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The University of Southern Mississippi

AN ELECTROPHYSIOLOGICAL INVESTIGATION OF THE COGNITIVE PROCESSES UNDERLYING PROVOKED AGGRESSION IN HUMANS

by

Jennifer Renee Fanning

Abstract of a Dissertation
Submitted to the Graduate School
of The University of Southern Mississippi
in Partial Fulfillment of the Requirements
for the Degree of Doctor of Philosophy

August 2011

ABSTRACT

AN ELECTROPHYSIOLOGICAL INVESTIGATION

OF THE COGNITIVE PROCESSES UNDERLYING

PROVOKED AGGRESSION IN HUMANS

by Jennifer Renee Fanning

August 2011

Recently, the event-related potential (ERP) technique has been applied to questions of social information processing. Several studies have examined standard and social information processing variables in aggressive individuals, but little is known about the neurophysiological processes that take place in real-time during an aggressive encounter. In this study, 48 men and women high and low in aggression history exchanged noise blasts of varying intensity with a (fictitious) opponent in a modified version of a well-validated laboratory-controlled behavioral measure of aggression, the Taylor Reaction-time Task (Taylor, 1967), while ERPs were simultaneously being recorded at scalp sites. Mixed model ANOVAs were used to analyze differences between aggressive and non-aggressive men and women in the neurophysiological processes related to perceiving provocation and responding to threat. Dependent variables included mean amplitude and latency for the following ERP components: P3 (to provocation), N2, No-Go P3, N450, and the negative slow wave (NSW). Aggressive participants were more likely to make errors in identifying the high provocation stimuli while non-aggressive participants were not. Non-aggressive participants showed greater and slower processing of provocative stimuli as evidenced by larger P3 amplitude and later P3 peak. During the aggression trials, aggressive participants were more likely to administer a "high" noise blast to the opponent under conditions of low provocation. Both the aggressive and non-aggressive groups made greater use of the "high" noise blast following high provocation. Effects were also observed for components

previously linked to inhibitory processes (N2, No-Go P3, N450, and NSW). The results suggest that aggressive and non-aggressive individuals process personally relevant threat information differently. More research is needed to understand how ERP components putatively linked to inhibitory cognitive processes relate to aggression in real-world encounters. Further implications and future directions are discussed.

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A Dissertation
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August 2011

DEDICATION

I dedicate this dissertation to my family, including my father, John, brother, Michael, and late mother, Linda.

ACKNOWLEDGMENTS

I would like to thank those who helped me through the process of completing this dissertation. In particular, I would like to thank my advisor, Dr. Mitchell Berman, for being a wonderful mentor throughout this project and throughout graduate school. I would also like to thank my committee members, Drs. Tammy Barry, Bradley Green, and Randolph Arnau (USM, Department of Psychology), for their advice, assistance, and feedback along the way. I would like to thank Eileen Todd and Nick Hammond for their assistance with technical aspects of the project. I owe thanks as well to Mr. Billy Scruggs for his generous assistance in recruiting participants. Very special thanks go to my family for their support.

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LIST OF ABBREVIATIONS

LL_P	-	Low provocation block (P) oddball stimulus. The stimulus is "LOW"
LM_P	-	Low provocation block (P) frequent stimulus. The stimulus is "MEDIUM"
HH_P	-	High provocation block (P) oddball stimulus. The stimulus is "HIGH"
HM_P	-	High provocation block (P) oddball stimulus. The stimulus is "MEDIUM"
LL_A	-	Aggression block (A) following the low provocation block (L). The participant's response choice is "LOW"
LM_A	-	Aggression block (A) following the low provocation block (L). The participant's response choice is "MEDIUM"
LH_A	-	Aggression block (A) following the low provocation block (L). The participant's response choice is "HIGH"
HL_A	-	Aggression block (A) following the high provocation block (H). The participant's response choice is "LOW"
HM_A	-	Aggression block (A) following the high provocation block (H). The participant's response choice is "MEDIUM"
HH_A	-	Aggression block (A) following the high provocation block (H). The participant's response choice is "HIGH"
f	-	Frequent stimuli. Comprising 80% of trials in the provocation blocks.
o	-	Oddball stimuli, Comprising 20% of trials in the provocation block.
C	-	Central electrode sites, comprised of sites C3, Cz, C4
F	-	Frontal electrode sites, comprised of sites F3, Fz, F4
P	-	Parietal electrode sties, comprised of sites P3, Pz, P4
A	-	Aggressive participants
NA	-	Non-aggressive participants

CHAPTER I

INTRODUCTION

Significance of the Study

Aggression is a serious and widespread social problem that affects millions of people each year in the United States and abroad. Aggression and violence incur enormous costs in both human and economic terms. Globally, approximately 3.2 million men and 1.9 million women are physically assaulted each year. In the United States, an estimated 51.9% of women and 66.4% of men have been assaulted at some point during their lifetime (NIJ/CDC, 2000). In 2004, The World Health Organization (WHO) estimated that the cost of interpersonal violence in the United States alone totals more than $261 billion per year, or approximately 3.3% of the country's gross domestic product. The average lifetime medical treatment cost for a single gun violence victim in the United States is between $37,000 and $42,000. Furthermore, victims of violence and their families are not the only ones who are affected. Much of the economic burden of violence is borne by government and taxpayer dollars. Intervention programs aiming to reduce violence are not only mandated ethically, they are also viable economically - prevention programs that are targeted at reducing interpersonal aggression and violence have been shown to save more money than they cost (WHO, 2004).

Biological, situational, cognitive, and emotional factors all play a role in the expression of aggressive behavior. Threat and provocation are among the most robust and replicated situational predictors of interpersonal, interspecies, and intergroup aggression. Biological influences on aggression include cerebral structural, functional, neurochemical, and hormonal factors (Garza-Trevino, 1994; Patrick & Verona, 2007; Saver, Salloway, Devinsky, & Bear, 2000; Volavka, 1990). Executive functioning (EF) and social information processing are cognitive factors which have been shown to relate to aggressive responding to provocation (Dodge & Crick, 1994; Giancola & Zeichner, 1994). Little is known, however, about the real-time neurocognitive processes that underlie the perception of and response to threat or

provocation. This study addresses a critical gap in the literature by examining electrophysiological differences between participants with and without significant histories of aggressive behavior in real-time physiological processes that are associated with the evaluation of provocative cues and the expression or inhibition of aggressive behavior under neutral and provoking conditions. First, the literature on aggression under conditions of provocation will be reviewed. Second, a brief description of the event related potential (ERP) technique will be given. Next, a review of studies on aggression and associated constructs using ERP techniques in both clinical and non-clinical samples will be provided. Finally, objectives, rationale, and hypotheses for the present study will be offered.

CHAPTER II

REVIEW OF LITERATURE

Aggression

Considerable research has been conducted in the field of psychology on interpersonal aggression, its precursors, and its consequences. Aggression has been defined as an overt behavior, which can be physical, indirect, or verbal, that has the intended goal of harming another person against their will (Berkowitz, 1990). Aggression is a multiply-determined behavior that manifests in many ways (Anderson & Bushman, 2002). Subtyping of aggression has been controversial and fraught with empirical and definitional difficulties, but researchers who study aggression have often distinguished at least two types of aggression: *reactive* aggression and *proactive* aggression (Anderson & Bushman, 2002). Reactive aggression (also called hostile, impulsive, or affective aggression) historically has been conceptualized as intentional aggression in response to anger or provocation. Reactive aggression has also been described as a failure in the ability to self-regulate one's behavior. Proactive aggression (also known as instrumental aggression) has been conceptualized as premeditated aggression carried out in the service of achieving some other goal (i.e., inflicting harm is typically not the primary goal; Anderson & Bushman, 2002). More recent conceptualizations of aggression suggest that that the distinction between reactive and proactive aggression may have limited usefulness for studying real-world aggressive acts, as most aggressive acts have both hostile and instrumental components (Bushman & Anderson, 2001). For example, a batterer may feel "angry" for perceived slights or insults while assaulting a significant other, but may also engage in the behavior to alter the target's behavior to comply with the batterer's goals or intent. The psychiatric and clinical literatures have generally focused on reactive or *impulsive* aggression. For the purpose of this study, behavioral aggression that occured in response to threat or provocation was examined without concern for the nosological and subtyping issues that plague the field.

Several theories of aggressive behavior have been developed, each citing different factors proposed to influence aggression, including cognitions, emotions, situational factors, interpersonal factors, and personality traits (Baron & Richardson, 1994; Berkowitz, 1990). Anderson and Bushman (2002) organized these theories into a comprehensive and trans-theoretical model of aggression that they called the General Aggression Model (GAM). The GAM model posits that person variables (such as personality traits) and situational variables (such as a provocation) set into motion cognitive, affective, and arousal processes that mediate the appraisal of situations and decisions about how to respond to a situation. These appraisals and decision-making processes in turn lead to either thoughtful or impulsive action which can be observed as either aggressive or non-aggressive behavior. In a recent meta-analytic review, Bettencourt and colleagues examined the influence of provocation on aggression in humans. They found that provocative or threatening cues are a robust predictor of aggression, but that this effect is moderated by having a pre-disposition to aggression as well as by other personality traits (Bettencourt, Talley, Benjamin, & Valentine, 2006). This appears to be true for both men and women (Bettencourt & Miller, 1996). In sum, to understand aggressive behavior, contextual and personality variables must be considered. Furthermore, to understand how provocation and disposition to aggression influence the expression of aggression, we must also understand the cognitive processes underlying the interpretation of provocative stimuli, as well as the cognitive decision-making processes that lead to an aggressive response. Indirect, or offline, measures of cognitive functioning have already shown that a group of processes called *executive functioning* moderates the relation between provocation and the expression of aggressive impulses (Giancola & Zeichner, 1994), with lower executive functioning being associated with higher levels of aggression under provoking conditions. The purpose of this study was to approach the role of provocation in aggression in a novel way by examining real-time changes in participants' physiological brain functioning in response to provocative stimuli. This was possible through the use of a laboratory paradigm for simulating aggressive interactions in naïve participants and

through the use of electrophysiological (e.g., ERP) recording techniques. Importantly, the relationship between aggressive behavior and these real-time physiological processes were also examined.

Social Information Processing and Aggression

Social information processing theories hold that social cognitions underlie social behaviors. Dodge and Crick (1994) proposed several stages and substages of social information processing, including: (a) encoding cues, both internal and external, (b) interpreting cues, (c) clarifying goals, (d) accessing potential responses, (e) selecting a response, and finally, (f) enacting a response. As one might hypothesize, aggressive children have been found to display different patterns of social information processing than non-aggressive children (Crick & Dodge, 1996). In a review of social information processing biases in children and adolescents, Dodge and Crick (1990) noted that aggressive children made more hostile attributions in response to hypothetical scenarios, accessed a smaller number of potential responses, and evaluated aggressive responses more favorably and as more likely to result in desired outcomes, than did non-aggressive youth. Social information processing biases have also been observed in adults. Taylor and Epstein (1967) observed that aggressive retaliation on the TRT was associated with the extent which the participant interpreted their opponent's behavior as provocative or hostile.

Tests of facial affect recognition provide another way to examine social information processing biases. In these tests, participants are shown a picture of a face, and asked to identify what emotion the person is experiencing based on the expression. The results of such studies in aggressive and non-aggressive populations have been mixed. Hoaken and colleagues (2007) found that violent offenders made greater errors in identifying facial affect compared to non-violent offenders and control subjects. Neutral faces were more often misidentified as "disgust" and less often identified as "sad" by the violent offenders compared to the other groups. A study by Bowen and colleagues (2010) found that young girls with conduct disorder were less accurate at identifying happy and sad faces compared to a control group, but showed no difference in their

accuracy for angry faces. These studies demonstrate that the relationship of facial threat perception and aggression is complex.

Laboratory Measures of Aggression

Historically, it has been difficult to study aggression experimentally due to ethical considerations related to exposing participants to actual harm. However, laboratory paradigms have been developed that allow researchers to elicit and observe lesser forms of aggressive behavior under various experimentally manipulated conditions. These paradigms have typically provided the participant opportunities to be provoked by and "aggress" against another participant (usually fictitious) in the form of subtracting points or money, or by administering a noxious stimulus such as loud noises or electric shocks. These exchanges are often carried out in the context of a competitive cover-task, such as a reaction time competition, or in an appetitive task, for example, attempting to earn money through button presses. The use of laboratory-based paradigms to study aggression provides a high degree of internal validity, and the correlates of aggression found in "real-world" encounters parallel those observed in the laboratory, providing support for their external validity (Anderson & Bushman, 1997; Chermack, Berman, & Taylor, 1997; Giancola & Chermack, 1998; McCloskey & Berman, 2003). Provocation can be manipulated in these paradigms to mimic a real-life aggressive encounter by having the "other participant" exhibit increasingly threatening behavior in the form of increasing levels of shock or noise, or by stealing points from the participant (Anderson & Bushman, 1997; Chermack et al., 1998; Giancola & Chermack, 1998; McCloskey & Berman, 2003).

The Taylor Reaction-Time Task (TRT; Taylor, 1967) is a classic laboratory paradigm for studying physical aggression in response to provocation. A modified version amenable to ERP data collection was developed for use in this study. Over forty years of research has provided evidence for the TRT's validity and robustness to various modifications (see Anderson & Bushman, 1997; Giancola & Chermack, 1998; McCloskey & Berman, 2003). In the original TRT, participants compete against an increasingly provocative (fictitious) opponent in a reaction time

game during which electric shock is administered and received across a series of trials. Aggression is defined by the level of shock the participant sets for the opponent across the trials. Responding on the TRT is generally stable across trials unless subjected to experimental manipulation. That is, when the level of provocation by the "opponent" remains low and consistent across trials, the shock levels selected by participants tend to remain stable throughout the task (Chermack et al., 1997). In contrast to other paradigms, the TRT permits the administration of a noxious, ostensibly harmful stimulus. This stimulus mimics physical aggression and is a clear and unequivocal provocation or attack by the opponent. For these reasons, a modification of the TRT paradigm was developed for this study. Our goal in creating this task was to retain the most important features of the TRT while making the task more amenable to measuring event-related potentials.

Provocation

Scientists have long recognized the role of situational variables in provoking aggressive encounters (Bettencourt et al., 2006; Chermack et al., 1997; Giancola & Zeichner, 1994; Giancola & Chermack, 1998). The relationship between biological functioning and situational factors is complex; however, the interaction between biological factors and provocation has been demonstrated in a recent study by Berman, McCloskey, Fanning, Schumacher, and Coccaro (2009). Results from non-experimental studies indicate that aggression is associated with blunted serotonin functioning. A small number of experimental studies have attempted to manipulate serotonin (5-hyroxytryptamine; 5-HT) functioning in humans to determine if 5-HT plays a direct and causal regulatory role in human aggression (see Berman, Tracy, & Coccaro, 1997, for a review). The experimental research in the area has been mixed with some studies showing that experimentally altered 5-HT affects aggression in humans (see Berman et al., in press, for a recent review). One explanation proposed for the mixed findings has been that 5-HT modulates response to provocation in individuals with a history of aggression, who presumably have 5-HT imbalances. According to this theoretical model, situational cues (i.e., provocation), individual

differences variables (i.e., aggression history), and biological factors (5-HT functioning) must be considered in complex social behaviors, such as aggression. To test this model, Berman et al. (2009) recruited eighty men and women with significant (n = 40) and non-significant (n = 40) histories of aggression and randomly assigned them to receive either a specific serotonin reuptake inhibitor (SSRI; 40 mg paroxetine to augment serotonergic activity) or a placebo. All participants then completed the TRT and the groups' aggressive responses were compared at low and high levels of provocation by the fictitious opponent. The highest level of provocation involved a unique manipulation that represented a clear and unequivocal attack. At low levels of provocation, all participants behaved non-aggressively. However, participants who had a marked history of aggressive behavior and who received a placebo retaliated to provocation with high levels of aggressive responding (as measured by the levels of shock they selected to administer to the opponent). Participants with a history of aggression who had been administered 40 mg paroxetine several hours prior to the TRT responded to the provocation no differently than did non-aggressive participants. Thus, individuals with ostensibly impaired 5-HT activity (that is, individuals with a history of aggression who reflected impaired 5-HT activity peripherally by blunted hormonal response to the pharmacochallenge) responded with heightened aggression after attack, but acute 5-HT augmentation normalized their response to provocation. This study provided clear and unambiguous support for the notions that (a) central nervous system (CNS) functioning plays an important role in aggression, (b) that provocation is interpreted differently by individuals with an aggressive history, and (c) that this interpretation is regulated by central nervous system processes.

Whereas Berman et al. (2009) provided considerable support for the role of neurochemical functioning (impaired 5-HT response) and situational variables (provocation) in eliciting aggressive behavior, very little is known about neurophysiological processes that take place in real time in the central nervous system during an aggressive encounter. Accordingly, there is a need to examine neurophysiological functioning as it relates to aggressive behavior. The

purpose of the current study is to examine psychophysiological processes during a simulated aggressive encounter, as reflected in rapid changes in electrical activity in the brain in real time. We suspect that the timing and magnitude of neural electrophysiological events plays an important role in the expression or inhibition of aggressive behavior.

Psychophysiology

Psychophysiology has been defined as "the study of the relationships between physiologic measures and psychological states" (Scarpa & Raine, 1997, p. 376). Psychophysiological measures include heart rate (HR), skin conductance (SC), electroencephalogram (EEG), event related potentials (ERP), and hemodynamic techniques such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI; Luck, 2005; Patrick & Verona, 2007; Scarpa & Raine, 1997). While hemodynamic techniques for examining brain functions provide a high degree of spatial resolution (e.g., fMRI), they tend to be quite expensive to use and provide limited insight into temporally rapid changes in brain functioning. In contrast, electroencephalographic (EEG) techniques can be used with a high degree of temporal resolution of neural processes as they occur. Broadly defined, EEG is the recording of electrical activity in the brain by electrodes placed on the scalp. Electrical activity recorded at specific sites on the scalp reflects the local net effect of an amalgam of electrical activity taking place throughout the brain. Indeed, the electrical potential observed at a single point on the scalp is often generated by many different sources. For this reason, inferring localization of the source is limited at best (see Luck, 2005, for a discussion). Thus, in contrast to hemodynamic approaches, EEG provides a high degree of temporal accuracy but limited spatial accuracy. This makes EEG an important complement to other techniques for studying neurophysiology (Luck, 2005).

Event Related Potentials

Event related potentials (ERPs) have been defined as changes in electrical activity in the brain in response to a stimulus or event averaged across multiple presentations of the event (Luck,

2005; Scarpa & Raine, 1997). ERPs are relatively stable and replicable waveforms that occur within a certain temporal proximity to an event (Luck, 2005). Typically, ERPs that are observed in the context of research studies are generated through the presentation of auditory or visual stimuli. These stimuli set into motion a range of neural activity reflecting sensory, attentional, and higher order cognitive processes, including executive functions. At any given point in time, an electrical potential can be measured by an electrode placed in a given location on the scalp. The electrical potential is measured in microvolts (μV) and is either positively (+) or negatively (-) valenced. When plotted two dimensionally, ERP waveforms represent the magnitude (*y*) of potentials recorded at a given scalp site (or sites) plotted over time (*x*). By convention, positive deflections of the waveform are plotted downward and negative deflections upward.

The ERP waveform is broken up by time frames (or epochs) into "components" for more detailed analysis. An ERP component has been defined by Luck as a single positive or negative deflection in the ERP waveform (Luck, 2005, p. 10). The naming of components is based on the latency of the component which is time-locked to the presentation of the stimulus. Thus, a negative deflection of the ERP that occurs 100 ms post-stimulus onset is called "N100" (i.e., "negative 100"). The last two digits are often truncated, so that N100 is often reported as "N1." P2 represents a positive deflection of the ERP occurring on average approximately 200 milliseconds (ms) after the presentation of a stimulus. Researchers generally consider variability in amplitude and latency of components to reflect variations in cognitive processing of stimuli (Luck, 2005). Due to extraneous electrical activity ("noise"), researchers average together the ERPs generated by repeated presentations of a stimulus, typically twenty presentations or more (Luck, 2005). This allows for more reliable measurement of components' amplitudes and latencies.

Various ERP components have been described in the literature, and a wealth of research has been done to identify the cognitive processes that are represented by various components. A classic task in ERP research is the "oddball" task. In the traditional version of the task at least two

stimuli or classes of stimuli are presented visually or auditorily, and one of the stimuli appears on the majority (e.g., 80%) of trials while the other or others appear on a minority (e.g., 20% or 10%) of trials. The participant is told to respond to the target stimulus (the rarer stimulus), by pressing a response button. The presentation of the rare stimuli elicits the P3 component of the ERP. This effect is one of the most robustly observed in the ERP literature and has been the basis of much ERP research through variations on the task.

The P3 component is a mid-latency positive deflection of the ERP that peaks between 250 and 850 ms post-stimulus. P3 is thought to reflect the completion of stimulus evaluation processes that include identifying a stimulus, discriminating the stimulus, categorizing the stimulus, and selecting the correct response to the stimulus. More specifically, P3 amplitude has been interpreted as an index of the extent to which stimulus evaluation processes are taking place, while P3 latency has been interpreted as reflecting the duration of stimulus evaluation processes. P3 is thought to reflect largely cognitive ("top down"), not motor or sensory, processes (Duncan-Johnson & Donchin, 1982). Through various manipulations, the P3 component has been found to be sensitive to the probability of a stimulus, the participant's expectation of the stimulus probability, the salience of the stimuli, the relevance of the stimulus to the task and to the participant, and the emotional salience of the stimulus (De Pascalis, Stippoli, Riccardi, & Vergari, 2004; Naumann, Bartussek, Diedrich, & Laufer, 1992). Other researchers have suggested that P3 is affected by arousal levels, such that smaller P3 amplitude reflects lower overall cortical arousal.

At least three subcomponents have been described as occurring during the P3 time frame. P3a is a subcomponent of P3 observed most prominently at frontal sites. This component is thought to be sensitive to the surprisingness of a stimulus (Stevens, Pearlson, & Kiehl, 2007). P3b is maximally observed at parietal locations and is thought to be sensitive to the salience of a stimulus (for example, whether a stimulus is a target or non-target). A positive deflection in the P3 range has also been observed in response to "stop" stimuli in Go/No-Go tasks. This

component has been termed the "No-Go P3." It is thought to reflect inhibitory cognitive processes. As will be reviewed below, externalizing psychopathology and aggression have been consistently linked to abnormalities in P3 amplitude and latency, specifically, smaller and later P3s.

