. McFarland & Ross, 1982; Gasper & Clore, 1998) and that this relationship is important in examining the PTSD symptomatology-violence link. Consider, for example, an individual who attributes his success to his hard work and dedication. This person is likely to feel a sense of accomplishment and generally positive affect. In other words, his internal attributions might be said to elicit positive affect. Conversely, a man who has just had a stressful day at work might attribute another man's accidentally bumping into him in a bar as an intentional threat, leading to anger and increased need to protect himself.

Put another way, his negative affect appears to be influencing the attributions he makes in social situations.

In contrast to Berkowitz's (1990) cognitive neoassociationistic model of aggression, Clore and Centerbar (2004) ascribe a central role to cognitive processes in the elicitation of anger. Whereas Berkowitz's model grants cognitive processes the role of having a quantitative impact on anger (e.g. cognitions can serve to increase or decrease anger that is already present), Clore and Centerbar (2004) posit that cognitions have a qualitative influence on anger (e.g. cognitions are responsible for creating anger from other emotions). Lending support to the qualitative influence of attributions on anger, this theoretically based work has been supported by empirical evidence.

Providing empirical support for the central role of cognitions, recent authors have examined the role of emotions in retaliation following an experience of injustice (Barclay, Skarlicki, & Pugh, 2005). Using a sample of 173 individuals recently laid off from their jobs, these researchers assessed self-reported emotions and divided them into two groups: 1) Inward-focused negative emotions, such as shame and guilt, and 2) outward-focused negative emotions, such as anger and hostility. These authors posit that the outward-focused negative emotions reflect external attributions such that others are blamed for the offense of being laid off. Results suggest that outward-focused negative emotions partially mediated the relationship between being laid off and seeking retaliation. Put simply, those who were laid off only showed a tendency to seek retaliation if they experienced outward-focused negative emotions such as anger. To add another degree of complexity to this relationship, these authors speculate that the

cognitive attributions one makes after being laid off influence their emotional experience, and only after this process has occurred does the individual proceed from feeling to behaving.

Previous research has supported the speculation of Barclay and colleagues (2005) that behavior follows attributions only after experiencing a mediating emotion. Over the course of six studies, Weiner (1980) investigated the temporal sequence of attributions, emotions, and behavior within the context of a help-giving situation. Overall, he found that attributions gave rise to the emotional reactions of participants in response to hypothetical vignettes involving a person in need. In turn, results of a test of mediation suggested that emotional reactions guide one's actual behavioral responses, demonstrating an attribution-emotion-action sequence. Although this work examined help-giving behaviors, Weiner theorized that the same pattern should emerge across other motivated behaviors, including aggression. For example, attributing failure to the hindrance of others will likely lead to anger, and one is motivated to aggress against another in order to eliminate this anger.

Lending further support to the view that attributions influence affect, McFarland and Ross (1982) manipulated whether female subjects succeeded or failed on a social accuracy test while concurrently manipulating whether participants made internal or external causal attributions. While success or failure evaluations on the task played a role in participants' affect, these authors found that positive affect followed positive outcomes only when internal causal attributions were induced. Therefore, outcomes alone were insufficient for eliciting positive affect. This finding indicates an interplay between the

meanings that people attribute to events and their emotional state. Further, it suggests that assessing attributions is necessary in understanding affect.

In contrast to the work of McFarland and Ross (1982), Gasper and Clore (1998) focus on the opposite direction of this affect-attributions relationship. Specifically, these authors used a median split to categorize participants as being either high or low in trait anxiety, after which they administered a questionnaire assessing the perceived likelihood of 10 negative events occurring. As predicted, those with higher trait anxiety endorsed a greater likelihood of these events actually occurring than the low trait anxiety group. The authors propose that individuals high in trait anxiety have a tendency to use their affect as information, thus suggesting that negative affect might influence attributions.

While much of the research examining the relationship between attributions and affect has taken place outside the realm of aggression research, there is a limited literature focusing explicitly on both attributions and affect within the context of aggression. For example, in a review of the literature examining the original frustration-aggression hypothesis set forth by Dollard et al. (1939), which posits that “Aggression is always a consequence of frustration” (p. 1), Berkowitz (1989) provides a reformulation that includes attributions in this sequence of events. Congruent with the cognitive neoassociationistic model, frustration is thought to generate aggressive inclinations to the extent that it produces negative affect. According to Berkowitz (1989), the attributions people make regarding their frustration presumably determines how badly they feel. Therefore, according to this theory, attributions appear to play a causal role in subsequent levels of negative affect, which in turn influences aggression.

Looking at attributions and affect from a different perspective, Pederson (2006) examined the impact of affect on subsequent attributions and how this relationship influences aggression. Using an experimental manipulation, one group was primed to feel positive affect towards another (bogus) participant, while the other group was primed to have neutral feelings regarding their ostensible partner. Following this manipulation, participants received a provocation from the partner, who participants later had the opportunity to aggress against. Results of this manipulation demonstrated that individuals with positive affect show a tendency to make more external (i.e. less hostile) attributions and behaved less aggressively towards the bogus participant. Participants in the neutral affect condition, however, tended to make more internal (i.e. more hostile) attributions regarding their partner and aggressed at higher levels than did members of the positive affect group. Related to the present investigation, it should be noted that PSAP procedures inherently establish neutrally valenced targets, thereby possibly increasing the likelihood of evoking internal attributions regarding the confederate's behavior.

Finally, the work of Zillmann (i.e. Zillmann, 1988; Zillmann & Bryant, 1974; Zillmann, Bryant, Cantor, & Day, 1975; Zillmann & Johnson, 1973) speaks to the relationship between attributions and affect within the context of interpersonal aggression. Congruent with Schacter's (1964) two-factor theory of emotion, Zillmann posits that anger and aggression have a dual source; cognition and excitation. Expanding on Schacter's two-factor theory, Zillmann (1988) emphasizes the interaction of cognitions and arousal as they influence anger and aggression. Speaking to the impact of

attributions on affect, he notes that to the extent that anger is a function of level of excitation, anger should be influenced by the attributions one makes.

In a study examining the influence of attributions on participants' reactions to an impatient and rude experimenter, Zillmann and Cantor (1976) exposed participants to interactions between a polite experimenter and an overtly rude experimenter. In two conditions, the polite experimenter mentioned in passing to the participant that the rude experimenter was stressed about an upcoming midterm exam, and in another condition the participant received no information from the polite experimenter. Participants were then given the opportunity to retaliate against the rude experimenter by voting on whether or not he should be reappointed as a research fellow. Results indicate that participants who received mitigating information regarding the experimenter's behavior showed less physiological arousal and felt less agonistic emotion. In contrast, groups receiving no mitigating information tended to interpret the experimenter's behavior as a personal attack and subsequently showed greater arousal and negative affect. Further, those receiving no mitigating information voted against the experimenter's reappointment significantly more than participants receiving mitigating information. These findings by Zillmann and Cantor (1976) provide support for the belief that attributions influence angry affect, which in turn influences aggression.

Similarly, Zillmann and Johnson (1973) investigated the impact of cognitions on maintaining anger. Working under the assumption that provoked individuals will tend to ruminate over a provocation unless cognitively interrupted, these authors provoked individuals by punishing them with an electric shock after voicing their opinions on

controversial issues and then either exposed them to a violent or non-violent film.

Supporting their hypotheses, results indicate that provoked individuals exposed to a non-violent film showed less arousal than those exposed to a violent film, presumably because the non-violent film had disrupted the rumination on anger-perpetuating cognitions.

Turning now to the opposite side of this relationship between attributions and affect, Zillmann et al. (1975) demonstrated that very high levels of arousal can interfere with the cognitive processing that occurs following provocation. Similar to the study by Zillmann and Cantor (1976) previously discussed, Zillmann et al. (1975) provoked participants by exposing them to an impatient and rude experimenter while providing mitigating information regarding the experimenter's behavior, including an explanation that the rude experimenter was under a lot of stress with school. In addition, however, some participants engaged in strenuous physical exercise prior to receiving the mitigating information. While mitigating information has been shown to decrease anger, this effect was not observed among highly aroused individuals. In essence, high levels of arousal (resulting from exercise) appeared to negate the de-escalatory effect of mitigating information, presumably due to the cognitive deficits that seemed to materialize at high levels of arousal. These findings suggest that information might be processed differently at different levels of excitation, in turn carrying implications for those with symptoms of PTSD who characteristically process incoming information through a lens of heightened arousal.

Based on the extant literature, there does not appear to be a consensus as to whether attributions precede affect or vice-versa, and it is beyond the scope of this investigation to

resolve this debate. By providing an overview of this literature, however, we find support for the inextricable interplay between cognition and affect. To examine affect in the absence of attributions (and vice-versa) is to ignore an essential component in the study of human aggression.

PTSD and Cognition

Previous researchers have examined the relationship between PTSD and attributions from several perspectives. From one perspective, past research has examined the how attributional biases might impact one's vulnerability to developing PTSD following a traumatic event (e.g., Amir, Coles, & Foa, 2002; McNally, 1998). More relevant to the present study, however, another line of research has examined the impact of PTSD on one's attributions of threat in a given situation (e.g., Warda & Bryant, 1998). Expanding upon previous research that has examined interpretive biases for lexical tasks among those with PTSD, Elwood et al. (2007) expanded these findings to visual tasks. Comparing victims of previous interpersonal violence to non-victims, participants viewed 12 brief film clips depicting ambiguous interpersonal interactions of various emotional valences (positive, neutral, and threatening), after which they rated these clips on five questions, including how the clip will end, how confident they are in the outcome, perception of escalation of threat, speed of escalating danger, and predictability. Results demonstrated that victims perceived threatening situations as increasing in risk more rapidly than non-victims, while no differences were found between groups regarding the outcome of the ambiguous situations. Although these results only found differences

between groups for the threatening video clips, it does suggest that a history of trauma might influence the attributions one makes in an ambiguous situation.

