

PSYCHOPATHY, SADISM, AND UNPROVOKED AGGRESSION: PLEASURE AS
INTRINSIC MOTIVATION TO AGGRESS

by

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(Under the Direction of Amos Zeichner)

ABSTRACT

Although psychopaths represent one percent of society, they are responsible for nearly fifty percent of all crime and are five times more likely to commit future violent offenses. Research has demonstrated that individuals who display psychopathic traits engage in a more violent and “cold-blooded” pattern of behavior. The present study replicates previous research that showed psychopathy levels predicted a greater probability of engaging in aggressive behavior prior to being provoked. It further expands upon this research by attempting to identify the affective motivation of the unprovoked aggressor and how this emotional experience is linked to the psychopathy-aggression relationship. Two models of association were tested in which emotion processing either mediated the relationship between psychopathy and unprovoked aggression or, alternatively, moderated the relationship between psychopathy and unprovoked aggression. Results supported a moderated model in which Happiness and Sadness Facilitation associated with violence interacted with psychopathy to predict a significantly greater probability of unprovoked aggression. Results are discussed in terms of classification of psychopathy typologies and utility for predicting violent behavior.

INDEX WORDS: Psychopathy, Emotion Facilitation, Sadism, Unprovoked Aggression.

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DEDICATION

This work is dedicated to all those individuals who want more out of life and go get it.

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CHAPTER 1

INTRODUCTION

In 2005, the Department of Defense reported 2,158 coalition soldiers had been killed between March 20, 2003 and December 19, 2005 in the war in Iraq. In 2000, an estimated 1,561 youths under the age of 18 were arrested for homicide which comprised nine percent of all homicides reported that year (Fox & Zawitz, 2002). A 2001 national survey of adolescents in grades 6 through 10 reported that 13 percent of students acknowledged bullying their peers, 11 percent endorsed being a victim of bullying, and an additional six percent said that they had been both a victim and a perpetrator of bullying (Nansel, Overpeck, Pilla, Ruan, Simons-Morton & Scheidt, 2001). In 2003, students between the ages of 12 and 18 were victims of approximately 740,000 violent crimes while nine percent of all students were threatened or injured with a weapon on school property (DeVoe et al., 2004). The National Child Abuse and Neglect Data System reported an estimated 1,400 child fatalities in 2002 resulting from abuse or maltreatment (Department of Health and Human Services, 2004). According to the American Psychological Association's Task Force on Family Violence (1996), nearly one in three adult women will experience at least one physical assault by a partner during adulthood. Between 50,000 and 100,000 lesbian women and as many as 500,000 gay men are battered each year (Murphy, 1995). In 2004, the Federal Bureau of Investigation announced that approximately 1.4 million violent crimes (i.e., robbery, forcible rape, murder, and assault) were reported to law enforcement agencies. Of these crimes, aggravated assault comprised 62.5 percent of violent offenses (Federal Bureau of Investigation-Uniform Crime Reports, 2004).

These empirical data, coupled with knowledge that there are numerous unreported violent crimes each year, confirm the pervasiveness of these deleterious phenomena in our society. It is a ubiquitous problem that has existed as long as humankind and it is evidenced in our historical and religious texts that detail the innumerable wars that have occurred among, between, and within nations. Our schools teach of the genocide committed by political leaders such as Mao Ze-Dong (49 million murdered), Joseph Stalin (13 million murdered), and Adolph Hitler (12 million murdered). We are educated about the numerous “holy wars” and crusades that were waged in the name of God, and we learn of the rebellion against the British government, a process that gave birth to this nation. These historical examples and empirical studies (e.g., Gondolf & Shestakov, 1997; Kageyama, Hara, Ishii, & Hasegawa, 1996; Painter & Farrington, 1998) aver that although American society may often be criticized for being a violent society and rife with crime, these phenomena are not exclusive to our culture. Perhaps those that strike us as the most horrific and, probably, most salient acts, are the instances of violence committed by one individual against another. Aggressive and violent behavior is a phenomenon that necessitates scientific examination, and neglect of this endeavor is at society's peril.

Aggression

The scientific investigation of aggression only began one century ago. The first attempt to define this behavior was proffered by Dollard, Doob, Miller, Mowrer, and Sears (1939) when they defined *aggression* as any sequence of behavior, the goal of which is to injure the person toward whom it is directed. Bandura (1973) expanded this definition by including any behavior that results in injury, which can be in the form of physical or psychological (e.g., degradation, devaluation, imposed fear), or results in the destruction of property. However, these definitions did not address the intent of the actor. For example, under this definition, doctors who perform

invasive procedures would be considered aggressive. Additionally, accidental harm would not be excluded under this definition and, therefore, an individual who bumps into another on a crowded street, knocking him to the ground, would be labeled as behaving aggressively.

Berkowitz (1993a) attempted to resolve this discrepancy by operationalizing aggression as “any form of behavior that is intended to injure someone physically or psychologically” (p. 22).

During the past century, numerous theorists have offered different conceptualizations of this phenomenon to describe aggression solely in terms of the behavior. Upon including assumptions about the provocation that may trigger aggression, the attributes of the provocateur as well as the aggressor and the emotional concomitants of the act were also examined (Bandura, 1973).

Anderson and Bushman (2002) consolidate and refine these theories to define human *aggression* as “any behavior directed toward another individual that is carried out with the proximate intent to cause harm, and during which, the perpetrator must believe that the behavior will harm the target, and the target is motivated to avoid the behavior (p.28).” They further define *violence* as aggression that has infliction of extreme harm as its goal (e.g., physical injury or death). All violence is considered aggression, but aggression may be present in the absence of violence as is the case of verbal aggression and indirect aggression. In the present discussion aggression and violence are used interchangeably to refer to only violent acts of aggression.

The evolution of the definition of aggression and different approaches of conceptualizing it, are likely a by-product of evolving theoretical paradigms that have been put forth in the past century. Perhaps the oldest theory in this field, *instinct theory*, was forwarded by Freud. Freud originally believed that all human behavior, including aggression, stems from libidinal drives to prolong life and reproduce (Bandura, 1973; Baron, 1977). However, Freud (1920) changed his views to a substantially more pejorative belief about behavior. He used the term *thanatos* (death

force) to refer to man's second major instinct of self-destruction and termination of life.

According to Baron (1977), Freud believed that all human behavior was a complex interplay between *eros* (life instinct) and *thanatos*. If the *thanatos* is unrestrained, life will be terminated and, therefore, through the mechanism of displacement, the energy of the death force is directed outward. Therefore, the basis of aggression is the redirection of the self-destructive death instinct toward others. The social implications of this theory reveal a negative prognosis for the prevention of aggressive acts due to the inevitability of aggression. As such, if *thanatos* is not externalized, the result is termination of the individual himself. However, Freud believed that the act of expressing some aggressive behaviors or emotions, primarily anger and hostility, reduces the destructive energy and attenuates the likelihood of more dangerous acts. This assertion has been criticized by Freud's followers (Hartman, Kris, & Loewenstein, 1949) and disputed by others (Doob & Wood, 1972; Geen, Stonner, & Shope, 1975). According to Baron (1977), Freud's assertion that the expression of aggressive acts would significantly mitigate future aggressive acts is being misinterpreted. He purports that Freud was not so optimistic and believed that cathartic effects of externalizing behaviors would be minimal and short lived.

A similar view was propagated by famed ethologist Konrad Lorenz (1966). He proposed the existence of an innate fighting instinct that is present in man as well as animals. This instinct, a result of evolution, spontaneously generates aggressive energy within an organism in a continuous and constant manner. With the passage of time, this energy accumulates until it is evoked by the presence of the necessary environmental stimuli. The stimulus threshold, necessary to release overt aggression, decreases as the level of accumulated aggressive energy increases due to passage of time. Lorenz, like Freud, believed that aggression is inevitable, and inherent in his theory is the process of catharsis. Although aggression cannot be prevented, it

can be reduced in intensity through repeated overt expressions preventing the accumulation of aggressive energy.

Although instinct theory remained extant for several decades, it never gained widespread acceptance from psychologists in the field. With the dismissal of this spontaneous and instinctual view, a more general theory arose suggesting that aggression is the result of a heightened state of arousal or *drive* that is attenuated through overt acts of aggression. *Drive theory* was formulated when Dollard and colleagues (1939) published *Frustration and Aggression*, introducing the *frustration-aggression hypothesis*. They suggest that frustration (i.e., blocking some form of goal-directed behavior) engenders an “instigation toward aggression” or aggressive drive, which facilitates aggressive behavior. According to Dollard and colleagues, frustration always leads to some form of aggression and aggression always stems from frustration. However, Baron (1977) notes several empirical studies that indicate frustration does not always lead to aggression (Berkowitz, 1969; Geen & O’neal, 1976), and additional studies show aggression may occur in the absence of frustration (Berkowitz, 1973). Miller (1941), one of the authors of the frustration-aggression model, relaxes the original constraints of the model by stating that frustration may lead to numerous behaviors among which aggression is dominant. Moreover, it is *instigation* rather than the behavior itself that is aroused by frustration. Berkowitz (1969) proposes several revisions to the frustration-aggression model the most significant of which is that frustration is not a sufficient condition for the elicitation of aggression. Aggressive cues must also be present and combine with frustration to produce an emotional state, such as anger, which will then produce a readiness to aggress (Baron, 1977). Furthermore, Berkowitz (1969) contends that highly frustrated individuals can reduce their aggressive drive only by inflicting harm upon the source of frustration. This view diverges from

Dollard and colleagues (1939) who, in their original assertion, state that the aggressive drive can be mitigated through assaults perpetrated upon individuals other than the provocateur.

As empirical research disputing the frustration-aggression hypothesis waxed, support for drive theory and its tenets began to wane. In their review of human aggression, Anderson and Bushman (2002) highlight five domain-specific theories of aggression that are paramount in guiding the bulk of current research. One of these extant theories, social learning theory, arose from its now extinct precursors, instinct theory and drive theory. According to social learning theory, aggressive behavior must be understood in terms of (1) acquisition of such behavior; (2) precipitants of this behavior; and (3) conditions which maintain the emission of aggression (Bandura, 1973). Acquisition of behavior occurs through processes of observational learning or by direct experience (Bandura, 1983; Mischel, 1973, 1999). Many youths of violent neighborhoods join gangs because they see members respected by peers, protected by peers, and able to afford luxuries that are otherwise out of reach to young men and women. When these individuals become members of a gang they learn that their peers have obtained that reverence and wealth through violence and they, too, may utilize violence to obtain these desired rewards. Studies demonstrate that incentives such as money, social status, or escape from aversive treatment by others increase the tendency of adults and children to use aggressive behaviors (e.g., Buss, 1971; Geen & Stonner, 1971; Patterson, Littman, & Bricker, 1967; Walters & Brown, 1963). As individuals directly experience the incentive to aggress, such acts become reinforced and the tendency to engage in such behavior is strengthened (Baron, 1977).

Another domain-specific theory discussed by Anderson and Bushman is proposed by Berkowitz (1989, 1990, 1993b). Cognitive neoassociation theory conceptualizes aggression as behavior triggered by an aversive event or noxious stimuli such as frustration, temperature, or

provocations (Anderson & Bushman, 2002). These aversive stimuli produce negative affect in the individual which, in turn, stimulates memories, thoughts, and motor reactions that are associated with the physiological arousal inherent in fight or flight responses. Those associations of fighting precipitate feelings of anger while the flight associations yield feelings of fear. Moreover, cues that are present during the arousal process become associated with the event and prove to be associations that may produce the same physiological arousal in response to future aversive events. This theory incorporates deliberate cognitive processing of appraisals and attributions. Individuals motivated to do so, may appraise what has caused them to experience physiological arousal and subsequent negative affect, and to evaluate the consequences of their aggressive reactions. This may result in the suppression of some aggressive behavior and the enhancement of others.

Anderson and Bushman also identify script theory (Tomkins, 1979) as one the five major current theories of aggression. Scripts are sets of particularly well-rehearsed, highly associated scenes in memory, often involving causal links, goals, and action plans (Abelson, 1981). Mosher and Tomkins (1988) explain that the most basic unit of the script is the scene, which is an actual event denoted by a beginning, an end, and an affective experience. When these scenes are organized and connected through a series of rules for interpreting, responding, and defending behavior, they are dubbed a script. These scripts are used to determine the appropriate behaviors to enact during social and interpersonal interactions. An individual selects a script that most appropriately represents a situation and then determines behavior by taking on one of the roles in that script. According to Anderson and Bushman, scripts that are frequently rehearsed become more readily accessible through the creation of new links to other concepts in memory. The more links that are generated the easier it is to access the script because the number of paths by

which it can be activated has increased. Additionally, each rehearsal or overt enactment of the script strengthens the existing links. Mosher and Tomkins (1988) argue that one script pervasive in society is the *machismo* script derived from the ideology of the warrior as a great hero. Under the guidance of this script, children learn that certain “masculine-linked” affects such as anger are seen as superior to the “inferior feminine-linked” affects of fear and distress. Furthermore, violence is viewed as a symbol of masculinity and those males who exhibit it are respected for their “manliness.”

Social interaction theory (Tedeschi & Felson, 1994) contends that aggression is a form of coercion intended to influence or change the behavior of the victim. These coercive acts can serve to achieve secondary gains such as money, sex, and services, or may influence social perceptions of the aggressor (e.g., toughness, respect, fear). The perpetrator decides to use aggression based upon the consideration of the costs of the behavior, the possible rewards of the behavior, and the probability that aggression will result in the desired outcome (Anderson & Bushman, 2002).

Zillmann (1983) proposes the excitation transfer theory which states that when two events are separated by a short latency, arousal, stimulated by the first event, may become associated with or attributed to the second event. Inherent in this model is the contention that arousal dissipates slowly and that anger may be extended over long periods of time if the arousal is attributed to that emotion. The residual arousal from one event may combine with arousal and anger experienced in a subsequent event to result in higher levels of anger. The maintenance of anger may predispose an individual to aggress even after the dissipation of arousal.

There is considerable overlap among these theories. For example, script theory could be considered a detailed and specific form of social learning theory in which scripts are developed

through observation of others and through direct experience. The neoassocialistic model incorporates cognitive associations between cues and concepts, while script theory details associations created between cognitive and physiological experiences. When we consider these converging theories it becomes apparent that in order to mitigate the pervasive and deleterious phenomenon that is aggression, we must undertake its scientific study from both a nomothetic and an idiographic perspective. Before the universal scientific principles that govern this behavior can be established, it must be understood that dispositional factors of the perpetrator, his/her characteristics and personal experiences, and the unique situational factors of each aggressive interaction play an integrative role in the occurrence of aggression. The authors of the General Aggression Model (GAM); (Anderson, 1997; Bushman & Anderson, 2001) argue that the occurrence of aggression is dependent upon both the individual differences (e.g., personality traits, attitudes, values) and situational variables (e.g., frustration, cognitive cues, pain or discomfort). The outcome (i.e., aggressive vs. constructive behavior) is determined by input (i.e., dispositional and situation variables) through interrelated routes. These routes are affect, cognition, and arousal experienced during the event as well as the consequent appraisal and decision making processes. These factors facilitate access to hostile feelings, aggressive thoughts, and physiological arousal, all of which are appraised by the individual before he or she engages in the aggressive behavior. Since its introduction, several empirical investigations have provided support for pathways contained within the GAM (e.g., Anderson, Anderson, Dill, & Deuser, 1998; Anderson & Dill, 2000; Lindsay & Anderson, 2000).

For the development and implementation of community interventions that aim to alleviate violence and aggression on a large scale, the scientific community must empirically identify person and situation factors that will engender, exacerbate, control, and maintain this

deleterious behavior. For example, the aggression-potentiating effects of situational variables like alcohol (for reviews, see Chermack & Giancola, 1997; Taylor & Chermack, 1993) and presence of aggressive cues (Bettencourt & Kernahan, 1997; Carlson, Marcus-Newhall, & Miller, 1990) have been firmly established in the research literature. These factors increase the likelihood of aggression across most settings. Also, the influence of gender of both perpetrator and victim on aggressive interactions has been reliably demonstrated to suggest that males are typically more aggressive and are targets of more aggression than are women (Bennett, Farrington, & Huesman, 2005; Bettencourt & Kernahan, 1997; Hammock & Richardson, 1992). In addition, a multitude of dispositional variables such as sexual prejudice (Parrott & Zeichner, 2005), hypermasculinity (Parrott & Zeichner, 2003), narcissism (Bushman & Baumeister, 1998), and impulsivity (Scarpa & Raine, 2000) have been studied and identified as consistent predictors of aggression. This is merely a small sample of the wealth of research that has been amassed to demonstrate the discrete influence different variables exert on aggressive behavior. However, to understand what instigates and mitigates aggressive behavior is significantly more complicated than identifying a handful of contributing variables. To prevent future occurrences of aggression, researchers must further identify the additive effects of variables and the interactive effects of the dispositional and situational variables that produce and maintain aggressive behavior. A literature search using the term *aggression* returns more than 24,000 articles that have attempted to undertake this formidable task.

Hammock and Richardson (1992) studied the relationship between aggression and dispositional variables that included the presence of an aggressive learning history, masculinity, and femininity as well as situational factors, gender of the perpetrator, gender of the victim, and provocation. The authors used a multi-factor approach to test both additive and multiplicative

models. Results suggested that multiple predictors improved prediction of aggression but did not demonstrate interactions among identified variables. Parrott and Giancola (2004) examined the interactive relations between trait anger, anger control, intoxication, and aggression. Trait anger significantly predicted aggression, but only among men who were intoxicated and reported low levels of anger control. The authors argued that anger control may be a crucial risk factor in determining whether one's anger proneness will lead to intoxicated aggression following provocation. Giancola, Helton, Osborne, Terry, Fuss, and Westerfield (2002) investigated the manner in which acute alcohol consumption and provocation interacted with gender to affect aggressive behavior. Under low provocation conditions, men were more aggressive than women, but under high provocation conditions men and women were equally aggressive. Alcohol increased aggression in men but not in women, and provocation proved to be the strongest predictor of aggressive responding. According to the authors, the results are important because they provided converging evidence that, whereas provocation and alcohol are effective in lifting inhibitions of aggression in men, only provocation seems to be an effective elicitor of aggression in women. This empirical research reveals that the composition of variables that contribute to violent interactions is extremely intricate and complicated. Moreover, it demonstrates that the study of aggression is a daunting task that can never be completely understood using only one avenue of investigation. Efficient and effective examination dictates that we begin by identifying and understanding variables with the greatest significance in the confluence of factors.

Provocation

Provocation can take the form of verbal insult, physical attack, or other noxious stimulus. It has been widely studied as it appears to be present in violent interactions and has been found to

have a large effect on aggression both inside and outside of the laboratory. Furthermore, crime statistics indicate that provocation is the major source of real-world aggression (Bushman & Anderson, 1998). Indeed, a major social determinant, and perhaps the most important determinant of aggressive behavior, is provocation (Lau, Pihl, & Peterson, 1995). Hammock and Richardson (1992) found in their study of seven different predictors of aggression that provocation proved to be the most powerful predictor, accounting for 81% the total explained variance. Its effect is well documented in the scientific literature: as the intensity of the provocation increases, so too, does the intensity of aggression with which the provoked participant responds (Baldwin & Randolph, 1982; Hoppe, 1979; Lau, Pihl, & Peterson, 1995; Santor, Ingram, & Kusumakar, 2003). Increases in provocation cause gender differences in perpetration to abate (Bettencourt & Miller, 1996; Bettencourt & Kernahan, 1997) as well as serving to weaken the inhibitory role played by the gender of the victim and to increase aggression toward women (Hammock & Richardson, 1992). Furthermore, theorists have argued that the effect of provocation is so powerful that it can suppress the influence of many personality traits that commonly inhibit aggressive behavior (Richardson, Green, & Lago, 1998).

In spite of numerous empirical studies that have affirmed the aggression-promoting effects of provocation, a dearth of empirical studies has investigated the occurrence of aggression in the absence of provocation. Perhaps this is because aggression that occurs under such conditions is significantly less prevalent and, therefore, difficult to observe in both natural and laboratory settings. According to Bushman and Anderson (1998), field investigations that examine the effect of provocation on aggression do not typically include a “no provocation” control condition. Bettencourt and Miller (1996) conducted a meta-analysis of sex differences in the effects of provocation on aggression. They were unable to identify a single field experiment

that included a “no provocation” condition. A recent literature search revealed only a handful of studies that addressed the topic of unprovoked aggression. These investigations were all conducted in laboratory setting and addressed unprovoked aggression either as a control condition or in supplemental hypotheses. In all studies, aggression was measured under the guise of providing feedback or punishment in a competitive reaction-time task. Participants competed against an ostensible opponent over multiple trials and were required to respond (i.e., provide verbal or physical feedback) to the opponent following each trial. Unprovoked aggression was measured via participants’ responses on the first trial of the interaction because it occurred prior to any indication of the opponent's intentions.

Hammock and Richardson (1992) found that masculinity and aggressive tendencies were positively correlated with unprovoked aggression. Men exhibited more overall unprovoked aggression and were more likely to enact aggression in the absence of provocation when the target was a man. In addition, femininity was negatively correlated with unprovoked aggression. Richardson, Green, and Lago (1998) examined the extent to which perspective-taking, a component of empathy, decreased aggressive but increased prosocial verbal responding in men and women. In this study, men emitted more verbally aggressive responses than women under conditions of provocation and no provocation. It is noteworthy that the authors found perspective-taking to be related to the frequency of sending positive, nonaggressive responses in the presence of aggressive provocation. Hence, high perspective-takers appear not only to inhibit aggressive behavior; they also maintain prosocial behavior when faced with aggressive provocation. Although this study assessed aggression in terms of verbal aggression and not violence, it is pertinent to the present examination. Measures of verbal aggression have been validated as comparable to other measures of physical aggression, and have been shown to be

correlated with physical aggression in naturalistic settings (Bushman & Anderson, 1998; Carlson 1989). Bushman (1995) examined the relationship among gender, the priming effect of violence in media, dispositional aggressivity, and aggressive behavior under provoked and unprovoked conditions. Results demonstrated that individuals who had been primed with a videotape of violent content and those who were higher in trait aggressiveness administered more aversive noise blasts to an ostensible opponent in the absence of provocation.