Other ERP components that have been studied in relation to aggression and disinhibitory psychopathology include N1, P1, N2, P2, N450, and the negative slow wave. The earlier N1, P1, and P2 components are thought to reflect bottom-up processes related to registering of sensory information and allocation of attentional resources. N2 is a negative deflection of the ERP peaking around 200 ms post-stimulus that is evident in tasks requiring the inhibition of a motor response. It is most frequently evident at fronto-central electrode sites. Competing accounts describe N2 as an index of inhibition or of detection of response conflict. N450 is a negative deflection of the ERP that peaks around 450 ms post-stimulus and is thought to reflect the detection of response conflict. The negative slow wave (NSW) is a late-going wave evident between 600 and 1,000 ms post-stimulus. It is thought to reflect the execution of inhibitory control processes. It has been found to be larger on trials in which inhibitory control was exercised in the presence of response conflict. Most of the research relating to aggression and externalizing disorders has focused on these components. It should be noted that the amplitudes and latencies of these components may be sensitive to the experimental paradigm by which they are induced and therefore may not reflect the same processes across studies. Furthermore, the cognitive processes underlying these components are better understood for some components than others.

Psychophysiology of Aggression and Related Constructs in Clinical Samples

Little research has been done to examine ERPs specifically as they relate to aggression. Most ERP research relevant to aggression has been done in the context of a group of disorders for which aggression is merely an associated feature. Studies of ERPs and aggression in the context of clinical disorders will be reviewed below, including for Intermittent Explosive Disorder (IED),

Episodic Dyscontrol Disorder (ED), Antisocial Personality Disorder (ASPD), Conduct Disorder (CD), psychopathy, Attention-Deficit/Hyperactivity Disorder (ADHD), and substance use disorders (SUDs). These disorders have all been described as "externalizing" disorders. In addition to sharing aggression as a diagnostic or associated feature, these disorders have been proposed to share a common biological diathesis and the behavioral phenotype of impulsivity (Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Krueger, Hicks, Patrick, Carlson, Iacono, & McGue, 2002; Krueger, Markon, Patrick, Benning, & Krämer, 2007). As such, these disorders have been described as forms of "disinhibitory psychopathology" (Patrick & Verona, 2007; Sher & Trull, 1994). This summary will be followed by a brief review of ERP studies on constructs related to aggression, including anger and impulsivity, and a review of ERP studies related to aggression in non-clinical populations.

Intermittent Explosive Disorder/Episodic Dyscontrol Disorder

Only one disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. Text revision; *DSM-IV-TR;* APA, 2000) identifies aggression as a cardinal feature: Intermittent Explosive Disorder (IED). IED (previously referred to in the literature as episodic dyscontrol; ED) has been characterized in the following way by the *DSM-IV-TR:* (a) as comprising multiple separate episodes of acting on aggressive impulses, resulting in serious assault or property destruction; and (b) the aggressive response is notably disproportionate to the precipitating event (APA, 2000). At one time thought to be extremely rare, recent studies have shown that IED has an estimated lifetime prevalence of 7.3% and 12-month prevalence of 3.9% in community samples (Kessler, Coccaro, Fava, Jaeger, Jin, & Walters, 2006). Furthermore, when less-stringent research criteria (Intermittent Explosive Disorder – Integrated Research Criteria; IED-IR; Coccaro, Kavoussi, Berman, & Lish, 1998) are used, lifetime prevalence estimates are about 11% of the community population (Coccaro, Schmidt, Samuels, & Nestadt, 2004). IED has been shown to be highly comorbid with other externalizing disorders of childhood and adulthood

(Kessler et al., 2006). In a community sample, higher rates of IED-IR were observed for women than for men (5:3 gender ratio; Coccaro et al., 2004).

Few electrophysiological studies have specifically examined individuals with IED, and these have largely been electroencephalogram (EEG) studies, rather than ERP studies. With EEG, brain wave activity is examined under "resting" conditions, rather than in response to a stimulus. In one ERP study, Bars and colleagues (2001) used a visual pattern reversal discrimination task to elicit ERPs in children and adolescents with (n = 267) and without (n = 59) features of IED (including explosive behavior, physical and verbal aggression). They also compared the groups on resting EEG activity. The group with explosive behaviors displayed higher P1 amplitude at occipital sites. This component is thought to be a biological marker of automatic sensory processing of incoming visual information. P1 amplitude was positively correlated with the number of past explosive episodes. The authors did not speculate what role P1amplitude may play in explosive behavior. They did, however, suggest that since P1 reflects automatic (bottom-up) sensory processes, the association between this component and explosive behavior may reflect a biological predisposition to explosive outbursts. The authors also observed that high delta frequency power (i.e., 1 to 4 hz) in the right hemisphere was associated with explosive behavior, a phenomenon that has previously been reported by other researchers (Bars, Heyrend, Simpson, & Munger, 2001). Delta waves have been interpreted as a marker of alertness and cortical activation or arousal (Houston & Stanford, 2005). As power is inversely associated with cortical activity, these results suggest that lower resting arousal may be associated with explosive behavior. P3 was measured in this study but not reported.

Drake, Hietter, and Pakalnis (1992) collected data on 23 patients with episodic dyscontrol who were undergoing testing for neurological abnormalities. The patients exhibited significant impulsivity, aggression, and violence, and most met criteria for IED. The patient group was compared to a group of depressed patients and a group of patients experiencing headaches. Brainstem auditory evoked potentials (BAEPs) and auditory evoked potentials (AEPs) were

elicited using an auditory oddball task. The authors observed lower N1 and N160 amplitudes in response to the stimuli in the ED group compared to the other groups, but no differences between groups in N2 or P3 amplitude. N1 has been described as an early sensory component reflecting attention allocation (Houston & Stanford, 2001). The authors provided no further interpretation of their null findings. N2 and P3 have previously been described as reflecting inhibitory cognitive processes and updating of working memory, respectively. The results of this study suggest that IED individuals may display deficits in allocation of attentional resources. Notably, the ED group was quite heterogenous, with several participants reporting a history of conduct disorder, learning disability, suspected seizures, alcohol abuse, and drug abuse.

Koelsch, Sammler, Jentschke, and Siebel (2007) compared individuals with and without moderate IED (mIED) on resting EEG and EEG in response to auditory stimuli (music) and visual stimuli (mIED questionnaire items appearing on the computer screen). The authors found higher levels of beta activity (13 to 20 hz) and lower levels of delta and alpha activity (1 to 4 hz and 8 to 13 hz, respectively) in individuals with mIED. This pattern of EEG activity has been linked to higher levels of cortical arousal and impulsiveness. The authors also found that mIED individuals scored higher on a measure of impulsive action (the Eysenck Impulsiveness/-Venturesomeness/Sensation-seeking Questionnaire; I7; Eysenck, Pearson, Easting, & Allsopp, 1985; Koelsch et al., 2007). The authors did not specifically assess EEG activity in relation to aggression.

An MRI study of 50 epilepsy patients (25 with IED and 25 without IED) that also included EEG data reported that right hemispheric brain abnormalities were more common in the IED than in non-IED patients (van Elst, Woermann, Lemieux, Thompson, & Trimble, 2000). The authors suggested the right hemispheric dysfunction may play a role in aggressive behavior in some individuals. Overall, the results of these studies suggest that individuals with IED evidence differences in early ERP components associated with sensory processing and allocation of attention. The results further support the notion that individuals with IED show differences in

arousal levels, which may in turn play a role in the expression of aggressive behavior. Finally, the evidence suggests that individuals with IED may display impairments in right hemispheric brain functioning.

Attention-Deficit/Hyperactivity Disorder (ADHD)

ADHD is an externalizing disorder characterized by symptoms of inattention and/or hyperactivity across multiple settings. Aggression is common in children with ADHD, due to their impaired ability to inhibit impulses (Klorman et al., 1988). Medications that are used to reduce symptomatology associated with ADHD (impulsivity) have also been found to reduce aggression. These medications have also been found to normalize P3 amplitude in ADHD children with aggressive behavior, adding further experimental support to the notion that P3 is involved in aggressive behavior. Klorman and colleagues (1988) recruited children ages 6 to 12 with either ADHD with aggression, ADHD without aggression, or borderline (i.e., subclinical) ADHD. The children were administered both placebo and methylphenidate (brand name Ritalin©) for two weeks each in counterbalanced order and were rated on their aggression and non-compliance, among other variables. ERPs were measured in the context of a test of continuous performance (Continuous Performance Test – double version; CPT-double). The CPT is a measure of attention and impulsivity. Although the task has a number of variations, the basic premise is that the person must continuously attend to stimuli that appear on the computer screen and respond to target stimuli while ignoring non-target stimuli. ADHD children with aggression showed decreased aggression and noncompliance (rated by parents and teachers) while taking methylphenidate compared to placebo and showed concomitant increases in the amplitude of their P3b components in the context of the CPT-double task (Klorman et al., 1988). This study provides support for the notion that interventions to reduce aggression have simultaneous effects on aggressive behavior and P3b amplitude, though the authors did not test for mediation and it is possible that a third variable may be accounting for both effects. Methylphenidate appeared to have no effect on P3b latency (Klorman et al., 1988).

A follow-up study of aggressive/oppositional ADHD children, non-aggressive ADHD children, and subclinical ADHD children ages 6 to 12 also suggested that children with clinically significant ADHD both with and without aggression showed greater reductions in aggression and oppositionality while taking methylphenidate relative to placebo compared to the subclinical ADHD group. Methylphenidate was associated with increased P3b amplitude, which the authors viewed as evidence of increased information transmission. Again, the authors did not test for mediation (Klorman, Brmaghim, Fitzpatrick, Borgstedt, & Strauss, 1994). Although P3b in these studies was analyzed as an indicator of cognitive processing in the context of a memory task, this component may also be relevant to other aspects of behavior, such as aggression (Klorman et al., 1994). Not all studies have replicated this finding. Smithee and colleagues (1998) administered methylphenidate and placebo in counterbalanced order for 14 days each to 26 children with ADHD. The drug had the effect of reducing aggression and oppositionality, but had no effect on P3s elicited during either of two oddball tasks (Smithee, Klorman, Brumaghim, & Borgstedt, 1998).

Children and adults with ADHD have also been studied extensively with regard to the neurophysiological substrates of impulsivity and inhibition. These studies are described later in a discussion of impulsivity and inhibition more broadly.

Conduct Disorder (CD)

DSM-IV-TR conduct disorder, which is usually diagnosed in individuals under the age of 18, comprises a constellation of symptoms that are characterized by a disregard for society's rules and by the violation of the rights of others. In short, it typically represents antisocial behavior in children and adolescents. Few electrophysiological studies have been done on conduct disorder (CD) and related traits, and little research has specifically assessed aggression in relation to ERP components in conduct disordered children and adolescents. Briefly, research on electrophysiological correlates of CD suggests that the disorder is associated with decrements in P3 amplitude (Bauer, 1997; Bauer & Hesselbrock, 1999, 1999b; Iacono, Carlson, Malone, &

McGue, 2002). Furthermore, retrospective reports of CD symptoms are inversely predictive of later P3 amplitude in adulthood (Bauer, 1997), such that a greater number of past CD criteria is associated with smaller P3 amplitude in the present. The exact topography of P3 differences appears to change over time as a function of age (Bauer, 1997). Specifically, it appears that CD is associated with posterior P3 decrements in younger individuals. These decrements seem to disappear in individuals who "outgrow" antisocial behavior. However, P3 decrements appear to shift from parietal to frontal areas in individuals who continue to show antisocial behavior into adulthood. These findings further support the relationship between P3 and antisocial behavior and suggest that P3 may be a trait-like marker of antisocial behavior. They also suggest that developmental factors play a role in both the physiological and behavioral manifestations of antisocial behavior (Bauer, 1997).

The findings of one study run counter to this perspective. Raine and Venables (1987) found that antisocial youth had larger P3 in response to a warning tone presented during a contingent negative variation (CNV) paradigm. In a CNV paradigm, two tones are presented. The second tone, S2, is followed by a highly aversive loud noise. The first tone, S1, is not followed by a noise. A negative deflection just after S2 and prior to the "punishment" is called the contingent negative variation. The CNV is thought to reflect the strength of the association between S2 and the punishment. Others researchers have suggested that CNV reflects sensitivity to punishment cues. Raine and Venables (1987) interpreted their findings as possibly reflecting greater processing of salient information in antisocial youths (in line with the idea that antisocial individuals have lower resting arousal and therefore seek stimulation by attending to salient information). It should be emphasized that P3 in this study was elicited in the context of a contingent negative variation paradigm, rather than the oddball paradigm. Furthermore, the groups were formed on the basis of a psychopathy rating scale, albeit one constructed to tap antisocial behavior. Indeed, the results of this study are similar to those obtained using psychopathic samples, described below. Raine and colleagues also found that antisocial youth did

not differ from prosocial youth in earlier, sensory-related components, such as N1 nor in contingent negative variation (CNV). N1 has been interpreted as an index of attention allocation. CNV has been interpreted as a measure of the readiness with which associations are formed and, alternatively, as a measure of the motivational significance of stimuli (Raine & Venables, 1987). The one study that attempted to link P3 amplitude to specific aspects of childhood antisocial behavior (CD) found that P3 amplitude (elicited by a Stroop task) was related to deceitfulness but not aggression or rule violations (Bauer & Hesselbrock, 1999b). P3 decrements have also been observed in adolescents with oppositional defiant disorder (ODD; Iacono et al., 2002).

Antisocial Personality Disorder (ASPD)

Antisocial personality disorder (ASPD) is defined by the *DSM-IV-TR* (APA, 2000) as a pervasive and inflexible pattern of relating to others and the world in a manner characterized by "disregard for, and violation of, the rights of others" (APA, 2000, p. 701). Aggression is included in the diagnostic criteria for ASPD, though physical aggression is not required to make a diagnosis. Aggression, criminality, impulsivity, and substance abuse are common features of individuals with ASPD.

To date, a number of studies have been conducted on ASPD and aggression using ERP techniques. Together, these studies show a pattern of reduced P3 amplitude in individuals with antisocial personality disorder, particularly in those with a history of violence (Barratt, Stanford, Felthous, & Kent, 1997; Barratt, Stanford, Kent, & Felthous, 1997b; Bauer, 1997; Bauer, O'Conner, & Hesselbrock, 1994; Bernat, Hall, Steffen, & Patrick, 2007; Branchey, Buydens-Branchey, & Lieber, 1988; Drake, Pakalnis, Brown, & Hietter, 1988; O'Conner, Bauer, Tasman, & Hesselbrock, 1994). These findings have been observed in samples of community residents with ASPD (Bauer et al., 1994; O'Conner et al., 1994), prisoners (Barratt et al., 1997, 1997b; Bernat et al., 2007; Drake et al., 1988), and alcohol-dependent (Branchey et al., 1988), and cocaine-dependent (Bauer, 1997) individuals using both auditory and visual oddball tasks. The evidence is mixed regarding differences in P3 latency (Drake et al., 1988) and early sensory

processing components (N1, P2, N2) in relation to antisocial behavior. Evidence for differences in hemispheric lateralization has also been mixed, with one study implicating right hemispheric abnormalities in aggressive behavior (Drake et al., 1988). The results of these studies cannot be attributed to the presence of alcohol dependence, family history of alcohol dependence, recent cocaine dependence, or IQ (Bernat et al., 2007; Branchey et al., 1988; Drake et al., 1988). Research has also shown that 15-year-olds observed as having larger N1 amplitudes and shorter P3 latencies during a contingent negative variation task were more likely to display criminal behavior at follow-up nine years later. These findings suggest that heightened attention to and faster information processing of salient stimuli may be associated with the development of antisocial and criminal tendencies over time (Raine, Venables, & Williams, 1990).

Research has shown that the administration of drugs to decrease aggression in ASPD individuals has concomitant effects on the P3 component of the ERP. For example, Barratt and colleagues (1997b) administered both phenytoin (Dilantin©; a drug with known aggression-reducing effects) and a placebo to prison inmates with a history of either impulsive or premeditated aggression in a crossover design. Impulsively aggressive offenders showed a decrease in aggressive behavior while taking phenytoin along with an increase in P3 amplitude to target stimuli during an oddball task, compared to when they received the placebo. Non-impulsive, premeditating offenders showed no decrease in aggression or increase in P3 amplitude while taking phenytoin. The results suggest that impulsively aggressive individuals may have abnormal P3 amplitudes that are normalized (increased) by drugs that reduce aggression, but that these effects are limited to individuals whose aggression is primarily impulsive rather than premeditated (Barratt et al., 1997b). The results highlight the interrelationship of impulsivity, P3 amplitude, and aggressive behavior in reactively aggressive individuals.

Overall, robust support exists for the notion that P3 differences are evident between antisocial adults and children and normal adults and children, and that, with a few exceptions, much of this evidence supports a link between history of violence and aggression and decreased

P3 amplitude. Mixed reports of differences in earlier, more automatic ERP components (e.g., N1, P1) tend to suggest that some antisocial individuals may differ from controls in their early sensory processing of incoming information (Raine & Venables, 1987). Taken together, the research suggests that antisocial individuals engage fewer cognitive resources in identifying, discriminating, and categorizing stimuli.

Psychopathy

In contrast to antisocial personality disorder, which is defined primarily in terms of behavior, psychopathy has been described as a personality structure that reflects primarily affective-interpersonal features, including lack of empathy, shallow affect, narcissism, impulsivity, sensation-seeking, and manipulativeness. Aggression is strongly associated with psychopathy and this association has been observed in both clinical and community samples (Hare & Neumann, 2008; Neumann & Hare, 2008; Patrick & Verona, 2007).

Much has been written about the physiology of psychopaths and a handful of ERP studies focusing on the P3 component have been conducted in psychopathic samples. The results have been at odds with the literature on P3 in antisocial (i.e., ASPD and CD) individuals. Specifically, increased P3 amplitudes have been observed in psychopathic criminals (compared to non-psychopathic criminals; Raine & Venables, 1988) and in adolescents with higher scores on a psychopathy checklist (Raine & Venables, 1987). Psychopaths showed no differences in P3 amplitudes in a study employing a linguistic oddball task (in which the stimuli to be discriminated were the sounds /t/ and /v/; Jutai & Hare, 1983), although they did show increased P3 latency during this task. These results are incongruent not only with other work on externalizing disorders, but also with the theoretical notion that psychopaths have impairments in sustained attention. However, the findings in psychopaths have been interpreted as reflecting psychopaths' enhanced ability to process information for short periods of time. Jutai and Hare (1987) speculated that psychopaths would show greater reductions in P3 amplitude if they were given a longer task that required more sustained attention. Overall, the tasks used in these studies

were brief and placed minimal demands on participants' information processing. Another factor that might account for the differences between psychopaths and antisocials may be the use of linguistic stimuli in the studies with psychopaths, which was done to test the hypothesis that psychopaths exhibit impaired linguistic processing.

One study of psychopathic and non-psychopathic prison inmates that used non-linguistic stimuli did find the usual pattern of reduced P3 amplitude to target stimuli. Kiehl and colleagues (1999b) presented participants with a visual oddball task in which larger and smaller squares presented on a computer screen served as the non-target and target stimuli, respectively. Psychopathic prisoners were found to have reduced P3 amplitude in response to target stimuli relative to non-psychopathic prisoners (Kiehl, Hare, Liddle, & McDonald, 1999b). Support for differences between psychophaths and non-psychopaths in early cognitive processes (sensory registration and attention) have been mixed (Jutai & Hare, 1987). To the author's knowledge, no published studies to date have examined psychopathy, aggression, and ERPs in a single study.

Other studies of psychopaths have focused on the N1 component of the ERP as a measure of central arousal, or alternatively, as a measure of attention allocation. Both of these constructs are important with regard to psychopathy, as psychopaths have been described as exhibiting both low levels of autonomic arousal and difficulty sustaining attention. Jutai and Hare (1983) found that psychopaths generally excelled early in focusing attention on task-relevant stimuli. However, as the task continued, psychopaths showed a tendency toward poorer performance and smaller N1 amplitude, suggesting poorer ability to maintain attention on a sustained task. Apparently, psychopaths found the distractor task (a military combat-themed video game) more stimulating than the assigned task and therefore allocated more attention to the interesting task and less to the boring (but relevant) task. Jutai and Hare (1987) also failed to find differences between psychopaths and controls in early sensory processing (the N1 component). One group of researchers found that adolescents with increased N1 amplitude and contingent negative variation (CNV) amplitude were more likely to develop psychopathic personality traits later on (Raine et

al., 1990). The authors suggest that heightened attention to stimuli (as evidenced by N1 amplitude) and greater attention to motivationally salient stimuli (as evidenced by CNV amplitude) in adolescents predicts (and perhaps contribute to the development of) psychopathic traits in adults assessed 9 years later (Raine et al., 1990).

In a review of electrophysiological studies of antisocial individuals (primarily psychopaths), Scarpa and Raine (1997) summarized the literature by noting that the research tends to highlight lower levels of arousal in psychopathic individuals (as evidenced by brainstem evoked potentials), increasing mid-latency ERPs to stimuli of increasing intensity (as in augmenting/reducing paradigms), and enhanced P3, suggesting greater processing of stimulating events by psychopathic individuals. As described above, the latter finding is particularly surprising, given that other groups of individuals that show high levels of aggression typically have reduced amplitude P3. One possible explanation for the unexpected findings is that the psychophysiological correlates of psychopathy are different from those of other externalizing disorders, and these differences may be manifested in the tendency of psychopaths to engage in different forms of aggression (e.g., more instrumental aggression; Patrick & Verona, 2007). Deficits in P3 amplitude have been associated primarily with impulsive, rather than premeditated, forms of aggressive behavior (Barratt et al., 1997, 1997b; Gerstle & Mathias, 1998). The relationship between P3 amplitude and aggression has been found primarily in samples of individuals with impulsive aggression, whereas psychopathy may be characterized more by instrumental or proactive aggression (Patrick & Verona, 2007). Indeed, in one study of adolescents, P3 amplitude was correlated positively with a measure of psychopathy but was uncorrelated with other measures of antisocial behavior (Raine & Venables, 1987).

Substance Abuse Disorders

Alcohol and drug abuse and dependence are common diagnostic and associated features of externalizing disorders. Indeed, impulsivity, disinhibition, and poor self-control are common traits of all of the externalizing disorders. It has even been said that “alcoholism is but one

possible endpoint of a cluster of etiological factors with genetic and environmental causes" (Porjesz, Rangaswamy, Kamarajan, Jones, & Padmanabhapillai, 2005, p. 994).

A considerable body of research has used ERP techniques to study the effects of family history of alcohol and cocaine dependence, as well as current and past use of substances, on psychophysiological measures. Decreased P3 amplitudes have been observed in alcohol-dependents and nicotine-dependent adolescents (Chen et al., 2007; Iacono et al., 2002). Smaller frontal P3 amplitudes have also been observed following the administration of an acute dose of alcohol, and this effect is particularly prominent in individuals with a family history of alcohol dependence (Porjesz et al., 2005). Smaller P3 amplitude has been found to predict later substance use in adolescents and young adults (Iacono et al., 2002), and relapse in cocaine-dependent individuals (Bauer, 1997).