In addition to the interpretive biases exhibited by those with PTSD noted by previous researchers, PTSD has been shown to be related to attentional biases as well. Specifically, it appears that anxiety disordered individuals process negative information more quickly than neutral or positively valenced stimuli compared to non-anxiety disordered individuals (Buckley, Blanchard, & Neill, 2000). Comparing 10 Vietnam combat veterans with PTSD to 10 combat veterans without PTSD and 10 non-combat veterans, McNally et al. (1987) implemented an auditory recognition task including Vietnam-related stress words with phonetically similar words (e.g. firefight vs. firefly) and neutral words. Measuring skin conductance levels during this procedure, only the PTSD group showed elevated skin conductance levels while identifying combat-related words. The authors suggested that a rapid stress response occurs during the automatic processing stage of cognition.

Other researchers have utilized a modified Stroop paradigm to compare the cognitive processing of those with PTSD to non-PTSD groups (i.e. Foa et al., 1991; McNally, Kaspi, Riemann, & Zeitlin, 1990). Examining 15 rape victims with PTSD to 13 rape victims without PTSD and 16 non-traumatized controls on response times to rape-specific words, general threat words, neutral words, and non-words, Foa et al. (1991) found that rape victims with PTSD took longer to respond to rape-specific threat words relative to the other categories. In contrast, the two non-PTSD groups did not show differential response times among the different types of words.

Comparing 15 Vietnam veterans with PTSD to 15 veterans without PTSD on a Stroop interference task, McNally et al. (1990) exposed participants to positive, neutral, PTSD-related, and obsessional words. Similar to Foa et al.'s (1991) findings, only the PTSD group evidenced delayed responding to PTSD-relevant words. This effect has been replicated with 30 Vietnam combat veterans compared to 30 non-PTSD combat veterans with slightly different words (Kaspi, McNally, & Amir, 1995). Taken together, these results suggest that individuals with PTSD show an attentional bias towards threatening cues compared to non-PTSD individuals. While these results seem to suggest that PTSD leads to altered information processing, methodological limitations prevent any conclusions regarding the directionality of this relationship.

PTSD and Affect

While not included in the diagnostic criteria, the DSM-IV-TR (APA, 2000) cites impulsivity as an associated feature of PTSD, leading some researchers to argue that antisocial tendencies among those with PTSD reflect general impulsivity (Goodwin & Guze, 1984). Examining this argument, Chemtob et al. (1994) compared three groups: Vietnam combat veterans with PTSD, Vietnam combat veterans without PTSD, and non-combat Vietnam era veterans with other psychiatric diagnoses. These authors found that combat veteran with PTSD scored significantly higher than the other two groups on several measures of anger, leading them to conclude that “veterans who have PTSD appear to be an extremely angry group of individuals” (p. 831). This finding is relevant to the present study, as anger as one form of negative affect that may increase the risk of

aggression, appears to be associated with PTSD. This finding was corroborated by Novaco and Chemtob (2002), who found that anger accounted for over 40% of the variance in PTSD symptomatology among combat veterans when controlling for age, education, and combat exposure. While the groups differed significantly on measures of anger, they did not differ on impulsivity factors, suggesting that impulsivity cannot account for the increased anger consistently found among veterans with PTSD (Chemtob et al., 1994).

Expanding on the work of Chemtob et al. (1994), Freuh et al. (1997) utilized a multimeasure assessment strategy to examine the link between PTSD and anger with a sample of combat veterans, with three measures of anger and two measures of PTSD. Results not only provided support for the relationship between PTSD and anger, but also showed that anger varies as a function of employment status, such that unemployed veterans with PTSD were particularly likely to evidence elevated levels of anger. Freuh et al. (1997) conclude that not only are veterans with PTSD particularly likely to show elevated anger, but they are also likely to show difficulties modulating their expression of anger. This unmodulated discharge of anger is congruent with previous research documenting the relationship between PTSD and aggression (i.e. Kulka et al., 1988).

Making the connection between combat-related PTSD symptomatology, anger, and interpersonal violence, Taft et al. (2007) found that veterans with elevated PTSD symptoms reported higher state anger across experimental conditions as well as higher levels of intimate partner violence compared to veterans without elevated PTSD symptoms. During an experimental procedure, participants were presented with either a

neutral (10 minutes of classical music) or trauma-related (10 minutes of combat-related sounds, including helicopters, gunfire, and Vietnamese voices) auditory prime. In addition to demonstrating higher anger across situations, participants with elevated PTSD symptomatology showed greater anger reactivity, defined as a greater increase in anger following the trauma-related auditory prime. Further, trait anger was found to mediate the effects of PTSD symptoms on physical assault, suggesting that trait anger represents a pathway through which PTSD symptoms lead to aggressive behavior.

In a longitudinal study of 20 female victims of rape, Kilpatrick, Resick, and Veronen (1981) assessed various aspects of victims' mood states at 1 month, 6 months, and 12 months post-assault. Relevant to the trauma-violence association, these authors found that victims showed greater hostility and anger than non-victims at each assessment period.

In an effort to synthesize these findings across trauma types, Orth and Weiland (2006) conducted a meta-analysis of 39 studies with trauma-exposed adults. As expected, studies consistently showed significant correlations between various trauma types with anger and hostility. Related to trauma type, samples with military combat experiences showed the strongest association with anger ($r = .56$), and the weakest association was found in samples experiencing criminal victimization ($r = .30$). All other trauma types yielded effect sizes (r 's) ranging from .43 to .48. The authors were appropriately quick to note, however, that these results do not indicate a causal relationship between combat-related trauma and anger, highlighting the possibility that those entering the military likely differ from those in the general population prior to the trauma. While Orth and

Weiland's (2006) meta-analysis provides impressive support for the association between PTSD and anger, the present study aims to advance this literature by examining anger and attributions as potential moderators of the PTSD symptomatology-aggression link.

In addition to finding support for the association between trauma and aggression, Orth and Weiland (2006) conducted two moderator analyses examining the effects of the length of time since the trauma and the type of traumatic event in relation to subsequent anger and hostility. As discussed previously, individuals with PTSD evidence heightened anger, which may increase risk for aggression. Interestingly, both length of time since the trauma and type of trauma were found to explain a substantial proportion of variance in effect sizes ($\beta_{\text{time}} = .47$, $\beta_{\text{trauma type}} = .42$). Based on these correlations, the authors concluded that the strength of association between anger and PTSD is lowest immediately following the traumatic event. During the first several months following the trauma the strength of association increases strongly and subsequently decreases slowly over time. Thus, the findings of Kilpatrick et al. (1981) discussed previously may underestimate the anger and hostility present in female rape victims due to the initial assessment taking place only one month following the assault.

Related to the present investigation, this study intends to examine the PTSD symptomatology-aggression relationship by examining potential moderators of this relationship. Specifically, this project will assess cognitive attributions and negative affect within the context of a laboratory-based aggression paradigm. To this point, the previous review has highlighted the literature regarding the variables of interest, with an emphasis on the PTSD symptomatology-violence link, the relationship between

attributions and violence, the relationship between affect and violence, and the bidirectional interplay between attributions and affect. The following section will provide a theoretical explanation for the PTSD symptomatology-violence relationship that incorporates the role of cognition and affect, thus providing a framework for the present investigation.

Theoretical Explanations Linking PTSD and Aggression

Based on the review of the literature, there appears to be substantial evidence suggesting a relationship between symptoms of PTSD and aggression. In addition, research indicates that the meanings people ascribe to events may play a role in their aggressive behavior and that negative affect appears to be related both to aggression as well as cognitive attributions. While a majority of the research linking PTSD to aggression has been correlational and largely atheoretical, one theory appears to be particularly relevant to better understanding this relationship and the role of cognition and affect in moderating the relationship.

Chemtob et al. (1997) proposed a theory of *survival mode processing* to describe the PTSD-aggression relationship. The theory suggests that individuals with PTSD evidence a substantially lowered threshold for perceiving situations as threatening. As a result, perceptions of threat activate both fear (i.e. flight) as well as anger (i.e. fight) responses. Given Zwemer and Deffenbacher's (1984) position that anger is of a more positive emotional valence (and is therefore preferable) and is incompatible with fear, the theory contends that individuals with PTSD enter survival mode processing in response

to perceived threat that potentiates anger rather than fear responses. Further, “survival mode” preempts other cognitive processes (e.g. searching for alternative explanations), thus detracting from one’s usual mental capacities to regulate affect and inappropriate behavior. Within this model, cognitive, affective, and behavioral domains are discussed in terms of their reciprocal relationships with one another. Recognizing that anger is often a necessary but not sufficient condition leading to aggressive behavior, Chemtob et al. (1997) describe the cognitive domain as playing a pivotal role in the regulation of arousal and anger. Previous research, however, has indicated that strong arousal can impair cognitive processing, thus decreasing cognitive control over behavior (Hamilton & Wartburton, 1979). According to Zillmann (1972), people draw causal attributions regarding their emotional state based on salient cues in their immediate environment. The excitation felt in a given situation may involve “residues from incompletely decayed, unrelated antecedent arousal” (p. 247) which in turn serves to intensify the presently felt emotion. Relevant to those with PTSD symptomatology, persistent arousal may serve to hinder the cognitive regulation of aggressive behavior due to faulty processing of interpersonal cues. Put another way, the residual arousal characteristic of PTSD might serve to intensify feelings of threat in everyday situations, thereby potentiating aggressive behavior.