Caprara and colleagues conducted research focused on the role of personality characteristics such as irritability and emotional susceptibility related to aggression instigated by a self-esteem provocation (Caprara, 1982; Caprara & Renzi, 1981; Caprara, Renzi, Amolini, D'Imperio, & Travaglia, 1983; Caprara, Renzi, Amolini, D'Imperio, & Travaglia, 1984). Provocation was defined in terms of *instigation to aggress*. Individuals participated in a learning task where instigation to aggress was manipulated by providing either positive feedback (no instigation) or negative feedback (instigation) as threat to the participant's self-esteem. Accordingly, provocation was operationalized as a negative judgment of the subject's performance on the learning task. Next, participants participated in a learner-teacher task which required them to administer electrical shocks to a confederate for each wrong response. This line of research is important because it demonstrated the provocative effects of negative comments to an individual and how they serve to increase aggressive behavior. Ego-threat seems to play an important role in precipitating aggression and is, probably, the "most common and effective form of provocation" (Baumeister & Boden, 1988, Pg 118). It is noteworthy that in the Caprara experiments, the target of aggressive behavior was not the provocateur but an innocent person. According to Geen (1998), "Caprara has shown that personality and situational variables only interact under specific combinations of personality variables. Furthermore, he demonstrated that

dispositional irritability was the variable that most significantly moderated the relationship between provocation and aggression” (pg. 14). Additionally, these studies are important in that they demonstrate that the presence of aggressive cues (i.e., the weapons effect) increases aggression in the absence of provocation (Caprara et al., 1984), a finding in contrast to earlier research suggesting that the “weapons effect” does not occur in the absence of provocation (Berkowitz & Lepage, 1967).

The type of aggression that arises in response to provocation has been designated as *hostile* or *reactive* because it is perpetrated as a response to an affective state (i.e., anger or hostility) precipitated by the provocation. This form of aggression has also been referred to as impulsive, affective, emotional, retaliatory, or angry aggression. Berkowitz (1983, 1989) identified *hostile/reactive* aggression as a form of aggression ensuing when an individual perceives a threatening situation and responds in self-defence. This type of aggression is an impulsive, immediate reaction to an emotionally-laden stimulus such as perceived insult, embarrassment, or physical danger and is juxtaposed to instrumental aggression conceptualized by Bandura (1983) as a highly goal-driven behavior motivated by the attainment of an external reward or reinforcement. Arguably, the primary goal of hostile/reactive aggression is to inflict harm, it lacks planning or thought, and is committed while angry. In contrast, the goal of instrumental aggression is to attain secondary gain (e.g., money), it involves more planning and forethought, and is committed without anger (Bushman & Anderson, 2001). The significant demarcation between the two types of aggression is the level of control the individual is believed to have over each type (i.e., instrumental being more premeditated and hostile/reactive more automatic). Hostile/reactive aggression seems to be an innate response that nearly all individuals could exhibit under necessary conditions (e.g., provocation, heightened arousal, and anger),

while instrumental may be more pathological as it is predicated on an external impetus. It could be argued that the presence of an incentive or secondary gain, while not actually a provocation, is similar to provocation; it serves as the impetus to aggress. However, this would likely be a milder form of provocation as it would not cause the affective experience of anger and ensuing physiological arousal. Therefore, instrumental aggression would likely require aberrant or pathological behavioral inhibition processes, whereas the hostile/reactive aggression displayed in response to provocation may be judged as more socially justified.

As previously mentioned, the power of provocation to induce aggressive responding is often strong enough to suppress many response-inhibiting traits an individual may possess (Giancola, 2003; Richardson et al., 1998; Richardson et al., 1994). Therefore, under manipulated conditions, a normally nonaggressive person could be driven to enact aggressive behaviors. Provocation may even be a necessary requirement for eliciting aggression in normal, healthy individuals (Berkowitz, 1988; Caprara, et al., 1983). As such it is less common and, arguably, more pathological for an individual to demonstrate violent behavior in absence of provocation, incentive, and other priming factors such as violent media. This is the theoretical impetus for a recent laboratory experiment that investigated the influence of psychopathic traits on the engagement in unprovoked aggression (Reidy, Zeichner, & Martinez, in press). In this study, participants competed against an ostensible opponent in a competitive reaction time task that offered them the choice to administer electric shocks to an opponent after each the 30 trials. They were told that their opponent would be given the same opportunity for each trial. Participants received no shocks from the ostensible opponent until the seventh trial of the task and any individual who administered a shock during the first six trials was considered an unprovoked aggressor. The authors found that individuals who endorsed higher levels of

emotional detachment and antisocial behavior were more likely to initiate shocking during the unprovoked trials rather than refraining from aggressing until provoked. Although psychopathy has been consistently associated with aggressive and violent behavior (Douglas, Ogloff, Nicholls, & Grant, 1999; Porter, Drugge, Fairweather, Hervé, Birt, & Boer, 2000; Salekin, Rogers, & Sewell, 1996; Skeem & Mulvey, 2001), this is the first study to demonstrate that psychopathy contributes to the engagement in unprovoked aggression. However, in this investigation, the authors did not address the motivation of the unprovoked aggressor. Although it appears that psychopaths are more likely to engage in unprovoked aggression, it is not evident why they do so.

Psychopathy

McCord and McCord (1964) described the psychopath as “an asocial, aggressive, highly impulsive person, who feels little or no guilt and is unable to form lasting bonds of affection with other people and represents a major danger to society” (p. 3). The construct of psychopathy is composed of two distinct factors (Hare, 2003; Harpur, Hakstian, & Hare, 1988; Harpur, Hare, & Hakstian, 1989). The first factor, *emotional detachment*, is comprised of emotional and interpersonal aspects, including affective shallowness, absence of empathy, lack of remorse, lack of shame, superficial charm, manipulation, grandiosity, and lying. The second factor, *antisocial behavioral style*, is marked by impulsivity, aggression, substance abuse, high sensation seeking, low socialization, proneness to boredom, irresponsibility, lack of concern or plans for the future, low motivation, and early life behavioral problems and delinquency (Kiehl, et al., 2001; Patrick, Cuthbert, & Lang, 1994; Woodworth & Porter, 2002). At present, this personality disorder is believed to affect approximately one percent of the general population and approximately 20 percent of the population of incarcerated individuals. The numbers of criminal acts, especially

the most violent, are disproportionately committed by psychopathic offenders who are responsible for nearly half of the most serious crimes (Hare, 1993; Hare & Mcpherson, 1984).

One of the most notable traits of the psychopath is his stunning lack of empathy and inability to understand another's perspectives. This may be the quintessential trait that delineates these individuals from other criminals and aggressive individuals. Experts have proposed that such a deficiency may be the result of interplay between biological deficits and poor socialization (Blair, 2001, 1995; Gorenstein & Newman, 1980). Blair's *violence inhibition mechanism model (VIM)* proposes that an innate biological system exists to respond to negative affect stimuli such as cues of sadness and fear. This system is considered to be essential to the process of moral socialization whereby an individual learns to inhibit behaviors that elicit sadness or fear from another individual in the presence of third party observers. Blair argued that this mechanism is not present or is deficient in the psychopath. This, in turn, leads to the underdevelopment of morality (Blair et al., 2002). The model predicts that psychopathic individuals would display impaired recognition of sad and fearful facial expressions. This prediction has been supported by studies of children with psychopathic characteristics who display this deficit (Blair, 2001). Moreover, Blair and colleagues (2002) found that recognition of fearful vocal affect by psychopathic inmates was impaired, and that there was an association between recognition of sad vocal affect and higher scores on the Psychopathy Checklist-Revised (PCL-R). Furthermore, the amygdala has been shown to be involved in the response to fearful and sad facial expressions (Blair, Morris, Frith, Perrett, & Dolan, 1999). To date, relatively few studies have directly assessed the relationship between the amygdala and psychopathy. However, two recent neuroimaging studies have confirmed the relationship between amygdala dysfunction and psychopathy. Tiihonen and colleagues (2000) used magnetic resonance

imaging to show that greater levels of psychopathy were associated with reduced amygdaloid volume. Kiehl and colleagues (2001) compared neural responses of psychopathic and nonpsychopathic individuals during an emotional memory task where participants were required to process neutral and negatively valenced words. The authors found a reduced response in the amygdala of the psychopathic group relative to the nonpsychopathic group during the processing of negatively valenced words.

It is held that the amygdala is central to fear conditioning (Blair & Frith, 2000; LeDoux, 1995). Patrick (1994) proposed a second model of socialization, the *low-fear model*, which purports that faulty socialization in the psychopathic individuals, is due to an inability to experience fear. Studies that show impaired startle reflex potentiation in psychopaths provide evidence for a deficit in fear (Levenston, Patrick, Bradley, & Lang, 2000; Patrick, 1994; Patrick, Bradley, & Lang, 1993). These and similar findings support the argument that the psychopath's violent and antisocial behavior may be due to a dysfunctional ability to experience emotion and to recognize it in others (Blair, 2001; Kosson, Suchy, Mayer, & Libby, 2002; Patrick, Cuthbert, & Lang, 1994). This research supports the VIM and low-fear model of psychopathy and aligns with other studies that have demonstrated the moderating effect of empathy and perspective taking on the relationship between aggressive behavior and situational variables such as provocation (Richardson et al., 1998; Richardson et al., 1994). Blair underscores the importance of empathy in the development of moral socialization and warns that the absence of this disposition may lead to deficiency of the VIM. This line of research evinces a significant relationship among the ability to experience emotion, consider and understand the probable emotional experience of the target, and the likelihood of perpetrating violence. Herpertz and colleagues (2001) indicated that the psychopath's profound level of hypo-emotionality could

effectively eliminate inhibition of violence if it served a selfish function. Because the psychopath has a dysfunctional emotional experience, he cannot accurately recognize or predict emotion in others and, therefore, receives no cues indicating that behavioral inhibition is called for. Indeed, it is quite likely that the emotionally-detached psychopath is unaware of the negative experience of his victim.

Based on this theory, psychopaths would be more likely to exhibit a more violent, “cold-blooded,” and pathological form of aggression. This hypothesis is bolstered by several studies that have shown a positive relationship between psychopathy and instrumental aggression (Cornell, Warren, Hawk, Stafford, Oram, & Pine, 1996; Serin 1991). Those who engage in instrumental aggression may not be reactive to provocation. If true, this would indicate that, in such individuals, provocation is not a necessary impetus for aggression. In fact, in the context of instrumental aggression, the perpetrator is likely to maintain a larger repertoire of aggressive scripts in response to needs, desires, and social problems (Huesmann, 1998). As such, he may endorse more normatively-appropriate appraisals of violence as a means of attaining a goal. Current research affirms this contention: psychopaths evince significantly more instrumental aggression than nonpsychopaths (Woodworth & Porter, 2002). Cornell and colleagues (1996) examined the relationship between psychopathy and violence in 106 men in a medium-security state prison. Instrumental violence was operationalized as violence that was goal-driven and required planning without antecedent provocation. They found that psychopaths were more likely to have committed instrumental violence than those not likewise diagnosed. Additionally, Factor 1 scores accounted for much of the variance associated with the instrumentality of the homicides, whereas Factor 2 scores did not significantly contribute to this dimension. That is, perpetrators who evinced severe emotional detachment were predominantly those who have

committed “cold-blooded,” instrumental homicides. These findings are congruent with further data suggesting that instrumental violence is commonly associated with self-reported low levels of arousal and anger as concomitants of aggression and violence. Although instrumental aggression appears to be a different form of aggression from hostile/reactive aggression, it is not analogous to unprovoked aggression in which the perpetrator chooses to enact violent behavior absent of incentive to do so.

Psychopathy & Sadism

Sadism has been defined as the derivation of pleasure from inflicting physical or emotional pain on another person (Porter & Woodworth, 2005). The term arose in reference to Marquis de Sade, a French philosopher who attempted to document all sexual perversions in literature and provide a philosophical explanation for their existence. The consequence of his endeavor: the process of deriving sexual pleasure from inflicting physical pain on another was given his name. However, sadism no longer refers specifically to sexual pleasure but rather the enjoyment of inflicting general harm upon another (Baumeister & Vohs, 2004).

No longer a formal diagnosis in the current DSM-IV-TR, sadism has a relatively small research base from which to draw conclusions about its relationship to psychopathy (Meloy, 1997; Murphy & Vess, 2003). Nevertheless, it has long been argued to be associated with psychopathy (Meloy, 2000). Holt, Meloy, and Strack (1999) used a nonrandom sample of inmates from a maximum security prison to demonstrate that psychopaths were more sadistic than nonpsychopaths. They found that psychopaths were not only more sadistic as measured by the Millon Clinical Multiaxial Inventory’s (MCMI-II) scale 6B and the Personality Disorder Examination (PDE), but that levels of psychopathy and sadism were linearly and positively associated. Additionally, violent and sexually violent groups of psychopathic offenders did not

differ in levels of sadistic traits indicating that these traits were not specifically linked to sexual pleasure. More recently, Porter, Woodworth, Earle, Drugge, and Boer (2003) investigated the use of sadistic violence in a group of sexual homicide offenders. Thirty-eight murderers were categorized as either psychopaths or nonpsychopaths and compared in terms of sadistic violence. Psychopaths were found to use significantly more sadistic violence than nonpsychopaths during the perpetration of homicides. Moreover, the authors found that the correlations between sadistic violence and the total scores and Factor 1 scores of the PCL-R significantly correlated at .35 and .34 respectively. Factor 2 did not significantly correlate with sadistic violence ($r = .20$). It is noteworthy that in this study, 83% of psychopaths committed sadistic acts against their victims as did 53% of nonpsychopaths. While these statistics are significantly different, they reveal that sadism is prevalent among nonpsychopaths and reflect the idea that it is possible for even the “normal” person to develop a propensity toward sadism.

Baumeister and Campbell (1999) offer an account for both the existence of sadism and its evolution within the individual. The Opponent-Process Theory (Solomon & Corbit, 1974) is based on the concept of physiological homeostasis and states that any process which takes the body away from its stable, resting state must be followed by an internal counter process to return to its normal state. Initially, the departing (A) process response is strong, whereas the restorative process is relatively weak and inefficient. However, with the passage of time, the restorative (B) process is exercised over many similar experiences and becomes increasingly efficient and powerful causing the departing (A) process to become weaker. Eventually, the (B) process becomes the dominant process leading to a shift in the stable homeostatic state. Therefore, the “normal” individual who is loathe to inflicting harm will have an extremely negative physiological response upon the commission of a violent act. The body will require a (B)

process to end the aversive, distressed response, and it would likely have a fairly pleasant, positive quality. At first, this positive response may be quite slight and weak, but the (B) process would strengthen, causing the overall quality of violent acts to take on a positive, pleasant nature.

The shortcoming of this theory is that it fails to indicate why most individuals do not become sadists but, rather, remain averse to the perpetration of violence. According to Baumeister and Campbell (1999), the development of a sadistic drive may be moderated by guilt. In normally socialized individuals, the presence of guilt which is linked to empathy and empathizing with a victim's distress would make empathic perpetrators experience the negative emotions themselves. Therefore only the minority of people who lack the experience of guilt and empathy could benefit from the opponent process that gradually produces a positive experience in response to inflicting harm. The authors argue that this is why people seek out activities that will allow violence with minimized guilt such as is the case with hunting. Hunting is considered morally acceptable by a much larger proportion of the population than that proportion which condones violence against people. Moreover, professional sports such as football and hockey that not only condone but promote sanctioned violence among athletes are giant industries in numerous nations' economies.

This theory appears to fit with the theory of psychopathy that suggests psychopaths do not experience guilt or empathy and would explain the aforementioned research finding psychopaths to be more sadistic. However, under this theoretical paradigm, it is unclear why an individual who experiences no guilt or empathy would experience the initial aversive (A) response when committing a violent act. In fact, psychopaths have been shown to have deficient physiological responses to depictions of violence (Patrick, Bradley, & Lang, 1993). Perhaps a more logical reason for the development of sadism is derived from sensation seeking and a need

to reduce boredom. A notable trait of the psychopath is his/her proneness to boredom and need for excitement (Kiehl, et al., 2001; Woodworth & Porter, 2002). Baumeister and Campbell (1999) identify this trait as a possible cause of “evil” separate from sadism which is identified as the “prototype of evil.” However, it seems logical, at least in reference to psychopaths, that this trait can be the seed from which the root of evil grows. Boredom and sensation-seeking has been the impetus of many violent acts seen in throughout history. In 1997, two teenage men living in a rural New Jersey town devised a plan to entertain themselves on an evening when they were unable to find stimulation. They ordered a late-night pizza and had it delivered to an abandoned house in a remote area. When the driver arrived the two men shot him to death making no attempt to rob him or achieve any form of rewards (Kornblut, Rutenberg, & McFarland as cited in Baumeister and Campbell, 1999). Historically, one of the most prolifically violent and original terrorist groups was originally founded as a fraternity to relieve boredom and play pranks on the public. The Ku Klux Klan was founded shortly after the end of the Civil War by a group of ex-Rebel soldiers in Tennessee. Their sole purpose was entertainment and to play pranks on the public. When they found that the recently freed slaves who were uneducated and superstitious were easy targets, they began to focus their attention on this group. Eventually, the KKK became one this country’s most notoriously violent organizations (for further review see Wade, 1987).

It is neither believed that every member of the KKK developed into a sadist nor that all were psychopaths. In its prime, a half-century ago, the KKK boasted nearly five million members. The pervasiveness of psychopathy is significantly less than this and the proportion of psychopaths that are sadistic is likely even smaller. However, the members of the KKK who committed violent acts did so in a social climate that condoned their actions. This likely

mitigated the guilt they experienced about their actions and definitely inhibited empathy for the targets of their violence. However, had they turned their violent actions toward a white protestant they would have been condemned and prosecuted for those actions. It is, therefore, unlikely that these five million members evolved into sadists. The psychopath is distinctly different from these individuals because his need for stimulation and lack of empathy drive him to commit violence indiscriminately without the experience of guilt. They, therefore, possess the necessary traits to develop an intrinsic motivation to aggress: to become sadists.

Another developmental argument for sadism may be linked to biological substrates. Berridge (2003) notes that pleasurable events elicit a positive affective reaction from the brain. He further indicates that although most researchers consider positive affect to be a conscious feeling of pleasure, unconscious affective psychological processes may sometimes occur in the brain independent of conscious feelings. As such, it is possible for an individual to develop a “liking” for or derive “pleasure” from a behavior or stimulus without conscious awareness of this process. This supposition is supported by emotion and cognition research that has demonstrated the existence of an affective priming action that facilitates performance in lexical decision tasks (Ferraro, King, Ronning, Pekarski, & Risan, 2003; Olafson & Ferraro, 2001). Similarly, the subliminal presentation of a happy facial expression fails to produce conscious report of positive affect yet increases the individual’s consumption of a fruit drink and subsequent subjective positive affective ratings of it later (Berridge & Winkielman, 2003).

Pecina & Berridge (2000) argue that there is sufficient evidence indicating that specific subsystems of the nucleus accumbens play a causal role in the processes of positive affect. The authors demonstrated that positive affect reactions are increased by the activation of opioid receptors within the medial caudal portion of this substrate. Moreover, microinjections of

opiates into this area increased consumption behavior of a bittersweet solution. The nucleus accumbens projects through the thalamus to the prefrontal cortex, which has been implicated in higher affective reactions (Zahm, 2000). This pathway may allow for the eventual transition of these unconscious processes into the conscious (Berridge, 2003).

The “normal” person is able to experience negative affect and is socialized to experience it at the appropriate times while the psychopath lacks this ability (Blair, Mitchell, & Blair, 2005). If the psychopath experiences dysfunction or dysregulation causing increased activation in the nucleus accumbens, he may develop subconscious positive affect in response to behaviors and stimuli that normally elicit negative affect. Perhaps through these related dysfunctions of the opposing affect systems, a psychopath can develop a conscious pleasure derived from inflicting harm upon another.

Statement of Purpose

The purpose of the present research is to replicate and expand upon the previous research that has demonstrated that individuals endorsing high levels of psychopathic traits are more likely to engage in unprovoked aggression (i.e., Reidy, Zeichner, and Martinez, in press). Specifically, the goal of the present study is to identify the affective motivation of the unprovoked aggressor and how this emotional experience is linked to the psychopathy-aggression relationship. Two models of association are tested in which emotion processing either mediates the relationship between psychopathy and unprovoked aggression or, alternatively, moderates the relationship between psychopathy and unprovoked aggression. To this end, we examine the relationship among levels of psychopathic traits, experience of pleasure upon the viewing of violent images, and engagement in direct physical aggression prior to provocation.

Similar to previous studies that have addressed unprovoked aggression, the study investigates aggressive responding in the context of a competitive interaction between participant and confederate. However, the present study is distinct from previous studies that did not provide participants a non-aggressive response option. In the bulk of empirical studies, participants are required to provide aversive feedback upon occurrence of a specified event (e.g., winning a competitive trial or the provision of an incorrect response in an evaluative task by a confederate). In the present study, all participants are given the choice to respond at the completion of each competitive trial if they so desire. Participants are allowed to deliver no aversive feedback to the confederate throughout the entirety of experiment if they so choose.

Hypotheses

1. Consistent with a growing body of research (e.g., Parrott & Zeichner, 2006; Reidy, Zeichner, Miller, & Martinez, in press), it was hypothesized that Psychopathy would be positively associated with trait and behavioral measures of aggression.
2. Following the findings of Reidy, Zeichner, and Martinez, (in press), it was hypothesized that Psychopathy would predict greater probability of engaging in unprovoked aggression.
3. It was hypothesized that when controlling for covariance between Factor 1 and Factor 2 of psychopathy, only Factor 1 would significantly predict increased probability of engaging in unprovoked aggression consistent with earlier findings (Reidy, Zeichner, & Martinez, in press).
4. Based on previous research indicating that Factor 1 is positively associated with “sadistic violence,” it was hypothesized that Factor 1 would be positively associated with facilitation of pleasure activation (i.e. Happiness Facilitation).
5. Pursuant to previous research in a nonforensic population indicating that Factor 1 is negatively associated with empathic concern and negatively associated with the

processing of sadness stimuli (i.e., Reidy, Zeichner, Hunnicutt-Ferguson, & Lilienfeld, in press), it was hypothesized that Factor 1 would predict less sadness (i.e., Sadness Facilitation) upon viewing violent images.

6. Consistent with the findings of Reidy et al, (in press), it was hypothesized that Unprovoked Aggressors would demonstrate more aggression than Provoked Aggressors.
7. It was expected that Happiness Facilitation in response to violent imagery would be positively associated with the probability of engaging in Unprovoked Aggression.
8. It was hypothesized that Sadness Facilitation in response to viewing of violent imagery would be negatively associated with the probability of engaging in Unprovoked Aggression.
9. We expected that the relationship between Factor 1 and Unprovoked Aggression would be mediated by Happiness and Sadness Facilitation.
10. We hypothesized that the relationship between Factor 1 and Unprovoked Aggression would be moderated, such that, a 3-way interaction would exist, in which individuals who are high on Factor 1, high on Happiness Facilitation, and low on Sadness Facilitation would be most likely to engage in Unprovoked Aggression.