A meta-analysis by Polich and Kok (1994) that included data from 22 studies concluded that P3 decrements are a marker of biological risk for alcohol dependence. The authors examined studies that included comparisons of individuals with and without a family history of alcohol dependence and found an overall effect size of family history of alcohol dependence of $d = .33$ on P3 amplitude. Furthermore, individuals with a positive family history of alcohol dependence showed smaller amplitude P3 even in the absence of any substance abuse history of their own (Polich & Kok, 2004). Polich interpreted these findings as evidence that individuals at risk for alcohol dependence due to a positive family history show abnormalities in their allocation of attention.

Porjesz and colleagues (2005) reviewed the literature on alcohol-risk, alcohol dependence, and ERPs. They reached similar conclusions to Polich and colleagues regarding P3 amplitude in high-risk individuals, and further noted that abstinent former-alcoholics show decrements in P3 amplitude that do not normalize with sobriety. Porjesz (2005) also noted that compared to controls, alcoholics do not show the normal pattern of P3b variations to "Go" and "No-Go" stimuli in a Go/No-Go task and the P3b component in the alcoholic group was not

localized to parietal scalp regions as it is in healthy individuals. Thus, alcoholic individuals showed less differentiated and more dispersed P3b components in response to the Go/No-Go task. This data provides further support for the notion that that P3 is a trait-like marker of a predisposition to alcohol dependence. Unlike Polich and Kok (1994), Porjesz et al. (2005) conceptualized P3 amplitude as a measure of central nervous system inhibition, with lower amplitudes reflecting lower inhibition. This interpretation is more consistent with the notion that impulsivity subsumes a range of externalizing disorders, and better accounts for the findings relating reduced P3 to aggressive and antisocial behavior reviewed in this paper.

Aside from P3, violent and non-violent alcoholics have been found to differ from non-violent, non-alcoholic control subjects in the amplitude of early ERP components reflecting arousal (N1) and sensory adaptation ($N1_{(tone\ 1)}$ / $N1_{(tone\ 2)}$ on a CNV task; Tarkka et al., 2001). Impaired inhibition in relation to alcohol has also been linked to the N450 component and the negative slow wave, which are thought to reflect stimulus evaluation processes and cognitive control. For example, the administration of an acute dose of alcohol has been linked to reduced N450 and NSW amplitudes and to impaired performance on a Stroop task, a task that specifically elicits response conflict, and the successful completion of which requires the ability to inhibit a response (Bartholow, Dickter, & Sestir, 2006; Curtin & Fairchild, 2003; see below).

Clearly, an abundance of research suggests that a number of ERP components are affected by substance use and are relevant to questions regarding inhibition and by extension aggression. In the case of P3, aberrant ERP components may actually be an endophenotype (a marker of risk) for alcohol dependence (Begleiter et al., 1998; Porjesz et al., 2005). Some researchers have even proposed that P3 has a genetic locus (Begleiter et al., 1998). Furthermore, as alcohol dependence is but one of several externalizing disorders (Hicks et al., 2004; Krueger et al., 2002, 2007), it is conceivable that P3 amplitude may be an endophenotype of a wider range of psychopathology, with the common trait being a tendency toward disinhibition (Chen et al., 2007; Polich et al., 1994; Porjesz et al., 2005; Sher & Trull, 1994).

Anger

The role of anger has been extensively studied in relation to aggressive behavior (Berkowitz, 1990). Anger is generally viewed as an emotional experience with behavioral, cognitive, and affective components. In contrast, aggression is an overt behavior, which can be physical, indirect, or verbal, that has the intended goal of harming another (Berkowitz, 1990). Clearly, anger experiences do not invariably lead to aggression, and aggressive behavior can occur in the absence of marked anger. However, given the role of anger in many instances of reactive aggression, electrophysiological studies of anger will briefly be reviewed.

Most psychophysiological research on anger has been conducted using EEG, as opposed to ERP, techniques. These studies have generally focused on the role of alpha waves in the frontal brain and hemispheric lateralization in the experience and expression of anger. One theory of hemispheric lateralization has proposed that left hemispheric activation is associated with positive mood and right hemispheric activation with negative mood (the *affective valence hypothesis*: Peterson et al., 2008). A competing theory suggests that left hemispheric activation is associated with approach motivation, and right hemispheric with avoidance motivation (the *motivational direction hypothesis;* Peterson et al., 2008). Explaining anger in terms of these theories is difficult, however, as anger is a negative emotion that is often associated with approach motivation. According to the first theory, anger should be associated with greater right hemispheric activation; according to the second it should be associated with greater left hemispheric activation.

The research on anger and EEG lateralization suggests that anger is associated with greater left than right hemispheric activation. This has been observed both for trait anger and state anger elicited through the presentation of anger-evoking images (Harmon-Jones & Allen, 1998; Harmon-Jones et al., 1997). The asymmetry has been attributed to a combination of increased left hemisphere activation and decreased right hemisphere activation (Harmon-Jones & Allen, 1998). Left hemispheric asymmetry has also been associated with increased aggression (Peterson,

Shackman, & Harmon-Jones, 2008). Participants in one study who were required to make right hand contractions (thus increasing left hemispheric activation) as opposed to left hand contractions, showed greater behavioral aggression during a modified Taylor Reaction-time Task paradigm, following an insult manipulation to increase anger (Peterson et al., 2008). The results suggest that hemispheric differences in cortical activity may play a role in the expression of anger and aggression.

A small number of studies on anger using ERP techniques were found. In one study, trait anger was unrelated to P3 amplitude in the context of both an oddball task and a continuous performance task among psychiatric and normal school children (Harmon-Jones, Barratt, & Wigg, 1997). In another study, trait anger was associated with reduced P3 amplitude in prison inmates who completed a modified oddball task (Barratt et al., 1997b).

Anger has been an important construct in the study of social information processing biases and aggression. A number of studies have demonstrated that participants with high trait anger, with experimentally induced high state anger, and with aggression histories all show biased attention to threat- or aggression-related material in the context of an emotional Stroop task or visual search task, as evidenced by slower reaction times (Cohen et al., 1998; Eckhardt & Cohen, 1997; Smith & Waterman, 2003; Smith & Waterman, 2004). Although ERPs were not recorded in these studies, the results are suggestive of differences in information processing as a result of state and trait anger. Other researchers have observed that participants who have been provoked with higher levels of provocation on a Taylor Reaction-Time Task show larger P3 amplitudes to facial stimuli reflecting different emotions (angry, sad, happy, fearful, and neutral), compared to participants who were not provoked during the task (Bertsch, Bohnke, Kruk, & Naumann, 2009). Overall, studies of ERPs in relation to anger are few in number but have yielded interesting findings that are relevant to the present study.

Impulsivity and Inhibition

The clinical disorders described above all share impulsivity (or disinhibition) as a common feature. Thus, it is reasonable to suspect that some of the common electrophysiological findings observed across studies (i.e., reduced P3 amplitude) may also covary with measures of impulsivity. In this section, only those studies which specifically assessed impulsivity in relation to ERP components are reviewed.

Impulsivity has been defined as the inability to inhibit an ongoing or pre-potent response, or to control behavior (Barratt, 1994). Impulsive actions are unplanned reactions to internal or external stimuli without regard for the consequences of those actions (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). Conversely, inhibition has been described as the *ability* to halt ongoing actions or already initiated actions (Kenemans, Bekker, Lijffijt, Overtoom, Jonkman, & Verbaten, 2005). The ability to adaptively initiate and inhibit actions is an important component of executive functioning, and one that is particularly relevant to the expression of aggressive behavior (Giancola & Zeichner, 1994). A recent meta-analysis by Bettencourt and colleagues (2006) illustrated that impulsivity is an important predictor of aggression under conditions of provocation.

Impulsivity and inhibition have been assessed using both self-report and behavioral measurement tools. One commonly used self-report measure of impulsivity is the Barratt Impulsiveness Scale (11^{th} edition; BIS-11; Patton & Barratt, 1995). The scale yields a total impulsivity score and three subscale scores: motor impulsiveness, nonplanning impulsiveness, and attentional impulsiveness. P3 amplitude has been found to correlate negatively with self-reported impulsivity as measured by the BIS-11, such that greater impulsivity is associated with smaller P3 amplitude, and this result has been found in normal individuals, impulsively aggressive and instrumentally aggressive prison inmates, and alcohol-dependent and formerly cocaine-dependent individuals (Barratt et al., 1997b; Branchey, Buydens-Branchey, & Hovrath, 1993; Chen et al., 2007). The relationship has been observed most often at parietal electrode sites

(Chen et al., 2007; Harmon-Jones et al., 1997). However, at least one research group failed to replicate this finding in a normal sample (Surguy & Bond, 2006), and another in an impulsively aggressive sample (Bond & Surguy, 2000). In another study, BIS-11 impulsivity showed no relation to the amplitude of earlier ERP components (e.g., N1, P2, or N2) nor to the latency of P3 in impulsively aggressive, non-impulsively aggressive, and normal control subjects (Barratt et al., 1997b). These results were all found in the context of oddball tasks.

In addition to self-report measures of impulsivity, some researchers have used behavioral impulsivity tasks in conjunction with the ERP technique in order to observe the real-time neurophysiological processes involved in the inhibition of a motor response. These behavioral tasks include go-stop (Go/No-Go) tasks, measures of continuous performance, and other variations on these tasks. In general, go-stop tasks present participants with a series of trials to which they must either respond (by pressing a key), or withhold a response. Often the cue to withhold a response (i.e., "stop") is given after a cue to respond, so that the participant must inhibit a response that has already begun.

Various theories have been proposed to explain why some people are better at successfully inhibiting a motor response than others. Gray (1987) proposed that two separate systems, a *Behavioral Activation System* (BAS) and a *Behavioral Inhibition System* (BIS) underlie the initiation and inhibition of behavior, respectively. These two systems have also broadly been described as mediating approach and avoidance behaviors. Problems with impulsivity might be a due to either deficient activation of the BIS or to hyper-activation of the BAS, or both. Another model of impulsivity/inhibition is the "race model" (Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004). According to this model, go-stop signals first generate "go" activation. The "stop" signal then generates a separate "stop" activation. These two processes then "race" to be expressed first behaviorally (Kok et al., 2004).

Studies using go-stop tasks in conjunction with ERP techniques have yielded considerable insight into the processes underlying impulsivity and inhibition. Dimoska and

Johnstone (2007) recruited participants with the top and bottom 15% of scores on the Eysenck Impulsiveness/Venturesomeness/ Sensation-seeking Questionnaire (I7; Eysenck et al., 1985) to create high and low impulsivity groups. Participants completed a go-stop task that included go trials and stop trials. Although the high impulsivity participants made no more errors (i.e., failing to inhibit on stop trials) than the low impulsivity participants, the two groups displayed differences in the amplitude and latency of the ERP components elicited during the task. High impulsive individuals demonstrated a larger amplitude (relative to the low impulsive group) of the lateralized readiness potential (LRP) of the ERP, which reflects cortical activity associated with the preparation of a motor response. LRP is typically observed approximately 250 and 600 ms post-stimulus onset maximally on the side of the scalp contralateral to the part of the body that is preparing to move, and is thought to be generated in the motor cortex. The larger amplitude of the LRP is thought to reflect greater activation of the motor cortex in the high self-reported impulsive group. The high impulsivity group also showed shorter latency of the LRP, indicating greater motor impulsivity, though the difference was not statistically significant. The high impulsive group was also observed to have larger stop-P3s (i.e., P3s elicited by successfully inhibited stop-trials) compared to the low impulsive group. Given that this group did not commit more impulsivity errors, this finding suggests that the highly impulsive individuals were able to inhibit their responses on stop trials by recruiting greater cognitive resources (Dimoska & Johnstone, 2007). Based on the pattern of results, the authors concluded that the primary impairment in impulsive individuals is in the inhibition of the motor response (as indexed by P3) rather than the activation of the response (as indexed by LRP), implicating problems with the Behavioral Inhibition System rather than the Behavioral Activation System in impulsivity. Impulsive individuals had larger N1 amplitudes, which have been linked in previous studies to sensation-seeking behavior (Dimoska & Johnstone, 2007). It has been proposed that impulsivity is more prevalent in individuals with low levels of arousal, and that these individuals engage in

impulsive and sensation-seeking behaviors to increase arousal to a more optimal level (Dimoska & Johnstone, 2007; Gray, 1987; Houston & Stanford, 2005).

Other researchers have found that P3s generated on successful stop trials have a larger amplitude and shorter latency compared to P3s generated on unsuccessful stop trials, supporting the race model of inhibition (Kok et al., 2004). This finding suggests that the No-Go P3 is directly related to the ability to inhibit a response. The researchers also observed through dipole source modeling that successful and unsuccessful stop P3s were generated by different cortical sites, a finding similar to that of Porjesz and colleagues (Kok et al., 2004; Porjesz et al., 2005). P3 in the context of go-stop tasks has been shown to be sensitive to advance cueing, suggesting that it indeed reflects inhibitory cognitive processes (Bruin, Wijers, & van Staveren, 2001).

In a novel approach to studying impulsivity, De Pascalis and colleagues tested the effect of positively and negatively valenced words on high and low impulsivity, and high and low anxiety participants (De Pascalis et al., 2004). The authors presented participants with a visual flanker task consisting of four conditions: positive target words embedded in a block of words that was primarily positive (positive target/positive standard words), positive target words embedded in negative standard words (positive target/negative standard words), negative target words embedded in positive standard words (negative target/positive standard), and negative target words embedded in negative standard words (negative target/negative standard). Anxious participants showed larger P3 amplitudes to negative target words than did non-anxious participants, suggesting that anxious individuals engage in greater information processing of negatively-valenced stimuli. P3 amplitude was found to be smaller (and the latency longer) to negative targets in impulsive participants relative to non-impulsive participants, suggesting that impulsive individuals engage in less information processing of negative stimuli, and are slower to commit cognitive resources to the processing of negative stimuli. No differences in cognitive processing (i.e., P3 amplitude or latency) of positive stimuli were found as a function of impulsivity or anxiety. This supports the notion that impairments in inhibition of responses are

more directly related to inhibitory control (BIS in Gray's theory) than to response activation (BAS; De Pascalis et al., 2004). This study also demonstrated that the emotional valence of the words employed interacted with participants personality predispositions to affect information processing.

The N2 component of the ERP is another component that has been implicated in inhibitory processing. N2 was initially conceptualized as a straightforward index of processes related to response inhibition after it was observed in several studies that the N2 component was larger for stop trials than for go trials (Bokura, 2001). More recently it has been suggested that N2 is actually a measure of response conflict, which is elicited most prominently when there is a conflict between a between a response being simultaneously activated and suppressed, as when a response has been primed on a stop trial (Bartholow, 2006). This notion is supported by the finding that N2 amplitudes are larger (more negative) for failed stop trials than for successful stop trials (Bartholow, 2006b). At least one author has also suggested that N2 actually reflects the activation process in a Go/No-Go task (Bruin, 2001).

Other components, such as N450, and the more tonic (long-lasting) negative slow wave (NSW) have also been associated with the successful inhibition on a Stroop task (Curtin & Fairchild, 2003). N450 has been proposed to reflect the detection of response conflict; NSW has been proposed to reflect "the activation and implementation of conflict resolution processes" (Curtin & Fairchild, 2003, p. 425), in other words, cognitive control processes. NSW has been shown to be sensitive to response conflict, such that greater inhibitory resources must be recruited to inhibit a strong response activation (Curtin & Fairchild, 2003).

The Stroop task is ideal for eliciting response conflict. Response conflict occurs when a task elicits two competing responses (for example, to press a button versus not to press the button). A modified Stroop task was employed by Curtain and Fairchild (2003) in a study involving alcohol administration. In the task, participants were presented with words in congruent (i.e., "blue-blue") and incongruent (i.e., "blue-red") ink and text combinations. The incongruent

words elicit response conflict. The administration of an acute dose of alcohol, which has known disinhibiting effects, was shown to reduce the evaluation of competing response information as well as cognitive control, as indicated by reduced N450 and NSW amplitudes (Curtin & Fairchild, 2003). In another study employing a different modified Stroop task, NSW amplitude was found to be larger on trials in which the competing response was successfully inhibited (i.e., cognitive control was exercised; Bartholow et al., 2006). At least one study, however, failed to find expected differences in N450 between high and low impulsive individuals during a Stroop task. Lansbergen and colleagues (2007) examined N450 and other ERP components generated by a Stroop task in both high and low trait impulsivity individuals. The Stroop task included both congruent and incongruent trials. In half the task, fewer trials were incongruent, leading participants to expect congruent trials. The rare incongruent trials during this half of the task thus caused greater Stroop interference, or response conflict. During the other half of the task, congruent and incongruent trials were more equally mixed. Incongruent trials during this half caused less interference because they were more expected. As expected, the authors found that N450 amplitude was larger on incongruent trials, particularly during the high interference half of the task. However, the high and low impulsivity groups (formed on the basis of scores on the I7 impulsivity questionnaire; Eysenck et al., 1985) did not differ in N450 amplitude. They also did not differ in the number of errors committed during the task. It would be expected that highly impulsive individuals would commit more errors on a task involving rapid decisions and responses. The N450 component was observed more strongly over the right hemisphere of the brain, which in previous research has been linked to inhibitory processes (Lansbergen, van Hell, & Kenemans, 2007).

A study on stereotype activation and response inhibition under conditions of alcohol intoxication revealed that alcohol had no impact on stereotype activation (as measured by P3 amplitude), but that alcohol did have an effect on successful response inhibition. In a modified version of a Stroop task, images of White or Black faces were followed by either White-racial or

Black-racial stereotype words in congruent (e.g., White-White) or non-congruent (e.g., White-Black) pairs. P3 amplitudes were larger following non-congruent pairs and were unaffected by alcohol dose (high, medium, or placebo). In a follow up stop task, the congruent and non-congruent pairs of stimuli were again presented but this time were followed by a "Go" signal (75% of trials) or a "Stop" signal (25% of trials). As expected, N2 was larger following incongruent trials, which was interpreted as greater detection of conflict, irrespective of alcohol dose. NSW was larger following "Stop" trials than "Go" trials, reflecting a greater need for inhibitory control on these trials. However, this finding was only observed in the placebo group but not in either of the alcohol groups, suggesting that acute alcohol intoxication impairs inhibition of responses but not conflict monitoring (Bartholow et al., 2006).

Other studies on the neurophysiological substrates of impulsivity and inhibition have been carried out in samples of ADHD children and adults. In the context of tests of continuous performance and go-stop tasks, ADHD adults and children have been found to demonstrate impairments in stopping, along with concomitant reductions in N2 and P3 amplitudes and delays in P3 latency on failed stop trials (Bekker, Overtoom, Kooij, Buitelaar, Verbatin, & Kenemans, 2005; Kenemans et al., 2005; Overtoom et al., 2002). A review of studies of attending, shifting attention, and stopping in ADHD children and adults suggests that deficits in attending (as evidenced by abnormalities in N1 amplitude) may actually account for considerable variability in impulsivity (Kenemans et al., 2005) in ADHD individuals. Specifically, control participants have been found to have larger N1 amplitude on successful stop trials and smaller N1 amplitude on unsuccessful stop trials. ADHD adults showed no difference in N1 amplitude between successful and unsuccessful stop trials (Bekker et al., 2005). At least one study also suggested that children with ADHD have deficits in orienting attention, as evidenced by reduced P1 amplitude (Perchet, Fourneret, Mauguière, & Garcia-Larrea, 2001).

Aggression and Psychophysiology in a Non-Clinical Sample

While considerable research has been done on electrophysiological correlates of externalizing behavior disorders more broadly, fewer studies have examined electrophysiological differences between aggressive and non-aggressive groups. Those studies of electrophysiological correlates (P3 and non-P3) of aggressive behavior in "normal" participants are briefly reviewed.

Mathias and Stanford (1999) recruited impulsively aggressive (n = 11) and non-aggressive (n = 11) college students to participate in an ERP study. The former group was both more impulsive and more aggressive, according to self-report measures. Participants completed a standard visual oddball task (80% non-target stimuli and 20% target stimuli), and a modified oddball task that including a high-frequency non-target stimuli (80% of trials), a low-frequency target stimuli (10%), and a low-frequency unexpected non-target stimuli (10%). The authors observed that the impulsive aggressive group showed reduced P3 amplitude to target stimuli in both tasks, as well as longer P3 latency in the standard task. This effect was more pronounced at posterior sites (i.e., Pz) compared to anterior sites (Fz or Cz). Interestingly, impulsively aggressive individuals in this study took longer to process stimulus information (as evidenced by longer P3 latency) though they showed shorter reaction times compared to the non-aggressive group, suggesting they responded to the stimuli without having fully processed it.

Harmon-Jones et al. (1997) examined the ERPs of nine psychiatric-inpatient adolescents (diagnoses not specified) and 25 community control adolescents. A modified oddball task and a continuous performance task (CPT) were used to elicit ERPs. Aggression history was positively related to impulsivity and negatively to reading skills and intelligence. The authors found that physical and verbal aggression and self-reported impulsivity were negatively related to the amplitude of the P3 elicited by the oddball task, but not the CPT task. Specifically, aggressive children had smaller P3s to target stimuli. No significant relationships were found between P3 latency and any measure of aggression or impulsiveness (Harmon-Jones et al., 1997).

Bond and Surguy (2000) examined P3 components elicited by a standard auditory oddball task, using a sample of 28 males from the community split into two groups (high and low aggression/hostility) based on their scores on the Buss-Durkee Hostility Inventory (BDHI; Buss & Durkey, 1957). The high aggressive group showed longer latency P3s compared to the low aggressive group. No differences were observed in P3 amplitude, which the authors attributed to the low level of aggression overall in the sample. Participants with high hostility scores also showed prolonged P3 latency compared to those with low hostility scores (Bond & Surguy, 2000). In a similar study, Gerstle and Mathias (1998) recruited 44 college students and classified them as non-aggressive or impulsively aggressive. Participants were classified as aggressive if they had had several episodes (including at least two in the prior month) of failing to resist aggressive impulses to either hurt someone else or damage property, and which were out of proportion to the instigating stressor. Participants also had to obtain a certain minimum score on the BDHI to be classified as aggressive. The authors found that impulsively aggressive participants showed smaller P3 amplitude, but no differences in P3 latency to the target stimuli in the oddball task (Gerstle & Mathias, 1998).