As a real world example, a man and his girlfriend were arguing with raised voices with one another, eliciting a state of emotional arousal in the man due to the intrusions of traumatic memories. Based on his family history of witnessing domestic violence, he re-experiences the danger he had experienced growing up in the present confrontation. From

his perspective, his aggressive challenge to his girlfriend probably seems fully appropriate. To others, however, this man's response may seem excessive; as though he had "jumped the gun" in escalating this verbal altercation. Within Chemtob et al.'s (1997) theory, the emotions elicited in him by this altercation overwhelmed his cognitive processes that might otherwise have informed him that this situation was distressing but not physically threatening. In other words, the activation of this man's survival mode processing impeded his ability to accurately interpret the social stimuli involved in this situation.

While previous research has conceptualized attributions as a mediator in the relationship between trauma and aggression (i.e. Dodge et al., 2005), Chemtob et al.'s (1997) survival mode processing theory conceptualizes attributions and negative affect as moderators in the PTSD-aggression relationship. Specifically, within this theoretical framework, any component of the threat system is capable of activating its respective counterpart in the anger system. Therefore, it is possible that one's threat seeking structure can directly activate one's aggressive motor scripts in the absence of activating anger arousal or anger-related cognitions. Although this direct activation of aggressive motor scripts in the absence of cognitive or affective components suggests that the relationship between PTSD and aggression is not explained by attributions or negative affect, Chemtob et al. (1997) suggest that the combination of these variables makes it particularly difficult for those with PTSD to inhibit their aggressive motor scripts.

Utilizing the framework provided by Chemtob et al.'s (1997) survival mode processing theory, we are better able to see the possibility of individuals with PTSD

being particularly susceptible to having either anger or fear activated when they perceive a threat. As it relates to the present study, it is our intention to utilize the conceptual framework provided by survival mode processing theory to examine the moderating role of negative affect and cognitive attributions in the PTSD-aggression relationship.

Rationale for the Proposed Study and Hypotheses

Based on the individual and societal costs of interpersonal violence, the need to identify risk factors is evident in order to combat this prevalent social concern. While the link between PTSD and violence has been established by numerous researchers across multiple trauma types, the potential intervening influences on this relationship have been under-researched. In order to identify potential treatment targets for reducing violence, it is imperative to move beyond the distal influence of PTSD symptomatology on aggression to the more proximal variables. The present investigation will examine the potential moderating role of cognitive attributions and affect in the PTSD symptomatology-violence relationship.

In order to assess these variables of interest, a laboratory-based aggression paradigm, the Point Subtraction Aggression Paradigm (PSAP), in addition to multiple self-report measures will be used with a sample comprised of individuals with high and low PTSD symptomatology. The PSAP procedure requires participants to participate against an ostensible competitor in a computer-based task in which they attempt to earn as much money as possible. While attempting to earn money, however, participants are confronted by having their money subtracted by this competitor (actually by the PSAP

program at predetermined intervals). Recalling Baron and Richardson's (1994) definition of aggression described previously, we see that this definition speaks to the intentionality of the aggressor. While many lab-based aggression paradigms suffer from the necessity of drawing inferences as to the aggressors' motivations, we believe that concurrently examining attributions about the intentionality of the aggressor as well as aggressive behaviors addresses this potential problem and allows us to more confidently conclude that behavior defined as aggressive (be it button-pressing, the administration of hot sauce, or any other behavior operationalized as aggression) represents a valid form of aggression.

Individuals from high and low PTSD symptomatology groups, recruited based on PTSD scores, will be compared on their aggressive responding in response to the provocation of having money subtracted by a competitor, after which they will complete measures assessing their affect and attributions during the PSAP procedure. Based on Chemtob et al.'s (1997) survival mode processing theory, the present study tested five hypotheses:

- 1) Individuals with elevated PTSD symptomatology would show more negative affect, endorse more negative intent attributions, and behave more aggressively in response to provocation than those with low PTSD symptomatology.
- 2) Individuals who make more negative intent attributions and report more negative affect will behave more aggressively in response to provocation

than those who make less hostile attributions and report experiencing less negative affect.

- 3) Negative intent attributions will moderate the relationship between PTSD symptomatology and aggression. Specifically, individuals with elevated PTSD symptomatology who also show more negative intent attributions will be more likely to engage in aggression in response to provocation compared to those with elevated PTSD symptomatology who do not show negative intent attributions (no differences were expected based on level of negative intent attributions for those low in PTSD symptomatology).
- 4) Negative affect will moderate the relationship between PTSD symptomatology and aggression. Specifically, individuals with elevated PTSD symptomatology who also show more negative affect will be more likely to engage in aggression in response to provocation compared to those with elevated PTSD symptomatology who do not show negative affect (no differences were expected based on level of negative affect for those low in PTSD symptomatology).
- 5) Negative intent attributions and negative affect will moderate the relationship between PTSD symptomatology and aggression. Specifically, individuals who make more negative intent attributions and show more negative affect who evidence elevated PTSD symptomatology will engage in more aggression in response to provocation than those with either lower negative intent attributions or negative affect with elevated PTSD

symptomatology or those with lower negative attributions and less negative affect who show elevated PTSD.

Method

Phase 1

Participants. Phase 1 included 628 undergraduates recruited from the university's online subject pool at a large Southeastern state university.

Measures. The Posttraumatic Stress Disorder Checklist (PCL) is a 17-item self report measure used to assess symptoms consistent with PTSD on a 1-5 Likert scale, with responses potentially ranging from 1 (not at all) to 5 (extremely). It has shown high internal consistency as measured by Cronbach's alpha coefficients of .94, .85, .85, and .87 for the PCL total, re-experiencing, avoidance, and hyperarousal scores, respectively (Ruggiero, Del Ben, Scotti, & Rabalais, 2003). Further, high correlations (i.e. $r > .75$) between PCL total scores and two other well-established measures of PTSD (the IES and MS-C) suggest adequate convergent validity and test-retest correlation coefficients of .92 ($p < .001$) suggest high reliability. Previous research suggests an optimal cut-off score of 30 to obtain maximum sensitivity and specificity among individuals in the general population (Walker et al., 2002).

Procedure. Participants from the university's subject pool completed the PCL to determine eligibility for participation. This screening measure was completed online at a website utilizing encryption technology to protect confidentiality. Participants scoring 30 or above on the PCL were placed in the High-PTSD group, and a control group was identified through random selection from those reporting few PTSD symptoms (i.e., Low-PTSD) in the past month. Based on these eligibility criteria, participants were contacted by researchers via email to participate in phase 2 of the study.

Phase 2

Participants. Phase 2 included 28 male and 67 female undergraduates who met eligibility criteria determined during phase 1, after four participants were excluded from the High-PTSD group because they reported never experiencing a traumatic event. Participants' average age was 18.96 years old and ranged from 18-39. Participants' ethnic backgrounds included 70.7% Caucasian, 17.2% African-American, 4% Asian, 4% Hispanic, and 1% Indian/Middle-Eastern.

Measures. A demographics questionnaire (See Appendix H) was used to obtain information such as age, academic level, family background, and intimate relationship history. The Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) is a 53-item self-report inventory used to assess symptoms associated with a wide range of psychological disorders and provides nine primary symptom dimensions and three global indices of distress. Responses could range from 1 (not at all) to 5 (extremely). Coefficient alphas of the nine symptom dimensions have been found to range from .71 to .85 and test-retest correlation coefficients ranged from .68 to .91 (Kellett, Beail, Newman, & Frankish, 2003), suggesting sound psychometric properties.

To assess history, type and frequency of trauma, the Trauma History Questionnaire (THQ; Green, 1996) was administered. This 24-item measure examines experiences with potentially traumatic events such as crime, general disaster, and sexual and physical assault using a yes/no format. For each event endorsed, participants were asked to provide the frequency of the event as well as their age at the time of the event. Information regarding the perpetrator was also assessed for relevant items. The THQ has

shown moderate to high test-retest reliability (Mueser et al., 2001). Kappas for traumatic events that occurred more frequently (i.e. in more than 20% of the sample) ranged from .57 (physical attack without a weapon) to .89 (natural or human-made disaster). However, kappas were low for the two less frequently endorsed categories (.36 for life-threatening illness and .43 for close friend or relative being killed). Also, the THQ demonstrates strong interrater reliability, with kappas ranging from .76 (sexual assault) to 1.0 (accidents and witnessing killing/serious injury of other).

In order to measure various aspects of violence perpetration, family environment, and attitudes relevant to violence, a variety of measures were used. The Revised Conflict Tactics Scale (CTS-2; Straus et al., 1996) was given to assess positive and negative relationship tactics used to resolve conflict within the context of intimate relationships and measures the behavior of the respondents as well as their partners. This 78-item measure contains five subscales: Negotiation, Psychological Aggression, Physical Assault, Sexual Coercion, and Injury. Participants not currently involved in an intimate relationship responded based on their most recent relationship. The CTS-2 has shown high reliability in cross-cultural samples, with alpha correlation coefficients ranging from .74 to .89, and has shown acceptable correlations with other relevant measures in a sample of university students from 17 countries, supporting adequate construct validity (Straus, 2004).

Attitudes specific to the acceptability of partner violence were assessed with the 12-item Intimate Violence Subscale of the Attitudes Towards Violence Scale (ATV; Velicer, Huckel, & Hansen, 1989), and responses could range from 1 (*not true*) to 7 (*very*

true). The ATV has high internal consistency with a reliability coefficient of .81 (Funk et al., 1999).