CHAPTER 2

METHODS

Participants & Experimental Design

One-hundred and sixty-six men were recruited from the University of Georgia to participate in a study advertised as “Effects of Cognition and Emotion on Reaction Time.” The average age of all participants was 19.2 (*1.5*) with an average of 14.3 (*1.3*) years of education. The sample was composed of 81.3% Caucasian, 7.2% Asian, 6% Black/African-American, 1.2% Hispanic/Latino, and 4.2% identified themselves as some other ethnicity. The modal family income was \$70,000+. Women were excluded from the study due to low prevalence rates of psychopathy in this gender (Forth et al., 1996; Levenson et al., 1995; Lilienfeld and Andrews, 1996; Weizmann-Henelius, Viemerö, & Eronen; 2004) and because the majority of violent crimes are committed by men (Federal Bureau of Investigation-Uniform Crime Reports, 1993 - 2001). All participants received research credit for their participation

The main focus of this study was to assess the relationships among: the Emotional Detachment factor of psychopathy, the Antisocial Impulsive Behavioral factor of psychopathy, affect, and physical aggression. All variables were treated as continuous variables, and physical aggression was also treated as an ordered categorical variable in which there are three categories: 1) Unprovoked Aggression, 2) Provoked Aggression, and 3) Inhibited Aggression. Participants were grouped into one of the three categories contingent on when they initiated their aggressive responding in the shock paradigm. Participants who aggressed before receiving provocation were considered “unprovoked aggressors” ($n = 69$), whereas participants who refrained from

responding until provoked were labeled “provoked aggressors” ($n = 46$). The third group comprised individuals who did not initiate aggressive responding at any point during the course of the task. These individuals were considered “provoked nonaggressors” ($n = 22$). Levels of psychopathic traits were derived from participants's responses on the Self-Report Psychopathy Scales Version Three (SRP-III; Paulhus, Hemphill, & Hare, in press), Levenson Self-Report Psychopathy Scales (LSRP; Levenson et al., 1995), and the Psychopathic Personality Inventory-Revised (PPI-R; Lilienfeld, 2005). Affect activation was assessed via the Positive and Negative Affective Schedule (PANAS; Watson, Clarke, & Tellegan, 1988) and through the completion of a lexical decision task after participants have viewed the stimulus material.

Materials

Demographic Form. Participants completed a brief demographic form assessing age, race, education level, and average yearly income to confirm that groups are equivalent on these variables.

Self-Report Psychopathy scale: Version III (SRP-III; Paulhus, et al., in press). The SRP-III is a 64 item self-report measure of psychopathic traits. It is comprised of four subscales titled Interpersonal Manipulation (IPM), Callous Affect (CA), Erratic Life Style (ELS), and Anti-Social Behavior (ASB). The four subscales combine to represent the traditional two-factor model of psychopathy derived from Hare’s conception of the PCL-R: IPM and CA (Factor 1) and ELS and ASB (Factor 2). Items are rated along a Likert-type scale from “1” (*disagree strongly*) to “5” (*agree strongly*). The range of possible values for the subscales is 16 to 80 and, for the total score, 64 to 320. Normative values based upon a sample of 194 undergraduates at the University of British Columbia demonstrate reliability of (.81) for the overall SRP-III. For the purposes of this study we were only interested in F1, F2, and Total Psychopathy scales. In

the present sample, Cronbach's alphas were .83, .79, and .88 for F1, F2, and Total Psychopathy respectively.

Levenson Self-Report Psychopathy Scales (LSRP; Levenson et al., 1995). The Levenson Self-Report Psychopathy Scale was patterned after the Hare (1990) Psychopathy Checklist but was designed for use in nonforensic settings to assess behavioral features of individuals not identified as criminals (Epstein et al., 2006). This 26-item Likert-type scale comprises two subscales that assess two domains of the psychopathic personality. F1 (Cronbach $\alpha = .82$) reflects a callous, manipulative, and selfish use of others (e.g., "For me, what's right is whatever I can get away with"). F2 (Cronbach $\alpha = .63$) assesses impulsivity and poor behavioral control (e.g., "When I get frustrated, I often let off steam by blowing my top"). In the current sample Cronbach's alphas were .88, .68, and .87 for F1, F2, and Total Psychopathy scores, respectively.

The LSRP was selected to assess psychopathy for several reasons. First, it was developed for the purpose of detecting psychopathic features in non-institutional samples such as this one. Second, the LSRP is distinct from other existing self-report measures of psychopathy which only measure the social deviance component of psychopathy, because it is designed to assess both social deviance and callous shallow affect (Miller, Flory, Lynam, & Leukefeld, 2003). Third, this measure has been validated in the assessment of psychopathy in non-forensic undergraduate populations (Lynam et al., 1999; Parrott and Zeichner, 2006) and offending populations (Epstein, Poythress, & Brandon, 2006). It has also been shown to correlate with psychopathy in forensic population (Brinkley, Schmitt, Smith, & Newman, 2001).

Psychopathic Personality Inventory-Short Form (PPI; Cale & Lilienfeld, 2006; Lilienfeld, 1990). The PPI-short form is a 56 item self-report measure of both global psychopathy and the component traits of psychopathy. The PPI measures the continuum of

psychopathic personality traits present in a range of individuals and can be used in both clinical (e.g., forensic) and nonclinical (e.g., student, community) settings. The PPI was developed to assess the core features of psychopathy in nonclinical samples, although it has also been used to assess psychopathy in incarcerated samples. In addition to the total score, the PPI contains eight factor-analytically developed subscales: 1) Social Potency, 2) Coldheartedness, 3) Fearlessness, 4) Impulsive Nonconformity, 5) Stress Immunity, 6) Machiavellian Egocentricity, 7) Blame Externalization, and Carefree Nonplanfulness. For this study, we used the 56-item form of the PPI, which has been found to correlate $r = .90$ or above with the full PPI in several samples (Lilienfeld & Andrews, 1996). The total scale score is composed of eight subscales. Internal consistencies (Cronbach's alphas) for PPI total scores ranged from .85 to .94 (Edens, Poythress, & Lilienfeld, 1999; Lilienfeld & Andrews, 1996). In this study, only the PPI-Short Form total scores were of interest and yielded an alpha of .77.

Positive and Negative Affective Schedule (PANAS; Watson, Clarke, & Tellegan, 1988). The Positive and Negative Affective Schedule will be administered to assess participants' affect prior to viewing the visual stimuli, after the presentation of stimuli, and after completion of the competitive reaction-time task in order to detect any pre and post-existing positive or negative affective states. The PANAS consists of 24 mood descriptors comprising a 10-item Positive Affect (PA), a 10-item Negative Affect (NA) scale, and a 6-item Anger/Hostility (AH) scale. Respondents rate the extent to which they are experiencing each item on a 5-point scale. Watson and colleagues have reported extensive data on scale reliability, with alpha reliability coefficients ranging from .36 to .90 for the PA scale, .84 to .87 for the NA scale, and PA-NA inter-correlations ranging from -.23 to -.12 (Watson, Clarke, & Tellegan, 1988).

Buss Aggression Questionnaire (BAQ; Buss & Perry, 1992). The Buss Aggression Questionnaire is a 29-item scale that is used to assess self-reported aggression on four different dimensions: *Physical Aggression*, *Verbal Aggression*, *Anger*, and *Hostility*. Respondents rate each item on a 5-point scale. Buss and Perry (1992) reported high internal consistency for *Physical Aggression* at .85, *Verbal Aggression* .72, *Anger*, .83, and *Hostility*, .77, with the total score alpha coefficient of .89. Test-retest reliabilities were .80 for *Physical Aggression*, .76 for *Verbal Aggression*, and .72 for both *Anger* and *Hostility*. Construct validity (Buss & Perry, 1992) was demonstrated by positive correlations between measures of assertiveness and *Anger* ($r = .40$), emotionality and *Hostility* ($r = .52$), competitiveness and *Physical Aggression* ($r = .36$), and impulsiveness and *Verbal Aggression* ($r = .31$). For the purposes of this study, the BAQ was included to provide convergent validity of the assessment of aggression and therefore only the total scale score was utilized. Pertinent literature has demonstrated that high scores on this instrument are related to high levels of laboratory-based physical aggression in men (Giancola, 2002). In the present sample the internal consistency was .91.

Reactive–Proactive Aggression Questionnaire (RPQ; Raine, Dodge, Loeber, Kopp, Lynam, Reynolds, et al. 2006). This scale consists of 23 items on a 0–2 scale (0 = never, 1 = sometimes, 2 = always) assessing Reactive and Proactive Aggression. Coefficient alphas for the reactive –proactive scales were .82 and .83, respectively and an alpha of .89 for the total scale. The means for the RA and PA scales were 19.16 (SD = 3.38; range: 11–31) and 14.06 (SD = 2.38; range: 12–28).

Lexical Decision Task. Prominent models of human emotion support the existence of discrete emotions such as anger or fear (e.g., Ekman, 1982). In contrast, theorists also espouse a position that identifies two broad dimensions of affective structure: positive affect and negative

affect (e.g., Russell, Weiss, & Mendelsohn, 1989). These two approaches are not necessarily incompatible (Ehrlichman & Halpern, 1988), and, in fact, a hierarchical model of emotion has been proposed by Watson and Tellegen (1985) and received empirical support (Watson & Clark, 1992). This model posits that two broad dimensions of affect (i.e., positive and negative affect) are composed of several correlated yet distinct emotional states (e.g., happiness and interest, anger and fear). Pertinently, because self-reports of discrete emotions have specific and nonspecific variance, it is necessary for each level of the hierarchical structure to be assessed in order to establish affect-specific relations to other constructs (Watson & Clark, 1992). As such, affects will be assessed beyond the two-structure model. Additionally, in order to assess the presence of subconscious affects the participant completes a lexical decision task.

The participant is seated facing a computer monitor and a keyboard with two keys labeled either “word” or “nonword.” He is informed that the purpose of the task is to identify, as quickly as possible, whether or not each character string is a legal English word. Each participant is instructed to respond “word” or “nonword” by pressing the appropriate key on the keyboard following the presentation of each character string. Prior to the experimental task, there are ten announced practice trials (five words and five nonwords), in which all word stimuli are of neutral emotional connotation. Experimental trials consist of 90 word and 90 non-word trials. The onset of each trial is marked by a horizontally and vertically centered plus sign (+), which serves as a fixation point. After a 500 ms latency, the fixation point is replaced by a character string. The stimulus item disappears after the participant responds or a latency of 3000 ms, whichever occurs first, and is followed by an intertrial interval of 200 ms. The word stimuli are presented in a randomized fashion that is controlled by computer software.

Each nonemotion (i.e., neutral) word is matched to each emotion word in terms of word frequency (Kucera & Francis, 1967). In addition, neutral and emotion words are matched for syllabic length to reduce any effect word length may have on response latency. Because the use of unpronounceable, orthographically- irregular nonwords (e.g., BNEO) can eliminate a robust word-frequency effect (James, 1975), pronounceable non-words created by changing a single letter in each of the 45 neutral words will be used. Moreover, as research has demonstrated that latency decreases and accuracy increases the more often given words are repeated within a single task (Grant & Logan, 1993), all stimulus words are presented only once during the LDT. Each affective word will represent one of five emotions; 1) Disgust, 2) Fear, 3) Sadness, 4) Anger, or 5) Happy. Research on cognition and emotion has demonstrated the validity of this task in assessing an individual's current affective state (Olafson & Ferarro, 2001). Moreover, this task has been used in similar laboratory investigations examining the relationship between affective states and aggression (Parrot, Zeichner, & Evces, 2005; Parrot, Zeichner, & Hoover, 2006).

Visual Stimuli [International Affective Picture System (IAPS); Lang, Bradley, & Cuthbert, 2005]. Each participant viewed a series of 10 color images depicting violent scenes in which a perpetrator was inflicting physical aggression upon a victim. The pictures were drawn from a pool of slides previously rated on dimensions of valence and arousal by nonforensic research participants. For examples of the visual stimuli, see Appendix I.

Response Choice Aggression Paradigm (RCAP; Zeichner, Frey, Parrott, & Butryn, 1999). Under the guise of a 30-trial reaction time competition, the participant uses an aggression apparatus consisting of a white metal box mounted with an assortment of electrical switches and light emitting diodes (LED's). Arranged on the console are push buttons labeled "1" through "10" provided for the ostensible administration of shocks by the participant to his opponent. A

reaction time key is located at the center of the console. Shocks are administered via two electrodes attached to two fingers on the participant's non-dominant hand. The experiment is controlled by a 3-unit peripheral system interfaced with a PC located in a control room separated from the participant chamber. The shock unit features series resistance-regulation, which can never deliver more current than the total circuit resistance predicates. The set accuracy is controlled by the use of a fixed series resistor. The unit does not require calibration. For added safety, a shock level tester is connected the output so that accuracy can be verified. In addition, electrodes are never placed to form a path across the chest, head, neck, or abdomen.

Aggressive behavior is measured via seven indices: 1) *Shock Intensity* (SI) is the mean shock intensity for trials in which the participant administers a shock; 2) *Shock Duration* (SD) is the mean shock-time duration for trials which the participant administers a shock and represents an indirect form of aggression; 3) *Proportion of Highest Shock* (P10) is the number of times the participant uses the highest shock available for trials in which a shock is administered relative to all shock trials; 4) *Flashpoint Latency* (FP) defines the number of trials that expires before the participant administers the first shock; 5) *Flashpoint Intensity* (FPI) defines the intensity of the first shock administered; 6) *Flashpoint duration* (FPD) is the shock-time duration of the first shock administered; and 7) *Shock Frequency* (SF), which is the number of trials that a shock is administered. The latter four indices of aggression relate to one's ability to refrain from responding aggressively. Similar aggression paradigms have been used in previous studies and demonstrated good external validity (e.g., Giancola & Zeichner, 1995; Giancola & Zeichner 1997, Giancola & Zeichner, 2003).

Procedure

Each participant was required to come to the laboratory on two occasions. During the first experimental session, the experimenter met the participant in a hall near where the experimental chamber was located. After providing informed consent, the participant was given background information that the purpose of the experiment is to understand the relationship between personality attributes, attitudes, and reaction time. Following this explanation, participants completed a battery of questionnaires and then viewed 10 violence images via an automated Power-Point presentation. Following the presentation of each individual visual stimulus, the participant was given 7 seconds to rate 1 of 2 dimensions before seeing the next photo. All participants rated the valence (i.e., positive or negative) of the image on a range of -4 to 4, and to “rate how much pain and distress the victim in the image experienced” on a 0 to 10 point scale. For purposes of uniformity, a participant rated all photos on one dimension, then viewed each photo again to rate them on the other dimension. Half of the participants rated the valence of the photo first and the other half rated “rate how much pain and distress the victim in the image experienced” first in order to prevent sequence effects. The purpose of asking participants to rate valence and perceived pain and distress was twofold. First, it serves to ensure that the participant attended to the stimuli and evaluated them cognitively and affectively. Second, it provides for the replication of previous research (Patrick et al., 1993) indicating that although psychopaths’ physiological responses were different, their self-report of affect did not differ from nonpsychopaths.

After viewing the 10 images, participants completed the LDT and then completed the PANAS for a second time. Upon completion of the second PANAS, participants were thanked and scheduled to return to a different laboratory on a second day to complete the second

experimental session with a different experimenter. The purpose of separating the experiment into two sessions is to prevent any confounding arousal or priming effects (see Berkowitz & Le Page, 1967; Zillman, 1983) of the visual stimuli that may serve to increase aggressive behavior. Additionally, the aggression data was collected in a different laboratory by a different experimenter to reduce any association of this task to the previous violent imagery viewed in session 1.

At the commencement of the second experimental session participants were greeted by the experimenter and asked if his name is Phil (the name of the fictitious opponent). In order to disguise the RCAP's purpose as a measure of aggression, the participant was told a fictitious cover story concerning the task. He was again informed that the study's aim is to understand the relationship between personality attributes, attitudes, and reaction time. The participant was told that he is competing in a reaction time task against an "opponent" who is seated in another chamber and that during the task, he has the opportunity to punish his opponent following each reaction time trial by administering a shock to the competitor. Moreover, he is informed that whoever reacts faster to the green light on the aggression console by releasing the reaction time button will win that trial. The task was explained to the participant as follows:

First, a red "get ready" light will illuminate, and you should get ready and place your finger over the reaction time button. Next, a yellow "press" light will illuminate, and you should press the reaction time button until the green "release" light illuminates. Once the green light illuminates, you must release the reaction time button as quickly as possible. Next, a computer will determine who was "faster." When you lose, a red "Lose" light will flash several times, and when your opponent loses, a green "Win" light will flash several times. After this, all three lights on the console will illuminate for six seconds, during which time both you and your "opponent" will have the opportunity to administer a shock. In order to administer a shock, you must press one of ten shock buttons, which increase in shock intensity, with the button labeled "1" being the "lowest" shock intensity and the button labeled "10" being the "highest" shock intensity.

However, the participant was repeatedly told that he could refrain, entirely, from administering shocks to his opponent as could his opponent. LED's provided visual feedback to the participant as to the level of shock (i.e. 1 through 10) they received from the confederate.

Prior to commencement of reaction time task, each participant's subjective pain threshold was determined via incrementally-increasing shocks so that no shock administered during the task was above the participant's reported subjective pain threshold. During this process the participant was informed that both he and his opponent would be able to hear one another as their pain threshold is determined. This was done to further the deception that the opponent is a "real person." After this, the participant competed in the sham reaction time task against the confederate. The confederate was described to the participant as a man to avoid confounding gender effects. In effect, the confederate was not a person, but a computer program by which a predetermined series of shocks was administered a maximum of 15 times. Once the competition was completed, the experimenter conducted a manipulation check in during which the participant was asked a series of questions to assess the success of the deception. Participant's were asked 1) to report their impression of their opponent, 2) whether they believed their opponent was "fair" during the task, 3) whether they believed the task was a good measure of their reaction time, 4) how they felt about administering shocks, and 5) whether they recognized the voice of their opponent as someone they knew. In addition to the manipulation check, the participant's behavior during the reaction time task was observed via a video camera. Behavior indicating belief that his opponent was real (e.g., cursing at the opponent, saying the opponent's name) was noted and used in determination of the deception's success. The purpose of this interaction with the experimenter is to provide the participant an opportunity to indicate that he did not believe his opponent was an actual person if such is the case. All participants were

thoroughly debriefed at the completion of the manipulation check, thanked for their participation, and given course credit.

Risk & Protection of Participants

Some discomfort may be experienced when receiving electric shocks. Each participant's subjective sensitivity to shocks was assessed and no shocks higher than the participant's reported pain threshold were administered. No long-term adverse consequences have been reported in connection with this procedure. Additionally, the participant was allowed to terminate his participation without prejudice at any time. In previous studies that used competition tasks such as this one, neither immediate nor subsequent problems were encountered. However, any participant reporting emotional distress will be referred to mental health providers.

CHAPTER 3

RESULTS

Data Reduction

Of the 166 men who completed the first experimental session, 149 returned to complete the second experimental session in which aggression data was obtained (attrition rate = 10%). Of the 149 men who completed the entire experiment, seven were excluded from behavioral aggression analyses because they reported that they were not misled by the experimental deception. Additionally, two men chose not to participate in the aggression paradigm when they read the consent form which indicated that electric shocks would be involved. Two men were deemed ineligible to participate in the aggression phase of the study because they had previously participated in similar studies and one individual's aggression data was lost due computer malfunction. This resulted in a sample of 137 men who completed both phases of the experiment and were eligible for analysis.

According to Shadish, Cook, and Campbell (2002) mono-operation bias (i.e., using only one measure of a construct) should be avoided in all areas of psychopathology research. Moreover, in research on psychopathic personality, measures of this construct are only weakly or moderately correlated (Cale & Lilienfeld, 2006) which can lead to questions about construct validity. Accordingly, the present study uses multiple self-report measures of psychopathy. In the analysis of these multiple indices, the practice of Cale and Lilienfeld (2006) was followed. To keep the total number of analyses to a minimum and thereby minimize the risk of Type I error, composite scores for the psychopathy measures were created from the LSRP and SRP-III.

Standardized (*z*-scores) of total psychopathy, Factor 1, and Factor 2 scores were computed for each measure and summed to create composite scores. These composite scores were used for all subsequent analyses. Because the PPI does not follow the same theoretical two-factor structure common to the PCL-R, LSRP, and the SRP-III, this measure was not aggregated with other psychopathy measures. Correlations among psychopathy measures can be seen in Appendix A, Table 1.

Following procedures of previous research (e.g., Bushman & Anderson, 1998; Reidy, Zeichner, & Martinez, in press; Reidy, Zeichner, Foster, & Martinez, 2008), aggression indices captured by the RCAP were combined to form four distinct measures. First, as prior research has suggested that shock intensity, duration, and frequency reflect a similar underlying phenomenon (i.e., General Aggression [GA]; Carlson, Marcus-Newhall & Miller, 1989), a GA index was created by summing the standardized values of these measures. A second composite index, Initial Aggression (IA), was created by summing the standardized values of first shock intensity and duration to indicate the level at which individuals initiated aggression. Finally, Extreme Aggression (P10) was analyzed individually as was Flashpoint Latency (FP). Correlations among indices of behavioral aggression and self-report trait aggression can be seen in Appendix A, Table 2.

The goal of the present study was to identify how the relationship between psychopathy and emotion-processing related to the perpetration of unprovoked aggression (i.e., a mediated or moderated relationship). To accomplish this, it was pertinent to 1) demonstrate that there is a relationship between psychopathy and aggression, 2) establish that there is relationship between psychopathy and unprovoked aggression, 3) analyze the relationship between psychopathy and affective responses to violent imagery, 4) analyze the predictive relationship of affective

responses to violent imagery on aggression, 5) analyze the predictive relationship of affective responses to violent imagery on unprovoked aggression, 6) test a mediation model in which happiness and/or sadness facilitation in response to violent imagery mediate the relationship between psychopathy and unprovoked aggression, and 7) test a moderation model in which happiness and/or sadness facilitation in response to violent imagery interact with psychopathy to predict unprovoked aggression. Finally, Unprovoked and Provoked Aggressors were compared on the amount of aggression they demonstrated.