Similar selection criteria were used to recruit undergraduate participants for a study of early ERPs. Houston and Stanford (2001) divided 30 undergraduates into two groups: impulsively aggressive individuals and non-aggressive controls, using the same criteria as Gerstle and Mathias (1998). All participants were exposed to three intensities of photic stimulation to elicit early latency ERP components. The authors found differences between the groups with impulsive aggressive individuals showing reduced P1 (indicating poorer sensory gaiting), and increased N1 amplitude (indicating greater orienting to novel stimuli). Impulsive aggressors also showed increased augmenting (P1-N1) to increasingly intense stimuli, which is often interpreted as enhanced tolerance of intense stimuli, as one might see in psychopaths. Finally, impulsive aggressors showed shorter latency of the P1, N1, and P2 components. Overall, the results are consistent with the notion that impulsive aggressive individuals display lower levels of arousal

that they seek to ameliorate through faster orienting to and greater processing of novel stimuli (i.e., sensation seeking). The authors hypothesized that the effects of these differences in early processing of stimuli may carry over to later information processing functions (Houston & Stanford, 2001).

In a follow-up study, the same group of authors recruited men who met partial criteria for Intermittent Explosive Disorder (IED) from the community for a study on the effects of phenytoin (an anti-aggression drug) on aggression and ERPs. Participants were administered placebo and phenytoin in counterbalanced order for two separate six-week periods. Along with decreases in self-reported aggressive outbursts during the drug period, participants were observed to have longer P1, N1, and P2 component latencies. There was also a statistical trend toward participants having smaller N1 amplitudes while taking phenytoin versus placebo. The authors suggested that the administration of phenytoin normalizes early sensory and attentional processes, such that participants are better able to filter extraneous sensory information (N1; sensory gaiting) while focusing on relevant information (P1 amplitude), and are better able to orient their attention (P1, N1, P2 latencies; Stanford, Houston, Mathias, Greve, Villemarette-Pittman, & Adams, 2001).

EEG differences in cortical activation have also been observed in aggressive individuals. For example, Santesso and colleagues (2005) found that children who had more externalizing behaviors including aggression showed greater relative right frontal resting EEG activation compared to a control group. The authors suggested that this finding might be due to greater negative affect in the externalizing group (Santesso, Reker, Schmidt, & Segalowitz, 2005), though it is also possible that the result might be related to deficits in inhibition, which is largely carried out by the right hemisphere of the brain (Peterson et al., 2008).

ERPs and Social/Emotional Information Processing

In a small number of studies, researchers have begun to test the effects of emotionally-valenced stimuli on P3 amplitude. These studies may help to shed light on the cognitive processes

involved in the perception of threat and aggressive cues in samples of normal and aggressive individuals.

Surguy and Bond (2006) recruited 32 healthy individuals and divided them into two groups (high and low hostile aggression) based on a mean-split of their Buss-Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) scores. The authors presented participants with neutral non-target words (80%), neutral target (food-related) words (10%), and aggression-themed non-target words (10%) in a modified oddball task. P3 amplitude in response to neutral (target and non-target) words did not differ between the two groups. However, the aggressive group showed lower P3 amplitude in response to the distractor aggression words compared to the non-aggressive group. The authors theorized that the reduced P3 amplitudes reflect a deficit of cognitive control (i.e., self-regulation) in reactively aggressive individuals (Gerstle et al., 1998).

De Pascalis and colleagues were interested in the effects of personality traits (anxiety and impulsivity) on P3 amplitude in response to positive and negative target words in a modified oddball task. They found that participants high in trait anxiety were more sensitive (as evidenced by larger P3 amplitude) to the negatively valenced emotional target words than were participants low in trait anxiety. They found the reverse pattern for impulsive participants, such that high impulsive individuals evidenced lower P3 amplitude and later P3 peak in response to negative target words than did low impulsive participants. The authors concluded that P3 amplitude is sensitive to the emotional content of the stimuli and that impulsive participants may allocate fewer cognitive resources to inhibitory processes and may be slower to allocate cognitive resources to inhibitory processing.

Thomas and colleagues (2007) used a variation on the emotional Stroop task to examine the effect of threat versus neutral words on reaction time and P3 amplitude and latency in 22 healthy college undergraduates. They found larger P3 amplitudes in response to the threat words (e.g., war, torture, abuse) than to the neutral words, but no difference in P3 latency. Interestingly, there were no observed differences in reaction time between the two classes of words. The

authors concluded that the threat words triggered greater processing due to the emotional salience of the words. The results also suggest that analyses of ERPs are more sensitive to differences in cognitive processes than is reaction time. This study provides useful information as to how "normal" (non-aggressive) individuals process threat-related stimuli.

Bertsch and colleagues (2009) investigated the effects of provocation on P3 amplitude. They used a modified Stroop task in which faces showing different emotions (happy, sad, angry, or neutral) were briefly displayed in one of four colors: red, blue, yellow, or green. Participants were instructed to name the color of the stimulus. Participants completed a Taylor Reaction-Time Task prior to the Stroop task and half were provoked by the opponent with high shocks while the other half received only low shocks. Provoked participants showed greater P3 amplitudes to all faces regardless of the emotion. They also showed larger P3 amplitudes to happy compared to neutral faces at all electrode sites and *lower* P3 amplitude to angry faces compared to neutral faces at right lateral sites. Their results suggest that recent provocation may serve to enhance processing of incoming emotional stimuli (in this case faces). In contrast to the previous studies cited, they did not observe larger P3 amplitudes for threatening or emotional stimuli compared to neutral stimuli.

ERPs and Laboratory Aggression

To date only one known study has examined event related potentials "online" in the context of a simulated aggressive encounter. Krämer and colleagues (2007) examined ERP waveforms generated during completion of a modified version of the TRT. In their study, 40 men and women participated in a competitive reaction time game against two distinct fictitious opponents, one low aggressive and one high aggressive opponent. In the context of the game, participants had the opportunity to exchange loud noise blasts with and deduct money from the opponent. Each trial in the task was broken into three stages for the purpose of analyzing ERPs, a *decision phase*, in which the participant selected a noise level to be administered in the event that they won the reaction time trial, the actual reaction time trial, and an *outcome phase*, in which the

participant was informed whether they "won" or "lost" the trial (i.e., whether they or their opponent would be receiving the loud noise and money deduction). The authors examined ERP components during the decision and outcome phases. Results of the study indicated that under high provocation, men who reported greater aggressive ideation showed greater N2 amplitude during the decision to inhibit aggressive responding (i.e., when highly aggressive individuals responded to high provocation with low levels of retaliation), which the authors interpreted as indicating that greater executive control resources are needed for aggressive men to inhibit aggressive responding when provoked. In addition, individuals low in aggressive ideation showed a similar magnitude negative deflection around 300 ms at frontal sites (FRN) irrespective of whether they or their opponent received the noise blast. The authors interpreted the FRN component as reflecting processes related to empathy (more specifically, the positive or negative valence of an outcome). In contrast, individuals high in aggressive ideation seemed to have higher FRN amplitudes when they were attacked, and lower amplitudes when they were aggressing. The authors interpreted this to mean that delivering and receiving aggressive attacks are equally aversive to individuals low in aggressive ideation, but that high aggressive ideation individuals are more averse to being aggressed upon compared to aggressing (Krämer, Büttner, Roth, & Münte, 2007).

Although the results of this study are suggestive, there are several limitations worth mentioning. A major limitation of this study was the formation of groups based on an aggression measure that assesses aggressive disposition rather than actual history of aggressive behavior. Items such as "If someone provokes me, I want to punish him badly" are evaluative statements that better reflect aggressive ideation which may or may not be associated with actual aggressive behaviors. Indeed, the authors found considerable overlap among high and low trait aggressive participants in the actual levels of aggression they displayed during the task, and aggressive ideation and aggressive behavior on the laboratory task were uncorrelated in women participants.

No study to date has examined ERP components in response to provocation as a function of aggression history, which is what the present study undertook to do.

Another major limitation of this study is that provocation intensity was minimal and confounded with opponent. Specifically, provocation was not equated to a highly noxious stimulus that was unequivocally provocative, but rather represented a rather mild irritant (a noise blast at the level of their tolerance threshold). Moreover, the two levels of provocation were administered by two different fictional opponents, confounding provocation with provocateur. This design is likely to underestimate effects as participants are likely to engage in less evaluative processing of the opponent's motive, and are more likely to simply label one opponent as "aggressive" and the other as "not aggressive."

The authors also interpret slight differences in *p* values (e.g., $p = .001$ versus $p = .005$) for significant effects in post-hoc analyses to deconstruct interactions. This approach to decomposing interactions is not at all informative, especially in the absence of effect size presentations, given that slight differences in *p* values can be affected by minor differences in variance components that are irrelevant to null hypothesis testing. Finally, the authors do not report P3 findings which is surprising given that P3 has been implicated in the evaluation of threat cues, impulsivity, and externalizing behavior problems.

Summary

As can be seen from the review above, there have been many studies to suggest differences in neurophysiological processes between individuals with externalizing disorders and those without, particularly on tasks that require participants to evaluate and categorize stimuli and to inhibit a motor response. Studies that have used aggressive and non-aggressive samples have been consistent with those of other externalizing disorders, with the exception of psychopathy. Individuals with externalizing disorders and aggression have been found to exhibit differences in information processing such as reduced P3 amplitude and later P3 latency, and this finding has been robust across different diagnostic groups, populations, and experimental tasks. Reduced P3

amplitudes may indicate that fewer cognitive resources are engaged in processes related to identifying, discriminating, categorizing, and responding to stimuli. With regard to the perception of provocation, individuals with P3 abnormalities may be impaired in their ability to distinguish provoking from non-provoking cues, relative to controls. In tasks that require participants to inhibit a prepotent response, reduced P3 amplitudes may indicate a reduced ability to recruit cognitive resources necessary to inhibit a response.

A small number of studies have used psychophysiological techniques to examine differences in how individuals process provocative information. One study by Kramer and colleagues even recorded ERPs while the participant completed a laboratory measure of aggression. Overall these studies suggest that (a) P3 is sensitive to the emotional salience of material presented; (b) healthy participants and anxious participants show larger P3 amplitudes to threat-related and negative emotion material than to neutral material; (c) impulsive participants show smaller P3 amplitude to negative emotion material than do non-impulsive participants; and (d) provocation may have a priming effect on ERPs such that larger P3 amplitudes are observed following provocation, at least in healthy participants.

A number of studies have linked certain ERP components to inhibitory processes (e.g., N2, No-Go P3, N450, and NSW) through the use of Go/Stop and related tasks. Differences in the amplitudes and latencies of these components have been observed between relevant clinical samples (e.g., individuals with ADHD) and controls. However, very little is known about how these components might function in the context of a realistic aggressive encounter.

Rationale

The purpose of this study was to examine ERP components that are relevant to the perception of provocation and the expression of aggressive behavior. Considerable research using self-report, behavioral, and ERP data suggests that aggressive and non-aggressive individuals display differences in how they process and respond to social cues. The literature to date suggests that the information reflected in event related potentials can provide insights into the cognitive

processes at work in disorders ranging from ADHD to substance use, personality traits such as psychopathy and impulsivity, and behaviors such as aggression. For the most part, this work has involved relating abnormalities in ERP components to static characteristics such as diagnosis or personality traits. Little work has examined the cognitive processes related to aggression that is reflected in real time as a behavior is being carried out. This is particularly true in the area of aggression, where only one study has examined ERPs "online" during a laboratory-controlled escalating aggressive encounter (Krämer et al., 2007). As reviewed above, the Krämer study suffers a number of limitations. The present study replicates and extends Krämer's study by including a more ecologically valid provocation (i.e., a noise that is purportedly twice the intensity of the participant's tolerance threshold). The study was carried out on participants with and without a history of aggression and provided participants with an extreme aggression response option.

Objectives

The purpose of this study was to examine the neurophysiological bases of aggression in real time in the context of an aggressive encounter as a function of provocation and aggression history. In order to examine the ERP components of interest, the classic Taylor Reaction-Time Task was modified. Provocation and aggression trials were separated by block to examine the relevant ERPs separately. In order to examine processing of threat-related stimuli, the provocation trials were presented in the form of a modified oddball task, in which the participant categorized what level of noise blast the opponent just set for them, based on the word (*low, medium,* or *high*) appearing on the computer screen. Inhibition-related ERPs were examined during the aggression trials. Participants were free to select any noise blast (*low, medium,* or *high*) for the opponent across a series of trials. Analysis of ERPs focused on components previously identified as related to inhibition in studies using Go/No-Go tasks and other tasks involving inhibition.

This study improves on previous work in several ways. First, we sampled high- and low-aggression participants using a broadband measure of clinically relevant history of actual aggressive behavior, rather than aggressive disposition. Second, we included a highly salient and unequivocal provocation or threat condition in a within-subjects design. One criticism that has been made about the original TRT is that administration of a threat stimulus no greater than a pre-determined "unpleasantness" threshold does not represent a true attempt to inflict harm (a cardinal feature in the definition of aggression; Berman et al., 2009). Previous research has also shown that biologic influences on the expression of aggression are more easily identified when a provocation is clear and salient (Berman et al., in press; McCloskey, Berman, Echevarria, & Coccaro, 2009).

Hypotheses

Based on the review of literature, several predictions were offered. First, we hypothesized that participants identified as aggressive by the LHA would display more behavioral aggression on the modified TRT as indexed by higher noise blast selection. Second, we hypothesized that during the *provocation blocks,* participants in the high aggressive group based on life history would show decreased P3 amplitude to provocative stimuli in the high provocation block relative to the low aggressive group. We predicted that non-aggressive participants would show larger amplitude P3s to the high provocation oddball than to the low provocation oddball and that aggressive participants would not show this difference (indicating that the decreased amplitude is not accounted for by standard oddball effects). Third, we hypothesized that during the *aggression blocks* of the oddball-TRT, the amplitudes of several components previously linked to inhibitory processing would vary as a function of previous provocation level, aggression history, and the response selected (high or low retaliatory aggression). Specifically, it was predicted that following the high provocation condition, greater aggressive life history would be associated with lower N2, P3, N450, and NSW amplitudes. Any differences in P3 latency were anticipated to be in the direction of longer latency for more aggressive responding. Furthermore, we expected that

highly aggressive individuals would exhibit larger N2, N450, NSW, and No-Go P3 amplitudes on trials in which they inhibited an aggressive response compared to those in which they displayed a high level of retaliation.

CHAPTER III

METHODS

Participants

Sixty-five men (n = 30) and women (n = 35) right-handed participants between the ages of 18 and 45 were recruited to participate in a study on "EEG and reaction-time." Changes in P3 have been observed in individuals older than 45 (Kutas, Iragui, & Hillyard, 1994); for this reason, the age limit was capped for participation. Participants were recruited from undergraduate psychology courses to earn course credit (N = 39; 13 males, 26 females) and from the community (N = 26; 17 males, nine females). Community residents were recruited through a campus-wide email announcement and from a local diversionary program for non-violent felony offenders. Community participants received either $25 for their participation or community service hours. Student participants were prescreened through online research and survey management websites (Sona-systems.com and Surveymonkey.com). Community participants were prescreened through Surveymonkey.com and telephone interviews. Approximately 500 people completed the prescreening for this study. Exclusionary criteria for the study included (a) age over 45 years; (b) left-handedness or ambidextrousness (due to potential cortical laterality differences); (c) current psychotropic medication use; (d) history of psychotic or bipolar disorder; (e) history of severe traumatic brain injury (with loss of consciousness and personality change); and (f) visual, auditory, reading, neurological, or motor problems that would interfere with the ability to perform the study tasks. Participants with corrected-to-normal vision (i.e., individuals wearing glasses or contacts) were admitted into the study. The Life History of Aggression scale (LHA; Coccaro, Berman, & Kavoussi, 1997) was administered with the prescreener in order to recruit approximately equal numbers of aggressive and non-aggressive males and females. A cut-score of 10 was used to create groups, with participants scoring 10 or higher being classified as aggressive.

Table 1

Descriptive Statistics and Group Differences for the Aggressive and Non-aggressive Groups

	Aggressive[a]		Non-Aggressive		Chi-square[b]	T-test
	n	*M (sd)*	*n*	*M (sd)*	χ^2 (*df*)	*t* (*df*)
Age (*46*)	--	22.5 (*2.5*)	--	22.3 (*4.3*)	--	-0.18
Gender					.72 (*1*)	--
Male	12	--	11	--		
Female	10	--	15	--		
Race					3.16 (*4*)	--
White/Cauc.	13	--	13	--		
African Am.	8	--	11	--		
Hispanic	1	--	0	--		
Native Am.	0	--	1	--		
Other	0	--	1	--		
Source					1.79 (*1*)	--
Student	12	--	19	--		
Community	10	--	7	--		
Marital status[c]					0.21 (*2*)	--
Single	20	--	23	--		
Married	1	--	2	--		
Divorced	1	--	1	--		
Education					1.76 (*3*)	--
High School	3	--	2	--		
Some college	16	--	21	--		
College deg.	3	--	2	--		
Some graduate	0	--	1	--		
Living Situation					0.44 (*3*)	--
Alone	4	--	3	--		
With partner	4	--	5	--		
Roommate(s)	9	--	12	--		
With Family	5	--	6	--		

*$p < .05$. [a] A cut-score of >= 10 was used to classify participants as aggressive or non-aggressive. [b] All Chi-squares are two-way Chi-squares. [c] Single = never married.

Several participants who completed part or all of the study had their data excluded from the final analyses for the following reasons: (a) problems with the stimulus protocol (seven participants); (b) incomplete data (nine participants); and (c) non-compliance with some aspect of the study (one participant). Overall, excluded participants did not differ from included participants as a function of gender ($\chi^2 = .23$, $p > .05$) but differed slightly in age ($M_i = 22.4$, $sd = 3.6$; $M_e = 24.8$, $sd = 5.6$; $t_{(63)} = .20$, $p < .05$). Excluded participants were also disproportionately drawn from the diversionary program ($\chi^2 = 22.8$, $p < .001$) which was due to the fact that these were the first participants in the study. In fact, all diversionary participants had their data excluded as a result of a technical problem with the stimulus program.

Forty-eight individuals' data were retained for the final analyses (Table 1). The final sample included 23 males and 25 females. Twenty-six participants (11 males; 15 females) were classified as aggressive based on a cut-score of 10 on the LHA. Twenty-two participants were classified as non-aggressive (12 males; ten females). Aggressive participants (defined by a cut-score of 10 on the Life History of Aggression Scale; Coccaro, Berman, & Kavoussi, 1997) displayed significantly higher scores on a self-report measure of aggression disposition, the BPAQ compared to the non-aggressive group (Buss-Perry Aggression Questionnaire, Total score; Buss & Perry, 1992; $M_{NA} = 57.42$, $sd = 14.3$, $M_A = 74.36$, $sd = 17.6$, $t_{(46)} = -3.67$, $p < .001$; Table 10), supporting the validity of the study groups. The average age of the final sample was 22.4 years ($sd = 3.6$). Thirty-one participants were students and 17 were community volunteers. Self-identified racial composition of the sample is as follows: 54% White/Caucasian, 40% Black/African American, and 2% each Hispanic, Native American, and bi- or multiracial. Ninety percent of the sample had never been married; 6% were married; and 4% were divorced. Approximately 15% of the participants lived alone, 19% lived with a spouse or partner, 44% lived with one or more roommates, and 23% lived with family (due to rounding percentages do not equal 100%). With regard to education 10% of participants had completed high school but no college; 77% had completed some college; 10% were college graduates; and 2% had completed

some graduate coursework. Current household income for participants in our sample ranged from under $10,000 per year (37.5%; modal income) to over $100,000 per year, with the mean and median income being between $30,000 and $40,000 per year. One participant did not provide current income data. Seventeen subjects (37%) had corrected-to-normal vision. Descriptive statistics for these variables (except for income) are presented separately for the aggressive and non-aggressive participants in Table 1. Overall, the two groups did not differ as a function of age, gender composition, racial composition, living arrangement, marital status, level of education, or source (community vs. student; all *p*s > .05).

Measures

Aggression History: The Life History of Aggression Scale

The Aggression subscale of the LHA (Coccaro et al., 1997) was used to assess the frequency and severity of aggression across the lifespan. The LHA consists of eleven items, each having six response options indicating how many times the participant has engaged in the specified behavior. Response options range from (0) *never happened* to (5) *happened so many times I couldn't give a number*. The aggression subscale was the measure of interest in this study. This subscale consists of five items assessing angry outbursts, physical fighting, verbal aggression, assaults, and aggression toward objects. Inter-rater agreement is good (ICC = .94), as are internal consistency (α =.87) and test-retest reliability (r > .80; Coccaro et al., 1997). Alpha for this study was .78. Scores on the Aggression subscale of the LHA have been shown to positively relate to aggressive behavior observed in the laboratory, as well as to biological variables theoretically associated with aggressive behavior (Berman et al., in press; Coccaro, Berman, Kavoussi, & Hauger, 1996).

Aggressive Tendencies: The Buss-Perry Aggression Questionnaire

The BPAQ (Buss & Perry, 1992) is a 29-item scale which measures aggressive feelings and disposition. Responses are on a 5-point Likert-type scale with options ranging from *Extremely unlike me* (1) to *Extremely like me* (5). Higher scores indicate greater self-perceived

aggressiveness. In addition to a total scale score, the BPAQ yields four factor analytically-derived subscales: physical aggression, verbal aggression, anger, and hostility. Test-retest reliability for the total scale score has been reported as .80, with alpha = .89 (Buss & Perry, 1992). Alpha for this sample was .91. Evidence for the validity of both the BPAQ subscales and the total scale score is supported by modest but reliable correlations between self-ratings on the BPAQ and peer-ratings of aggressive tendencies (Buss & Perry, 1992). The total scale score also seems to robustly correlate with theoretically meaningful and related constructs, and less so with unrelated constructs (Buss & Perry, 1992). For this reason, the total scale score was used for this study.

Mood Rating Scales

In order to examine the potential effects of mood on behavior during the TRT, participants completed visual analog mood rating scales at five different time points during the task. The scales consisted of a series of mood-related adjectives including nervous, calm, depressed, energetic, fearful, irritable, angry, tired, and bored. Each adjective was associated with a 10 cm horizontal line with the anchors *Not at all* and *Most ever*. Instructions to participants were to mark the point on the line best corresponding to their mood "at this minute." Later, the experimenter measured the marks for each adjective and recorded the number of millimeters from the lower anchor (range 0 to 100) as the score for that adjective. Self-ratings of "nervous" and "angry" were of particular interest in this study.

Procedure

Upon arrival at the Clinical Research Lab, participants were led into a noise-attenuated experiment room where they were given a consent form to read and sign. Participants had the opportunity to ask the experimenter any questions they had before beginning the study. Participants were prepped for the ERP recording (see below). Participants were seated in an armchair about 24 inches in front of a 17-inch cathode-ray computer monitor. Participants completed a demographic and health questionnaire as they were being prepped.