The General Violence Conflict Tactics Scale (CTS-G; Stuart et al., 2003) was used to assess non-intimate violence. This measure assesses the frequency of physical aggression during the past year towards adult friends, bosses, co-workers, adult relatives, acquaintances, strangers, police officers, gang/groups, and others. The Parent-Child Conflict Tactics Scale (CTS-PC; Straus, Hamby, Finkelhor, & Runyan, 1998) assessed the frequency of mother-to-child and father-to-child family of origin violence victimization across five subscales. Overall, the CTS-PC evidences modest to poor reliability across the Physical Assault Scale ($\alpha = .55$), Psychological Aggression Scale ($\alpha = .60$), Nonviolent Discipline Scale ($\alpha = .70$), and Neglect Scale ($\alpha = .22$). The extremely low α ($-.02$) for the Severe Physical Assault Scale may be due to the fact that it measures rare events, resulting in an extremely skewed distribution which lowers the correlations which factor into the α value (Straus et al., 1998).

In addition, the 16-item Family of Origin Violence Questionnaire (FOVQ; Holtzworth-Munroe, Meehan, Herron, Rehman, & Stuart, 2000) assessed the frequency of witnessing family of origin violence from mother-to-father and from father-to-mother. This measure is comprised of items from the CTS2 (Straus et al., 1996) involving the frequency of interparental violence ($\alpha = .86$ for father-to-mother violence, $.65$ for mother-to-father violence) as well as items from the CTS-PC (Straus et al., 1995) to report the number of abusive-neglectful parental behaviors experienced as a child ($\alpha = .92$ for father-to-mother, $.87$ for mother-to-father) (Holtzworth-Munroe et al., 2000).

The Positive and Negative Affect Schedule (PANAS-X; Watson, Clark, & Tellegen, 1988) will assess the participant's affective state following the PSAP procedure. The PANAS-X is a reliable measure, with internal consistency reliabilities (Cronbach alpha's) of .89 for the Positive Affect Scale and .85 for the Negative Affect Scale (Watson, Clark, & Tellegen, 1988). It has also been found by these authors to correlate adequately with other measures of distress, such as the Beck Depression Inventory and State Trait Anxiety Inventory, as well as evidence convergent and discriminant validity. Responses on this 24-item measure could range from 1 (*very slightly or not at all*) to 5 (*extremely*).

To assess attributions regarding the meanings and motivations participants ascribe to the fictitious competitor, participants will complete the PSAP Attributions Questionnaire. This measure was developed by the researchers based upon widely used measures of interpersonal attributions, including items from the Relationship Attributions Measure (RAM; Fincham & Bradbury, 1992) and the Negative Intentions Questionnaire (NIQ; Holtzworth-Munroe & Hutchinson, 1993). Possible responses range from 1 (*not at all*) to 6 (*extremely*). From the PSAP Attributions Questionnaire, a Negative Intent Attributions subscale (NIA; $\alpha = .83$) was created from items 1, 2, 5, 6, and 9.

Procedure. Phase 2 was comprised of participation in a web-based survey where participants completed the aforementioned measures, as well as a lab-based aggression paradigm followed by two self-report measures. When participants arrived in the laboratory they were given an informed consent form to review. While reading this form a confederate entered indicating he/she was there to participate in the study. This

increased the likelihood that participants would believe they were indeed competing against a “real” person. The researcher informed the participant and confederate that they would be competing in a computer task to earn money in adjacent rooms through networked computers, at which point the confederate was escorted to the adjacent room. Wires running from the participant’s computer through the ceiling contributed to this deception. After completing the informed consent form, the participant was given a handout with instructions for the PSAP task. At this point the researcher went to the adjacent room, ostensibly to review the informed consent and PSAP instructions with the other participant.

After several minutes the researcher returned to the participant and answered any questions regarding the computer task and informed the participant that the fictitious opponent was ready to begin. After beginning the procedure, the researcher left the room to monitor the progress of the participant via a networked computer monitor. By adopting this procedural adaptation from Kortynk and Perkins (1983), the risk of potential demand characteristics was minimized.

The PSAP (Cherek, 1992) procedure is a 25-minute computer-based task in which the participant ostensibly competes against another participant, who is actually a confederate. The PSAP is a free operant procedure that employs a 3-button response panel that corresponds to the letters A, B, and C presented on a computer monitor. By pressing button A, the participant earns \$0.10 on an FR-100 schedule. By pressing button B, participants can subtract \$0.10 from the fictitious opponent on an FR-10 schedule. Button B responses serve as the dependent measure of aggression. This operationalized

definition is congruent with Baron and Richardson's (1994) definition of aggression in that it is directed towards harming the other person, through monetary subtractions, and this other person would be motivated to avoid this treatment. Button C responses, also set on an FR-10 schedule, serve to protect the participants' earnings for a variable period of time from point subtractions by the fictitious opponent. While participants are led to believe that they are competing against somebody else, point subtractions actually occur on a predetermined basis. These predictable point subtractions serve as provocation for the participant. The PSAP has demonstrated external validity (Cherek, Moeller, Schnapp, & Dougherty, 1997) as well as group discrimination (Moe, King, & Bailly, 2004), lending support to the study of human aggression under highly controlled laboratory conditions.

While there are a variety of measures for assessing aggression, the PSAP has several unique strengths. First, many measures employ a retrospective self-report format which may be influenced by other variables. Among other potential confounds, past research has shown that people tend to underreport their own aggression (Gregoski, Malone, & Richardson, 2005), which may be due in part to issues of social desirability (Harris, 1997). In contrast, the PSAP examines aggressive responding to current provocation. Put simply, the PSAP examines behaviors rather than asking about them.

Comparing the PSAP to other laboratory-based aggression paradigms, we find that the PSAP has evolved to address concerns that have arisen over time. For instance, in an influential critique of the field, Tedeschi and Quigley (1996) noted that many laboratory-based paradigms did not offer a non-aggressive response alternative. In recent

years, the PSAP has added a third (C) button, serving as a second non-aggressive response option. Next, Tedeschi and Quigley (1996) noted that many laboratory-based paradigms might be influenced by demand characteristics. In response to this criticism, Kortynk & Perkins (1983) slightly modified the PSAP procedure by leaving the participant alone in the room to circumvent this problem. Finally, Tedeschi and Quigley (1996) contend that we cannot necessarily infer that the operationalized definitions of aggression in the existing paradigms are in fact measuring aggression because there is no measure of participants' intentions or motivations. Addressing this concern, the present study will assess the attributions made by participants that ostensibly influenced their aggressive responding.

Canonically, the PSAP is administered over a series of experimental sessions until aggressive responding plateaus. While some contend that multiple sessions are necessary to accurately assess an individual's aggressive behavior (e.g. Cherek, 1992), recent researchers have questioned the utility of this approach for examining group-level differences (Golomb, Cortez-Perez, Jaworski, Mednick, & Dimsdale, 2007). Examining the validity of a PSAP-First Session (PSAP-FS) paradigm which requires only a single 25-minute session, Golomb et al. (2007) found high convergent validity for the PSAP-FS, as this paradigm correlated highly with other measures of aggression. Therefore, these authors suggest that a single 25-minute PSAP session has satisfactory construct validity properties for generating findings based on group-level data.

Upon completion of the PSAP procedure the researcher returned to the room with the participant, who then completed the PANAS-X and PSAP Attributions

Questionnaires described above. After completing these two measures, the participant was debriefed regarding the nature of the study and the deception involved. During debriefing, participants were asked whether they believed that they had been competing against the fictitious competitor in order to ensure that the cover-story was effective. Participants were compensated with extra credit in the psychology class in which they were currently enrolled and received \$10 for their participation in Phase 2.

Results

Demographic Information

As noted above, the present sample was comprised of 28 male and 67 female undergraduates from a large Southeastern state university with an average age of 18.96 years ($SD = 2.70$). Of the participants, 71.9% were freshman, 14.6% sophomores, 7.3 % juniors, and 5.3% seniors. Further, in line with regional demographics, 80.2% of the present sample were Christian, 3.1% Jewish, 2.1% Muslim, 2.1% Hindi, and 9.4% endorsed “other.” Socioeconomically, 27.1% reported annual family incomes under \$50,000, 37.5% were between \$50,000-100,000, 15.6% were between \$100,000-150,000, 9.4% were between \$150,000-200,000, and 7.3% were greater than \$200,000. In regard to dating status, 54.2% reported they were not currently involved in a romantic relationship, while 40.6% were dating, 2.1% were married, and 1.0% were widowed/divorced. Finally, 88.5% of the present sample endorsed a heterosexual orientation, with 4.1% endorsing a gay/lesbian orientation, and 2.1% endorsing a bisexual orientation.

We first examined characteristics of the PTSD groups. The mean PCL score for the 50 Low-PTSD participants ($M = 22.52$, $SD = 3.78$) was significantly lower than the mean PCL score for the High-PTSD participants ($M = 51.62$, $SD = 7.06$), $t(90) = -25.18$, $p < .001$. Multiple t-tests and chi-square analyses were conducted to examine whether High-PTSD participants differed from Low-PTSD participants on any demographic variables. Results indicate no differences for age, academic level, gender, religion, family income, sexual orientation, or relationship status based on PTSD grouping. A significant

difference in PTSD grouping based on race did emerge, $\chi^2(5, N = 95) = 10.98, p = .05$. A visual examination showed that all of the Hispanic participants ($n = 4$) were in the High-PTSD group. Analyses conducted after removing these participants did not change the results and therefore, we retained these participants in all analyses.

PSAP Responding by Gender

For the total sample, participants pressed the A button (Earn) an average of 5,388 ($SD = 1056$) times, the B button (Aggression) 247 ($SD = 182$) times, and the C button (Protect) 443 ($SD = 262$) times per session.

In regard to A-Button responding (Earning), males ($M = 6,124, SD = 997$) pressed the A-button significantly more often than did females ($M = 5,080, SD = 933$), $t(93) = -4.87, p < .001$. Participants did not engage in greater A-Button responding when competing against a male ($M = 5,247, SD = 1133$) confederate compared to a female ($M = 5,512, SD = 977$) confederate, $t(94) = -1.23, p = .22$. Examining the potential interaction between gender of participant and gender of confederate on A-Button responding, results did not yield a significant interaction, $F(1, 91) = .013, p = .91$.