Psychopathy & Aggression

Trait Aggression. To establish both convergent and concurrent validity of the RCAP as a measure of aggression, the relationship between psychopathy indices and self-reported trait aggression were analyzed. The BAQ yields a total trait aggression score that encompasses the use of verbal and physical aggression as well as the experience of associated anger and hostility. The second measure of self-report trait aggression, the RPQ, yields two subscales that reflect the use of Proactive (PA) and Reactive (RA) forms of aggression.

We first computed Pearson-product moment correlations between indices of total psychopathy and trait aggression. The Total Psychopathy composite score (derived from the LSRP and SRP) correlated positively with all measures of trait aggression: BAQ ($r = .56, p < .001$), RA ($r = .56, p < .001$), PA ($r = .65, p < .001$). Additionally, correlations between the PPI total score and trait aggression were computed to demonstrate convergent validity of the composite index. Psychopathy as assessed by the PPI correlated with all measures of trait aggression: BAQ ($r = .35, p < .001$), RA ($r = .31, p < .001$), and PA ($r = .40, p < .001$). Overall, these analyses indicate that the construct of psychopathy is positively associated with trait aggression.

We next computed regression equations to demonstrate the relationship between the two subfactors of psychopathy (F1 & F2) and trait aggression. Regression analyses were employed to control for covariance between the two factors, account for the presence of suppressor effects, and to test for interaction between the two factors. When BAQ trait aggression was regressed on F1, F2, and the product term of F1 and F2, the model was significant $F(3, 132) = 21.99; p < .001; R^2 = .33$. Examination of the individual coefficients indicated that there was no interaction between the two factors. However, both F1 ($B = .32; p = .001$) and F2 ($B = .35; p < .001$) predicted greater levels of trait aggression endorsement.

When PA was regressed on F1, F2, and the product term of the two factors, the model was significant, $F(3, 132) = 52.70; p < .001; R^2 = .55$. Examination of the individual coefficients indicated that there was a significant main effect for F1, ($B = .24; p = .002$) and F2, ($B = .48; p < .001$). Additionally, the coefficient for the product term indicated that there was a significant interaction between the two factors, ($B = .25; p < .001$). A series of simple regression equations were computed to explicate the relationship between each factor and PA at one SD above and one SD below the mean of the opposing factor. When F2 was treated as the moderator, F1 significantly predicted PA at high levels of F2, (1 SD above mean) $B = .45, p < .001$ but not at low levels of F2, (1 SD below the mean) $B = -.04, p > .10$. When F1 was treated as the moderator, F2 significantly predicted PA at high levels of F1, $B = .71, p < .001$, as well as at low levels of F1, $B = .21, p < .05$, although the magnitude of prediction was smaller. Plots of the interactions can be seen in Appendix B, Figure 1A and 1B.

In the next regression equation, RA was regressed on the F1 and F2 and the interaction term. The overall model was significant, $F(3, 132) = 35.59; p < .001; R^2 = .37$. The regression coefficient indicated that both F1 ($B = .29, p = .001$) and F2 ($B = .38; p < .001$) significantly

predicted greater levels of endorsement of reactive aggression. Additionally, the interaction term was significant ($B = .14, p < .05$). Simple regression analyses indicated that F1 predicted more RA at high levels of F2 ($B = .40, p < .001$) but not at low levels of F2 ($B = .13, p > .10$). At high levels of F1, F2 significantly predicted greater endorsement of RA ($B = .51, p < .001$). At low levels of F1, F2 significantly predicted greater RA ($B = .24, p < .05$), but at a significantly smaller magnitude. Plots of the interactions can be seen in Appendix B, Figure 2A and 2B. Refer to Appendix A, Table 3 for a summary of coefficients between psychopathy and aggression indices.

RCAP Aggression. The RCAP proffered four indices of behavioral aggression: General Aggression (GA; a general index of degree of aggression), Initial Aggression (IA; the intensity and duration with which an individual initiates aggression), Extreme Aggression (P10; the proportion of maximal aggression an individual demonstrates), and Flashpoint Latency (FP; how long an individual inhibits aggressive responding). Pearson-product moment correlations were first computed between the four RCAP aggression indices and the two indices of general psychopathy (Total Psychopathy and PPI). The composite Total Psychopathy score correlated with GA ($r = .31, p < .001$), IA ($r = .29, p < .001$), and FP ($r = -.36, p < .001$). Additionally, there was a trend toward significance when correlated with P10 ($r = .14, p = .055$). The PPI significantly and positively correlated with GA ($r = .22, p = .01$), IA ($r = .24, p < .001$), and FP ($r = -.32, p < .001$).

In the next set of analyses, regression equations were utilized to assess the relationship between the psychopathy factors and behavioral aggression. In the first equation, GA was regressed on F1, F2, and the product-term of the two factors. The overall model was significant $F(3, 132) = 6.19, p = .001, R^2 = .12$ and regression coefficients indicated that F1 had a marginal

positive effect on GA ($B = .19, p = .06$) while F2 did not achieve significance ($B = .17, p = .10$). Additionally, analyses indicated a significant interaction between the two factors on GA ($B = -.14, p = .05$). Simple regression analyses indicated that F1 did not predict GA at high levels of F2 ($B = .05, p > .10$) but did predict GA when the participant was low on F2 ($B = .23, p < .01$). Similarly, at high levels of F1, F2 was unrelated to GA ($B = .02, p > .10$) but significantly predicted GA at low levels of F1 ($B = .21, p = .01$). Plots of the interactions between F1 and F2 on GA can be seen in Appendix B, Figure 3A and 3B.

In the next equation, IA was regressed on the two factors and their interaction term. The overall model was significant, $F(3, 132) = 7.11, p = .001, R^2 = .10$, and the coefficients indicated that only the main effect of F1 was significant ($B = .31, p < .005$) suggesting that individuals who endorse more F1 than their counterparts initiate aggression at greater intensity and duration. In the third regression, P10 was regressed on the predictor variables and interaction term. The overall model was did not predict Extreme Aggression $F(3, 132) = 2.04, ns$.

In the final regression, FP was entered as the outcome variable. The overall model was significant, $F(3, 132) = 9.50, p < .001, R^2 = .18$. F1 demonstrated a significant main effect ($B = -.24, p < .05$) while F2 demonstrated a trend toward significance ($B = -.18, p = .08$) suggesting that individuals who endorse higher levels of F1 and F2 initiate aggression sooner than their counterparts. Additionally, the model indicated a significant interaction between F1 and F2 ($B = .18, p = .01$). For individuals high on trait F2, F1 did not predict FP ($B = -.09, p > .10$), however, at low levels of F2, F1 significantly predicted initiating aggression sooner ($B = -.44, p < .001$). Plots of the interactions can be seen in Appendix B, Figure 4A and 4B. Refer to Appendix A, Table 3 for a summary of coefficients between psychopathy and aggression indices.

Psychopathy & Unprovoked Aggression

One of the primary goals of the present study was to replicate previous research that has demonstrated that psychopathy is associated with an increased probability to engage in unprovoked aggression. To replicate this finding, a series of ordinal logistic regression equations were computed. Owing to the discrete sequential manner in which category assignment was made, the dependent variable was considered representative of an underlying ordinal construct. We used this approach in keeping with Cliff (1993), who suggested that the use of ordinal analyses offers greater robustness and power, considers the scale properties of the variable, and more readily addresses the research question at hand. Moreover, the use of ordinal regression provides relative risk likelihood estimates. The cumulative continuation ratio model was used by selecting a complimentary log–log function, as it is ideal for failure time data and measurement of threshold points. Additionally, this approach seemed appropriate as the present categories of the response variable did not reflect an arbitrary grouping of an underlying continuous variable (Anath and Kleinbaum, 1997; Scott et al., 1997). This model is based on probability estimates of inclusion in category j conditional on inclusion in a category greater than j . Hence, this model can be formulated as follows:

$$\log[Pr(Y = y_j | x) / Pr(Y > y_j | x)]$$

Psychopathy scores were treated as continuous predictors of group assignment. The ordered arrangement of the groups was unprovoked aggressors, provoked aggressors, and nonaggressors. Following the recommendations of West, Aiken, and Krull, (1996), psychopathy scores were standardized to allow for meaningful interpretation of effects. That is, as it is not possible to attain a score of zero for any of the scales on the LSRP, interpretation of regression coefficients would be rendered meaningless. In contrast, standardization of these variables produces a mean,

which has a value of zero and, thus, allows for the interpretation of coefficients at the average presentation of these traits.

We first computed a pair of regression equations using two models of general psychopathy (i.e., PPI and Total Scale Psychopathy) to demonstrate that this construct predicts an increased risk for unprovoked aggression. In the first equation, the PPI total score was entered as the predictor variable and aggressor type (1 = unprovoked aggressor, 2 = provoked aggressor, 3 = nonaggressor) was entered as the outcome variable. The model proved to be significant, $\chi^2(1, N = 137) = 6.52; p = .01, R^2_N = .05$. The negative coefficient for psychopathy, $Wald \chi^2(1, N = 137) = 5.29; B = -.23, p < .05$, indicated that as PPI psychopathy scores increase the probability of being an unprovoked aggressor (i.e., lower ordinal grouping) also increases. The test of parallel lines indicated that the assumption of proportional odds (i.e., the change in probabilities across groups is linear) was not violated $\chi^2(1, N = 137) = .13; p > .10$. To establish concurrent validity, we computed a second equation using general psychopathy to predict unprovoked aggression. When Total Psychopathy was entered as the predictor, the model was again significant $\chi^2(1, N = 137) = 14.12; p < .001, R^2_N = .11$. The negative coefficient for psychopathy, $Wald \chi^2(1, N = 137) = 11.32; B = -.35, p < .05$, indicates that Total Psychopathy predicts increased probability of being an unprovoked aggressor.

The next goal of the study was to address the contribution of the two factors (emotional detachment and antisocial behavior) to the prediction of unprovoked aggression. Previously, Reidy, Zeichner, and Martinez, (in press) demonstrated that F1 predicted Unprovoked Aggression while F2 did not. To replicate these findings, we simultaneously entered F1 and F2 into the regression equation as predictor variables. The overall model significantly predicted the probability of engaging in unprovoked aggression $\chi^2(2, N = 137) = 18.58; p < .001, R^2_N = .15$.

The assumption of proportional odds was met $\chi^2 (2, N = 137) = 1.05; p > .10$; and Goodness-of-Fit statistics indicate an appropriate fit to the data, $Pearson \chi^2 (270, N = 137) = 278.99; p > .10$. The significant negative coefficient for F1, $Wald \chi^2 (1, N = 137) = 7.14; B = -.43, p < .01$, indicates that greater emotional detachment predicts a greater probability of engaging in unprovoked aggression. The coefficient for F2 did not predict Unprovoked Aggression when controlling for F1, $Wald \chi^2 (1, N = 137) = 2.64; B = -.26, p = .10$. To test for an interaction between the two factors in the prediction of unprovoked aggression we conducted another ordinal regression in which we included a product term of the two factors. The coefficient indicated that there was a significant interaction between the two factors, $Wald \chi^2 (1, N = 137) = 7.2; B = .29, p < .01$. For individuals low in F2, F1 significantly predicted a greater probability of engaging in unprovoked aggression, $Wald \chi^2 (1, N = 137) = 12.72; B = -.72, p < .001$, but not for individuals who endorse high levels of F2, $Wald \chi^2 (1, N = 137) = .71; B = -.15, p > .10$. Similarly, for individuals who endorsed low levels of F1, F2 significantly predicted a greater probability of engaging in Unprovoked Aggression, $Wald \chi^2 (1, N = 137) = 7.28; B = -.50, p < .01$. However, for those men high on F1, F2 was not associated with risk for unprovoked aggression, $Wald \chi^2 (1, N = 137) = .11; B = .07, p > .10$. Plots of these interactions can be seen in Appendix B, Figure 5A and 5B.

Psychopathy & Affective Responses to Violent Imagery

A second goal of the present investigation was to assess the relationship between psychopathy and individuals' affective responses to violent imagery. To do this, we employed three measures of cognitive-emotional responding: 1) asking participants to rate valence and perceived pain/distress of the victim, 2) examining change scores on the PANAS, and 3) assessing response patterns to the LDT.

Ratings of violent imagery. The order in which participants rated image valence and perceived pain/distress was counterbalanced to control for sequence effects. The group that rated valence first had an average pleasantness rating of -2.92 versus the group that rated valence second who had an average rating of -2.51. An independent samples *t*-test indicated that those who rated images on valence (i.e., pleasant or unpleasant) first, rated the images as significantly more unpleasant $t(163) = -3.33, p = .001, d = .52$. No differences were found for ratings of perceived pain/distress ($M = 7.63$ vs. $M = 7.91$).

The relationship between general psychopathy and image ratings was first assessed by computing a set of Pearson product-moment correlations. The PPI was related to ratings of less perceived victim pain and distress ($r = -.22, p = .005$) as well as rating the images as less unpleasant ($r = .23, p < .005$). Similarly, the Total Psychopathy composite score related to less perceived pain and distress ($r = -.34, p < .001$) and less unpleasantness ($r = .47, p < .001$). A simultaneous regression was conducted with F1 and F2 as the predictors for ratings of pleasantness. The overall model significantly predicted valence, $F(2, 133) = 18.93, p < .001, R^2 = .22$, and the coefficients for F1 ($B = .23, p < .005$) and F2 ($B = .21, p < .01$) indicate that both independently predict rating the violent imagery as less unpleasant. When perceived pain/distress was entered as the outcome variable, the model was significant, $F(2, 133) = 9.24, p < .001, R^2 = .12$, and the coefficients indicated that individuals high on F1 ($B = -.31, p < .05$) perceived less pain and distress experienced by the victim while individuals high in F2 ($B = -.26, p = .07$) demonstrated a trend toward perceiving less pain and distress.

Changes in positive and negative affect. To first demonstrate that the viewing of violent imagery influenced the mood of the participants, dependent samples *t*-tests were computed comparing participants' on the PANAS before and after completing the task. Results indicated

that participants' negative affect did not change, $t(164) = 1.42, p > .10, d = .22$, but they did demonstrate a large significant decrease in levels of positive affect, $t(164) = 8.49, p < .001, d = 1.33$. Additionally, participants' anger/hostility demonstrated a slight nonsignificant increase, $t(164) = -1.74, p = .08, d = .27$. Overall, the violent content of the imagery appeared to have a large negative effect on participants' positive mood. Next, the relationship between general psychopathy and changes in mood was assessed via correlation coefficients. Both the PPI and Total Psychopathy composite score proved to be unrelated to changes in self-reported mood states. Similarly a series of simultaneous regressions indicated that F1 and F2 were unrelated to changes in mood as reported on the PANAS.

Lexical-Decision-Task. Trials reflecting incorrect responses were deleted. Additionally, any response latency greater than 1500ms or less than 350ms were considered extreme responses that reflected random responding (i.e., reaction-time outliers) and were consequently deleted. The aim of the present study was to examine change in processing speed of discrete affect words relative to (1) neutral words and (2) other affect words. Comparison of discrete affects to neutral words considers each affect as if it was orthogonal and had no influence on the operation of the other affective networks. To make the comparison, we first computed emotion facilitation scores by subtracting the mean response latency for the 15 emotion words in each discrete affect from the mean response latency to neutral words. Additionally, based on theory that emotion represents activation of an interconnected network of nodes (Bower, 1981) in which the functioning of one affect influences the functioning of another, we also created a set affect composite scores to which each individual affect was compared. This allowed for the nonorthogonal analysis of affective processing. To accomplish this, mean RT's for the 15 emotion words in each discrete affect were subtracted from the composite of the remaining 60

emotion words (i.e., four affects) such that, the average response time to sad words was subtracted from the average of the average RT's to angry, happy, fear, and disgust words. This process was repeated for each discrete affect category to yield a set of 5 composite affect difference scores. For both types of emotion facilitation scores (i.e., orthogonal and nonorthogonal), more negative RT values indicate less facilitation and, as such, decreased activation of that emotion “network.” Conversely, more positive RT values would suggest increased activation of that emotion “network.”

Response times to words were Angry $M = 680.78$; Happy $M = 716.12$; Disgust $M = 774.89$; Sad $M = 754.14$; Fear $M = 660.63$; Neutral $M = 737.96$; and nonwords $M = 753.95$. Dependent t -tests indicated that each discrete affect significantly differed from neutral words (all t 's $>$ absolute 4, all p 's $< .001$), and from all words of the other discrete affects (all t 's $>$ absolute 4, all p 's $< .005$).

Pearson product-moment correlations were computed between the general measures of psychopathy and the orthogonal affect scores. All correlation coefficients were nonsignificant for Total Psychopathy and PPI. Next, to control for general motor response time, a set of partial correlations for general psychopathy measures and composite affects difference scores (i.e., affect composite – individual affect) were computed controlling for response latencies to neutral words. Similar to the orthogonal analyses, the PPI and Total Psychopathy were unrelated to all affect scores. As a whole, general psychopathy was unrelated to emotion facilitation scores. Correlation coefficients for the PPI and Total Psychopathy can be seen in Appendix A, Table 4.

A series of regression equations entering F1, F2, and their interaction term as predictors of each orthogonal emotion facilitation score were computed. Results indicated that F1 and F2 were unrelated to response latencies for the Angry, Happy, and Disgust emotions. When the

response latencies to Sadness words were entered as the outcome variable, the overall model was nonsignificant but results indicated that F1 significantly predicted slower response times, $B = -.22, p < .05$. When response times to fear words were entered as the outcome variable, the overall model significantly predicted response latencies, $F(3, 133) = 3.26, p < .05, R^2 = .07$. F1 demonstrated a trend toward slower response latencies, $B = -.17, p = .08$, while F2 was significantly related to faster response times, $B = .19, p = .05$. Moreover, the interaction between F1 and F2 was significant, $B = .16, p < .05$. Simple regression analyses indicated that for high F2 individuals, F1 did not predict response times to Fear words, $B = -.01, p > .10$, but for individuals endorsing low levels of F2, F1 was related to significantly slower response times to Fear words, $B = -.32, p = .01$. Conversely, for individuals endorsing high levels of F1, F2 significantly predicted faster response times to fear words, $B = .34, p < .005$, but not at low levels of F1, $B = .03, p > .10$. Plots of the interaction can be seen in Appendix B, Figure 6A and 6B.

Regression analyses were repeated using the nonorthogonal affect composite difference scores as the outcome variable. To control for general motor response time, response latencies to neutral words were used as a baseline by entering them into step 1 of a hierarchical regression. Results indicated no relation between F1 and F2 for response latencies to Angry, Disgust, and Fear words. When Happy words were entered as the outcome variable, the model was nonsignificant, but F2 demonstrated a decreased response time, $B = -11.92, p < .05$. Similarly, when Sadness words were entered as the outcome variable, F1 predicted slower response latencies, $B = -12.33, p < .05$. A summary of regression coefficients for F1, F2, and their interaction terms can be found in Appendix A, Table 5.

Affective Responses to Violent Imagery as Predictors of Aggression

PANAS Change Scores and Trait Aggression. Pearson product-moment correlations indicated that changes in self-reported positive and negative affect were unrelated to the BAQ, PA and RA.

LDT Emotion Facilitation Scores and Trait Aggression. A simultaneous regression was computed using all orthogonal emotion facilitation scores (i.e., neutral words – discrete affect words) as predictors of the BAQ trait aggression. The overall model was nonsignificant as were each of the regression coefficients. The same pattern of results was found when RA and PA were entered as the outcome variable. As this process allows for examination of the relationship between each affect and trait aggression while controlling neutral words and all other affects, it was expected that using the nonorthogonal emotion facilitation scores should produce a similar pattern of results. Moreover, a simultaneous regression would be inappropriate as it would create redundancy in the model because each affect score inherently controls for the variance of other affects. Consequently, partial correlations controlling for neutral words were deemed the most appropriate analysis for this portion of the data. Contrary to expectation, proactive aggression (PA) correlated with two affective responses to the viewing of violent imagery. Individuals who demonstrated more disgust facilitation in response to the images endorsed more PA ($r = .15, p = .055$) while those who demonstrated more happiness facilitation endorsed less PA ($r = -.16, p < .05$).

PANAS Change Scores and RCAP Aggression. Pearson product-moment correlations indicated that changes in self-reported positive and negative affect were unrelated to GA, IA, P10, and FP.

LDT Emotion Facilitation Scores and RCAP Aggression. A series of simultaneous regressions were computed using all orthogonal emotion facilitation scores (i.e., neutral words – discrete affect words) as predictors of the four RCAP indices of aggression. When GA was entered as the outcome measure, the overall model was nonsignificant and response latencies to Sadness words demonstrated a trend toward significance, $B = -.12, p = .07$, suggesting that individuals who had less sadness facilitation following exposure to violent images demonstrated more aggression. When IA was entered as the outcome variable, the overall model approached significance, $F(5, 130) = 2.22, p = .056, R^2 = .08$. Again, a trend appeared in which less Sadness Facilitation predicted more aggression, $B = -.14, p = .08$. Additionally, response latencies to Disgust words demonstrated a trend in which more Disgust Facilitation predicted more IA, $B = .16, p = .07$, and response to fear words significantly predicted less IA $B = -.17, p < .05$. When P10 was entered as the outcome variable, the model was significant, $F(5, 130) = 2.37, p < .05, R^2 = .08$, and only disgust facilitation predicted aggression, $B = .35, p < .005$. In the final regression equation, when FP was entered as the outcome variable, the model was nonsignificant and both greater Happiness Facilitation ($B = -.18, p = .06$) and Less Sadness Facilitation ($B = .18, p = .06$) demonstrated a trend toward initiating aggression sooner. A summary of regression coefficients for emotion facilitation scores and the four RCAP indices can be seen in Appendix A, Table 6.

Partial correlation analysis between composite affect scores and RCAP aggression controlling for neutral words indicated that Anger Facilitation and Fear Facilitation were unrelated to all indices of behavioral aggression. Happiness Facilitation correlated with FP ($r_{\text{partial}} = -.18, p < .05$). Disgust Facilitation correlated with IA ($r_{\text{partial}} = .18, p < .05$) and P10 ($r_{\text{partial}} = .25, p < .005$). Again, decreased Sadness Facilitation demonstrated a trend toward more aggression for GA ($r_{\text{partial}} = -.16, p = .07$) and FP ($r_{\text{partial}} = .15, p = .08$). A summary of partial

correlation coefficients for emotion facilitation scores and the four RCAP indices can be seen in Table 5.