After prepping the participant, the experimenter read the task instructions for the modified oddball-TRT over an intercom system to the participant and ostensible opponent and checked with the participant before starting the task to ensure that the participant understood the task. Participants were given instructions to minimize blinking and other movements during the actual task, but were told they could blink and move about in a limited way during the break between block. The experimenter then left to an adjacent room to run the ERP stimulus and acquisition programs. The experimenter checked in with the participant at each break and provided further instructions or feedback as necessary. During the task, the experimenter monitored the participant for excessive eye and body movement and compliance via the EEG recording and a camera situated in the experiment room. The experimenter paused the experiment as needed to give feedback over the intercom or face-to-face.

Modified Taylor Reaction-Time Task (TRT)

For the modified oddball-TRT, participants wore a pair of earphones and sat in front of a computer monitor on which the visual stimuli for the task were presented. The participant was told that he or she would be competing in a task against another (actually fictitious) "opponent" in an adjoining room. After a short delay, a sound tolerance threshold was administered to determine auditory perception and discomfort threshold by administering sounds of exponentially increasing intensity up to a maximum of 115 dB, stopping when the participant reported that the sound was "definitely unpleasant" and that they wished to stop the procedure. To enhance the credibility of the experimental situation, this procedure was repeated with the other "subject."

Before beginning the reaction time task, instructions for the task were provided via intercom to both "subjects." The experimental protocol consisted of four blocks: two *provocation blocks* and two *aggression blocks.* One of the provocation blocks was a "high provocation" block; the other was a "low provocation" block. Participants completed all four blocks in the following order: (a) provocation block; (b) aggression block; (c) provocation block; and (d) aggression block. Provocation was counterbalanced to control for potential order effects. Participants were

randomly assigned to receive either the low provocation block or the high provocation block first, and the other provocation block later. All blocks consisted of 200 trials (for a total of 800 trials across the task). A two-minute break was administered halfway through each block and between each block. A three-minute break was provided between blocks two and three, which was the halfway point of the protocol. Printed instructions were also presented on the computer screen before the start of each block.

During the provocation blocks, participants were told that their "opponent" would be selecting noise blasts (low, medium, or high) that the participant would receive later in the experiment. They were also informed that the low and medium noise blasts would be below their previously determined pain threshold (25% and 75% of their threshold, respectively), but that the high noise blast would be twice the intensity of their threshold, and that this level of noise might cause headache, ringing in the ears, and difficulty hearing for a few hours. In reality, participants never received a noise blast above their threshold. Participants were told that their opponent's selection would appear on the computer screen (i.e., the words "low," "medium," and "high") and that they should identify their opponent's selection by pressing the corresponding button (low, medium, or high) on the button-press box. A pseudo-randomly varying stimulus onset asynchrony (SOA) between 3.7 and 4.1 seconds long separated each stimulus onset. If participants did not respond within 1.5 seconds of the presentation of the stimulus, a tone sounded indicating they should pay closer attention to the task. All stimuli in the experiment were presented on the screen for 200 ms. The pattern of stimuli appearing on the screen differed as a function of whether the block was the low provocation block or the high provocation block. In keeping with the standard oddball design, 80% of the stimuli appearing on the screen (i.e., the opponent's "choice") during each block were the word "medium." In the low provocation block, the remaining 20% of the trials were the word "low." In the high provocation block, the remaining 20% were the word "high." ERP waveforms were averaged separately for each stimulus (low, medium, and high) and for each block separately (low vs. high provocation and aggression vs. provocation). Trials

on which the participant did not respond or responded with an incorrect button press were discarded from any analyses.

During the *aggression blocks*, participants were instructed that the word "select" would appear on the screen and that this word was their cue to select a noise tone (low, medium, or high) for the opponent. Both blocks contained 200 trials with the same SOAs as the provocation blocks. Participants had 1.5 seconds to respond before an attention beep sounded. The two aggression blocks appeared identical; however, ERP waveforms were averaged separately for the two blocks, with one block representing aggression following low provocation and the other aggression following high provocation. ERP waveforms were also averaged separately for the participant's low, medium, and high selections. Trials in which the participant did not select a noise were discarded from analyses. Following each aggression block, the participant was informed that "the computer" would "randomly select" some of the noise blasts selected by the opponent to administer to the participant (and vice versa). Ten noise blasts (all below the participant's pain threshold) were delivered binaurally for 1020 ms (10 ms rise time/1000 ms plateau/10 ms fall time). The high noise blast was never actually administered during the protocol.

Event Related Potentials

Task stimuli were presented and event related potentials were recorded using custom-built equipment manufactured by the James Long Company©. This equipment includes a 16-channel bio-amplifier, a stimulus computer, a data acquisition computer, and a button-pad for key presses. Electrodes (F3/4, C3/4, P3/4, F7/8, M1/2, AFz, Fz, Cz, and Pz) were placed on the scalp according to the 10/20 International System using an elastic electrode cap. A conducive gel (ElectroGel©) was applied to the scalp using a blunt needle that was also used to abrade the skin's surface in order to reduce impedances between the scalp and electrode. Individual electrodes were also placed 0.5 cm above and below the left eye and 1 cm from the outer canthi of each eye to collect EOG data (HEOG and VEOG, respectively) so that eye movement artifact could be either

corrected or the data discarded from the final ERP waveforms. Before collecting any data, impedances were assessed and recorded for each electrode site. All scalp electrode impedances were below 5 kΩ and EOG electrode impedances were below 10 kΩ. Calibration data were collected before the task. Scalp electrodes were referenced online to the left mastoid (M1) and re-referenced offline to the average of the right and left mastoid (M1+M2/2). O1 served as a ground. A band pass filter of 0.05 to 100 Hz was used online and data were collected using a 50,000 Hz sampling rate. A low-pass filter of 30 Hz was applied offline along using a Sinc filter. This was done to eliminate 60 Hz noise. Artifact was managed in three stages. First, eye blink regression was used to correct for eyeblink artifacts. Next, automatic artifact scoring was applied to all artifacts exceeding a certain threshold in mV on channels F3 and F4 (these data were discarded). Finally, the data were reviewed manually to discard any significant remaining artifacts. Trials containing any artifact were discarded.

Statistical Analyses

Statistical analyses were conducted using SPSS (Version 16.0). Data were examined for data entry errors and outliers prior to conducting any analyses. Average waveforms were calculated for each trial type for each participant. Any error trials (trials on which the participant did not respond) were excluded from analyses. Grand mean waveforms were also computed across all participants in each group (high and low aggression and gender) for each type of trial. Exploratory analyses were conducted to identify any data abnormalities such as skew or outliers. Gender was included as a moderator in all analyses. For the ERP data, separate Mixed Model ANOVAs were conducted with P3, N2, N450, and NSW amplitudes and latencies. For examination of components in the *aggression blocks* (all components), separate Mixed Model ANOVAs were conducted with aggression history (high vs. low) and gender as between subjects factors, and scalp region (frontal, central, parietal), provocation (high vs. low), and behavioral response (selection of low, medium, or high noise blast) as within-subjects variables. For the component to be examined in the *provocation blocks* (P3 amplitude and latency), aggression

history and gender again served as between-subjects variables, while scalp region, provocation, and stimulus probability (frequent versus oddball) served as within subjects variables. Interaction terms were decomposed using simple effects analyses, followed by planned comparisons. Mauchley's test (*W*) was used to test for violations of sphericity, and the Greenhouse-Geisser correction was used to correct violations of the assumption of sphericity (Mathias & Stanford, 1999).

CHAPTER IV

RESULTS

Data Management

Before conducting the main analyses, ERP data were examined and cleaned to eliminate invalid trials. The data management procedures, which varied by type of block, are described below.

Provocation Blocks

In the provocation blocks, participants were instructed to identify the level of noise blasts set by the "opponent" by pressing a button on the response panel. Omission errors and identification errors were excluded. The majority of trials (98%) were correctly identified across all participants. Data on the percentage of correct trials as a function of aggression group status, provocation, and stimulus probability are presented in Table 2. A 2 (aggression group) × 2 (gender) × 2 (provocation) × 2 (stimulus probability) mixed-model ANOVA revealed a main effect of stimulus probability ($F_{(1, 46)} = 44.66$, $p < .001$) and interaction effects between aggression group and provocation ($F_{(1, 46)} = 5.76$, $p < .05$) and provocation and stimulus probability ($F_{(1, 46)} = 4.24$, $p < .05$) on errors/omissions. The interaction between aggression group, provocation, and stimulus probability approached significance on the percentage of errors and omissions, $F_{(1, 46)} = 3.91$, $p = .054$.

With regard to the main effect of stimulus probability, more errors and omissions were committed for oddball than for frequent stimuli, $M_o = .08$, $sd = .07$; $M_f = .01$, $sd = .01$, $t_{(47)} = 6.80$, $p < .001$. Participants committed more errors in response to the high provocation oddball than to the low provocation oddball, $M_H = .09$, $sd = .08$, $M_L = .07$, $sd = .09$, $t_{(47)} = -2.00$, $p = .051$, which accounted for the 2-way provocation × stimulus probability interaction. Decomposition of the 2-way aggression group × provocation interaction revealed that aggressive participants committed twice as many errors in the high provocation ($M = .06$, $sd = .04$) as in the low provocation block ($M = .03$, $sd = .04$, $t_{(25)} = -3.54$, $p < .01$), whereas non-aggressive

participants made similar numbers of errors across both provocation blocks (M_L =.04 , *sd* = .06; M_H =.04 , *sd* = .04; $t_{(21)}$ = .39, *p* = .70).

Table 2

Provocation Blocks Excluded and Retained Trials

	Aggressive[a]		Non-Aggressive[b]		Difference
	%[c]	*(Range)*	*%*[c]	*(Range)*	*t*[d]
LL_P[e]					
Total	100%		100%		
Errors/Omissions	-6%	*(0-25%)*	-7%	*(0-45%)*	0.30
Artifacts	-9%	*(0-45%)*	-8%	*(0-30%)*	0.09
Total retained	85%	*(55-100%)*	84%	*(50-100%)*	-0.31
LM_P					
Total	100%		100%		
Errors/Omissions	-1%	*(0-2%)*	-1%	*(0-4%)*	1.35
Artifacts	-9%	*(1-31%)*	-9%	*(1-40%)*	0.04
Total retained	91%	*(68-98%)*	90%	*(59-98%)*	-0.18
HM_P					
Total	100%		100%		
Errors/Omissions	-1%	*(0-4%)*	-1%	*(0-3%)*	-1.18
Artifacts	-7%	*(0-18%)*	-10%	*(1-70%)*	0.79
Total retained	92%	*(82-100%)*	89%	*(27-99%)*	-0.64
HH_P					
Total	100%		100%		
Errors/Omissions	-10%	*(70-100%)*	-7%	*(0-25%)*	-1.38
Artifacts	-8%	*(0-30%)*	-8%	*(0-50%)*	0.79
Total retained	82%	*(68-95%)*	85%	*(35-98%)*	0.27

*$p < .05$; [a] $n = 26$. [b] $n = 22$. [c] Mean percentage. Numbers have been rounded and therefore may not equal 100%. [d] $df = 46$. [e] See List of Abbreviations for definition of terms.

Follow up of the 3-way aggression × provocation × stimulus probability trend revealed that the two way interaction between stimulus probability and provocation was significant in aggressive participants for the oddball stimuli (M_H = .10, *sd* = .08; M_L = .06, *sd* = .07; $t_{(25)}$ = -3.68, $p < .001$) but not in non-aggressive participants (M_H = .07, *sd* = .07; M_L = .08, *sd* = .11; $t_{(21)}$ = .18, $p > .05$). This effect appears to account for the interaction effect of aggression group and provocation, as aggressive participants did not show any difference in errors when responding to low provocation frequent stimuli (M_L = .01, *sd* = .01) versus high provocation frequent stimuli (M_H = .01, *sd* = .01; $t_{(25)}$ = -0.97, $p > .05$).

Trials containing significant artifact due to blinking, body movement, or other anomalies were also discarded. Across all participants, 8.7% of valid provocation trials were discarded due to artifact contamination (range 0-41%). A mixed-model ANOVA was used to examine the effects of aggression group, provocation, and stimulus probability on percent of trials excluded due to artifact. No significant main effects or interactions emerged. Data on the percent of provocation trials eliminated due to artifact are presented in Table 2.

Across all participants 89% of all provocation trials were retained for calculation of ERPs (range 54-99%). The number of trials retained in each condition (provocation × stimulus probability) did not differ between the aggressive and non-aggressive groups. Percent of retained trials by aggression group and trial type are presented in Table 2.

Aggression Blocks

For each of the 400 trials contained in the aggression blocks, participants were free to select any noise blast (low, medium, or high) for the opponent. Therefore, there was no opportunity for errors. Omissions were possible but rare (less than 2% of trials overall; range 0-25%). The percent of omissions did not differ between aggressive and non-aggressive participants, $t_{(46)}$ = .25, $p > .05$.

Table 3

Aggression Blocks Excluded and Retained Trials

	Aggressive[a]		Non-Aggressive[b]		Difference
	%[c]	*(Range)*	*%*[c]	*(Range)*	*t (df)*
LL_A[d]					
Total	100%		100%		
Artifact	-12%	*(1-50%)*	-12%	*(0-44%)*	-0.02 (46)
Total retained	88%	*(50-99%)*	88%	*(56-100%)*	
LM_A					
Total	100%		100%		
Artifact	-10%	*(0-42%)*	-10%	*(0-37%)*	0.24 (43)
Total retained	90%	*(58-100%)*	89%	*(63-100%)*	
LH_A					
Total	100%		100%		
Artifact	-9%	*(0-57%)*	-2%	*(0-19%)*	-0.82 (23)
Total retained	87%	*(43-100%)*	95%	*(81-100%)*	
HL_A					
Total	100%		100%		
Artifact	-10%	*(0-35%)*	-8%	*(0-63%)*	-0.07 (46)
Total retained	90%	*(65-100%)*	92%	*(37-100%)*	
HM_A					
Total	100%		100%		
Artifact	-10%	*(0-26%)*	-8%	*(0-60%)*	0.34 (44)
Total retained	90%	*(74-100%)*	91%	*(40-100%)*	
HH_A					
Total	100%		100%		
Artifact	-9%	*(0-33%)*	-8%	*(0-67%)*	0.23 (42)
Total retained	91%	*(67-100%)*	91%	*(33-00%)*	

*$p < .05$; [a] $n = 26$. [b] $n = 22$. [c] Mean percentage. Numbers have been rounded and therefore may not equal 100%. [d] See List of Abbreviations for definition of terms.

Across all participants, 10.4% of valid aggression trials were discarded due to artifact (range 1-50%). A mixed-model ANOVA was used to examine the effects of aggression group, provocation, and response on percent of trials excluded due to artifact. There was a significant main effect of response on artifact, $F_{(1.48, 46)} = 8.73$, $p < .01$; an interaction between provocation and response, $F_{(1.35, 46)} = 4.95$, $p < .05$; and an interaction trend between aggression group, provocation, and response, $F_{(1.35, 46)} = 3.35$, $p = .06$, on artifact trials.

Analysis of the higher order 3-way interaction revealed that among non-aggressive participants following the low provocation block, there was significantly less artifact associated with the "high" response ($M = .02$, $sd = .05$) compared to the "medium" response ($M = .10$, $sd = .10$; $t_{(21)} = 3.69$, $p < .01$) or the "low" response ($M = .12$, $sd = .11$; $t_{(21)} = 2.95$, $p < .01$). Among aggressive participants there was significantly more artifact associated with the "low" response to low provocation than with the "medium" response ($M = .12$, $sd = .12$; $M = .10$, $sd = .02$; $t_{(25)} = 2.20$, $p < .05$). Data on the percent of artifact by aggression group, provocation, and response are presented in Table 3.

Across all participants 88% of aggression trials were retained for calculation of ERPs (range 49-99%). The number of trials retained in each condition (provocation × response) did not differ between the aggressive and non-aggressive groups. Data on the percentage of retained trials by trial type and group are presented in Table 3.

Self-Report and Behavioral Data

Order Effects

As described previously, participants were randomly assigned to receive the high provocation or low provocation block first. We tested for ordering effects on participants' response choices in the aggression blocks with a 1 (order) × 2 (provocation) × 2 (response) mixed model ANOVA. There were no significant main effects or interactions involving the order of counterbalancing. Main effects and interactions involving order are displayed in Table 4.

Table 4

Effect of Counterbalancing (Provocation) Order on Aggressive Responses

	F	*(df)*
Main Effects		
Order	1.88	(1)
Provocation	1.43	(1)
Response	46.03	(2)***
Two-Way Interactions		
Order*Provocation	1.93	(1)
Order*Response	2.14	(2)
Response*Provocation	17.98	(2)***
Three-Way Interactions		
Order*Provocation*Response	2.00	(2)

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend).

Reaction Time

Provocation blocks. Analyses were carried out on the effects of between-subjects variables (gender and aggression group) and within-subjects variables (e.g., provocation level) on mean reaction time. For the provocation blocks, a 2 (aggression) × 2 (gender) × 2 (provocation) × 2 (stimulus probability) mixed-model ANOVA was conducted for mean reaction time (in seconds). Provocation and stimulus probability each had an effect on reaction time, $F_{(1, 44)} = 16.68$, $p < .001$ and $F_{(1, 44)} = 246.42$, $p < .001$, respectively (see Table 5). Stimuli in the high provocation block were associated with longer reaction times compared to the low provocation block ($M_L = .47$, $sd = .05$; $M_H = .50$, $sd = .07$; $t_{(47)} = -3.56$, $p < .01$), and oddball stimuli were associated with longer reaction times compared to frequent stimuli ($M_o = .52$, $sd = .06$; $M_f = .44$, $sd = .06$; $t_{(47)} = 15.09$, $p < .001$).

Table 5

Reaction Times in Provocation Blocks

	F	*(df)*
Main Effects		
Aggression	2.13	(1)
Gender	3.85	(1) †
Provocation	16.68	(1)***
Stimulus Probability	246.42	(1)***
Two-Way Interactions		
Agg*Gend	0.42	(1)
Agg*Prov	0.44	(1)
Agg*Stim	2.84	(1) †
Gend*Prov	0.11	(1)
Gend*Stim	3.45	(1) †
Prov*Stim	10.33	(1)**
Three-Way Interactions		
Agg*Gend*Prov	7.93	(1)**
Agg*Gend*Stim	1.28	(1)
Agg*Prov*Stim	0.17	(1)
Gend*Prov*Stim	2.07	(1)
Four-Way Interactions		
Agg*Gend*Prov*Stim	1.92	(1)

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend).

There was a significant interaction between gender, aggression group, and provocation level on mean reaction time, $F_{(1, 44)} = 7.39$, $p < .01$. Under low provocation conditions, aggressive and non-aggressive females had similar reaction times, while under *high* provocation aggressive females responded later than did non-aggressive females, $M = .55$, $sd = .05$, $M = .49$, $sd = .05$, $t_{(23)}$

= -3.06, $p < .01$. Aggressive females also showed longer reaction times compared to non-aggressive males, $M = .49$, $sd = .06$, $t_{(19)} = -2.63$, $p < .05$. This effect can be seen in Figure 1.

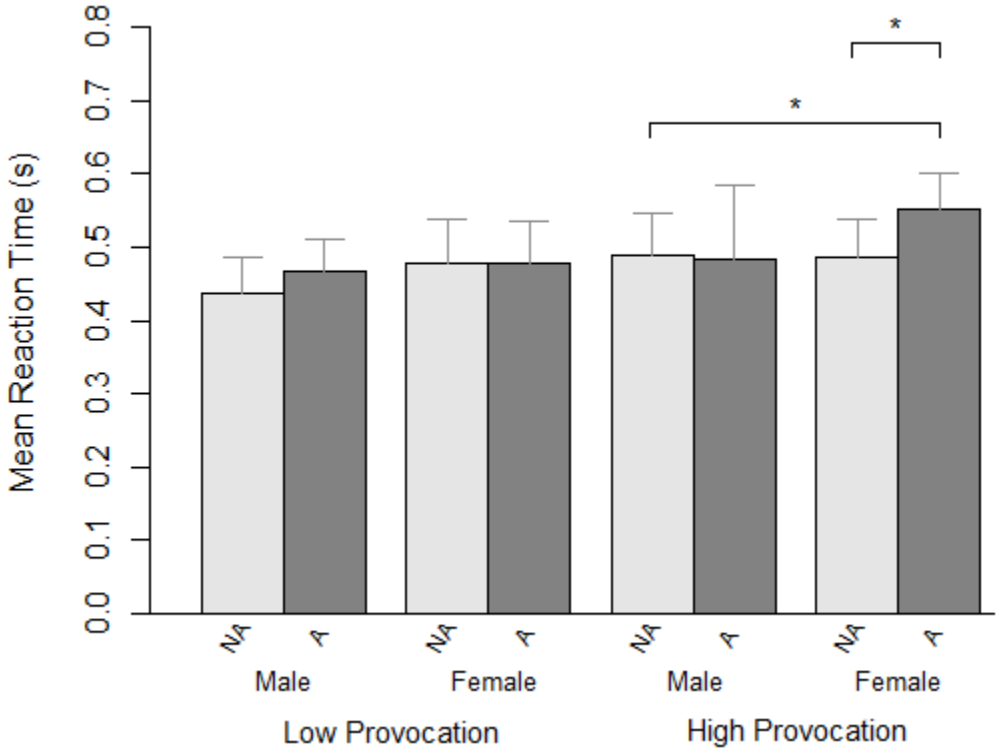

Figure 1. Reaction times in the provocation blocks as a function of aggression group, gender, and provocation. Note: * $p < .05$.

An interaction between provocation and stimulus probability also had a significant effect on reaction time, $F_{(1, 44)} = 10.33$, $p < .01$. Mean reaction times for each stimulus are as follows: high provocation oddball ($M = .55$, $sd = .08$), low provocation oddball ($M = .50$, $sd = .06$), high provocation frequent ($M = .45$, $sd = .07$), and low provocation frequent. ($M = .43$, $sd = .05$). All pairwise comparisons were significant (see Appendix D, Table D1). On average, participants responded significantly faster on error trials ($M = .38$, $sd = .09$) than on correctly identified trials ($M = .48$, $sd = .05$; $t_{(37)} = 7.98$, $p < .001$), suggesting that impulsive responding was a factor in the number of commission errors.

Aggression blocks. For the aggression blocks, a 2 (aggression) × 2 (gender) × 2 (response) × 2 (provocation) mixed model ANOVA was conducted for mean reaction time. No significant main effects or interactions emerged (see Table 6). There was a non-significant trend toward provocation affecting reaction time in the aggression blocks, $F_{(1, 22)} = 3.05$, $p < .10$, with reactions times tending to be slower in the high provocation block than the low provocation block, $M = .30$, $sd = .05$, $M = .29$, $sd = .04$. Means and standard deviations for the reaction time data are displayed in Table C1 (Appendix C).