No differences were found when comparing males ($M = 247, SD = 185$) to females ($M = 249, SD = 185$) on aggressive responding, $t(93) = .04, p = .97$. Further, results showed that participants' aggressive responding did not differ when competing against a male ($M = 250, SD = 158$) relative to a female ($M = 244, SD = 203$) confederate, $t(94) = -.16, p = .88$. Examining the potential interaction between gender of participant and gender of confederate, a univariate ANOVA was conducted with gender of

participant and gender of confederate as independent fixed factors and aggressive responding serving as the dependent variable. Results did not yield a significant interaction, $F(1, 91) = .21, p = .65$.

A trend approaching significance indicated that females ($M = 477, SD = 260$) engaged in somewhat more protective responding (C button) than males ($M = 375, SD = 255$), $t(93) = 1.77, p = .08$. Participants did not respond to a female ($M = 455, SD = 249$) confederate with more protective responding compared to a male ($M = 431, SD = 280$) confederate, $t(94) = .44, p = .66$. Examining the potential interaction between gender of participant and gender of confederate, a univariate AVOVA was conducted with gender of participant and gender of confederate as independent fixed factors and protective responding serving as the dependent variable. Results did not yield a significant interaction, $F(1, 91) = .52, p = .47$.

Hypothesis 1

The first hypothesis stated that High-PTSD participants would endorse greater negative affect and negative intent attributions and would respond more aggressively than would Low-PTSD participants. The data generally supported this hypothesis. Results showed that High-PTSD participants reported greater negative affect ($M = 59.89, SD = 15.63$) than Low-PTSD participants ($M = 51.06, SD = 9.19$) in response to the lab procedure, $t(94) = -3.42, p < .01$, and showed a trend approaching significance wherein High-PTSD individuals ($M = 11.36, SD = 5.49$) attributed more negative intentions to their opponents' behavior than Low PTSD individuals ($M = 9.69, SD = 4.24$), $t(94) = -$

1.68, $p = .09$. The data also indicated that High-PTSD individuals were more aggressive than Low-PTSD individuals, with High-PTSD participants responding with an average of 286.80 B-button responses ($SD = 197.88$) compared to an average of 212.02 B-button responses for Low-PTSD participants ($SD = 160.87$), $t(94) = -2.04$, $p < .05$. Results showed that the level of provocation (i.e. removal of money) was not significantly different between the two groups, $t(94) = -.67$, $p = .51$.

Hypothesis 2

The second hypothesis stated that individuals reporting more negative affect in response to the PSAP procedure would respond more aggressively than individuals reporting less negative affect. In order to examine the relationship between negative affect and aggressive responding, we first analyzed the bivariate correlation between aggressive responding on the PSAP and the 35-item negative affect scale of the PANAS-X. Results of this analysis failed to demonstrate a relationship between overall negative affect and aggressive responding, $r = -.011$, $p = .92$. Results were similar when examining this correlation within High-PTSD ($r = -.116$, $p = .45$) and Low-PTSD ($r = -.034$, $p = .81$) groups. Given the possibility that many of the items comprising negative affect might be differentially related to aggressive responding (i.e. “sluggish” versus “angry”), correlations were examined between each of the 35 items comprising the PANAS-X negative affect scale and aggressive responding. Results revealed no significant relationships between any of the negative affect items and aggressive responding (r 's ranged from $-.13$ to $.16$).

Examining the second component of hypothesis two, negative intent attributions regarding the confederate were hypothesized to be positively correlated with aggressive responding. Overall, results demonstrated that participants' negative intent attributions were unrelated to aggression, $r = -.117, p = .26$. Results were similar when examining this correlation within High-PTSD ($r = -.133, p = .38$) and Low-PTSD ($r = -.193, p = .18$) groups. Follow-up tests were conducted to assess the relationship between individual items on the attribution measure with aggressive responding. The only item that demonstrated a significant relationship, following Bonferroni adjustment for multiple comparisons, was item 1 ("The other participant intended to have a negative effect on me"), $r = -.290, p < .01$, indicating counter-intuitively that individuals who perceived their competitor as intending to have a negative effect on them engaged in less aggression relative to participants who perceived less negative intent.

Hypothesis 3

The third hypothesis stated that negative intent attributions would moderate the relationship between PTSD symptomatology and aggression. Specifically, High-PTSD individuals who also show more negative intent attributions were hypothesized to be more likely to engage in aggression in response to provocation compared to High-PTSD individuals who did not show negative intent attributions, while no differences were expected in the Low-PTSD group based on level of negative intent attributions. In order to conduct this regression analysis, negative intent attribution scores were centered and a cross-product term was created between centered negative intent attribution scores and

the dichotomous variable of High- and Low-PTSD groups. Regression analyses showed that PTSD grouping was significantly related to aggressive responding, $F(1, 94) = 4.16, p < .05$. However, the test for moderation revealed that the interaction term for PTSD groups and negative intent attributions failed to improve the model, $F(1, 92) = .106, p = .75$ (See Table 1, Appendix A). Follow-up analyses were conducted to assess the moderating role of each individual item comprising the attributions scale. The results of these 14 tests of moderation showed no significant effects for the interaction terms between participants' PTSD status and negative intent attributions.

Hypothesis 4

The fourth hypothesis proposed that negative affect would moderate the relationship between PTSD symptomatology and aggression, such that High-PTSD individuals who also showed more negative affect would be more likely to engage in aggression in response to provocation compared to High-PTSD individuals who did not show negative affect. No differences were expected in the Low-PTSD group based on negative affect. Using the same procedure as above to conduct a regression analysis, the test for moderation revealed that the interaction term for PTSD symptomatology and negative affect failed to improve the model, $F(1, 92) = .071, p = .79$ (See Table 2, Appendix B). Similar to the approach used above, individual items from the PANAS-X that were theoretically related to aggression (i.e. scornful, irritable, upset, angry, hostile, and loathing) were then examined to determine any potentially moderating items in the relationship between PTSD symptomatology and aggression. Results of this series of

linear regressions suggest that none of these items moderated the relationship between PTSD symptomatology and aggression.

Hypothesis 5

The fifth hypothesis proposed that negative intent attributions and negative affect would moderate the relationship between PTSD grouping and aggression, with High-PTSD individuals who make more negative intent attributions and evidence greater negative affect engaging in more aggression than High-PTSD individuals with lower negative intent attributions and/or negative affect. No differences were expected in Low-PTSD individuals based on level of negative intent attributions or negative affect. In order to conduct this regression analysis, negative intent attributions and negative affect scores were centered, and cross-product terms were created between PTSD grouping and centered negative intent attributions, PTSD grouping and centered negative affect, centered negative intent attributions and centered negative affect, and PTSD grouping, centered negative intent attributions, and centered negative affect scores. PTSD grouping, centered negative intent attributions, and centered negative affect was entered into step one of the regression equation, followed by the cross-product of PTSD grouping and negative intent attributions, the cross-product of PTSD grouping and negative affect, and the cross-product of negative intent attributions and negative affect in step two, and the cross-product of PTSD grouping, negative intent attributions, and negative affect in step three. Examining the potential 3-way interaction between PTSD grouping, negative intent

attributions, and negative affect on aggressive responding, results did not yield a significant interaction, $F(1, 84) = 1.231, p = .30$ (See Table 3, Appendix C).

Exploratory Analyses

While the main-effect for PTSD symptomatology on aggression is in the expected direction, the null findings for the relationships between negative affect and aggression and the non-significant trend in the opposite direction between negative intent attributions and aggression might raise questions about the validity of the PSAP paradigm. Indeed, previous research consistently supports the relationship between negative intent attributions and aggression and, therefore, the present finding that those who made more negative intent attributions showed a trend towards responding less aggressively necessitates further examination. To this end, bivariate correlations between aggressive responding during the PSAP procedure and other self-report measures of aggression (i.e. CTS, CTS-G, Velicer) were examined to assess the concurrent validity of the present lab-based paradigm. Results show that the PSAP measure of aggression was not related to any of the self-report measures of previous violent behavior or attitudes regarding the acceptability of violence (See Table 4, Appendix D).

Given that this data have demonstrated a relationship between PTSD symptomatology and aggressive responding on the PSAP, while failing to demonstrate a relationship between aggression on the PSAP and a history of self-reported violent behavior, we decided to assess the relationship between PTSD symptomatology and self-reported measures of aggression. Using the CTS-G as the primary measure of actual acts

of general violence, High-PTSD individuals showed a trend approaching significance suggesting that they engaged in more aggressive acts than Low-PTSD individuals, $t(90) = -1.83, p = .07$.

Exploratory PSAP Analyses

The role of protective responding. Because the present study utilized a three-button PSAP response panel, as opposed to the more common two-button panel, it is possible that this alteration resulted in a different profile of responding than that documented by previous research. The introduction of the “C-Button” response, which protects one’s earnings from subtractions, necessitates further analyses in order to determine what influences might play a role in one’s decision to assume a protective stance. A similar analytic plan to that applied to aggressive responding was applied to protective responding. Comparing High- and Low-PTSD individuals to one another regarding their protective responding on the PSAP, we find no differences between these groups, $t(94) = .312, p = .76$. In addition, there was no relationship between one’s level of negative affect and protective responding, $r = .015, p = .89$. Further, after Bonferroni correction, no individual items from the PANAS-X were significantly related to protective responding. However, there is evidence of a non-significant trend suggesting that one’s level of negative intent attributions is related to their level of protective responding, such that as one interprets their competitors as intending to have a negative effect on them they are more likely to respond protectively, $r = .171, p = .09$. While no single item is significantly related to protective responding following Bonferroni corrections for the 14 items comprising the attributions measure (requiring $\alpha \leq .004$),

several approached significance. Most closely approaching a significant relationship with protective responding is item 7 (“The other participant was trying to motivate me to compete harder”), $r = -.249, p = .01$. In addition, item 5 (“The other participant was trying to make me angry”) showed a relationship with protective responding prior to Bonferroni correction, $r = 1.98, p = .05$.