Affective Responses to Violent Imagery as Predictors of Unprovoked Aggression

PANAS Change Scores and Unprovoked Aggression. A logistic ordinal regression equation was computed in which aggressor type (i.e., Unprovoked, Provoked, or Nonaggressor) was entered as the outcome variable and PANAS changes scores for positive and negative affect were entered as the predictor variables. Results indicated that changes in self-reported affect did not predict Unprovoked Aggression. The full model, $\chi^2 (1, N = 137) = 3.67; p > .10, R^2_N = .03$, was nonsignificant and the coefficients for each predictor indicated that neither predicted aggressor type: positive affect, $Wald \chi^2 (1, N = 137) = 1.97, B = -.03, p > .10$; negative affect, $Wald \chi^2 (1, N = 137) = 1.86; B = -.05, p > .10$.

LDT Emotion Facilitation Scores and Unprovoked Aggression. An ordinal regression using the five orthogonal emotion facilitation scores as predictors indicated that emotion facilitation was unrelated to unprovoked aggression, $\chi^2 (1, N = 137) = 8.20; p > .10, R^2_N = .07$, (all $Wald \chi^2 < 2.5$, all B 's < absolute .01, all p 's > .10).

To compute ordinal regression analyses using the nonorthogonal emotion facilitation scores, a series of linear regressions were conducted in which each emotion facilitation score was regressed onto response times for neutral words. The standardized residual was then saved to create a composite score with the variance of the neutral words partialled out. This, in effect, controls for the neutral baseline. Each residualized emotion facilitation composite score was then independently entered into an ordinal regression equation in which aggressor type was regressed onto the emotion facilitation score. A simultaneous regression using all partialled emotion facilitation scores as predictors would create redundancy in the equation as each

emotion facilitation score inherently controls for the variance of the other four affects. As such, the maximum number of predictors that could be entered into the equation at one time would be $K - 1$, where K = the number of affects. Consequently, the outcome variable was regressed on each emotion facilitation score separately. For Fear Facilitation, Anger Facilitation, Disgust Facilitation, and Sadness Facilitation, all regression models and B 's were nonsignificant, indicating no relationship with Unprovoked Aggression. However, when Happiness Facilitation was entered as the predictor variable, the model was significant, $\chi^2(1, N = 137) = 3.90; p < .05$, $R^2_N = .03$; and the coefficient indicated that the more happiness individuals experienced in response to viewing the violent imagery the greater their probability of engaging in unprovoked aggression, $Wald \chi^2(1, N = 137) = 3.62; B = -.23, p = .057$.

Does Emotion Facilitation Mediate the F1-Unprovoked Aggression Relationship?

The main hypotheses of this study stated that the emotional detachment factor of psychopathy would be positively associated with facilitation of pleasure activation and less sadness activation upon viewing violent images. Furthermore, it was believed that this relationship would predict the probability of engaging in Unprovoked Aggression. The purpose of the present investigation was to identify the relationship between emotional experience, psychopathy, and the perpetration of Unprovoked Aggression. One proposed model that could explain the relationship is a causal model in which emotional facilitation of positive affect in response to violence mediates the F1-Unprovoked Aggression relationship. However, because F1 did not predict positive affective responses to violent imagery, a mediated relationship is not possible. Additionally, F1 demonstrated a pattern of predicting less sadness in response to violent imagery, but less Sadness Facilitation did not predict Unprovoked Aggression again indicating no mediated relationship. Consequently, this causal model was rejected and tests of mediation were not conducted.

Does Emotion Facilitation Moderate the F1-Unprovoked Aggression Relationship?

A second model of the F1-Unprovoked Aggression relationship that could explain the relationship is a moderated model in which the emotional detachment factor interacts with emotion facilitation to predict who engages in unprovoked aggression. In this model, it is hypothesized that individuals who were high in F1, demonstrated more Happiness Facilitation, and less Sadness Facilitation would demonstrate an increased propensity to engage in Unprovoked Aggression. To test this hypothesis, an ordinal regression containing F1, the residualized Happiness Facilitation composite score, and the residualized Sadness Facilitation composite score were entered with the 3-way and all 2-way interactions as predictors of unprovoked aggression. This model proved to fit the data well, $\chi^2 (1, N = 137) = 26.21, p < .0001, R^2_N = .20$. Regression coefficients indicated a significant main effect for F1, $Wald \chi^2 (1, N = 137) = 14.79; B = -.61, p < .001$, and a trend toward significance for the main effect of Happiness Facilitation, $Wald \chi^2 (1, N = 137) = 3.30; B = -.24, p = .069$, while Sadness Facilitation was unrelated, $Wald \chi^2 (1, N = 137) = .01; B = .01, p > .10$. Contrary to expectation, the interaction term for F1 x Happy was nonsignificant, $Wald \chi^2 (1, N = 137) = 1.77; B = -.21, p > .10$, as was the interaction between Sad and Happy, $Wald \chi^2 (1, N = 137) = .27; B = .27, p > .10$. However, the interaction between F1 x Sad proved to be significant, $Wald \chi^2 (1, N = 137) = 4.41; B = -.32, p < .05$. The 3-way interaction was nonsignificant, $Wald \chi^2 (1, N = 137) = .01; B = -.01, p > .10$.

Simple ordinal regression analyses indicated that for individuals high on Happiness Facilitation and high on Sadness Facilitation, F1 strongly predicted increased probability of Unprovoked Aggression, $Wald \chi^2 (1, N = 137) = 15.59; B = -1.12, p = .001$. When individuals were high on Happiness Facilitation but low on Sadness Facilitation, F1, contrary to original expectations, did not predict engaging in Unprovoked Aggression, $Wald \chi^2 (1, N = 137) = .03; B$

= -.06, $p > .10$. For individuals low on Happiness Facilitation and low on Sadness Facilitation, the relationship between F1 and Unprovoked Aggression was again nonsignificant $Wald \chi^2 (1, N = 137) = .32$; $B = -.12$, $p > .10$. For individuals low on Happiness Facilitation but High on Sadness Facilitation, F1 did predict Unprovoked Aggression $Wald \chi^2 (1, N = 137) = 5.16$; $B = -.73$, $p < .05$. A plot of this interaction can be seen in Appendix B, Figure 7.

Contrary to expectation, interaction analyses indicated that while a positive affective response to violence had an effect on predicting greater propensity to engage in Unprovoked Aggression, it did not interact with F1. Moreover, while Sadness Facilitation did interact with F1, it was in the opposite direction expected. Individuals who were high on F1 and Sadness Facilitation were more likely to shock their opponents before being shocked.

To test the consistency of this finding, another ordinal regression using orthogonal emotion facilitation scores to test for the 3-way and all 2-way interactions was conducted. Similar to the nonorthogonal ordinal regression, the orthogonal model proved to fit the data well, $\chi^2 (7, N = 137) = 30.22$, $p < .001$, $R^2_N = .23$. The Wald Statistic indicated a significant conditional effect for F1, $Wald \chi^2 (1, N = 137) = 11.17$; $B = -.51$, $p = .001$; but not for Happiness Facilitation, $Wald \chi^2 (1, N = 137) = 2.41$; $B = -.21$, $p > .10$; or Sadness Facilitation, $Wald \chi^2 (1, N = 137) = .72$; $B = .12$, $p > .10$. Similar to the nonorthogonal model, the interaction between F1 and Happiness Facilitation did not achieve significance, $Wald \chi^2 (1, N = 137) = .24$; $B = -.081$, $p > .10$; but the interaction between F1 and Sadness Facilitation did, $Wald \chi^2 (1, N = 137) = 7.93$; $B = -.42$, $p = .005$. Additionally, the interaction between Happiness and Sadness Facilitation approached significance, $Wald \chi^2 (1, N = 137) = 3.04$; $B = .20$, $p = .08$. The 3-way interaction was nonsignificant, $Wald \chi^2 (1, N = 137) = .56$; $B = -.11$, $p > .10$.

Simple ordinal regression analyses indicated that for individuals high on Happiness Facilitation and high on Sadness Facilitation, F1 strongly predicted increased probability of Unprovoked Aggression, $Wald \chi^2 (1, N = 137) = 12.00; B = -1.49, p = .001$. When individuals were high on Happiness Facilitation but low on Sadness Facilitation, the relationship between F1 and Unprovoked Aggression was nonsignificant, $Wald \chi^2 (1, N = 137) = .00; B = -.03, p > .10$. For individuals low on both Happiness Facilitation and Sadness Facilitation, the relationship between F1 and Unprovoked Aggression was again nonsignificant, $Wald \chi^2 (1, N = 137) = .26; B = -.16, p > .10$. For individuals low on Happiness Facilitation and High on Sadness Facilitation, F1 strongly predicted Unprovoked Aggression, $Wald \chi^2 (1, N = 137) = 4.71; B = -.99, p < .05$. A plot of this interaction can be seen in Appendix B, Figure 8.

Post-hoc Exploratory Analyses

Pleasantness Ratings of Violent Images. Overall, the model using affect composite scores (comparing each discrete affect to all other affects) and the model using orthogonal affect facilitation scores (comparing each discrete affect directly to neutral words) produced a highly similar pattern of results with similar statistical coefficients. A noteworthy finding was that for high F1 individuals, greater Sadness Facilitation predicted a greater propensity toward Unprovoked Aggression rather than less facilitation as had been hypothesized. If, in fact, greater Sadness Facilitation in high F1 individuals represents a greater danger to commit more severe types of violence, then it would follow that these individuals would be likely to rate violence as less negative. Therefore, a linear regression model using valence ratings of the violent imagery as the outcome variable was conducted with the same three predictors and all possible 2-way interactions plus the 3-way interaction. The overall model was significant $F(7, 128) = 5.14, p <$

.001, $R^2 = .22$. Regression coefficients indicated an average effect of F1, $t(129) = 5.60$, $B = .46$, $p < .001$, and an interaction between F1 and Sadness Facilitation, $t(129) = 2.00$, $B = .17$, $p < .05$.

Simple regression analyses indicated that for individuals high on Happiness Facilitation and high on Sadness Facilitation, F1 significantly predicted rating images as less unpleasant, $t(129) = 3.88$, $B = .81$, $p < .001$; whereas, for individuals high on Happiness Facilitation and low on Sadness Facilitation, F1 did not predict image ratings $t(129) = .82$, $B = .17$, $p > .10$. For individuals low on both Happiness and Sadness Facilitation, F1 predicted rating images as less unpleasant, $t(129) = 2.95$, $B = .42$, $p < .005$; and for individuals low on Happiness Facilitation and high on Sadness Facilitation, F1 also predicted rating the images as less unpleasant, $t(129) = 2.87$, $B = .46$, $p = .005$. A plot of this interaction can be seen in Appendix B, Figure 9.

When this analysis was repeated using the orthogonal affect scores, the pattern of results and regression coefficients were nearly identical. The full model containing the 3-way interaction and all 2-way interactions plus first-order terms was significant, $F(7, 128) = 5.30$, $p < .001$, $R^2 = .23$. Regression coefficients indicated an average effect of F1, $t(129) = 5.07$, $B = .43$, $p < .001$, and the interaction between F1 and Sadness Facilitation approached significance, $t(129) = 1.89$, $B = .18$, $p = .06$.

Simple regression analyses indicated that for individuals high on Happiness Facilitation and high on Sadness Facilitation, F1 significantly predicted rating images as less unpleasant, $t(129) = 4.85$, $B = .72$, $p < .001$; whereas, for individuals high on Happiness Facilitation and low on Sadness Facilitation F1 did not predict image ratings, $t(129) = .83$, $B = .19$, $p > .10$. For individuals low on both Happiness and Sadness Facilitation, F1 predicted rating images as less unpleasant, $t(129) = 2.02$, $B = .29$, $p < .05$; and for individuals low on Happiness Facilitation and

high on Sadness Facilitation, F1 also predicted rating the images as less unpleasant, $t(129) = 2.42$, $B = .48$, $p < .05$. A plot of this interaction can be seen in Appendix B, Figure 10.

Ratings of Perceived Pain/Distress of the Victim in Violent Images. The complex regression model for the 3-way interaction was computed using ratings of perceived pain/distress as the criterion variable and F1, plus the composite affect scores as the predictors. The full model was significant, $F(7, 128) = 5.30$, $p < .005$, $R^2 = .16$. Regression coefficients indicated an average effect of F1, $t(129) = -4.26$, $B = -.36$, $p < .001$, and the 3-way interaction among F1, Happiness Facilitation, and Sadness Facilitation achieved significance, $t(129) = -2.39$, $B = -.22$, $p < .05$. Additionally, the interaction between F1 and Sadness Facilitation approached significance, $t(129) = -1.69$, $B = -.15$, $p = .09$.

Simple regression analyses indicated that for individuals high on Happiness Facilitation and high on Sadness Facilitation, F1 strongly predicted ratings of less pain/distress, $t(129) = -3.94$, $B = -.83$, $p < .001$; whereas, for individuals high on Happiness Facilitation and low on Sadness Facilitation, F1 did not predict ratings of pain/distress, $t(129) = -.45$, $B = -.09$, $p > .10$. For individuals low on both Happiness and Sadness Facilitation, F1 predicted rating images as portraying less pain/distress experienced by the victims, $t(129) = -2.28$, $B = -.33$, $p < .05$; and for individuals low on Happiness Facilitation and high on Sadness Facilitation, F1 did not predict ratings of perceived pain/distress, $t(129) = -1.11$, $B = -.18$, $p > .10$. A plot of this interaction can be seen in Appendix B, Figure 11.

The analyses were repeated using the orthogonal affect scores. The full model was significant, $F(7, 128) = 3.72$, $p = .001$, $R^2 = .17$. Regression coefficients indicated an average effect of F1 $t(129) = -3.84$, $B = -.33$, $p < .001$ and a significant interaction between Happiness Facilitation and Sadness Facilitation, $t(129) = -2.35$, $B = -.17$, $p < .05$.

Simple regression analyses indicated that for individuals high on Happiness Facilitation and high on Sadness Facilitation, F1 strongly predicted ratings of less pain/distress, $t(129) = -4.10$, $B = -.62$, $p < .001$; whereas, for individuals high on Happiness Facilitation and low on Sadness Facilitation F1 did not predict ratings of pain/distress, $t(129) = -1.39$, $B = -.33$, $p > .10$. For individuals low on both Happiness and Sadness Facilitation, F1 did not predict pain/distress experienced by the victims, $t(129) = -1.25$, $B = -.19$, $p > .10$; and for individuals low on Happiness Facilitation and high on Sadness Facilitation F1 did not predict ratings of perceived pain/distress, $t(129) = -.93$, $B = -.19$, $p > .10$. A plot of this interaction can be seen in Appendix B, Figure 12.

Unprovoked vs. Provoked Aggressors

General Aggression. To test the hypothesis that Unprovoked Aggressors were more aggressive relative to their provoked counterparts, a contrast was calculated using GA. However, as Unprovoked Aggressors, by nature of the task, had a larger range of opportunities to aggress, it is possible that the statistical difference between the two groups could be attributed to the first five trials. Consequently, aggression variance of unprovoked trials had to be removed. Therefore, to remove the variance contributed during the unprovoked block, aggression of Unprovoked and Provoked Aggressors evinced during the final 25 trials only was compared. Results indicated that Unprovoked Aggressors evinced more aggressive behavior than their provoked counterparts, $t(112) = 4.94$, $p < .001$, $d = .93$. See Appendix B, Figure 13.

Initial Aggression. To test whether Unprovoked Aggressors demonstrate significant levels of aggression in the absence of provocation, levels of IA were compared between Unprovoked and Provoked Aggressors. Results indicated that Unprovoked Aggressors' IA was not significantly less than Provoked Aggressors' IA, $t(112) = .64$, $p > .10$, $d = .12$. As such, data

suggest that Unprovoked Aggressors did not merely administer a swift, mild shock. In fact, although not significantly greater, Unprovoked Aggressors IA was actually more than Provoked Aggressors ($z = .2371$ vs. $z = .1488$).

Unprovoked vs. Provoked Aggression. Unprovoked Aggressors level of IA indicated that they employ significant aggression in the absence of provocation. Moreover, their overall aggression in the unprovoked block was as great as their aggression in the provoked block, $t(67) = -.91, p > .10, d = .17$. These data suggest that Unprovoked Aggressors did not merely administer a single shock as a warning; but rather, initiated a pattern of persistent aggression. In fact, Unprovoked Aggressors GA during the 5-trial unprovoked block was greater than Provoked Aggressors GA during 25-trial provoked block, $t(113) = 4.39, p < .001, d = .83$. See Appendix B, Figure 14.

CHAPTER 4

DISCUSSION

The purpose of the present investigation was to replicate and expand on previous research which demonstrated that, in a nonforensic population of men, psychopathy, in particular emotional detachment, predicts greater probability of engaging in unprovoked aggression. Specifically, we aimed to identify the emotional experience which may motivate psychopathic individuals to engage in unprovoked aggression. In doing so, we tested two proposed statistical models in which emotional experience either 1) mediates or, 2) moderates the relationship between the emotional detachment factor of psychopathy and unprovoked aggression. The results of the present investigation supported many of the proposed hypotheses.

First, the present findings shed light on the nature of unprovoked aggression and the risk for sustained aggression that it carries. Although psychopathic traits of participants predict unprovoked aggression, the present data also indicate that Unprovoked Aggressors evinced more aggression than those who waited until provocation materialized, even when variance from unprovoked trials was removed. Moreover, the nonsignificant difference between Unprovoked and Provoked Aggressors on IA indicates that Unprovoked Aggressors initiated aggression absent of provocation at levels commensurate to Provoked Aggressors' initial response to provocation. As such, Unprovoked Aggressors were not merely "testing the waters" by administering one minor shock, rather, their aggression was severe and sustained. Hence, the unprovoked aggressor may also be the perpetrator whose aggression leads to the most severe consequences.

As expected, psychopathic traits were positively related to the use of both trait aggression and behavioral indices aggression. Additionally, the present results replicate the findings by Reidy, Zeichner, and Martinez (in press) and confirm hypotheses that psychopathy is significantly related to the engagement in unprovoked aggression; and, that when controlling for factor covariance, Factor 1 predicted greater probability of Unprovoked Aggression while Factor 2 was nonsignificant. Contrary to expectation, Factor 1 did not predict greater Happiness Facilitation in response to the viewing of violent imagery; however, as predicted, Factor 1 did appear to be related to a decrease in Sadness Facilitation following the viewing of violent imagery. Moreover, while decreased Sadness Facilitation after viewing violent imagery was not related to Unprovoked Aggression, increased Happiness Facilitation in response to viewing violent imagery was related to an increased propensity toward engaging in Unprovoked Aggression.

These findings are notable for several reasons. The happiness facilitation – unprovoked aggression relationship suggests the existence of a sadistic motivation for perpetration of aggressive behavior. That is, individuals who demonstrated greater happiness in response to violence were more likely to engage in unprovoked aggression suggesting that the pleasure they experience from violence motivates its occurrence. Moreover, the lack of relationship between Factor 1 and Happiness Facilitation in response to violent imagery suggests that this sadistic motive for aggression exists independent of psychopathy. In addition, the relationship between Factor 1 and decreased Sadness Facilitation did not predict an increased propensity for Unprovoked Aggression. Taken as a whole, these findings indicate that the relationship between Factor 1 and unprovoked aggression is not mediated by these particular emotional experiences.

Consequently, the first model was rejected and testing of the moderation model in which Factor 1 interacts with Happiness and Sadness Facilitation was initiated.

The results of the moderation model provide consistent but mixed support for the interaction of the three variables. First, in the full model, contrary to expectation, the 2-way interaction between Factor 1 and Happiness Facilitation did not reach significance nor did the interaction between Happiness and Sadness Facilitation. Perhaps the most notable finding was the significant interaction between Factor 1 and Sadness Facilitation. Although Factor 1 was expected to be moderated by Sadness Facilitation, it was hypothesized that a negative interaction would exist in which decreases in Sadness Facilitation would increase the relationship between Factor 1 and Unprovoked Aggression. However, the interaction term indicated that increases in Sadness Facilitation significantly increased the relationship between Factor 1 and Unprovoked Aggression. The lack of significance for the regression coefficient of the hypothesized 3-way interaction suggests that the Factor 1 – unprovoked aggression relationship should be equivalent for individuals high on both Sadness and Happiness Facilitation and individuals high on Sadness Facilitation but low on Happiness Facilitation. However, when simple regressions were computed, a consistent pattern appeared in which the strongest relationship between Factor 1 and Unprovoked Aggression existed when individuals were high on both Sadness and Happiness Facilitation. It is noteworthy that these results were strikingly similar when the model was tested using nonorthogonal emotion facilitation scores and orthogonal emotion facilitation scores (see Figures 7 and 8).

The implications of these findings for the two models may be pertinent to the theoretical and practical assessment and classification of psychopathic individuals. For example, if the mediated model had been confirmed it would lend support to potential theory of homogeneity

among psychopathic violent offenders and their motivations for perpetrating violent crime. However, evidence for the moderated model supports the argument that there may be multiple psychopathy typologies in which the offender, his violent crimes, and motivations may differ drastically. Skeem, Poythress, Edens, Lilienfeld, and Cale, (2003) note that “If variants of psychopathy can be identified reliably and supported empirically, they may improve our ability to understand, treat, and manage a class of individual who have largely been regarded as dangerous, incurable cases” (p. 515). The notion that there may be variants of psychopathic personalities has existed since Cleckley (1941) first described the core deficits of this disorder (for a review see Skeem, et al., 2003). Karpman (1941) postulated that psychopaths could be identified as either primary or secondary. According to Karpman, both primary and secondary psychopaths are typified by hostile and antisocial behavior, but are drastically differentiated by their etiology and motivation for such behavior. Primary psychopaths are thought to reflect a biological predisposition to an emotional deficit whereas secondary psychopaths suffer a conditioned emotional neurosis attributable to environmental causes such as physical abuse perpetrated by a parent (Karpman, 1948). Moreover, Karpman believed that secondary psychopaths possess a conscience, markedly absent in primary psychopaths, which allow the secondary psychopath to demonstrate occasional displays of guilt or need for acceptance.