Table 6

Reaction Times in Aggression Blocks

	F	*(df)*
Main Effects		
Aggression	1.02	(1)
Gender	.10	(1)
Provocation	3.05	(1) †
Response [a]	0.57	(1.36)
Two-Way Interactions		
Agg*Gend	0.36	(1)
Agg*Prov	0.57	(1)
Agg*Resp [a]	0.79	(1.36)
Gend*Prov	0.12	(1)
Gend*Resp [a]	0.33	(1.36)
Prov*Resp [a]	0.79	(1.17)
Three-Way Interactions		
Agg*Gend*Prov	1.71	(1)
Agg*Gend*Resp [a]	0.92	(1.36)
Agg*Prov*Resp [a]	0.81	(1.17)
Gend*Prov*Resp [a]	0.19	(1.17)
Four-Way Interactions		
Agg*Gend*Prov*Resp [a]	0.09	(1.17)

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Self-Reported and Behavioral Aggressive Behavior

Self-reported aggression and aggressive behavior on the task were examined. Correlations between self-report and behavioral aggression indices are shown in Table 7. Life History of Aggression scale (Aggression subscale) scores and Buss Perry Aggression Questionnaire total scores were highly correlated (r = .60, p < .001). BPAQ (r = .38, p < .01) and LHA (r = .40, p < .01) scores were also correlated with the use of the "high" noise blast against the opponent under low provocation. Use of the "low" noise blast following low provocation was highly correlated with use of the "low" following high provocation (r = .77, p < .001), and highly *negatively* correlated with use of the "high" noise blast in either block (see Table 7). Likewise, use of the "high" blast under low provocation was strongly predictive of the use of the "high" under high provocation (r = .53, p < .001), and negatively related to use of the "low" noise blast under high provocation (r = -.30, p < .05). Other significant relationships and trends are shown in Table 7. Planned comparisons between aggressive and non-aggressive participants are shown in Table 8. As one would expect, aggressive participants scored significantly higher than non-aggressive participants on the BPAQ Physical Aggression subscale and total scores (BPAQ Physical Aggression: M_{NA} = 17.31, sd = 5.11; M_A = 23.36, sd = 7.63; $t_{A(46)}$ = -3.27, p < .01;BPAQ Total: M_{NA} = 57.42, sd = 14.33; M_A = 74.36, sd = 17.64; $t_{T(46)}$ = -3.67, p < .001). With regard to aggressive behavior during the competitive task, aggressive participants used the "high" noise blast against the opponent significantly more times than did non-aggressive participants, M_{NA} = 7.73, sd = 11.67; M_A = 19.55, sd = 25.92, $t_{(46)}$ = -2.09, p < .05. Aggressive participants were also more likely to use the "high" noise blast at all in both the low provocation and high provocation blocks (Low provocation block: M_{NA} = 0.46, sd = 0.51; M_A = 0.73, sd = 0.46; $t_{L(46)}$ = -1.89, p < .05; High provocation block: M_{NA} = 0.85, sd = 0.37; M_A = 1.00, sd = 0.00; $t_{H(46)}$ = -1.96, p < .05). Aggressive participants used the "high" aggressive option more than non-aggressive participants under high provocation, but the difference was not statistically significant, M_{NA} = 30.69, sd = 22.32; M_A = 34.23, sd = 20.78; $t_{(46)}$ = -0.56, p > .05.

Table 7

Correlations Among Aggression Measures

	BPAQ[a]	LHA[b]	LL_A	LM_A	LH_A	HL_A	HM_A	HH_A
BPAQ	--							
LHA	.60***	--						
LL_A	-.03	-.27†	--					
LM_A	-.25†	.06	-.78***	--				
LH_A	.38**	.40**	-.54***	.05	--			
HL_A	.08	-.09	.77***	-.70***	-.30*	--		
HM_A	-.22	.09	-.64***	.71***	.06	-.89***	--	
HH_A	.18	.06	-.60***	.34*	.53***	-.69***	.28†	--

*$p < .05$; **$p < .01$; ***$p < .001$, † < .10 (trend); [a] BPAQ = Buss Perry Aggression Questionnaire, Total score; [b] LHA = Life History of Aggression scale, Aggression subscale. See List of Abbreviations for definition of terms.

Table 8

Comparison of Aggression Measures for Aggressive and Non-Aggressive Participants

	Non-Aggressive		Aggressive		Difference
	M	*(sd)*	*M*	*(sd)*	*t* (df)[h]
LHA – Aggression[a]	5.58	(2.70)	13.14	(3.71)	-8.16 (46)***
LHA – Total[a]	7.08	(3.97)	17.41	(7.50)	-6.10 (46)***
BPAQ – Physical[b]	17.31	(5.11)	23.36	(7.63)	-3.27 (46)**
BPAQ – Total[b]	57.42	(14.33)	74.36	(17.64)	-3.67 (46)***
mTRT – LL_A[c]	95.54	(39.43)	87.45	(36.32)	0.73 (46)
mTRT – LM_A	93.23	(39.9)	86.82	(32.87)	0.60 (46)
mTRT – LH_A	7.73	(11.67)	19.55	(25.92)	-2.09 (46)*
mTRT – HL_A	76.62	(55.70)	69.50	(31.32)	0.53 (46)
mTRT – HM_A	91.04	(44.27)	94.59	(19.88)	-0.35 (46)
mTRT – HH_A	30.69	(22.32)	34.23	(20.78)	-0.56 (46)
mTRT – Any LH[d]	0.46	(0.51)	0.73	(0.46)	-1.89 (46)*
mTRT – Any HH[e]	0.85	(0.37)	1. 00	(0. 00)	-1.96 (46)*
mTRT – LH_A + HH_A[f]	38.42	(26.95)	53.77	(44.42)	-1.47 (46)
mTRT – HH_A - LH_A[g]	22.96	(23.29)	14.68	(15.31)	1.43 (46)

*$p < .05$; **$p < .01$; ***$p < .001$; [a] = Life History of Aggression, Aggression subscale/Total subscale; [b] = Buss Perry Aggression Questionnaire, Physical Aggression subscale/Total subscale; [c] = modified Taylor Reaction-Time Task, see List of Abbreviations for definitions; [d] = any administration of LH; [e] = any administration of HH; [f] = total number of "high" noise blasts administered; [g] = number of "highs" selected under high provocation minus number of "highs" under low provocation; [h] = one-tailed.

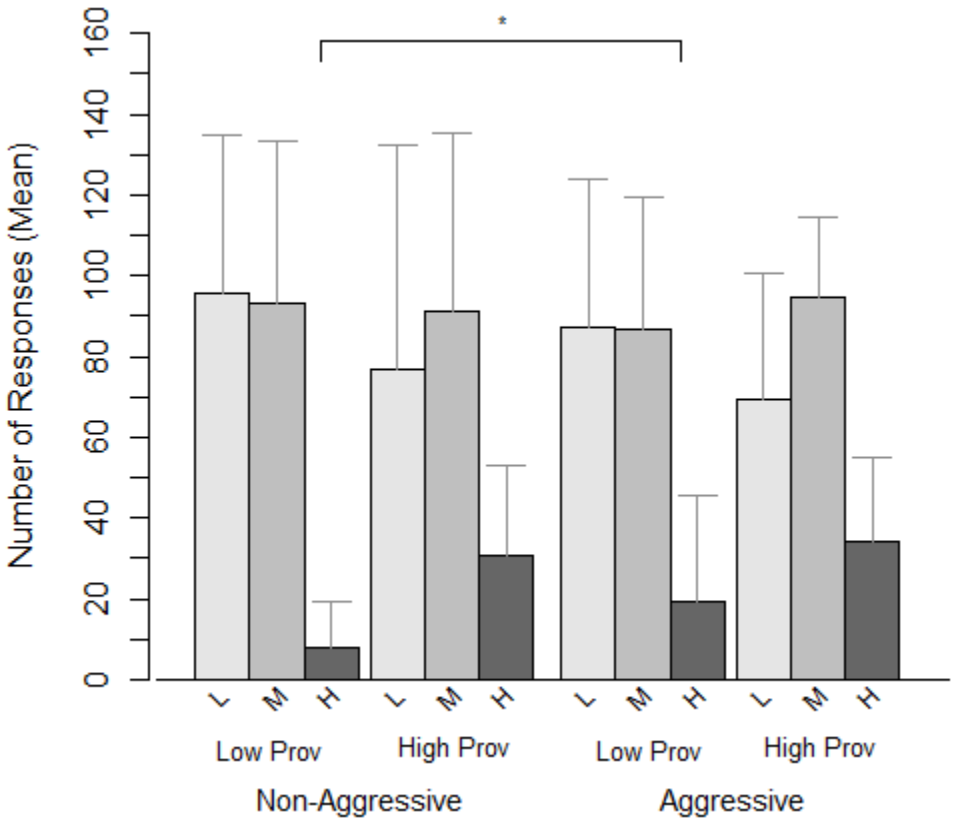

Figure 2. Responses (low, medium, or high) as a function of provocation, aggression group, and gender. Note: * $p < .05$.

Finally, a 2 (aggression group) × 2 (gender) × 2 (provocation) × 2 (response) mixed model ANOVA was conducted to examine the separate and interactive effects of the variables of interest on response selection (Table 9). The response data are illustrated in Figure 2 as a function of aggression group, gender, and provocation. A significant main effect of response ($F_{(1.28, 88)}$ = 41.17, $p < .001$) reflected that participant used the "high" response (M = 45.46, sd = 36.44) less frequently than either the "low" (M = 165.19, sd = 78.73; $t_{(47)}$ = 7.82, $p < .001$) or "medium" (M = 182.96, sd = 66.18; $t_{(47)}$ = 14.06, $p < .001$) response.

Table 9

Main and Interaction Effects on Aggressive Behavior

	F	*(df)*
Main Effects		
Gender	0.59	(1)
Aggression	0.38	(1)
Provocation	2.38	(1)
Response[a]	41.17	(1.28)***
Two-Way Interactions		
Gender*Agg	2.16	(1)
Provocation*Response[a]	16.85	(1.70)***
Gender*Provocation	0.18	(1)
Gender*Response[a]	0.14	(1.28)
Aggression*Provocation	0.31	(1)
Aggression*Response[a]	0.41	(1.28)
Three-Way Interactions		
Gender*Agg*Provocation	3.05	(1) †
Gender*Agg*Response[a]	0.06	(1.28)
Gender*Provocation*Resp[a]	0.14	(1.70)
Agg*Provocation*Resp[a]	1.08	(1.70)
Four-Way Interactions		
Gender*Agg*Prov*Resp[a]	0.23	(1.70)

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Response interacted with provocation, $F_{(1.70, 88)} = 16.85$, $p < .001$. Participants were significantly more likely to choose the "low" response in the low provocation block ($M = 91.83$, $sd = 37.85$) than in the high provocation block ($M = 73.35$, $sd = 45.84$; $t_{(47)} = 4.34$, $p < .001$), and were more likely to choose the "high" response in the high provocation block ($M = 32.31$, $sd = 21.47$) than in the low provocation block ($M = 13.15$, $sd = 20.20$; $t_{(47)} = -6.55$, $p < .001$).

Mood Rating Scales

Participants completed visual analog mood rating scales at five points during the aggression task. A 2 (aggression group) × 2 (gender) × 2 (provocation order) × 5 (assessment point) mixed model ANOVA was used to determine whether these variables affected mood fluctuations during the task. This was done for self-ratings of "nervous" and "angry" as these seemed to be the two emotions most relevant to the task and the questions of interest for this study. The results are displayed in Table 10 (means in Table C2) and Figures 3 and 4.

Self-rated nervousness was found to vary only as a function of assessment point, $F_{(2.27, 156)} = 36.01$, $p < .001$. Nervousness decreased in a curvilinear fashion over the course of the task, decreasing significantly at each time point until Time 3, after which, nervousness no longer differed between consecutive assessment points, most likely due to a floor effect in the scores (see Table C2). None of the variables examined were found to affect self-ratings of anger during the task. There was a trend for gender differences in self-ratings of anger, $F_{(1, 156)} = 3.15$, $p < .10$. Women rated themselves slightly higher on anger over the course of the task overall, $M_F = 11.68$, $sd = 14.31$; $M_M = 5.63$, $sd = 7.60$, $t_{(45)} = -1.77$, $p < .10$. Due to random assignment into the counterbalanced conditions (high provocation vs. low provocation block first), some of the cell sizes in this analysis were quite small, and therefore these results should be interpreted with caution.

Table 10

Mood Data Collected at Five Time Points During the Task

	"Nervous"		"Angry"	
	F	*(df)*	*F*	*(df)*
Main Effects				
Time [a]	36.01	(2.27)***	0.26	(2.69)
Order	1.12	(1)	0.22	(1)
Aggression	0.01	(1)	0.13	(1)
Gender	1.21	(1)	3.15	(1) †
Two-Way Interactions				
Order*Time[a]	2.15	(2.27)	1.16	(2.69)
Time*Aggression[a]	0.19	(2.27)	1.56	(2.69)
Time*Gender[a]	0.99	(2.27)	2.13	(2.69)
Order*Aggression	0.05	(1)	0.21	(1)
Order*Gender	0.24	(1)	0.04	(1)
Aggression*Gender	0.04	(1)	0.46	(1)
Three-Way Interactions				
Time*Order*Aggression[a]	0.23	(2.27)	1.77	(2.69)
Time*Order*Gender[a]	1.67	(2.27)	1.79	(2.69)
Time*Aggression*Gender[a]	1.36	(2.27)	0.90	(2.69)
Order*Aggression*Gender	1.06	(1)	0.00	(1)
Four-Way Interactions				
Time*Order*Agg*Gend[a]	0.23	(2.27)	1.54	(2.69)

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Nervous

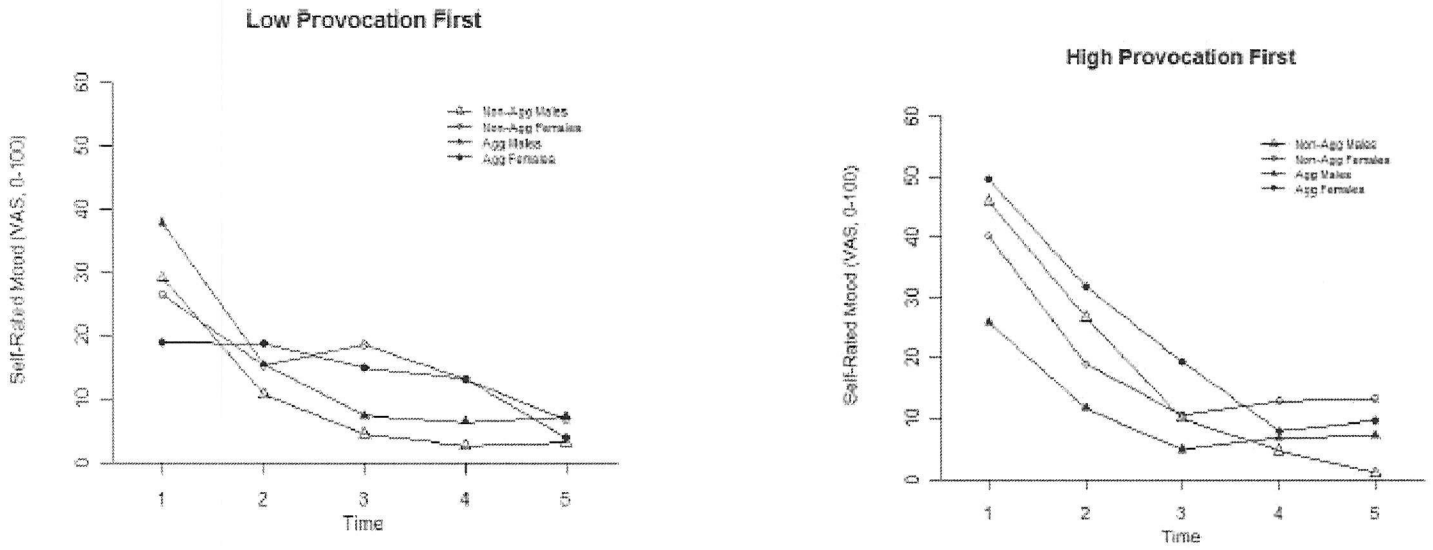

Figure 3. Mood ratings across five time points for "nervous" and "angry." Time 1 is pre-task. Times 2 and 4 are following provocation blocks (low or high). Times 3 and 5 are following aggression blocks.

Angry

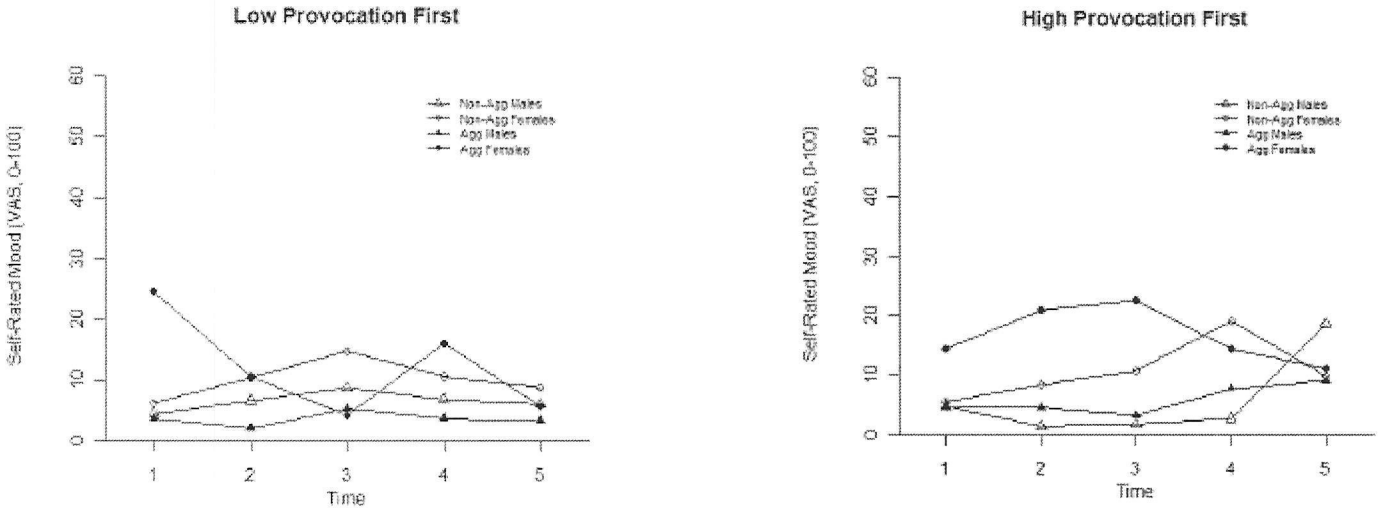

Figure 4. Mood ratings across five time points for "angry." Time 1 is pre-task. Times 2 and 4 are following provocation blocks (low or high). Times 3 and 5 are following aggression blocks.

Event Related Potentials

P3 and N2 were the primary components of interest in this study. Results and discussion for the N450 and NSW components are presented in Appendix B.

Provocation P3

In order to examine the effects of interest on ERPs, two separate 2 (aggression) × 2 (gender) × 3 (electrode group), × 2 (provocation) ×2 (stimulus probability) mixed model ANOVAs were conducted for mean P3 amplitude and mean P3 latency in the provocation blocks (Table 11). Mean P3 amplitude represents the average ERP amplitude between 300 and 600 ms post-stimulus onset. P3 latency represents the time between stimulus onset and peak amplitude during the 300 to 600 ms post-stimulus window.

Mean provocation P3 amplitude. For mean P3 amplitude, significant main effects emerged for electrode group and stimulus probability, but not for provocation, aggression group, or gender (Table 11). For the effect of electrode, the largest mean P3 amplitudes were observed at parietal sites (P: M = 9.60, sd = 3.88), followed by central sites (C: M = 7.99, sd = 4.13), and lastly frontal sites (F: M = 3.98, sd = 4.25; $F_{(1.31, 57.73)} = 121.56$, $p < .001$). Follow-up paired-sample t-tests revealed that these differences are significant, $t_{FC(47)} = -11.69$; $t_{FP(47)} = -10.37$; $t_{CP(47)} = -5.51$; all ps < .001. With regard to stimulus probability, participants displayed higher mean P3 amplitude to oddball stimuli (o: M = 8.61, sd = 4.23) than to frequent stimuli (f: M = 5.39, sd = 2.89, $F_{(1, 44)} = 100.51$, $p < .001$). This finding reflects the classic "oddball effect" of larger amplitude P3 to rare stimuli.

A significant 2-way interaction between electrode group and stimulus probability was also observed, $F_{(1.12, 49.04)} = 31.79$, $p < .001$. The primary comparisons of interest were consistent with the main effects. A significant 3-way interaction between electrode group, provocation level, and stimulus probability limited the main effects and 2-way interaction, $F_{(1.17, 51.53)} = 5.18$, $p < .05$. To decompose this interaction, separate stimulus probability × provocation repeated measures ANOVAs were conducted for each electrode group. At frontal sites, stimulus

probability ($F_{(1, 47)}$ = 24.22, p < .001), provocation ($F_{(1, 47)}$ = 4.22, p < .05), and their interaction ($F_{(1, 47)}$ = 6.26, p < .05) exerted an effect on mean P3 amplitudes. High provocation stimuli generated larger mean P3 amplitudes at frontal sites (M = 3.98, sd = 4.25) compared to low provocation stimuli (M = 3.25, sd = 4.26; $t_{(47)}$ = -2.06, p < .05). This appears to be accounted for by the fact that the high provocation oddball (M = 5.33, sd = 5.51) generated a significantly higher mean P3 amplitude at frontal sites than did the low provocation oddball (M = 4.02, sd = 5.41; $t_{(47)}$ = -2.46, p < .05), as high and low provocation frequent stimuli did not generate different mean P3 amplitudes at frontal sites, M_L = 2.48, sd = 3.54; M_H = 2.63, sd = 3.42; $t_{(47)}$ = -0.54, p > .05. This effect can be seen in Figure 5. The 2-way ANOVAs for central and parietal sites only showed an effect of stimulus probability, $F_{C(1, 47)}$ = 93.90, p < .001; $F_{P(1, 47)}$ = 189.92, p < .001, consistent with the main effect of stimulus probability overall.

The only significant effect to emerge from this analysis that involved either gender or aggression group was a gender × aggression group × electrode × provocation interaction, $F_{(1.44, 63.22)}$ = 3.91, p < .05. Follow-up reduced ANOVAs of electrode × provocation level at each gender × aggression group revealed an interaction between electrode and provocation for non-aggressive women, $F_{(2, 88)}$ = 8.23, p < .05, in addition to the main effect of electrode. As can be seen below in Figure 6, non-aggressive women showed significantly larger mean P3 amplitude in the high provocation condition (M = 5.06, sd = 4.69) compared to low provocation condition (M = 3.27, sd = 5.49; $t_{(14)}$ = 2.80, p < .05) at frontal sites and across all stimuli (oddball and frequent). Between-subjects simple effects were not significant.