We next sought to examine the potentially moderating role of negative affect and negative intent attributions on the relationship between PTSD symptomatology and protective responding. Looking first at the role of negative affect on the relationship between PTSD symptomatology and protective responding, negative affect scores were centered and a cross-product term was created between these centered scores and PTSD grouping. Results showed that negative affect moderated the relationship between PTSD symptomatology and protective responding, $F(1, 92) = 4.82, p < .05$ (See Table 5, Appendix E & Figure 1, Appendix F). In order to decompose this interaction, two new variables were created to examine the role of negative affect in High- and Low-PTSD. To examine the role of negative affect in Low-PTSD individuals, Low-PTSD was dummy-coded as 0 and High-PTSD was dummy-coded as 1. To examine the role of negative affect in High-PTSD, Low-PTSD was dummy-coded as -1 and High-PTSD was dummy-coded as 0. For Low-PTSD individuals, those endorsing greater negative affect responded protectively more than Low-PTSD individuals with low negative affect, $t(93) = 2.00, p = .05$. For High-PTSD individuals, negative affect did not influence protective responding, $t(93) = -.94, p = .35$.

After finding an interaction between negative affect and PTSD symptomatology, the role of negative intent attributions in the relationship between PTSD symptomatology and protective responding was examined. Negative intent attribution scores were centered and a cross-product term was created between these centered scores and PTSD grouping. Regression analyses conducted to test for moderation revealed that the interaction term for PTSD grouping and negative intent attributions was not significant, $F(1, 92) = .55, p = .46$.

Engaged responding

Further analyses were conducted in which aggressive (B-button) and protective (C-button) responding were grouped together into a variable we labeled “Engaged Responding,” suggesting that the participant had elected to respond in a manner reflecting their engagement with their competitor (via aggression or protection), thus deviating from the strict adherence to their goal of earning money (A-button responding). Comparing High- and Low-PTSD individuals on engaged responding, no significant differences emerged between groups, $t(94) = -.83, p = .41$. Further, neither negative affect, $r = .006, p = .96$ nor negative intent attributions, $r = .069, p = .50$ were related to engaged responding. To conduct regression analyses, the sum of aggressive and protective responding served as the dependent variable, PTSD grouping was entered into step one of the equation, centered negative affect scores were entered into step two (and centered negative intent attributions scores were entered for the second regression equation), and the cross-product between PTSD grouping and negative affect (and

negative intent attributions for the second equation) were entered into step three. Results of a linear regression suggest that neither negative affect, $F(1, 92) = 3.30, p = .07$ nor negative intent attributions, $F(1, 92) = .15, p = .70$, moderates the relationship between PTSD grouping and engaged responding.

Early Trauma and Aggression

In light of previous research examining the association between childhood histories of witnessing domestic violence and heightened aggressive responding on the PSAP paradigm, further analyses were conducted examining the role of both witnessing early domestic violence as well as experiences of being the victim of childhood abuse on later aggression. Looking first at experiences of witnessing domestic violence, 27% of our sample reported witnessing at least one act of domestic violence during childhood as assessed by the FOVQ, and scores ranged from 0 to 95, suggesting a wide range of experiences among this sample. Looking at our High- and Low-PTSD groups, we see that High-PTSD individuals endorse witnessing more domestic violence ($M = 8.84, SD = 18.64$) as children than Low-PTSD individuals ($M = 1.00, SD = 3.38$), $t(91) = -2.92, p < .01$. Further, the present data showed that family of origin violence was related to aggressive responding on the PSAP, $r = .240, p < .05$. However, it appears that a history of witnessing family violence is not associated with negative affect or negative intent attributions during the PSAP procedure, $r = -.113, p = .28$ and $r = -.164, p = .12$, respectively.

Expanding upon this previous research, our data allowed us to examine whether present symptoms of PTSD moderated this observed relationship between witnessing violence in one's family of origin and aggressive responding on the PSAP. To conduct regression analyses, PCL scores were centered and a cross-product term was created between PCL scores and FOVQ scores. Results of linear regression showed that current PTSD symptomatology does not moderate this relationship between witnessing family violence and aggression, $F(1, 81) = .003, p = .96$. Breaking PTSD symptomatology into the three clusters utilized in the DSM classification, we again see that none of these factors moderates this relationship: Re-experiencing factor, $F(1, 81) = .025, p = .88$; Avoidance/Numbing factor, $F(1, 81) = .175, p = .68$; Hyperarousal factor, $F(1, 81) = .855, p = .36$.

Turning now to the relationship between a history of childhood physical abuse and aggression, High- and Low-PTSD individuals did not differ in the extent to which they were the victims of childhood physical abuse as assessed by the CTS-PC, $t(91) = -1.83, p = .07$, and a history of childhood abuse was not related to aggressive responding on the PSAP, $r = -.032, p = .76$. Subsequent analyses examined the potentially moderating role of current PTSD symptomatology on the relationship between childhood physical abuse and aggressive responding. First examining the Pearson coefficient between High- and Low-PTSD groups with aggressive responding among individuals reporting no childhood physical abuse (CTS-PC = 0), we find no relationship between PTSD group and aggression, $r = -.016, p = .95$. In contrast, there appears to be a trend approaching significance suggesting an association between PTSD grouping and

aggression for those reporting at least one experience with childhood physical abuse (CTS-PC >0), $r = .21$, $p = .06$. However, linear regression reveals that this interaction effect does not reach significance, $F(1, 89) = 1.65$, $p = .20$.

Similar to the analyses examining the role of witnessing family of origin violence and experiencing childhood physical abuse, we next sought to examine how one's trauma history, as assessed by the THQ, relates to aggression. Overall, participants endorsed a range of traumatic experiences (See Table 6; Appendix G). Examining the prevalence of traumatic experiences, 22.2% ($n = 10$) of Low-PTSD individuals report never experiencing a traumatic event, compared to none of the High-PTSD individuals who report never experiencing a traumatic event. Looking first at the relationship between one's overall trauma history and PTSD grouping, we find that High-PTSD individuals ($M = 5.42$; $SD = 5.45$) endorsed more trauma overall than Low-PTSD individuals ($M = 2.20$; $SD = 1.90$), $t(76) = -3.68$, $p < .001$. In addition, trauma history was related to other measures of emotional distress, including the BSI Global Severity Index, $r = .307$, $p < .01$, BSI Anxiety Index, $r = .323$, $p < .01$, BSI Depression Index, $r = .233$, $p < .05$, and BSI Interpersonal Sensitivity Index, $r = .273$, $p < .05$. Looking at the subscales of the THQ, we find that High-PTSD individuals have experienced more general disaster trauma (i.e. hurricanes, fires, etc), $t(83) = -3.66$, $p < .001$, and crime-related trauma, $t(91) = -2.04$, $p < .05$, than Low-PTSD individuals. While one's trauma history appears to be related to their current PTSD symptomatology, the present data showed no main effect between one's general trauma history and aggressive responding on the PSAP, $r = -.031$, $p = .78$, although general trauma history was related to aggression assessed by the CTS-

G, $r = .259$, $p = .02$. Similarly, THQ subscales showed no relationship with aggressive responding on the PSAP.

Based upon the observed relationship between one's trauma history and current symptoms of PTSD, in conjunction with the lack of main effects evident in this data between trauma history and aggression, we next sought to assess PTSD grouping as a potential moderator of the relationship between trauma history and aggression.

Regression analyses conducted to test for moderation revealed that the interaction term for trauma history and PTSD grouping failed to improve the model, $F(1, 74) = 1.47$, $p = .23$. Similar tests of moderation were conducted, with general disaster trauma, crime-related trauma, and physical/sexual trauma subscales replacing overall trauma history scores, each of which failed to improve the model, $F(1, 81) = .69$, $p = .41$; $F(1, 89) = 1.22$, $p = .27$; $F(1, 81) = .69$, $p = .41$.

Discussion

The present study sought to examine the effect of PTSD symptomatology on aggression using a lab-based aggression paradigm, as well as examine the potential moderating role of negative affect and negative intent attributions in the PTSD-aggression link. While previous research demonstrating the relationship between PTSD and violence often utilizes retrospective self-report measures of aggression with clinical samples (i.e. Castillo et al., 2002; McFall et al., 1999), the present study extended prior research by investigating this relationship with a non-clinical sample using a lab-based behavioral measure of aggression.

In line with previous findings supporting the PTSD-aggression link, the present study found that individuals high in PTSD symptomatology were more aggressive than individuals low in PTSD symptomatology using a lab-based aggression procedure. While previous research has consistently documented this relationship through retrospective self-report methods, often with male combat veterans (i.e., Kulka et al., 1988), the present study expands upon these findings in several notable ways. First, to our knowledge, it is the first lab-based aggression study to document the relationship between PTSD symptomatology and aggression, suggesting that the PTSD-aggression link is amenable to study under highly controlled laboratory conditions. Further, by examining this relationship with a non-clinical college student sample, we find further support for this relationship across genders and types of trauma beyond combat-related trauma. Finally, the highly controlled conditions of a lab-based aggression paradigm were desirable to glean a better understanding of the factors that might link PTSD to aggression.