Karpman’s distinction between the two types of psychopaths resembles the current conceptualization of psychopathy, in which, Factor 1 is analogous to primary psychopathy and Factor 2 is analogous to secondary psychopathy. Likewise, the distinction between primary and secondary psychopathy may be pertinent to the distinctions between Unprovoked and Provoked Aggressors. For example, Karpman (1955) suggests that the primary psychopath often acts deliberately and instrumentally to maximize gains or excitement in contrast to the secondary

psychopath who acts impulsively and hostilely in a reactive manner. The primary psychopath is a cold-blooded aggressor while the secondary psychopath is a “hot-headed, impulsive” aggressor (Skeem et al., 2003). In the present sample, the differentiating disposition between Unprovoked Aggressors and Provoked Aggressors may represent the same dispositions that differentiate primary from secondary psychopaths. Moreover, Karpman (1946, 1955) distinguished between two forms of primary psychopaths: the aggressive predatory and the passive-parasitic variants. The predatory psychopath actively seeks out his victim against whom he can perpetrate his violent inclinations whereas the passive-parasitic psychopath only resorts to violent persuasion when a easily exploitable victim is not readily available. In the present sample, individuals high on Factor 1 who displayed more Sadness Facilitation after viewing violent imagery were at significantly greater risk to perpetrate Unprovoked Aggression. It is possible that these unexpected results reflect the presence of a predatory typology of psychopathy traits, under which the individual is guided by recognizing socially-appropriate responses to emotional and interpersonal interactions. These individuals may not experience empathy, but may be able to access and display the appropriate cognitions to feign compassion and sincerity. This ability to feign the appropriate emotional responses may facilitate the perpetration of the predatory acts. In fact, this speculation is supported by treatment outcome research indicating that treatment programs aimed at teaching empathy and prosocial coping skills to psychopaths actually leads to increased violent recidivism rates in this population (e.g., Rice, Harris, & Cormier, 1992; Seto & Barbaree, 1999). Moreover, this paradoxical outcome may be related specifically to the emotional detachment factor of psychopathy (Hare, Clark, Grann, & Thornton, 2000).

The literature on the treatment of psychopathy indicates that when the highly emotionally detached individuals has the necessary skills to cognitively access and demonstrate emotionally-

prosocial responses despite the inability to actually feel them, they may use this skill to commit more egregious and violent crimes. This speculation is consistent with the present results which indicate that the high Factor 1 - high Sadness Facilitation individuals were significantly more likely to initiate Unprovoked Aggression. Moreover, this pattern of trait and emotion facilitation predicted that these individuals' ratings of violence as being significantly less unpleasant. Additionally, this pattern emerged when both the orthogonal and nonorthogonal emotion facilitation scores were used in the model. In other words, the emotionally-detached individuals who were able to demonstrate sadness facilitation consistently rated the violent images as less unpleasant.

This pattern was again found when ratings of victim pain/distress were entered as the criterion variable. Using nonorthogonal emotion facilitation scores yielded a significant positive interaction between Factor 1 and Sadness Facilitation. High Factor 1 men who demonstrated greater Sadness Facilitation rated victim pain/distress at significantly lower levels. Furthermore, when this model was repeated using nonorthogonal emotion facilitation scores, a similar trend appeared between Factor 1 and Sadness Facilitation. However, most notable was the significant outcome of the 3-way interaction. The emotionally detached individual who demonstrated a concurrent increase in Happiness and Sadness Facilitation rated the victims' pain and distress as significantly less. Although this finding differs statistically from the prediction of unprovoked aggression and ratings of pleasantness, it is highly consistent with the pattern revealed by the simple regression analysis, in which, the strongest relationship between Factor 1 and the criterion variable (i.e., Unprovoked Aggression and Pleasantness ratings) existed for individuals who demonstrated a concomitant positive facilitation of Happiness and Sadness (see Figures 7 through 12).

This finding may lend further support to the existence of a socially skilled, “predatory” typology of psychopathic personality. It may suggest, similar to Karpman’s two variants of predatory-violent primary psychopaths, that there were two variants in the present sample. These two variants may have had two different underlying emotional motivations for the perpetration of aggression. The first type (high Factor 1, high Sadness Facilitation, and low Happiness Facilitation), characterized by high emotional detachment and the necessary skills to demonstrate feigned prosocial emotional responses, is a predator who may utilize aggression to satisfy his needs or desires such as in the case of instrumental violence. The second variant (high Factor 1, high Sadness Facilitation, and high Happiness Facilitation) differs from the first in that it combines the predatory nature with sadistic motives for engaging in aggression. These individuals may, not only possess the core emotional deficit that precludes the development of empathy, and the manipulative and exploitive skills necessary to feign empathy, they may also be conditioned to experience intrinsic pleasure in response to violence which exacerbates their risk for violence. Although there was not a statistical difference between the “nonsadistic predatory” psychopathy type and the “sadistic predatory” psychopathy type in the present sample, inspection of the standardized coefficients for the simple regressions predicting Unprovoked Aggression reveals that the “sadistic predatory” individual had coefficients nearly one half standard deviation larger than the “nonsadistic predatory” individual. Moreover, these large differences in the magnitude of prediction existed on ratings of pleasantness and ratings of victim pain/distress.

We are not the first to propose the existence of sadistic psychopathic subtype. For example, Holt, et al., (1999) postulated that sadism was an endogenous component common to all psychopaths, but that this was only a minor component of the trait combination. In contrast,

Stone (1998) theorized that there is only a subtype of sadistic psychopaths which exists.

Notably, based on their clinical observations at a forensic psychiatric hospital, Murphy and Vess (2003) identified four classes of psychopaths, one of which was a sadistic type. According to these authors, the sadistic variant displays prominent evidence of deriving pleasure from the suffering of others. His distinguishing characteristic is the capacity to recognize the suffering of others and the corresponding pleasure associated with that suffering (Murphy & Vess, 2003).

This speculation may be congruent with the results of the present investigation in which “sadistic predatory” type participants presented with increased Sadness and Happiness Facilitation. In the present sample, the increased Sadness Facilitation may reflect the capacity to recognize the suffering of others while the increased Happiness Facilitation might reflect pleasure derived from suffering. Similarly, while this “sadistic predator” subtype individual did not rate images as pleasant, such individuals rated these stimuli as significantly less unpleasant. This finding may signify an interaction between the presence of exploitive skills required for faking prosocial responses (preventing them from giving ratings on the positive side of the scale) and the presence of sadistic traits which mitigates the negative ratings of pleasantness.

The present results indicate that, in terms of the GAM, varied inputs may have differential importance in the determination of unprovoked aggression. Theorists have espoused provocation as one of the most important or, perhaps, a necessary determinant of aggressive behavior (e.g., Anderson and Bushman, 2002; Berkowitz, 1993; Caprara et al., 1983; Geen, 2001; Lau et al., 1995). However, the present investigation, in which participants were truly unprovoked and unprimed for aggression, demonstrated that trait factors may be more important than provocation in the perpetration of this type of aggression. These findings are congruent with Zillman and Weaver (2007) who found that highly hostile men committed socially-unprovoked

aggression regardless of a violent or nonviolent prime. Relatedly, Richardson et al. (1998) found that dispositional perspective-taking outweighed the influence of aggressive provocation in inhibiting aggressive responding. This may suggest that certain dispositional factors (e.g., psychopathy, empathy, hostility) comprise a more pathological combination of traits that is of greater importance in the perpetration of unprovoked aggression.

In the present sample, the best indicator of increased probability of unprovoked aggression was the degree of Factor 1 psychopathy traits (i.e., emotional detachment and lack of empathy) the individual possessed. This pattern of results is congruent with recent research suggesting that distinct personality factors may have specific relationships to different types of aggression. For example, Reidy, Zeichner, Miller, and Martinez (2007) found that Factor 1 traits positively related to both instrumental and hostile/reactive aggression, whereas Factor 2 psychopathy positively related only to hostile/reactive aggression. Similarly, Barry, Thompson, Barry, Lochman, Adler, and Hill (2007) examined the relationship between subdimensions of psychopathy (narcissism, callous/unemotional traits, and impulsivity) and found that narcissism predicted both proactive and reactive aggression, whereas impulsivity predicted only reactive aggression. However, the latter authors found that when other traits were controlled, the callous/unemotional dimension did not relate to either form of aggression. The similarity between findings linking psychopathy and narcissism to aggression in general may indicate that narcissism could be linked to increased risk of unprovoked aggression. Congruent with this speculation, some evidence suggests that narcissism is positively associated with primary but not secondary psychopathy (Hare, 1991; McHoskey, Worzel, & Szyarto, 1998; Reidy, Zeichner, Hunnicutt-Ferguson, & Lilienfeld, in press), most likely the same factor related to perpetration of unprovoked aggression.

Murphy and Vess (2003) identified a narcissistic subtype of psychopathic personality in addition to the sadistic subtype. According to these authors, the narcissistic variant can be seen in an individual who embodies the characteristics of psychopathy but whose clinical presentation includes primarily narcissistic features of a pathological degree. Grandiosity, entitlement, and callous disregard for the feelings of others are likely to be the most evident features in this individual. Congruent with this view, Reidy, Zeichner, Foster, and Martinez (2008) recently found that subfactors of narcissism related to aggression are Entitlement and Exploitativeness. These subfactors were also found to positively correlate with Factor 1 psychopathy ($r = .46$ and $r = .53$ respectively) but not Factor 2 (Reidy, Zeichner, Hunnicutt-Ferguson, and Lilienfeld, in press). In fact, displaced aggression (i.e., aggression directed at an “innocent” party uninvolved in provocation), a form of unprovoked aggression, has been seen in individuals who endorse high levels of narcissistic traits (Martinez, Zeichner, Reidy, and Miller, 2008; Twenge and Campbell, 2003). Consequently, a narcissistic variant of psychopathy may exist in which a particularly potent combination of traits may lead to unprovoked aggression. Future research may seek to investigate the existence of a narcissistic variant of psychopathy and compare its relative prediction to the “sadistic” and “nonsadistic predatory” variants of psychopathy.

The findings of the present investigation must be interpreted with caution for several reasons. First, the obtained sample of participants was relatively homogeneous, as participants were predominantly single, Caucasian high school graduates who are enrolled in a university. Certainly, such a sample does not fully represent the general population. This study would be strengthened by the inclusion of women, non-collegiate participants, and greater ethnic diversity. Second, this investigation does not allow determination of causality, and other factors that were not accounted for may have affected the relationship between psychopathy and aggressive

behavior. Third, in considering levels of psychopathic traits obtained in this study, clinically pathological levels of psychopathy were not represented in the sample. Expanding this area of research to forensic populations would increase the predictive validity of the findings. This may also improve identification of at-risk individuals across settings and who perpetrate varied degrees of violence. Finally, in this paradigm, we created conditions in which experimental provocation (in terms of shocks administered to a participant) was absent. However, it is likely that each situation is replete with stimuli a potential perpetrator may find provoking, and that any stimulus may have a specific interactive effect with a given disposition. For example, although participants were not provoked in the initial block of trials, they all believed that they could be shocked. Thus, the unprovoked portion of the task could have been “threat-laden” for those individuals with a hostile attribution bias. As such, future laboratory studies should seek to investigate the complex nature of provocation in other paradigms to help elucidate the causes of this form of aggression.

Nevertheless, the present study adds to the literature in several ways. First, it supports previous research that indicates that psychopathy is positively associated with aggression using both trait and behavioral indices. Second, it replicates the results of Reidy, Zeichner, & Martinez, (in press) which indicate that psychopathy, in particular Factor 1, predicts an increased probability of initiating unprovoked aggression. Moreover, it expands this research by replicating it with multiple measures of psychopathy and assessing affective motives of the unprovoked aggressor. Furthermore, it advances the literature because it is, to our knowledge, the only experimental investigation of psychopathy and sadistic motives for aggression. Third, patterns of emotional responding lend support for the speculation that there are multiple typologies of psychopathic personalities that may have a heterogeneous symptomatology,

patterns of violence, and amenability to treatment. Moreover, the present methodology offers a method of assessing and classifying the personality variants based on differential emotional functioning. We endorse the assertion made by Skeem and Colleagues who said that, “Clearly, if variants were characterized by different risk factors for, and pathways to, antisocial and violent behavior, this finding would have key implications for violence risk assessment, management, and treatment. Before assessment and treatment can be tailored to individuals as a function of differential psychopathic/antisocial features, their feature constellations must be identified” (2003, p. 515).

This study and others like it have the potential to expand our understanding of aggression, violent crimes, and the personality characteristics that predict these variables. Researchers have indicated that identifying the motivation of the violent acts a person commits may provide invaluable insight into the likelihood of future violence (Woodworth & Porter, 2002). Law-enforcement agencies, court systems, and parole boards could potentially use this knowledge to facilitate criminal investigations, determine prison sentences, and decide who is least likely to recidivate if paroled. Furthermore, it is hoped that information gained from this and similar studies may serve to inspire new treatment plans and inform therapeutic programs to diminish intervention-client mismatch. Illumination of the intrinsic motivators of aggression will inform the assessment of, and development of interventions for, individuals at risk to commit future violent acts.

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APPENDIX A

TABLES

TABLE 1
Correlations among Psychopathy Indices

	1	2	3	4	5	6	7
1. PPI Total	-	.55*	.65*	.68*	.49*	.39*	.53*
2. SRP F1		-	.57*	.89*	.73*	.41*	.73*
3. SRP F2			-	.88*	.55*	.58*	.64*
4. SRP Total				-	.72*	.56*	.77*
5. LSRP F1					-	.41*	.94*
6. LSRP F2						-	.69*
7. LSRP Total							-

Note. PPI = Psychopathic Personality Inventory; SRP = Self-Report Psychopathy Scale - III; LSRP = Levenson Self-Report Psychopathy Scales; F1 = Factor 1; F2 = Factor 2.

* $p < .001$.

TABLE 2
Correlations among Aggression Indices

	1	2	3	4	5	6	7
1. GA	-	.54***	.42***	-.78***	.28***	.20*	.10
2. IA		-	.57***	-.55***	.10	.20*	.22*
3. P10			-	-.34***	.16 [†]	.19*	.13
4. FP				-	-.28***	-.20*	-.16 [†]
5. BAQ					-	.66***	.53***
6. RA						-	.67***
7. PA							-

Note. GA = General Aggression; IA = Initial Aggression; P10 = Extreme Aggression; FP = Flashpoint Latency; BAQ = total scale self-reported trait aggression derived from BAQ; RA = Reactive Aggression; PA = Proactive Aggression.

* $p < .05$; ** $p < .001$; *** $p < .0001$

Table 3
Coefficients for Psychopathy & Aggression Indices

Variable	GA	IA	P10	FP	BAQ	RA	PA
PPI	.22**	.24**	.09	-.32***	.35***	.34***	.40***
TP	.31***	.29***	.14 [†]	-.36***	.56***	.56***	.65***
F1 ^a	.19 [†]	.31***	-.02	-.24*	.32***	.29***	.24**
F2 ^a	.17	.01	.18 [†]	-.18 [†]	.35***	.38***	.48***

Note. PPI = Psychopathic Personality Inventory; TP = Total Psychopathy Composite Score; F1 = Factor 1 Psychopathy Composite Score; F2 = Factor 2 Psychopathy Composite Score; GA = General Aggression; IA = Initial Aggression; P10 = Extreme Aggression; FP = Flashpoint Latency; ^a = Coefficients from Simultaneous Regressions.

[†] < .10; * $p < .05$; ** $p < .01$; *** $p < .001$

Table 4

Correlation Coefficients for General Psychopathy & LDT Affect Indices

Variable	Fear	Anger	Sadness	Happiness	Disgust
<u>Neutral – Discrete Affect</u>					
PPI	.02	.00	-.13	-.09	-.03
TP	.03	.00	-.08	-.05	.06
<u>Affect Composite – Discrete Affect^a</u>					
PPI	.09	.04	-.12	-.03	.02
TP	.05	.02	-.10	-.06	.08

Note. PPI = Psychopathic Personality Inventory; TP = Total Psychopathy Composite Score; ^a = Represents Partial Correlation Coefficients.

Table 5

Regression Coefficients for Psychopathy Factors & LDT Affect Indices

Variable	Fear	Anger	Sadness	Happiness	Disgust
<u>Neutral – Discrete Affect</u>					
F1	-.17 [†]	-.02	-.22 [*]	.02	-.01
F2	.19 [*]	.03	.14	-.09	.06
X	.16 [*]	.05	.02	.01	.05
<u>Affect Composite – Discrete Affect</u>					
F1	-.10	.09	-.18 [†]	.12	.06
F2	.14	-.06	.09	-.20 [*]	.02
X	.11	-.02	-.05	-.06	.01

Note. F1 = Factor 1 Psychopathy Composite Score; F2 = Factor 2 Psychopathy Composite Score; X = Interaction term between F1 and F2.

[†] < .10; * p < .05.

Table 6
Coefficients for LDT Affect Indices & RCAP Indices

Variable	Fear	Anger	Sadness	Happiness	Disgust
<u>Neutral – Discrete Affect</u>^a					
GA	-.09	-.04	-.12 [†]	.10	.10
IA	-.17 [*]	.04	-.14 [†]	-.03	.16 [†]
P10	.07	-.12	-.14	-.05	.35 ^{**}
FP	.15	.03	.18 [†]	-.18 [†]	.02
<u>Affect Composite – Discrete Affect</u>^b					
GA	-.08	-.01	-.16 [†]	.12	.10
IA	-.11	.07	-.14	-.03	.18
P10	.01	-.10	-.12	-.11	.25 ^{**}
FP	.04	-.04	.15 [†]	-.18 [*]	.03

Note. GA = General Aggression; IA = Initial Aggression; P10 = Extreme Aggression; FP = Flashpoint Latency; ^a = Represents Regression Coefficients; ^b = Represents Partial Correlation Coefficients.
[†] < .10; * $p < .05$; ** $p < .01$.

APPENDIX B

FIGURES

FIGURE 1A

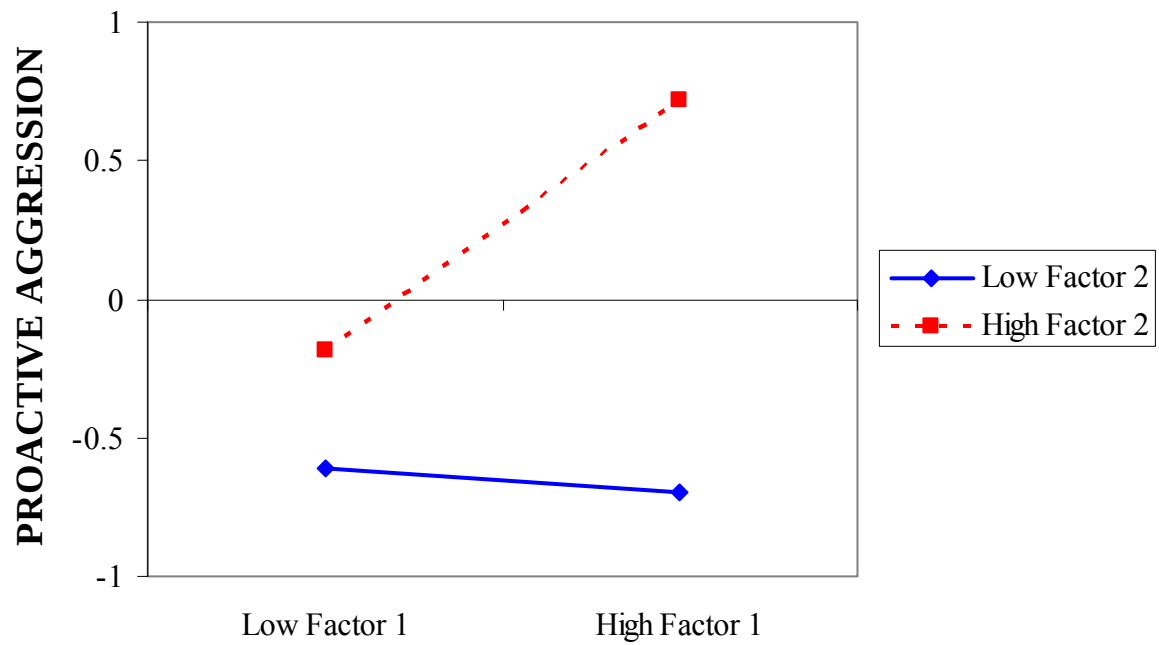
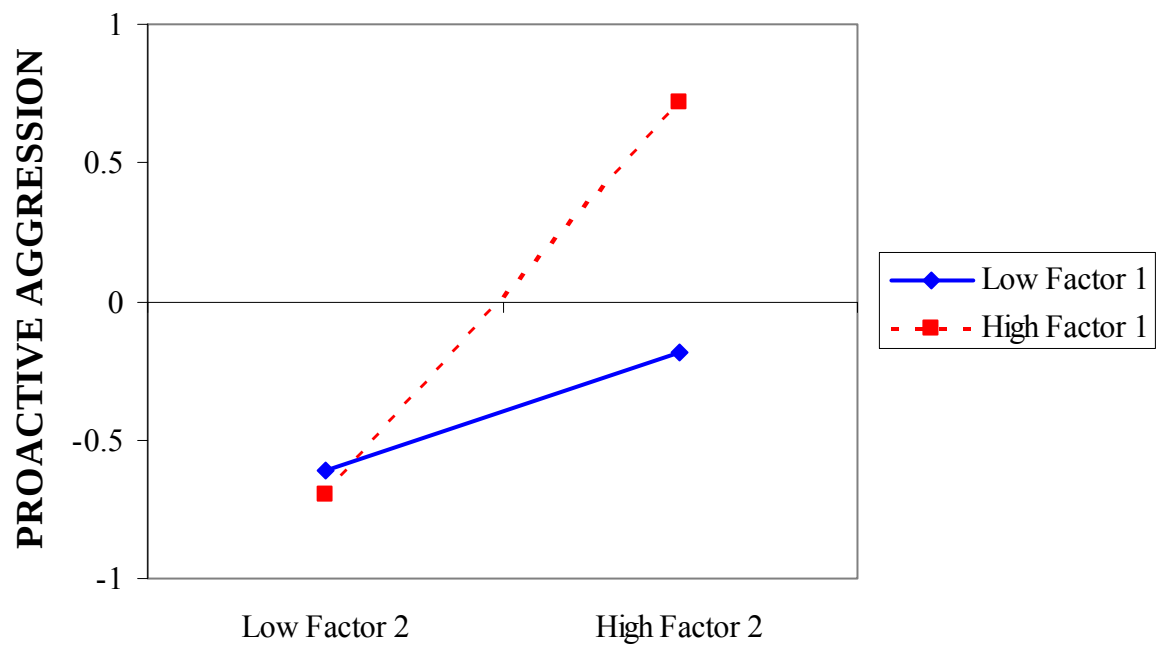


FIGURE 1B



Interaction between F1 & F2 on Proactive Aggression.