Table 11

Main and Interaction Effects on Provocation P3 Mean Amplitude (300-600 ms)

	F	*(df)*	η^2
Main Effects			
Gender	0.57	(1)	.01
Aggression	1.59	(1)	.03
Electrode[a]	121.56	(1.31)***	.73
Provocation	1.24	(1)	.03
Stimulus	100.51	(1)***	.68
Two-Way Interactions			
Gender*Agg	0.76	(1)	.02
Electrode*Prov[a]	3.45	(4.71)†	.07
Electrode*Stim[a]	31.79	(1.12)***	.40
Provocation*Stim	1.74	(1)	.04
Gender*Electrode	0.64	(2)	.00
Gender*Prov	1.86	(1)	.04
Gender*Stim	0.33	(1)	.00
Aggression*Elec	0.11	(2)	.00
Aggression*Prov	0.92	(1)	.02
Aggression*Stim	2.77	(1)	.02
Three-Way Interactions			
Elec*Prov*Stim[a]	5.18	(1.17)*	.10
Gender*Elec*Prov	0.66	(2)	.01
Gender*Elec*Stim	1.99	(2)	.02
Gender*Prov*Stim	1.74	(1)	.00
Agg*Elec*Prov	0.83	(2)	.02
Agg*Elec*Stim	0.08	(2)	.00
Agg*Prov*Stim	2.94	(1)†	.06
Gender*Agg*Elec	1.03	(2)	.01
Gender*Agg*Prov	0.16	(1)	.00
Gender*Agg*Stim	0.90	(1)	.01
Four-Way Interactions			
Agg*Gender*Elec*Stim	2.13	(2)	.03
Agg*Gender*Elec*Prov[a]	3.91	(1.44)*	.07
Agg*Gender*Stim*Prov	1.74	(1)	.01
Agg*Elec*Stim*Prov	0.02	(2)	.00
Gender*Elec*Stim*Prov	0.38	(2)	.01
Five-Way Interactions			
Gend*Agg*Elec*Stim*Prov	1.67	(2)	.03

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

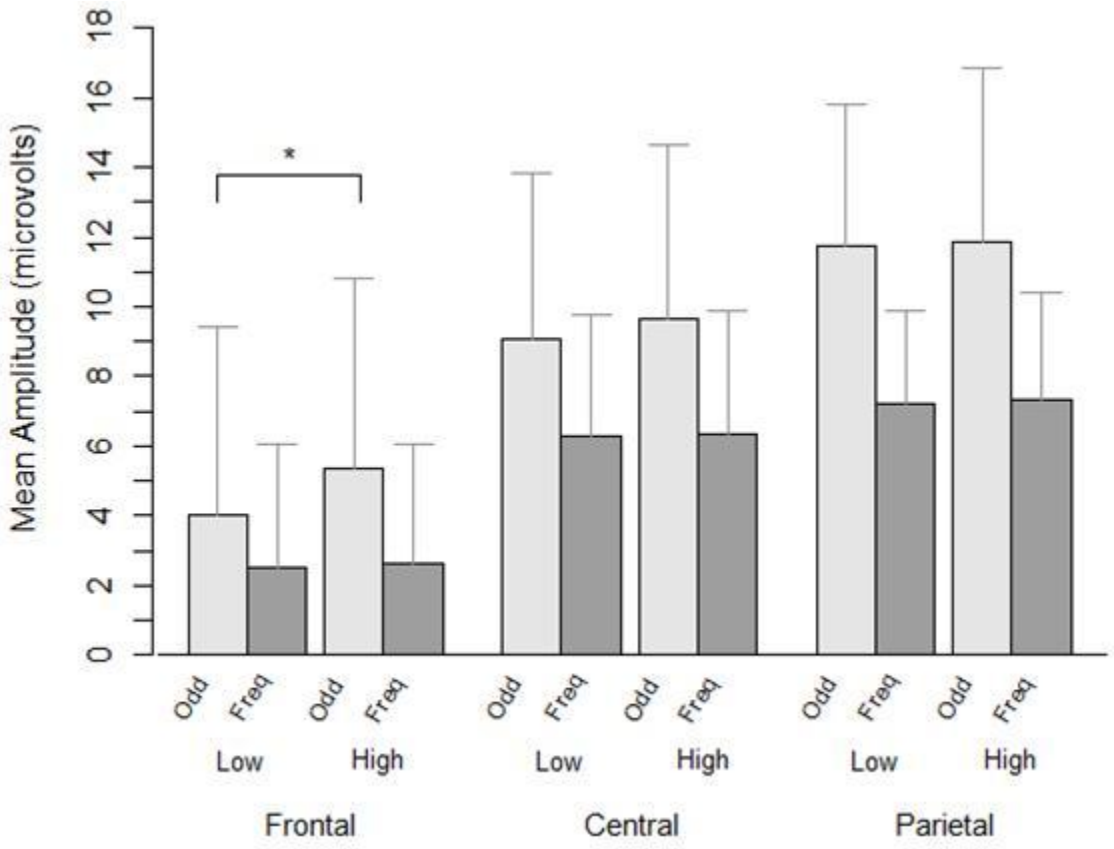

Figure 5. Three-way interaction effect of electrode group, provocation, and stimulus probability on mean provocation P3 amplitude. Note: * $p < .05$.

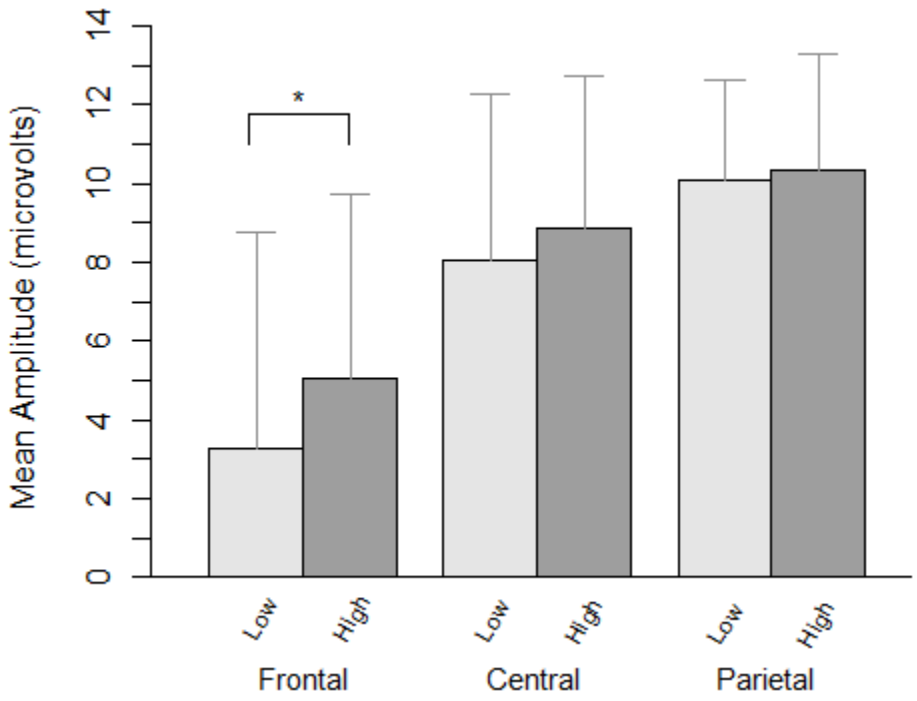

Figure 6. Four-way interaction effect of gender, aggression group, electrode group, and provocation on mean provocation P3 amplitude in non-aggressive females. Note: * $p < .05$.

A non-significant trend was found for the aggression group × provocation × stimulus probability interaction, $F_{(1, 44)} = 16.25$, $p < .10$ (illustrated in Figure 7). A reduced ANOVA (provocation × stimulus probability within each aggression group) revealed an interaction of provocation and stimulus probability that was also a trend in non-aggressive participants, $F_{(1, 44)} = 3.87$, $p = .06$. Follow-up exploratory analyses indicated that non-aggressive participants showed significantly higher mean P3 amplitude in response to the *high provocation* oddball ($M_H = 10.04$, $sd = 4.94$) than to the *low provocation* oddball ($M_L = 8.77$, $sd = 4.55$; $t_{(25)} = -2.20$, $p < .05$; see Figure 7). Aggressive participants showed no such difference in P3 amplitude ($M_H = 7.73$, $sd = 4.07$; $M_L = 7.64$, $sd = 4.13$; $t_{(21)} = 0.13$, $p = .90$). There was also a trend of non-aggressive participants having higher mean P3 amplitude for the high provocation oddball than aggressive participants ($M_{NA} = 10.04$, $sd = 4.94$; $M_A = 7.64$, $sd = 4.13$; $t_{(46)} = 1.81$, $p < .08$). The waveforms

for this trend are shown in Figure 8. Means and sds for the provocation P3 amplitudes can be found in Table C3 (Appendix C).

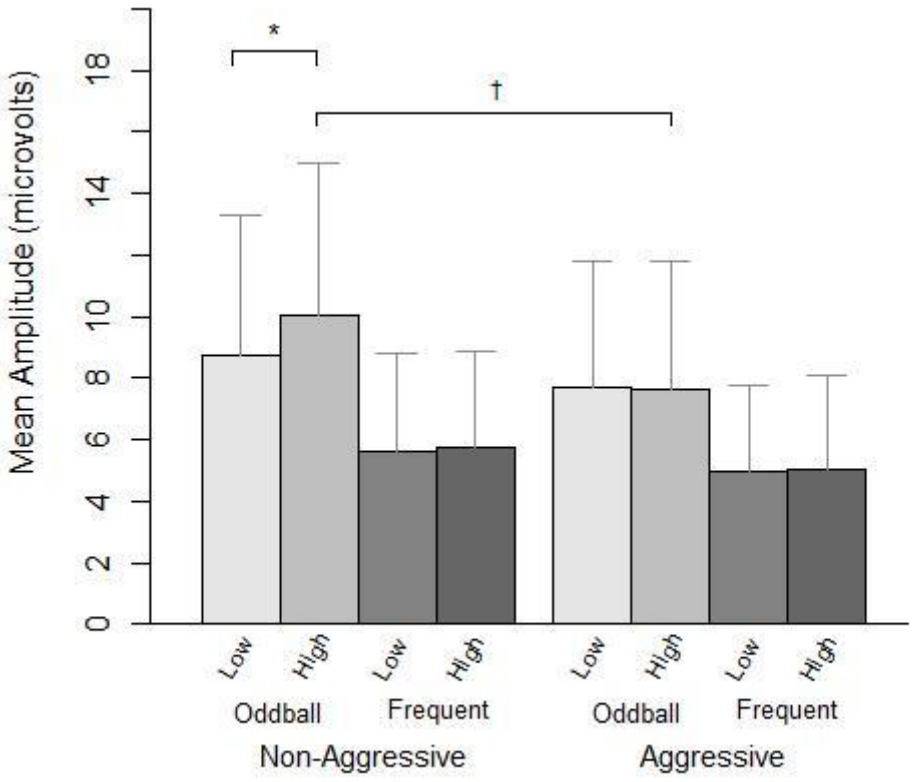

Figure 7. Trend of 3-way interaction between aggression group, stimulus probability, and provocation on provocation P3 mean. Note: * $p < .05$, † $p < .10$.

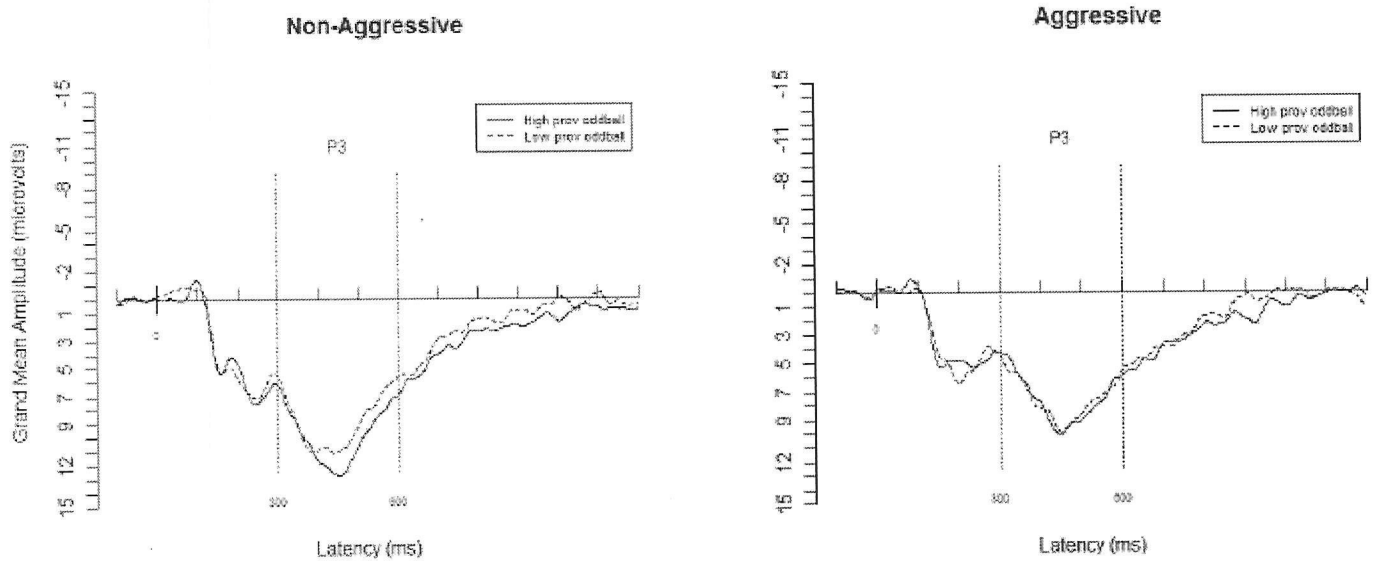

Figure 8. Low and high provocation oddball waveforms for aggressive and non-aggressive participants. The dotted lines indicate the range of mean provocation P3 amplitude.

Mean provocation P3 latency. Main effects for latency were found for electrode group ($F_{(1.46, 64.17)} = 12.80$, $p < .001$) and stimulus probability, $F_{(1, 44)} = 22.73$, $p < .001$ (see Table 12). Across all participants, P3 amplitudes peaked earliest at parietal sites ($M = .41$ seconds, $sd = .04$), then at frontal sites ($M = .43$, $sd = .05$), and lastly at central sites ($M = .44$, $sd = .06$). Parietal P3 peaked significantly earlier than both central ($t_{(47)} = 5.44$, $p < .001$) and frontal sites ($t_{(47)} = 2.64$, $p < .05$). The difference between frontal and central sites was also significant ($t_{(47)} = -2.11$, $p < .05$). P3 peaked later across all participants for oddball stimuli ($M_o = .44$, $sd = .05$) than frequent stimuli ($M_f = .42$, $sd = .05$; $t_{(47)} = -5.05$, $p < .001$).

These main effects were qualified by a 2-way interaction between stimulus probability and electrode site on P3 latency, $F_{(1.59, 69.80)} = 32.18$, $p < .001$. Oddball stimuli had a significantly later latency compared to frequent stimuli at both central sites ($M_o = .45$, $sd = .06$, $M_f = .43$, $sd = .06$, $t_{(47)} = 2.49$, $p < .05$) and parietal sites ($M_o = .44$, $sd = .05$, $M_f = .38$, $sd = .05$, $t_{(47)} = 8.28$, $p < .001$), but not at frontal sites ($M_o = .44$, $sd = .06$, $M_f = .43$, $sd = .06$, $t_{(47)} = .66$, $p > .05$).

Two 3-way interactions were observed. The first was an interaction between gender, aggression group, and provocation on P3 latency, $F_{(1, 44)} = 6.97$, $p < .05$. To follow-up, separate 2 (gender) × 2 (aggression) ANOVAs were conducted at each level of provocation, revealing a significant effect of provocation among aggressive females. Specifically, aggressive females showed later peak P3 ($M = .47$, $sd = .04$) compared to non-aggressive females ($M = .42$, $sd = .03$; $t_{(23)} = -2.90$, $p < .01$) and to aggressive males ($M = .42$, $sd = .04$; $t_{(20)} = -2.74$, $p < .05$) in the high provocation block. Differences in P3 latency as a function of provocation level were also examined through paired sample t-tests for each combination of gender and aggression. Aggressive female participants showed later latency P3 for provocative stimuli than non-provocative stimuli, $M = .47$, $sd = .04$, $M = .44$, $sd = .06$, $t_{(9)} = -2.73$, $p < .05$. This effect is illustrated in Figure 9. No other group showed this difference.

Table 12

Main and Interaction Effects on Provocation P3 Latency (300-600 ms)

	F	*(df)*	η^2
Main Effects			
Gender	1.36	(1)	.03
Aggression	1.17	(1)	.02
Electrode[a]	12.80	(1.46)***	.22
Provocation	2.79	(1)	.05
Stimulus	22.73	(1)***	.33
Two-Way Interactions			
Gender*Agg	1.43	(1)	.03
Electrode*Prov[a]	0.54	(1.69)	.01
Electrode*Stim[a]	32.18	(1.59)***	.91
Provocation*Stim	0.04	(1)	.00
Gender*Electrode[a]	0.57	(1.46)	.01
Gender*Prov	0.53	(1)	.01
Gender*Stim	0.34	(1)	.00
Aggression*Elec[a]	1.35	(1.46)	.02
Aggression*Prov	0.04	(1)	.00
Aggression*Stim	0.50	(1)	.00
Three-Way Interactions			
Elec*Prov*Stim[a]	0.82	(1.72)	.01
Gender*Elec*Prov[a]	0.71	(1.69)	.02
Gender*Elec*Stim[a]	0.25	(1.59)	.01
Gender*Prov*Stim	0.24	(1)	.01
Agg*Elec*Prov[a]	1.46	(1.69)	.03
Agg*Elec*Stim[a]	1.14	(1.59)	.03
Agg*Prov*Stim	6.41	(1)*	.13
Gender*Agg*Elec[a]	0.23	(1.46)	.00
Gender*Agg*Prov	6.97	(1)*	.13
Gender*Agg*Stim	0.88	(1)	.01
Four-Way Interactions			
Agg*Gender*Elec*Stim[a]	0.91	(1.59)	.02
Agg*Gender*Elec*Prov[a]	0.14	(1.69)	.00
Agg*Gender*Stim*Prov	0.04	(1)	.00
Agg*Elec*Stim*Prov[a]	0.01	(1.72)	.00
Gender*Elec*Stim*Prov[a]	0.30	(1.72)	.01
Five-Way Interactions			
Gend*Agg*Elec*Stim*Prov[a]	0.62	(1.72)	.01

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

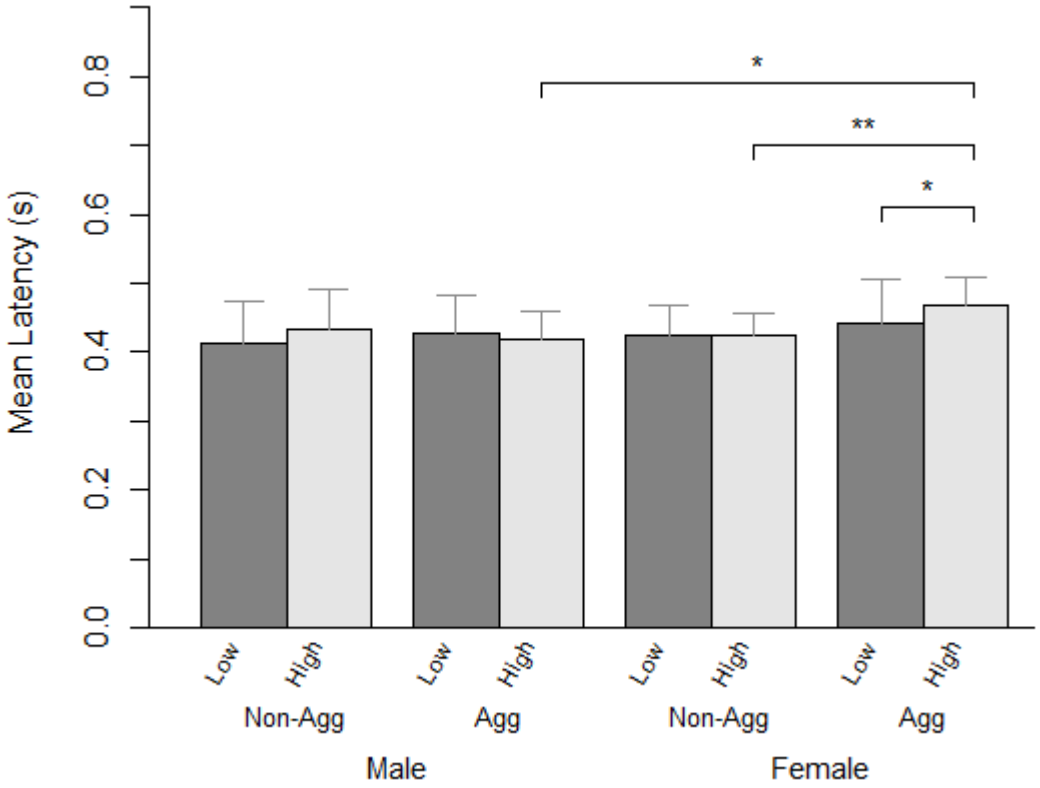

Figure 9. Three-way interaction of gender, aggression group, and provocation on provocation P3 latency. Note: * $p < .05$, ** $p < .01$.

Second, there was an interaction between aggression group, provocation level, and stimulus probability on P3 latency, $F_{(1, 44)} = 6.41$, $p < .05$, illustrated below in Figure 10. Means for these analyses can be found in Appendix C, Table C4. Follow-up t-tests to evaluate the effect of aggression group on latency for different stimuli (i.e., between-subjects simple effects) were non-significant. Repeated-measures ANOVAs (provocation × stimulus probability) at aggression group level revealed significant effects of stimulus probability in both aggressive ($F_{(1, 21)} = 7.65$, $p < .05$) and non-aggressive ($F_{(1, 25)} = 18.85$, $p < .001$) participants, and an interaction between stimulus probability and provocation among non-aggressive participants, $F_{(1, 25)} = 4.70$, $p < .05$. The effect of stimulus probability in aggressive participants was consistent with the oddball effect of longer latency for rarer stimuli ($M_o = .45$, $sd = .05$; $M_f = .43$, $sd = .06$). Non-aggressive

participants, on the other hand, had significantly longer latencies to *high provocation* oddball stimuli (M_o = .45, *sd* = .05) compared to high provocation frequent stimuli (M_f = .41, *sd* = .05; $t_{(25)}$ = 5.68, p < .001), compared to *low* provocation frequent stimuli (M_f = .41, *sd* = .05; $t_{(25)}$ = -4.34, p < .001), and to low provocation oddball stimuli (M_o = .43, *sd* = .06; $t_{(25)}$ = -2.64, p < .05; see Figure 10 below). The low provocation oddball was also later in non-aggressive participants than the high provocation frequent stimuli (M_{Lo} = .43, *sd* = .06; M_{Hf} = .41, *sd* = .05; $t_{(25)}$ = 2.28, p < .05; not shown in Figure 10). There was no difference in latency between the low provocation oddball and low provocation frequent ($t_{(25)}$ = 1.91, p > .05), or between the two frequent stimuli ($t_{(25)}$ = .38, p > .05). Provocation P3 latency was correlated with reaction time, r = .34, p < .01.