Specifically, the role of negative affect and negative intent attributions were examined as potentially meaningful variables in the observed PTSD-aggression relationship. Consistent with expectations, those high in PTSD symptomatology reported more negative affect in response to the PSAP than those low in PTSD symptomatology. Further, there was a trend approaching significance suggesting that those high in PTSD symptomatology made more negative intent attributions during the PSAP regarding their competitor's behavior.

According to Chemtob et al.'s (1997) survival mode processing theory, individuals with PTSD tend to perceive threat more readily, in turn activating either fear (flight) or anger (fight) responses more readily than individuals without PTSD. Congruent with Chemtob et al.'s theory, the present study demonstrated that individuals high in PTSD symptomatology reported greater negative affect in response to the potentially threatening lab-based procedure, suggesting the possibility that they more readily enter into "survival mode" processing compared to those low in PTSD symptomatology. Chemtob et al.'s theory further states that individuals' cognitive, affective, and behavioral systems are reciprocally related, suggesting that people's attributions might be influenced by their affect. The trend suggesting that those high in PTSD also endorsed more negative intent attributions regarding their competitor's behavior in the present study is congruent with such a reciprocal relationship between one's cognitive, affective, and behavioral domains.

Based on a substantial amount of empirical support indicating that negative affect, including anger and depression, are related to self-reported aggression (Bjork, Dougherty,

& Moeller, 1997; Taft et al., 2007; Verona, Patrick, & Lang, 2002), we expected to find this relationship in the present lab-based study. However, the data did not support this hypothesis, as aggressive responding was unrelated to negative affect during the PSAP procedure. In addition, based on consistent support for the relationship between attributional biases and aggression noted by previous researchers (Berkowitz, 1983; Chemtob et al., 1997; Dodge, Pettit, Bates, & Valente, 1995; Lochman & Dodge, 1994; McNiel, Eisner, & Binder, 2003; Nader, Coles, & Foa, 2002), we attempted to replicate this relationship by assessing attributions immediately following completion of the PSAP task. Again, this relationship was not supported in the present sample. It is possible, however, that negative affect and attributions are, in fact, related to aggression, but only among individuals with PTSD. In order to examine this possibility, tests of moderation were conducted.

In light of the independent relationships between PTSD, negative affect, and negative attributions with aggression noted by previous research, we sought to integrate these variables using Chemtob et al.'s (1997) survival mode processing theory. According to Chemtob et al.'s model, PTSD increases sensitivity to threat, which in turn influences attributions, affect, and aggression. Due to the reciprocal relationship posited by Chemtob et al. (1997) between the cognitive, affective, and behavioral domains, we expected to see attributions and affect play a moderating role in the relationship between PTSD and aggression. However, neither negative affect nor negative intent attributions moderated the relationship between PTSD symptomatology and aggression in the present

study, nor did the three-way interaction between PTSD symptomatology, negative affect, and negative intent attributions.

The lack of significant moderation effects raises several questions. Specifically, how might we explain this discrepancy between our data and the robust literature documenting these effects? Looking first at the non-significant relationship between negative affect and aggression as well as the finding that negative affect did not moderate the PTSD-aggression relationship, it is possible that our methodology did not truly capture participants' negative affect "in the moment" because they completed the PANAS-X immediately following the PSAP procedure. In addition, although we hypothesized negative affect in general to be related to aggression, the present study might have benefited from a specific measure assessing state anger following the PSAP in order to assess this particular emotion in greater detail.

Future research in this area might benefit from including physiological measures to obtain more temporally valid measures of negative affect. Scarpa and Raine's (1997) review of the literature on the relationship between aggression and the physiology of negative emotionality suggests that some combination of skin conductance, heart rate, EEG, and event-related potential measurements might serve to further explicate the underpinnings of the relationship between PTSD and aggression. Further, Scarpa and Raine (1997) note that physiological underarousal is often associated with instrumental/proactive aggression, whereas physiological overarousal is often associated with emotional/reactive aggression. Gerra et al. (1997) have noted an increase in heart rate and systolic blood pressure during the PSAP procedure, thought to reflect

engagement by participants in this conflict simulation. As would be expected, Gerra et al.'s (1997) findings suggest that individuals with PTSD demonstrate physiological overarousal, suggesting the possibility that the PTSD-aggression relationship might be conceptualized more specifically to the domain of emotional/reactive aggression. Within the context of future research implementing the PSAP, measures of physiological arousal might elucidate overarousal to be another risk factor for aggressive responding while providing a temporally valid measure of affect in response to the lab-based procedure.

Further, the non-significant main-effect for negative intent attributions on aggression as well as the lack of moderation evidenced by negative intent attributions on the PTSD-aggression relationship was unexpected. Because it is possible that the methodology carries similar retrospective biases to those described above, it is likely that the present study would have benefited from a more temporally valid measure. Future research might involve assessing attributions verbally during the task rather than assessing them following the procedure. Another possibility might entail the use of random computer prompts throughout the task that attempt to gauge participants' attributions during the task.

In addition to the a priori hypotheses, a variety of exploratory analyses were conducted. First, we examined potential gender differences in PSAP responding. In light of meta-analytic research suggesting that gender differences in aggression may not be very large (Eagly & Steffen, 1986; Hyde, 1984), our lack of findings for differences between genders on aggressive and protective responding is not surprising. Further, Allen et al. (1996) found no gender differences on PSAP aggressive responding or protective

responding and suggested that gender differences on aggressive behavior are minimal under laboratory conditions.

Because a majority of research utilizing the PSAP has used a two-button response panel, this study's use of a three-button response panel allows for additional analyses beyond those typically addressed in the literature. Unfortunately, because there is, to our knowledge, no empirical basis for protective responding among those with elevated PTSD symptomatology, we are left only with theory to guide our analyses of protective responding. While the primary purpose of this study was to assess the influence of PTSD symptomatology on aggression, Chemtob et al.'s (1997) survival mode processing theory would suggest that some individuals with PTSD become protective (i.e., flight opposed to fight) when confronted by a threatening situation. While the mechanism underlying whether an individual with PTSD adopts an approach or avoidant style is complex and in need of future research, Dempsey, Overstreet, and Moely (2000) suggest that as behavioral avoidance decreases, physiological arousal increases, suggesting that arousal is influenced by whether one adopts an approach or avoidant style. Therefore, with individuals with PTSD who evidence an approach style, we might expect to find heightened arousal that serves to trigger survival mode processing. Conversely, it is possible that we would not observe these survival mode processes in individuals with PTSD who evidence an avoidant style due to the effects of avoidance on minimizing arousal. The present study demonstrated that negative affect moderated this relationship between PTSD symptomatology and protective responding.

Overall, the present study carries several implications. First, while previous research has consistently supported the robust association between PTSD and aggression, this is, to our knowledge, the first lab-based aggression procedure to demonstrate this effect. While some might contend that the use of a sub-clinical sample is problematic, it is intriguing that sub-clinical differences in PTSD symptomatology were still able to differentiate groups on their levels of aggressive responding. Similar findings linking sub-clinical PTSD symptomatology to aggression have also been noted by others (Taft et al., 2007). While PTSD is relatively common among college samples, the use of a symptomatology measure opposed to a diagnostic measure is a limitation to the present study. While we found participants' trauma histories to be highly correlated with PTSD symptomatology, suggesting that we were examining genuine PTSD symptoms, future research would benefit from the use of true diagnostic tools such as the Standard Clinical Interview for DSM Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1997).

Second, by virtue of utilizing the three-button response panel, the present study was able to assess both sides of the "fight or flight" response to threat. While we did not find any main effects for PTSD symptomatology on protective responding, future research would likely benefit from the inclusion of this response option in order to more fully capture the behavioral tendencies of individuals responding to a threatening situation. Finally, this research expands the empirical foundation for the PSAP in differentiating groups based on trauma and negative affect. Previous researchers have found individuals experiencing childhood physical abuse to be more aggressive on the PSAP (Moe, King, & Bailly, 2004). The present study provided support for this finding

while elucidating the independent role of PTSD symptomatology on aggressive responding. Additionally, in line with previous research (Moretti et al., 2006), the present study provided support for the relationship between witnessing parental domestic violence and aggression. Given the observed relationship between witnessing family of origin violence and PTSD, coupled with what we know about the association between PTSD and aggression, this relationship between witnessing family of origin violence and aggression is not surprising in that witnessing family of origin violence might serve as a traumatic event contributing to PTSD. We examined whether PTSD symptomatology mediated the relationship between witnessing family of origin violence and aggression, but our data did not support this. Future research is needed to more carefully examine the relationship between witnessing family of origin violence and PTSD related to aggression.

Based on the non-significant relationship between negative affect and negative intent attributions with aggression in the present study, one might conclude that the PSAP is insufficient to elicit these reactions that would influence aggressive responding. However, while the present study did not attempt to assess the qualitative reactions of participants in response to the provocation of the PSAP, informal observations of participants following the procedure seem to suggest that the PSAP was indeed provocative. Notably, many participants appeared dismayed when the researcher re-entered the room following completion of the PSAP, expressing shock at the confederates' unexpected behavior during the task. Based on this observation, future

research using visual coding during the PSAP might begin to elucidate the relationship between affect and aggression on the PSAP in a more reliable way.

Another possibility for the lack of findings regarding the role of negative affect and negative intent attributions in the PTSD-aggression relationship involves the degree of threat experienced by participants in response to the PSAP procedure. While the present study exposed individuals to the highest degree of threat available with the PSAP paradigm, it is important to remember that the manipulation of threat involves altering the schedule of point subtractions. It is possible that, even at the highest rate of point subtractions, participants are not experiencing the situation as sufficiently threatening to trigger the reactivity characterizing survival mode processing, and therefore the situation does not elicit the reciprocal effects between cognition, affect, and behavior described by Chemtob et al. (1997). Future research might attempt to modify the PSAP procedure in an effort to increase participants' perceived threat. One possibility might be the addition of instant messaging between participant and confederate during the task on an adjacent computer screen, allowing the participant to receive affectively-charged messages in real-time during the PSAP procedure.