FIGURE 2A

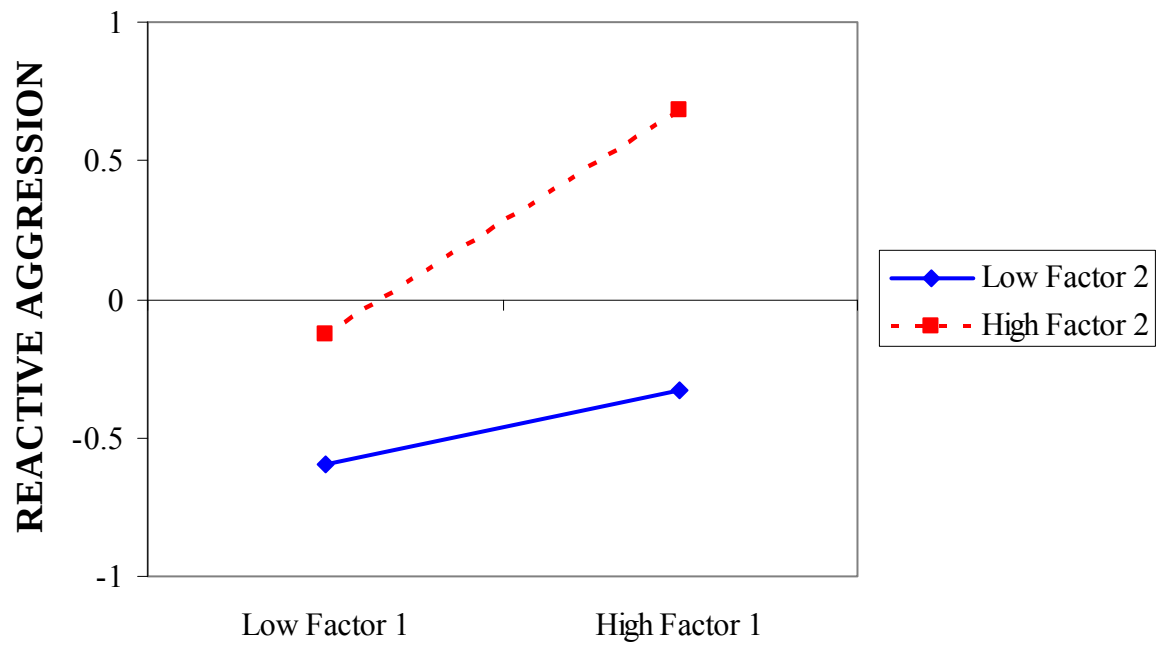
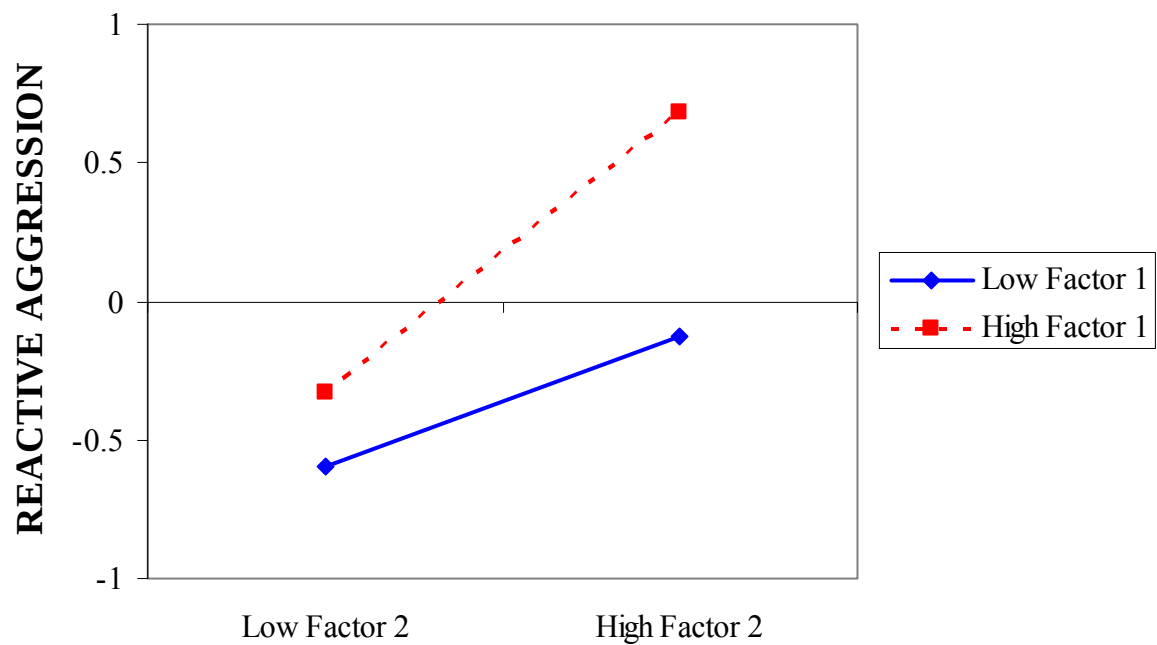


FIGURE 2B



Interaction between F1 & F2 on Reactive Aggression.

FIGURE 3A

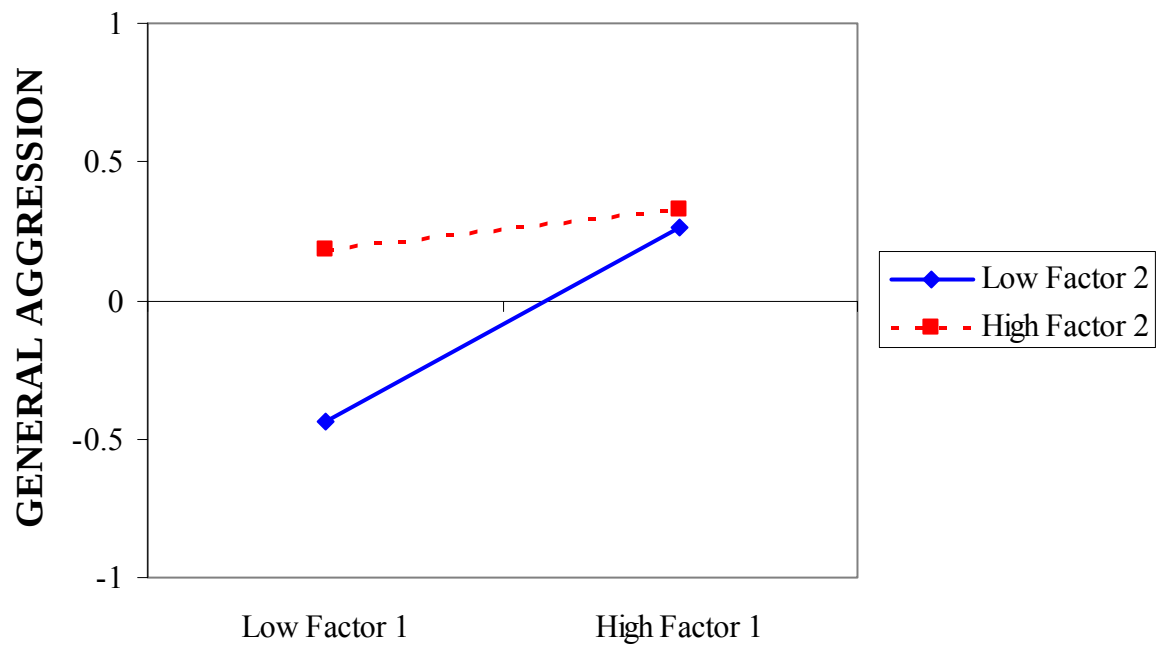
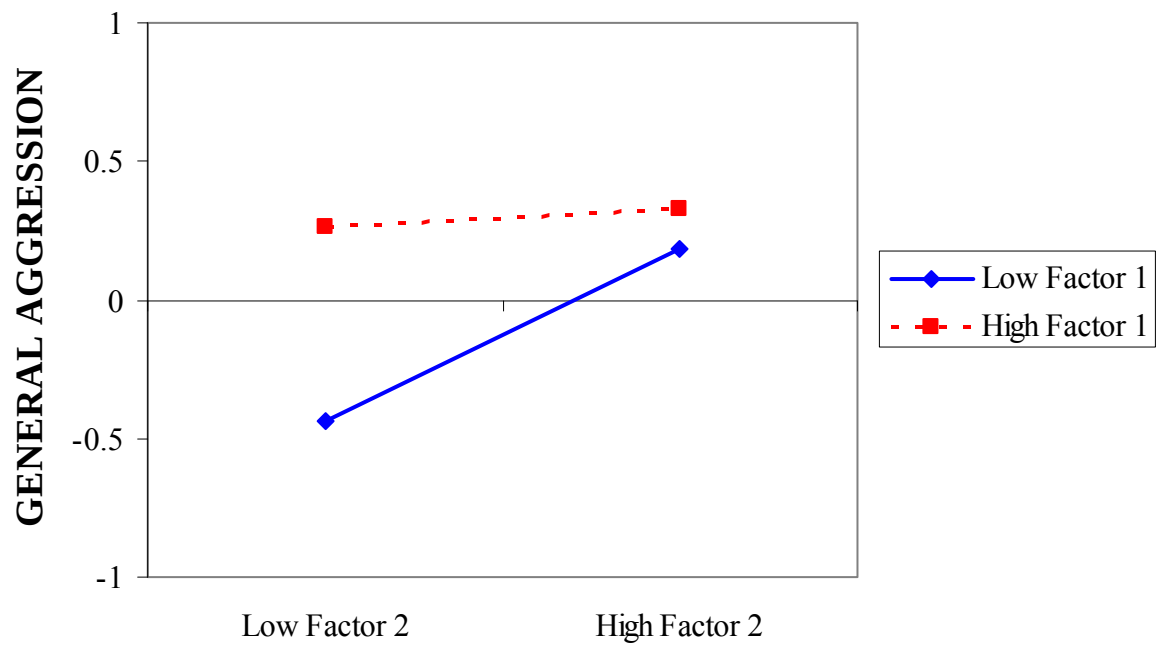


FIGURE 3B



Interaction between F1 & F2 on General Aggression.

FIGURE 4A

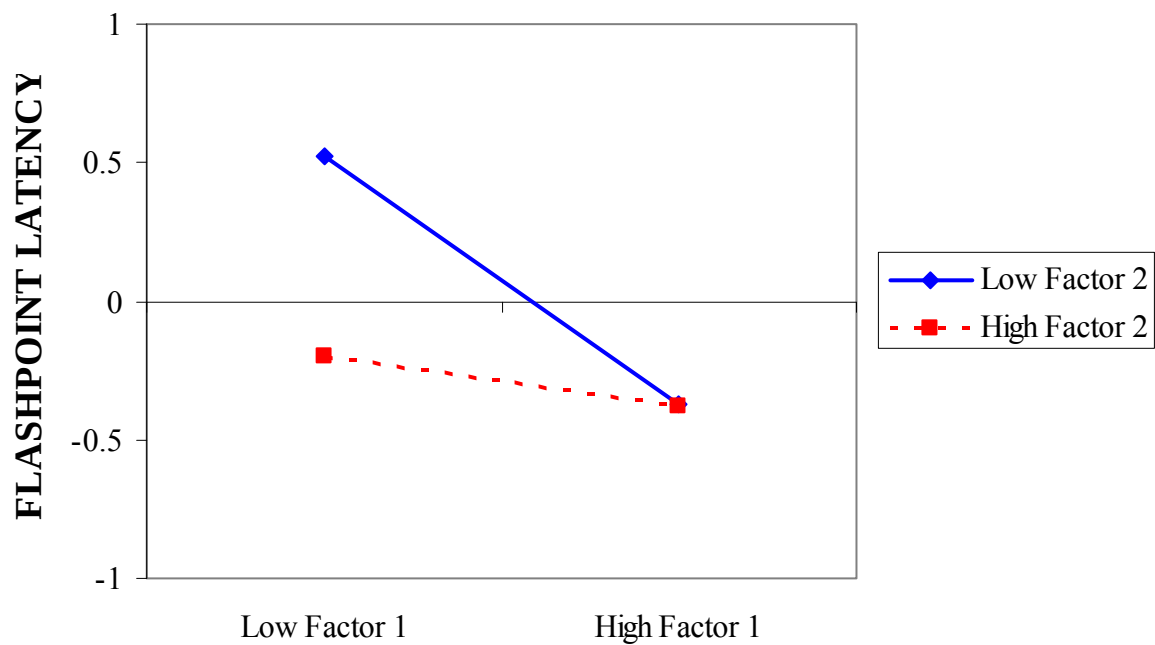
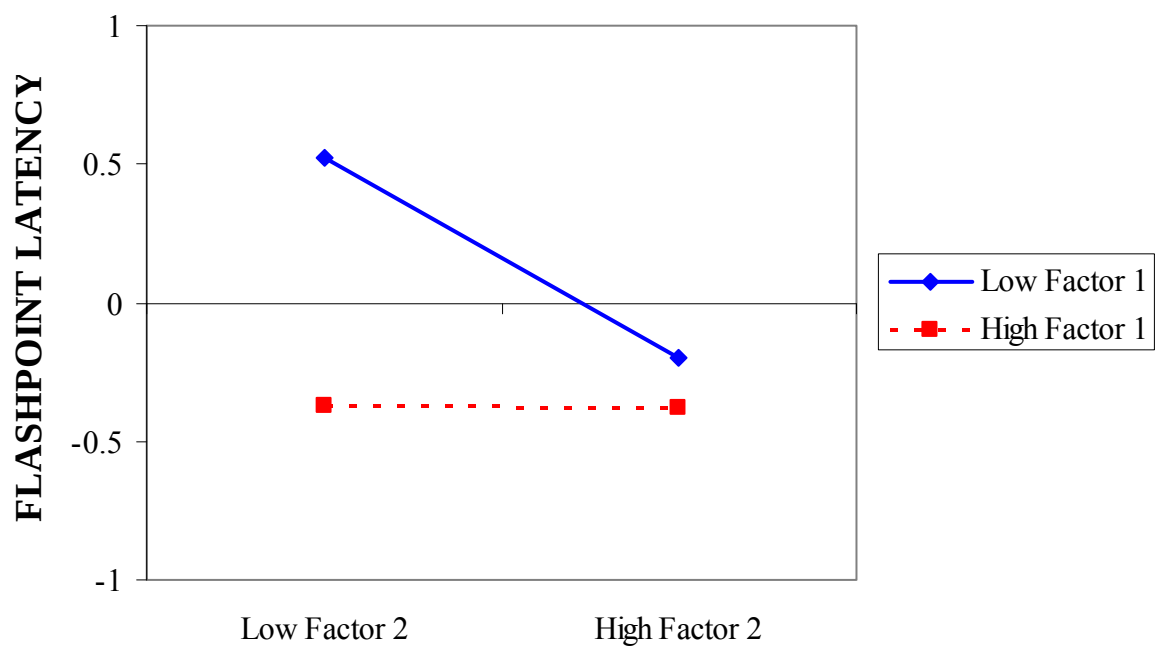


FIGURE 4B



Interaction between F1 & F2 on Flashpoint Latency.

FIGURE 5A

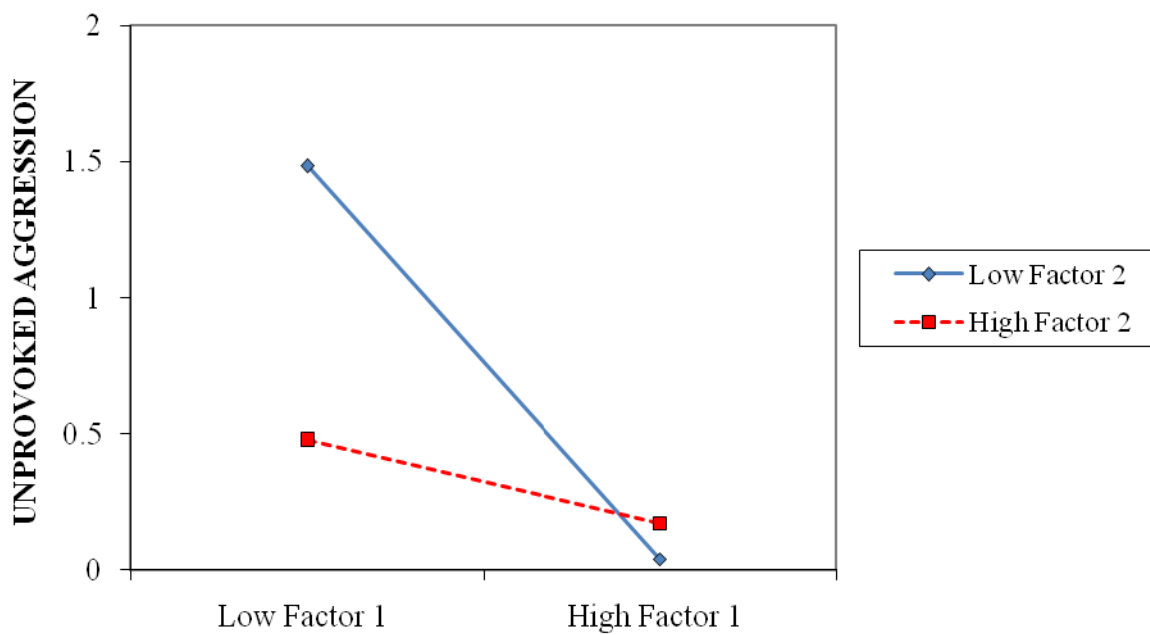
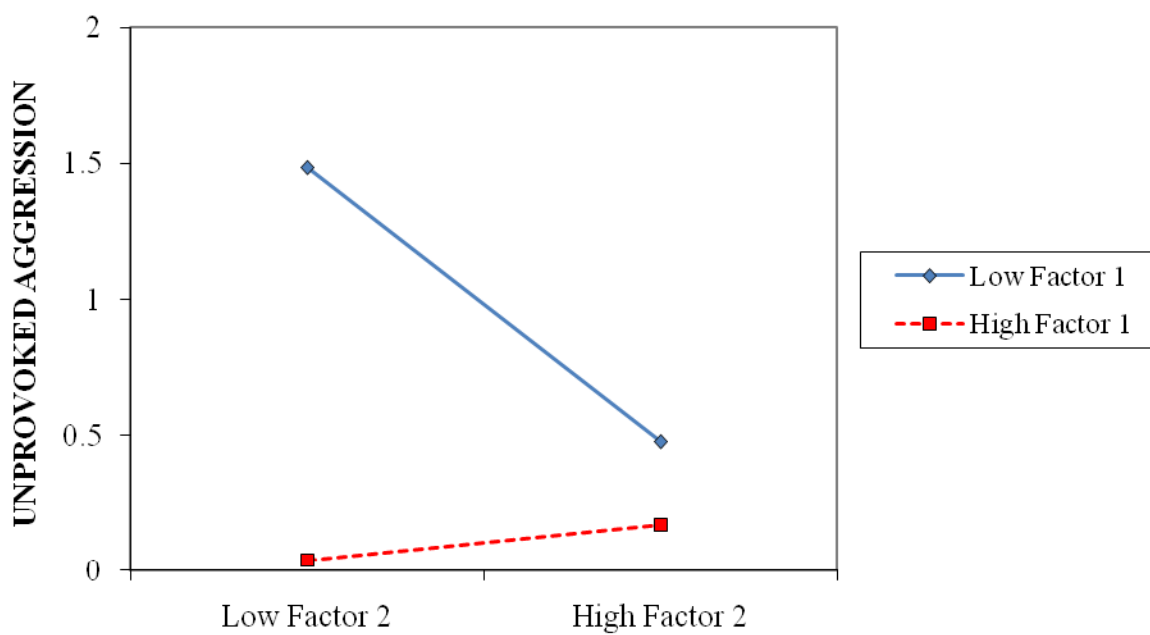


FIGURE 5B



Interaction between F1 & F2 on Unprovoked Aggression.