While lab-based aggression paradigms offer the experimental control lacking in self-report measures of aggression, future research is needed to elucidate the relationship between negative affect, attributions, and aggression under these conditions. While the PSAP and other lab-based aggression paradigms lead us to infer aggression, future research would benefit from the introduction of physiological measures in order to further enhance our understanding of the underlying processes involved in these tasks. Further, it

is important to note that the PSAP does not provide sufficient output to ascertain whether the participant aggressed first or in response to being aggressed against. As such, it is difficult to determine whether some aggressive responding might best be viewed as proactive aggression, while other aggressive responses might better be conceptualized as reactive aggression. Future research might examine this potential distinction in the relationship between proactive versus reactive aggression among those with PTSD.

In summary, the present study was novel in utilizing a lab-based aggression paradigm to examine the relationship between PTSD symptomatology and aggression, as well as the potentially moderating role of negative affect and negative intent attributions in the PTSD symptomatology-aggression relationship. Using this method, we found that PTSD symptomatology was related to aggression. Further, a significant relationship between PTSD symptomatology and negative affect and a trend approaching significance linking PTSD symptomatology and negative intent attributions was evident. However, the link between PTSD symptomatology and aggression might be particularly complex, as we did not find initial support for the potentially moderating role of negative affect or negative intent attributions in this relationship. Still, this study was a vital first step in examining this complex association. Future research targeted at further specifying the PTSD-aggression relationship will be critical, and the use of lab-based aggression paradigms may hold promise.

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Appendix

Predictor Variables	R	R ²	Adj. R ²	ΔR ²	F	ΔF	β
Step 1							
PTSD-Grouping	.20	.04	.03	.04	4.17*	4.17*	.23*
Step 2							
NI Attributions	.26	.07	.05	.02	3.30*	2.37	-.20
Step 3							
PTSD x Attributions	.26	.07	.04	.00	2.21	.11	.05
<i>Note:</i> N = 96 *p < .05 Δ = change in							

Predictor Variables	R	R ²	Adj. R ²	ΔR ²	F	ΔF	β
Step 1							
PTSD-Grouping	.21	.04	.03	.04	4.17*	4.17*	.24*
Step 2							
Negative Affect	.22	.05	.03	.01	2.42	.70	-.04
Step 3							
PTSD x Affect	.22	.05	.02	.00	1.62	.07	-.06
Note: N = 96	* p < .05	Δ = change in					

Predictor Variables	R	R ²	Adj. R ²	ΔR ²	F	ΔF	β
Step 1	.25	.07	.03	.07	2.02	2.02	
PTSD-Grouping							.31 ^{**}
Negative Affect							.02
NI Attributions							-.27
Step 2	.28	.08	.01	.07	1.17	.36	
PTSD x Affect							-.09
PTSD x Attributions							.10
Affect x Attributions							.57
Step 3							
PTSD x Affect x Attributions	.31	.09	.02	.02	1.23	1.56	-.50

Note: N = 92 * p < .05 ** p < .01 Δ = change in

Appendix D

Table 4.

Bivariate Correlations Between PSAP Aggression and Self-Reported Aggression

	PSAP	Psy Abuse	Phys Assault	Sexual Coercion	Injury to Partner	ATV	BSI Hos	CTS-G
PSAP	1	.092	.023	-.011	.052	-.169	.134	-.013
Psy Abuse		1	.617**	.130	.424**	.020	.294**	.238*
Phys Assault			1	.582**	.443**	-.027	.151	.006
Sexual Coercion				1	-.034	.055	.129	.115
Injury to Partner					1	-.046	.138	.485**
ATV						1	.068	.275*
BSI-Hos							1	.261**
CTS-G								1

*p < .05 **p < .01

PSAP=PSAP B-Button Responses; Psy Abuse=CTS Psychological Abuse; Phys Assault=CTS Physical Assault; Injury to Partner=CTS Injury to Partner; ATV=Velicer Attitudes Towards Violence Scale; BSI Hos=Basic Symptom Inventory-Hostility Index; CTS-G=CTS General Violence Scale

Predictor Variables	R	R ²	Adj. R ²	ΔR ²	F	ΔF	β
Step 1							
PTSD-Grouping	.03	.00	-.01	.00	.10	0.10	11.69
Step 2							
Negative Affect	.04	.00	-.02	.00	.08	0.07	9.32*
Step 3							
PTSD x Affect	.23	.05	.02	.05	1.66	4.82*	-11.97*
<i>Note:</i> N = 96 * p < .05 Δ = change in							

Appendix F

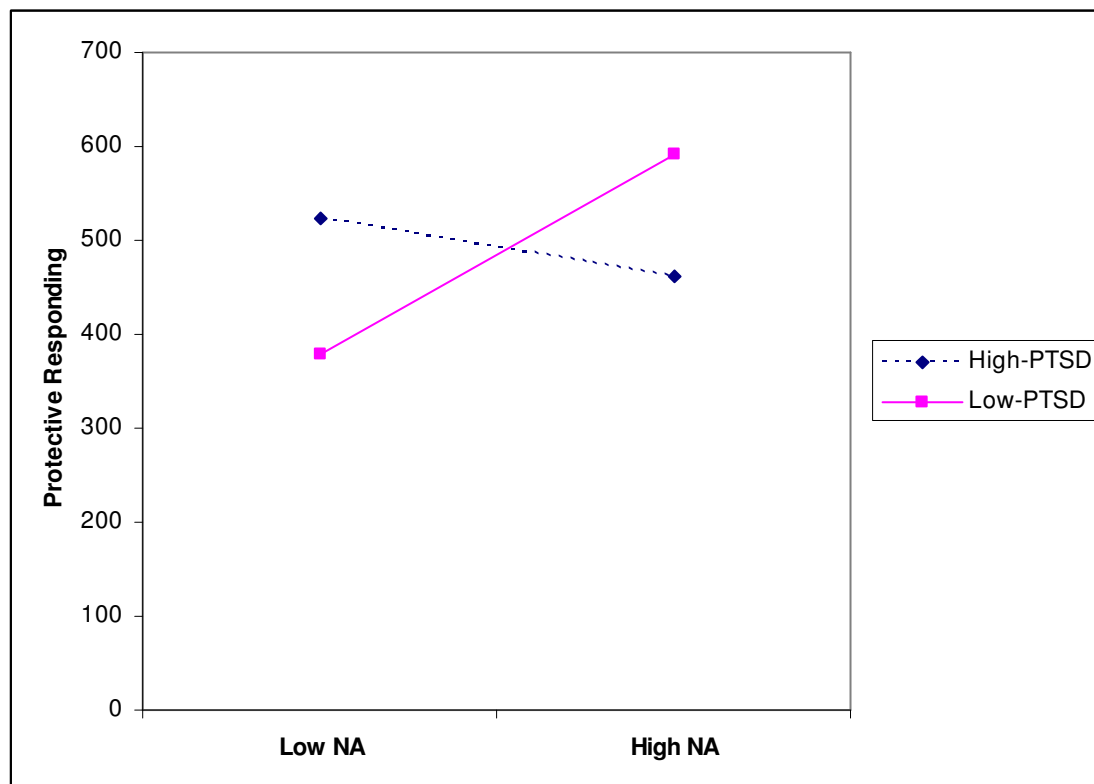


Figure 1. *PTSD x Negative Affect Interaction on Protective Responding*

Appendix G

Table 6.

Frequency of Overall THQ Trauma and Trauma Sub-Type

	General Disaster	Crime	Physical/Sexual Experiences	Total
0	16	64	58	10
1	17	20	16	10
2	21	7	8	14
3	11		2	11
4	14	1	1	14
5	3			9
6	2			3
7				1
8				3
11	1			
12				1
13				1
21		1		
32				1

Note: N = 96

THQ = Trauma History Questionnaire

Appendix H

Demographics Questionnaire

1. Age: _____
2. Sex/Gender: Female / Male
3. Academic Level: Circle One
 1. Freshman
 2. Sophomore
 3. Junior
 4. Senior
 5. Post-Baccalaureate/Graduate Student
4. Ethnic/Racial Background: Circle One
 1. White/Caucasian
 2. Black/African-American
 3. Hispanic/Latino
 4. Asian-American
 5. Native American
 6. Indian/Middle Eastern
 7. Other (please list): _____
5. Religious Background/Affiliation: Circle One
 1. Catholic
 2. Christian
 3. Jewish
 4. Muslim
 5. Buddhist
 6. Hindi
 7. Other (please list): _____
6. Family Income Level: Circle One
 1. Less than \$50,000
 2. \$50,000 - \$100,000
 3. \$100,000 - \$150,000
 4. \$150,000 - \$200,000
 5. Greater than \$200,000
7. What is your current marital status?
 1. Not dating anyone right now
 2. Dating
 3. Married
 4. Divorced/Widowed
8. If you are currently dating someone or are married, how long have you been with this person?
 *Total number of MONTHS together _____
9. Sexual Orientation: Heterosexual / Gay / Lesbian / Bisexual
10. If you answered "bisexual" to question 9 AND you are currently dating someone, what is the gender of your current dating partner? Male / Female

Vita

Aaron Kivisto graduated from Augustana College in Rock Island, Illinois in 2004 and received a B.A. in psychology. In 2006 he entered the doctoral program in clinical psychology at the University of Tennessee, Knoxville. Since 2006 he has worked as a graduate research assistant under the supervision of Dr. Todd Moore studying aggression, intimate partner violence, and the role of substance use in intimate partner violence. In addition to his research pursuits, Aaron has been working as a graduate student clinician at the University of Tennessee Psychological Clinic from 2007-present.