FIGURE 6A

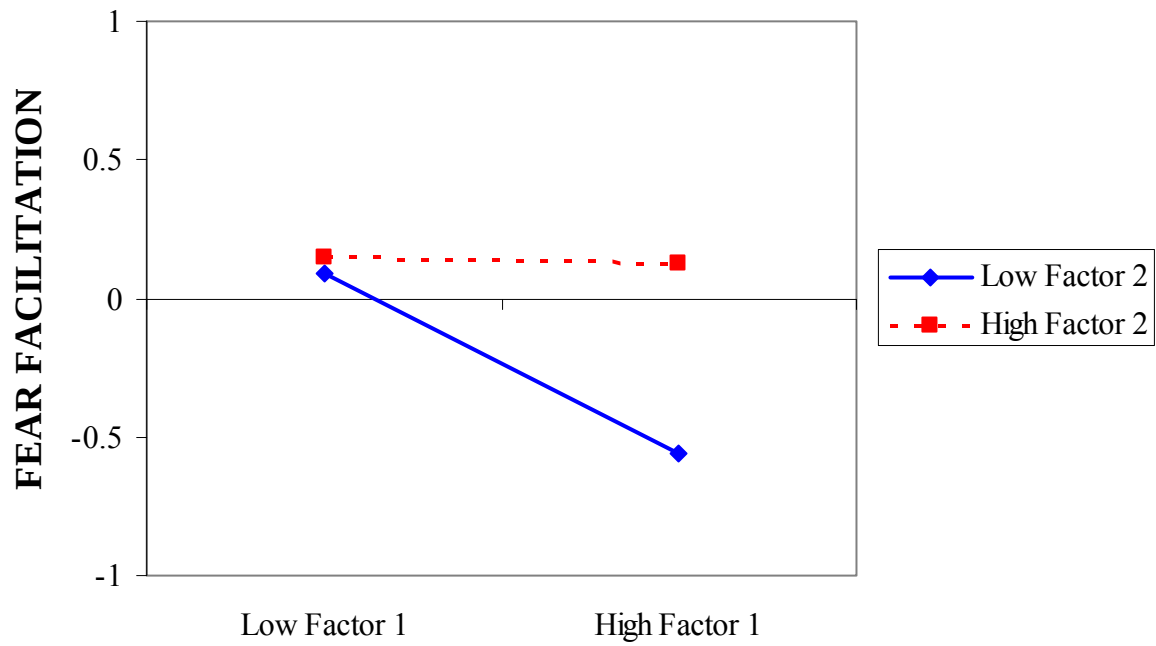
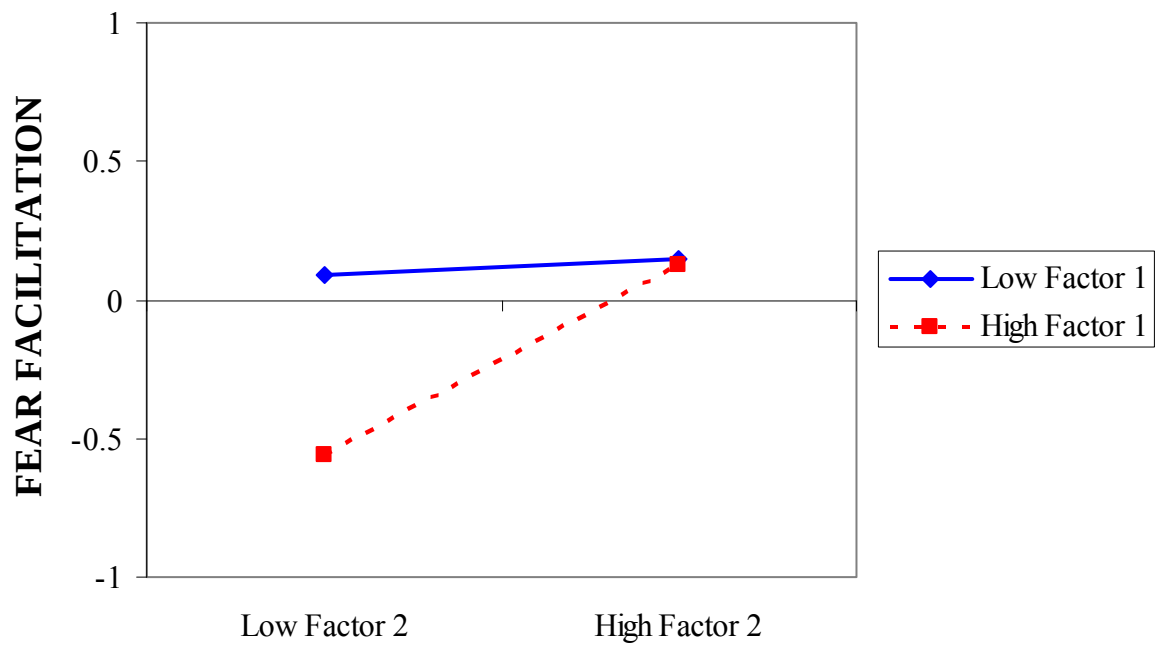
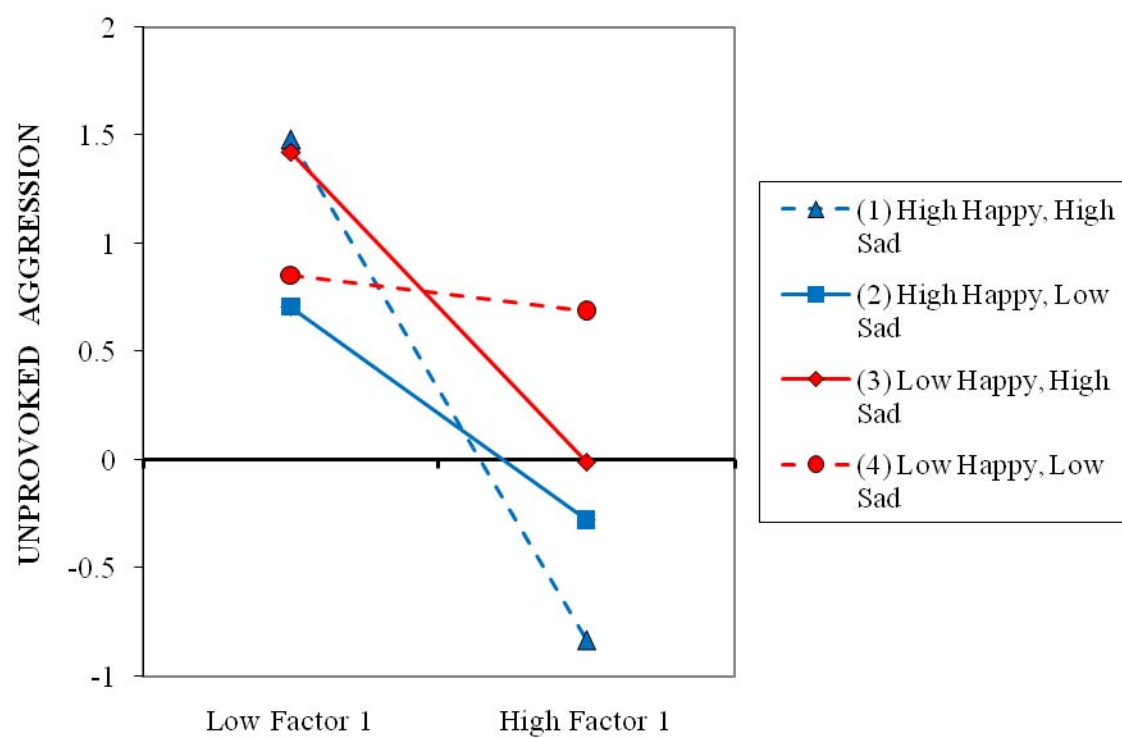


FIGURE 6B



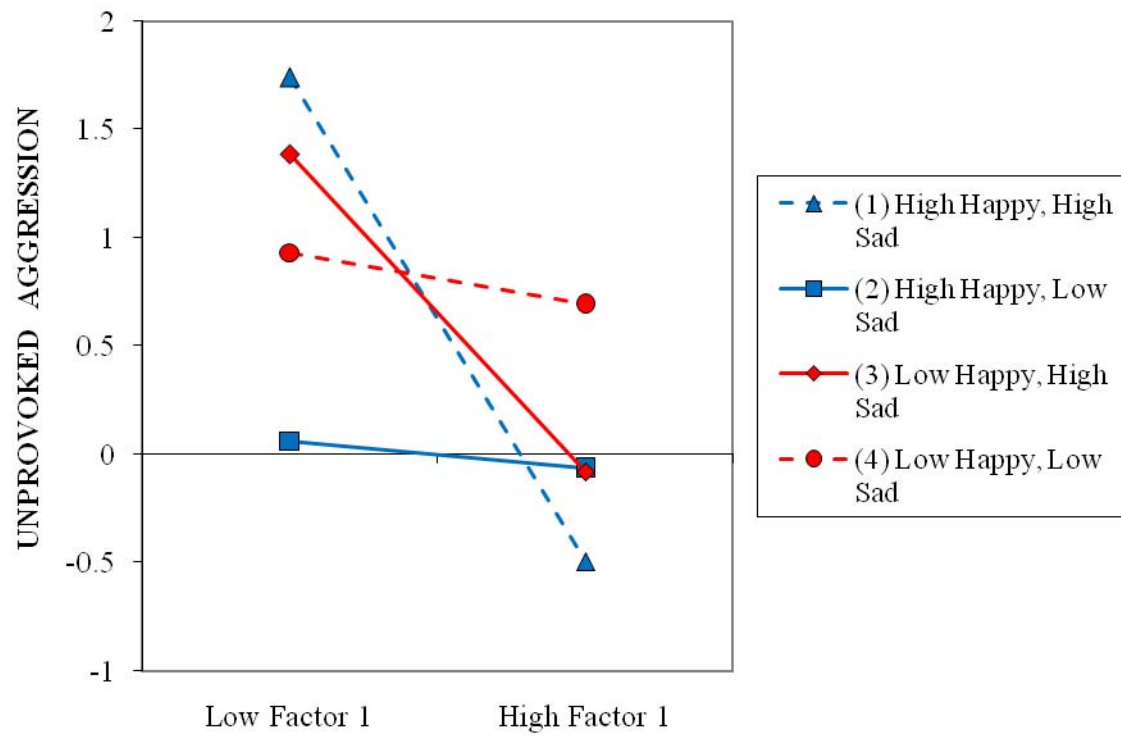
Interaction between F1 & F2 on Orthogonal Fear Facilitation.

FIGURE 7



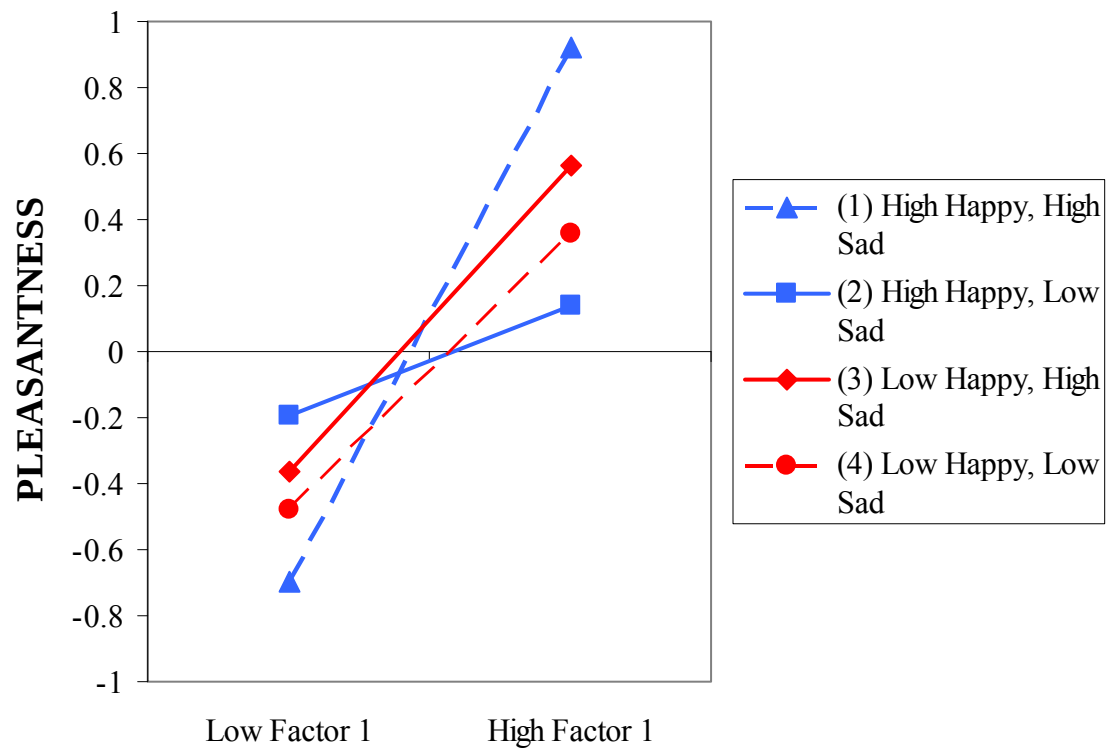
Interaction among F1, Happiness Facilitation, & Sadness Facilitation on Unprovoked Aggression Using Composite Affect Facilitation Scores.

FIGURE 8



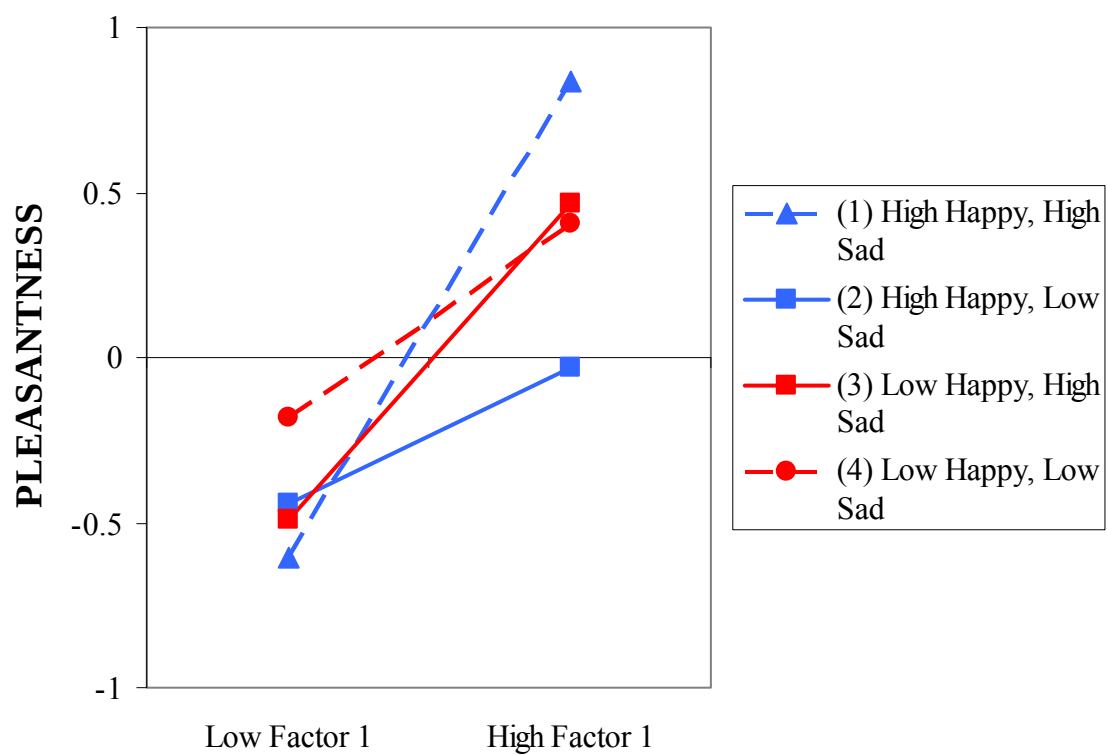
Interaction among F1, Happiness Facilitation, & Sadness Facilitation on Unprovoked Aggression Using Orthogonal Affect Facilitation Scores.

FIGURE 9



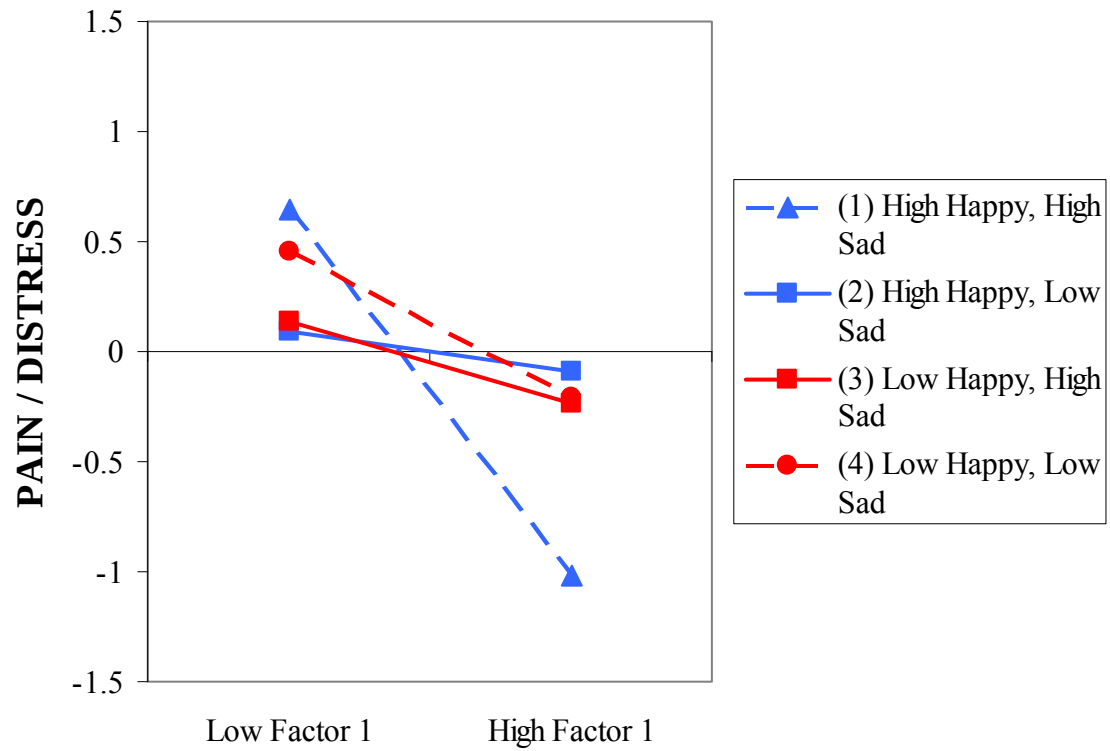
Interaction among F1, Happiness Facilitation, & Sadness Facilitation on Ratings of Valence of Violent Images Using Composite Affect Facilitation Scores.

FIGURE 10



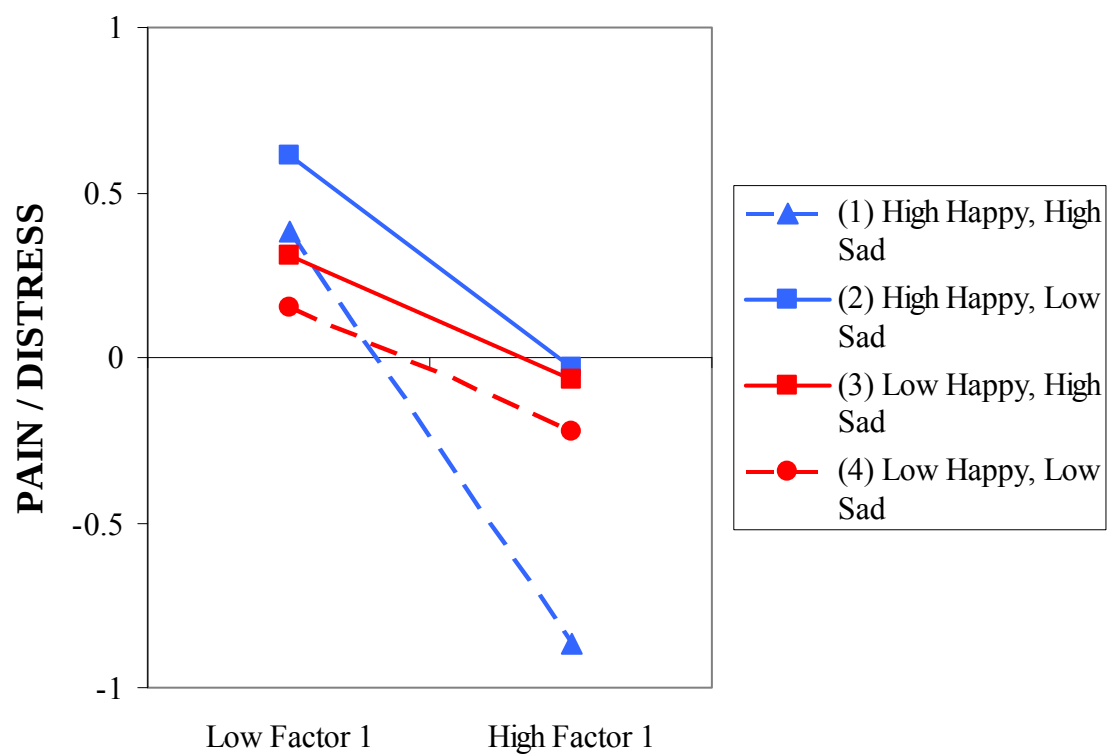
Interaction among F1, Happiness Facilitation, & Sadness Facilitation on Ratings of Valence of Violent Images Using Orthogonal Affect Facilitation Scores.

FIGURE 11



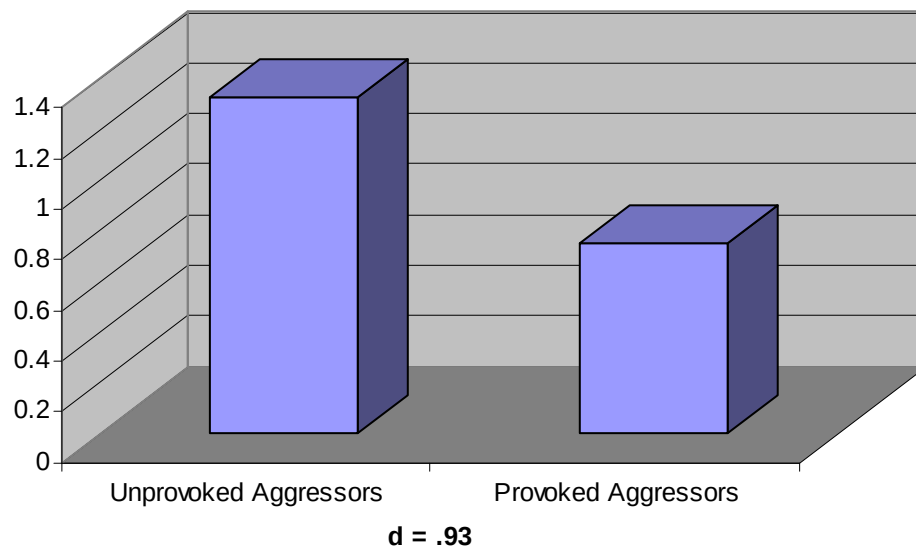
Interaction among F1, Happiness Facilitation, & Sadness Facilitation on Ratings of Victims' Pain/Distress Using Composite Affect Facilitation Scores.

FIGURE 12



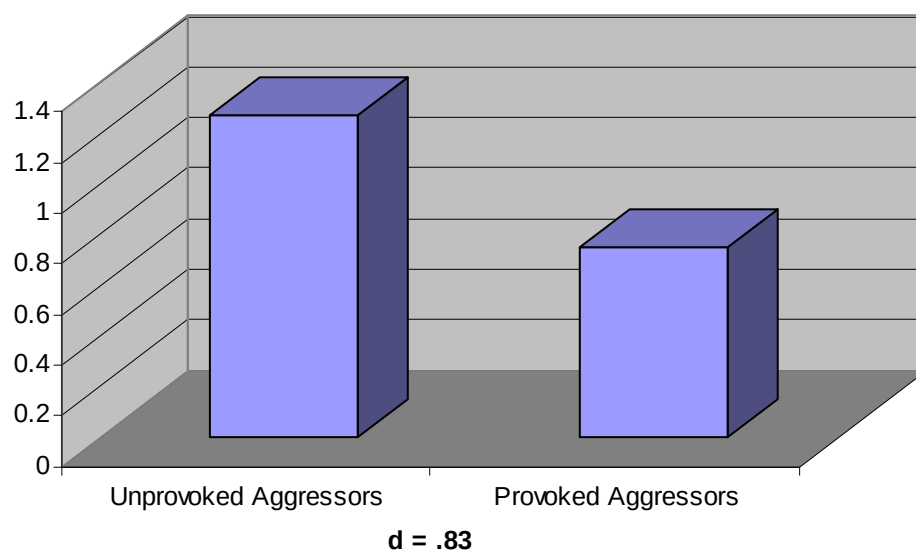
Interaction among F1, Happiness Facilitation, & Sadness Facilitation on Ratings of Victims' Pain/Distress Using Orthogonal Affect Facilitation Scores.

FIGURE 13



Mean Difference of Unprovoked & Provoked Aggressors' Level of GA during the Provoked Block of Trials.

FIGURE 14



Mean Difference of Unprovoked Aggressors' GA during Unprovoked Block & Provoked Aggressors' GA during Provoked Block.