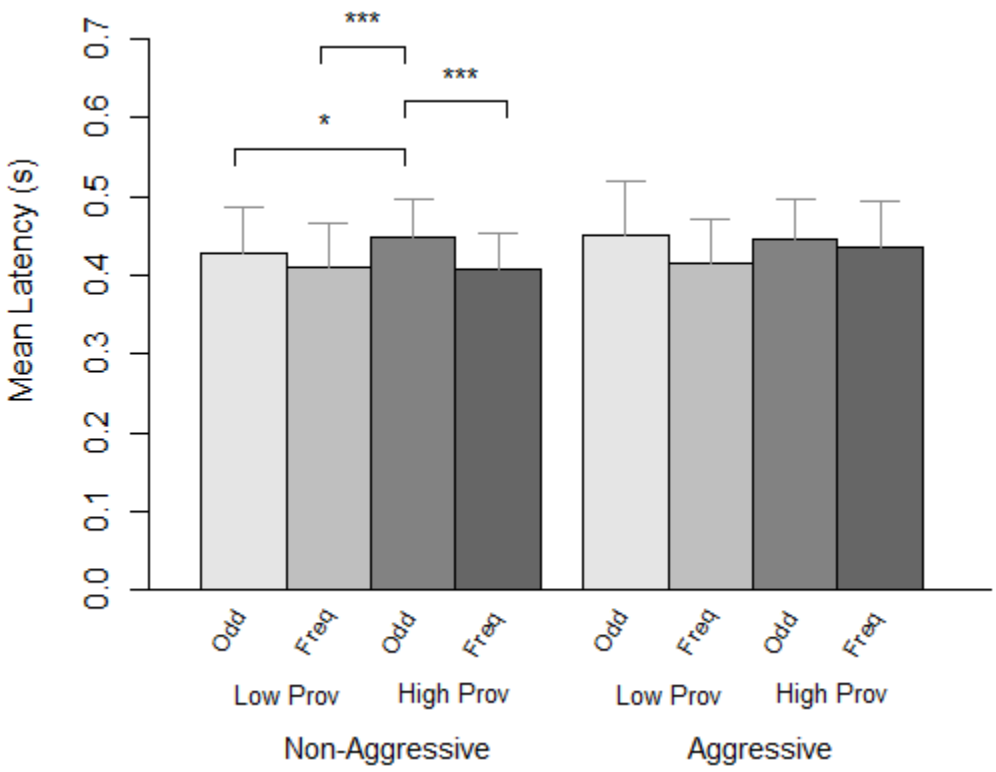

Figure 10. Three-way interaction between aggression group, provocation level, and stimulus probability on mean provocation P3 latency. Note: * p < .05, ** p < .01, *** p < .001.

Aggression ERPs

As described previously, two of the four blocks completed by participants were aggression blocks. Across these blocks, participants had 400 opportunities to select a noise blast (low, medium, or high) for their opponent. The two aggression blocks are distinguished by whether they followed the "low provocation" block or the "high provocation" block. As one would expect, more aggressive responses were chosen in the aggression block following the high provocation block than following the low provocation block. The particular aggression block is denoted by the first letter in the trial type, such that LL_A, LM_A, and LH_A represent behavioral selections (low, medium, and high) following the low provocation block, and HL_A, HM_A, and HH_A represent selections (low, medium, and high) following the high provocation block. These blocks are distinct from the provocation blocks in that button presses reflect free choices, not categorization of stimuli. Because the button presses in these blocks represent free responses, cell sizes varied considerably. Indeed, it was so rare for participants to select the high noise blast for the opponent following the low provocation block (LH_A) that ERPs for this category of data could not be analyzed. Because of this missing data it was not possible to conduct a full mixed model ANOVA that included responses from both provocation blocks (LL_A, LM_A, LH_A, HL_A, HM_A, and HH_A). To circumvent this problem, statistics are presented for the responses made under high provocation only (e.g., HL_A, HM_A, and HH_A).

Aggression N2

In order to examine the effects of interest on the N2 component, two separate 2 (aggression) × 2 (gender) × 3 (electrode) × 3 (response) mixed model ANOVAs were conducted for mean N2 amplitude and mean N2 latency in the aggression block following high provocation. Mean amplitude represents the average ERP amplitude between 150 and 300 ms post-stimulus onset. Latency represents the time between stimulus onset and peak amplitude during the 150 to 300 ms post-stimulus window.

Mean N2 amplitude. Electrode group exerted a main effect on mean N2 amplitude, $F_{(1.23, 49.09)} = 70.55$, $p < .001$ (Table 13). The largest (i.e., most negative) mean N2 was observed at the frontal electrode sites ($M = 1.63$, $sd = 2.67$), followed by central sites ($M = 3.80$, $sd = 2.39$), and finally parietal sites ($M = 5.33$, $sd = 2.36$). The differences between these mean amplitudes were significant between each site ($t_{FC(43)} = -9.30$; $t_{FP(43)} = -9.08$; $t_{CP(43)} = -6.27$; all ps $< .001$).

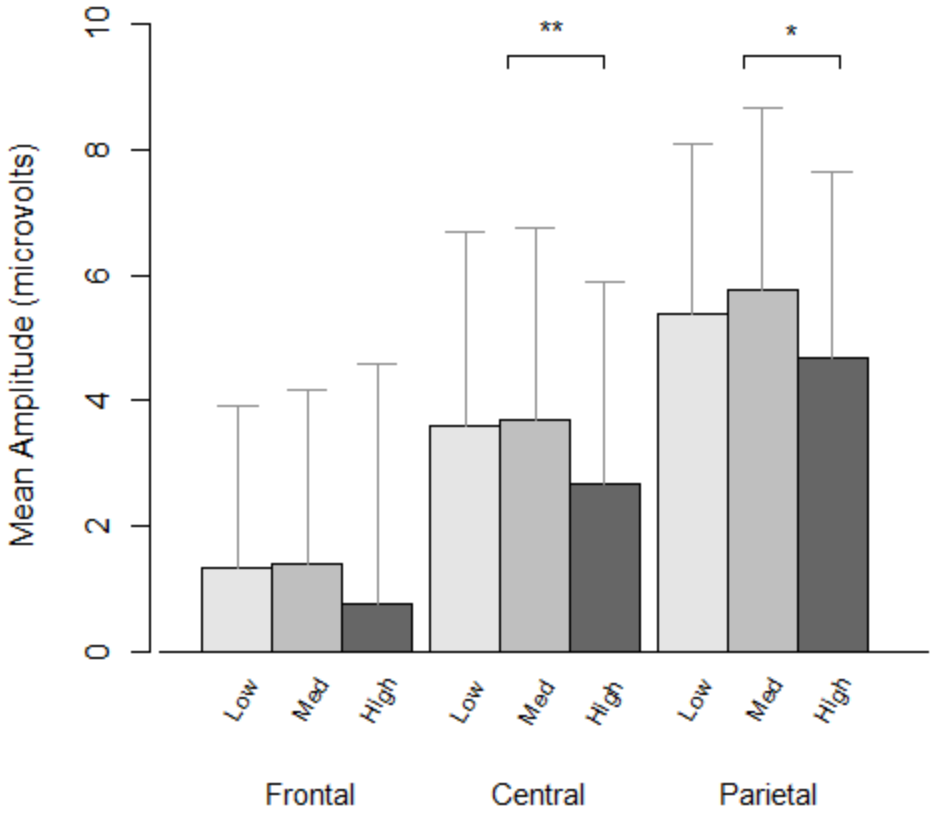

Figure 11. Four-way interaction of aggression group, gender, response, and electrode site on mean N2 amplitude in aggressive males. Note that means closer to 0 μv are considered larger (more negative). Note: * $p < .05$, ** $p < .01$.

A 4-way interaction between gender, aggression group, electrode site, and response was also observed, $F_{(2.24, 89.61)} = 3.45$, $p < .05$. Follow-up 2 (gender) × 2 (aggression) ANOVAs on each response by electrode site combination were non-significant. To examine the within-subjects

effects, pairwise comparisons of responses at each electrode site were conducted separately for aggressive and non-aggressive men and women. As can be seen below in Figure 11, aggressive males showed larger deflection of N2 for the "high" versus "medium" responses at both central (M_{HM} = 3.69, *sd* = 3.08; M_{HH} = 2.66, *sd* = 3.23; $t_{(11)}$ = 3.21, $p < .01$) and parietal (M_{HM} = 5.78, *sd* = 2.89; M_{HH} = 4.70, *sd* = 2.94; $t_{(11)}$ = 2.40, $p < .05$) sites. The waveforms for this effect are illustrated in Figure 12.

Table 13

Main and Interaction Effects on Aggression N2 Mean Amplitude (150-300 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.45	(1)	.01
Aggression	1.53	(1)	.04
Electrode[a]	70.55	(1.23)***	.63
Response	1.53	(2)	.03
Two-Way Interactions			
Gender*Agg	0.28	(1)	.01
Electrode*Response[a]	1.39	(2.24)	.03
Gender*Electrode[a]	0.61	(1.23)	.01
Gender*Response	0.55	(2)	.01
Aggression*Electrode[a]	0.32	(1.23)	.00
Aggression*Response	2.72	(2) †	.06
Three-Way Interactions			
Gender*Agg*Electrode[a]	0.21	(1.23)	.00
Gender*Agg*Response	1.22	(2)	.03
Gender*Electrode*Resp[a]	0.25	(2.24)	.01
Agg*Electrode*Resp[a]	0.39	(2.24)	.01
Four-Way Interactions			
Gender*Agg*Elec*Resp[a]	3.45	(2.24)*	.08

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

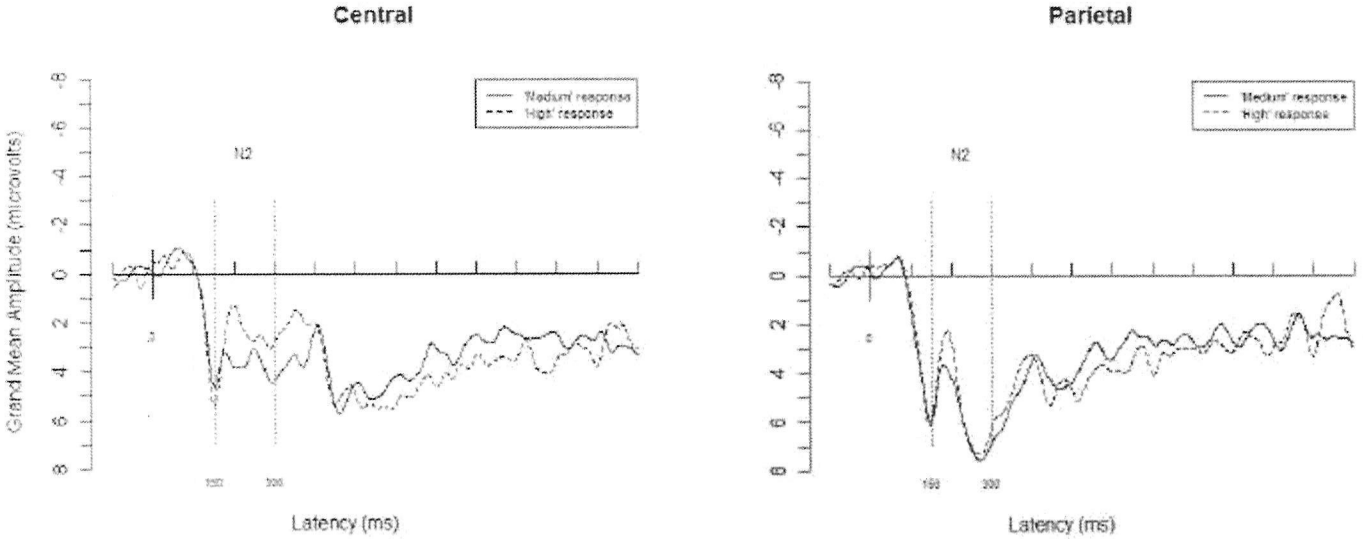

Figure 12. Grand mean waveforms for aggressive males showing difference in mean N2 amplitude for "medium" and "high" responses at central and parietal electrode sites.

Mean N2 latency. A main effect of electrode on N2 latency was observed, $F_{(2, 80)} = 74.38$, $p < .001$ (Table 14). N2 latency was shortest at parietal sites ($M = .20$, $sd = .02$), followed by central sites ($M = .23$, $sd = .03$), and latest at frontal sites ($M = .26$, $sd = .03$). The differences in latencies between sites were significant at ($t_{FC(43)} = 5.94$; $t_{FP(43)} = 11.24$; $t_{CP(43)} = 6.44$; ps $< .001$).

As illustrated in Figure 13, a 2-way interaction between aggression group and electrode showed that N2 latency was later among aggressive participants compared to non-aggressive participants at parietal sites ($M_A = .20$, $sd = .03$; $M_{NA} = .19$, $sd = .02$; $t_{(42)} = -2.18$, $p < .05$). This different was not significant at frontal ($M_A = .26$, $sd = .03$; $M_{NA} = .25$, $sd = .03$) or central ($M_A = .22$, $sd = .04$; $M_{NA} = .23$, $sd = .03$) sites (ps $> .05$). N2 latency was uncorrelated with reaction time, $r = .24$, $p = .24$).

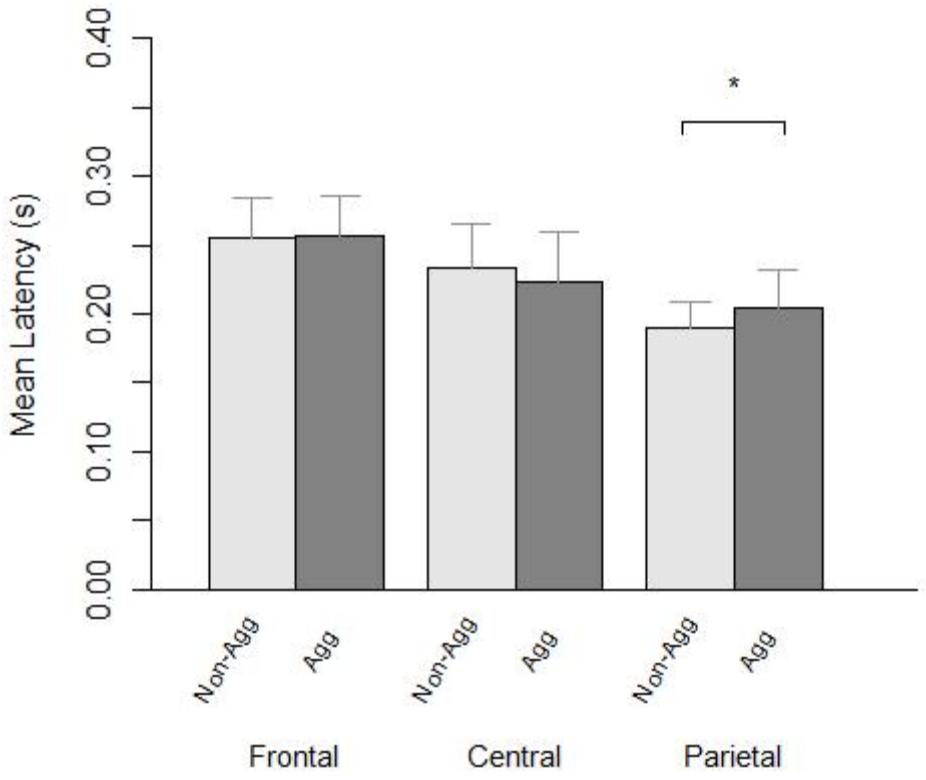

Figure 13. Two-way interaction between aggression group and electrode site on mean N2 latency. Note: * $p < .05$.

Table 14

Main and Interaction Effects on Aggression N2 Latency (150-300 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.00	(1)	.00
Aggression	0.08	(1)	.00
Electrode	74.38	(2)***	.49
Response	0.14	(2)	.13
Two-Way Interactions			
Gender*Agg	2.69	(1)	.01
Electrode*Response	0.91	(4)	.00
Gender*Electrode	0.32	(2)	.00
Gender*Response	0.70	(2)	.00
Aggression*Electrode	3.77	(2)*	.03
Aggression*Response	2.48	(2) †	.03
Three-Way Interactions			
Gender*Agg*Electrode	0.60	(2)	.00
Gender*Agg*Response	1.13	(2)	.00
Gender*Electrode*Resp	0.75	(4)	.00
Agg*Electrode*Resp	0.78	(4)	.05
Four-Way Interactions			
Gender*Agg*Elec*Resp	0.49	(4)	.05

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Aggression P3

In order to examine the factors influencing the P3 component of the ERP, two separate 2 (aggression) × 2 (gender) × 3 (electrode) × 3 (response) mixed model ANOVAs were conducted for mean aggression P3 amplitude and aggression P3 latency in the aggression blocks. Mean amplitude represents the average ERP amplitude between 300 and 600 ms post-stimulus onset.

Latency represents the time between stimulus onset and peak amplitude during the 300 to 600 ms post-stimulus window.

Table 15

Main and Interaction Effects on Aggression P3 Mean Amplitude (300-600 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.33	(1)	.00
Aggression	0.11	(1)	.01
Electrode[a]	89.11	(1.40)***	.67
Response [a]	0.53	(1.61)	.01
Two-Way Interactions			
Gender*Agg	1.30	(1)	.03
Electrode*Response [a]	0.33	(1.99)	.01
Gender*Electrode [a]	0.33	(1.40)	.00
Gender*Response [a]	0.26	(1.61)	.01
Aggression*Electrode[a]	0.87	(1.40)	.01
Aggression*Response [a]	0.70	(1.61)	.02
Three-Way Interactions			
Gender*Agg*Electrode [a]	1.93	(1.40)	.01
Gender*Agg*Response [a]	0.22	(1.61)	.01
Gender*Electrode*Resp[a]	1.26	(1.99)	.03
Agg*Electrode*Resp [a]	0.21	(1.99)	.00
Four-Way Interactions			
Gender*Agg*Elec*Resp [a]	3.05	(1.99) †	.07

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Mean aggression P3 amplitude. A significant main effect of electrode was observed on mean P3 amplitude, $F_{(1.40, 56.01)} = 89.11$, $p < .001$ (Table 15, above). Mean P3 amplitudes for each group are as follows: frontal sites ($M = 1.50$, $sd = 2.59$), central sites ($M = 4.58$, $sd = 2.81$), and parietal sites ($M = 4.70$, $sd = 2.47$). Aggression P3 amplitude was greater at central and parietal sites than at frontal sites, $t_{FC(43)} = -12.45$, $p < .001$ and $t_{FP(43)} = -9.13$, $p < .001$, respectively.

There was no significant difference in amplitude between central and parietal sites. A 4-way interaction between gender, aggression group, electrode group, and response was a statistical trend, $F_{(1.99, 79.52)} = 3.05$, $p < .10$.

Mean aggression P3 latency. A main effect of electrode site was observed on P3 latency, $F_{(1.46, 58.19)} = 40.67$, $p < .001$ (Table 16). P3 latency was shortest at parietal sites ($M = .41$, $sd = .07$), followed by central sites ($M = .47$, $sd = .07$), and longest at frontal sites ($M = .50$, $sd = .07$). The differences between these sites were all significant, $t_{FC(43)} = 4.86$, $t_{FP(43)} = 6.95$, $t_{CP(43)} = 5.35$, $ps < .001$. A 2-way interaction between gender and aggression was observed for P3 latency, $F_{(1, 40)} = 4.31$, $p < .05$. As can be seen in Figure 14, non-aggressive males showed significantly later P3 latencies ($M = .49$, $sd = .04$) across responses and electrode sites than did aggressive males ($M = .44$, $sd = .06$; $t_{(19)} = 2.23$, $p < .05$). There was also a strong trend of non-aggressive males having longer P3 latencies than non-aggressive females, $M = .45$, $sd = .06$, $t_{(20)} = 2.09$, $p = .05$.

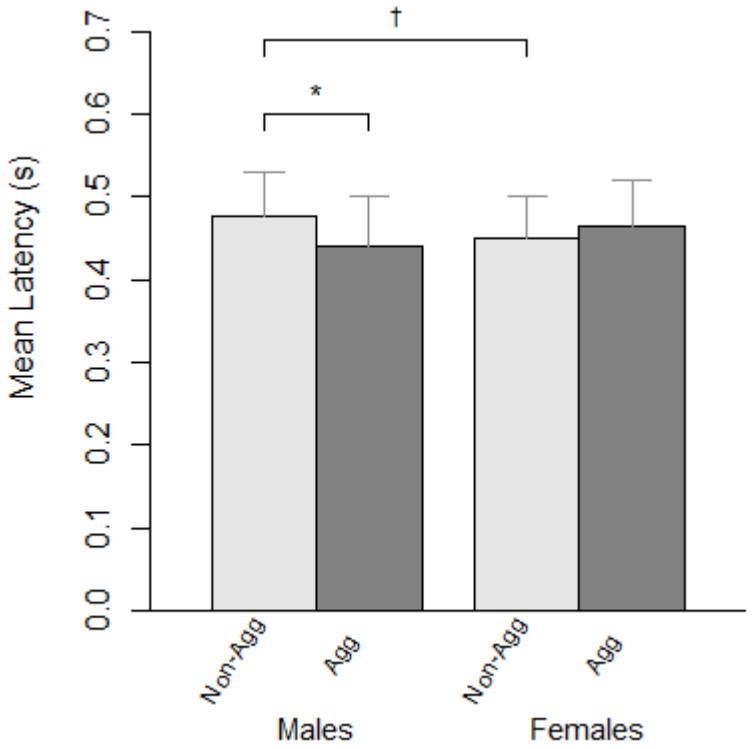

Figure 14. Interaction between gender and aggression group on mean aggression P3 latency. Note: * $p < .05$, † $< .10$.

Table 16

Main and Interaction Effects on Aggression P3 Latency (300-600 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.41	(1)	.00
Aggression	1.28	(1)	.03
Electrode[a]	40.67	(1.46)***	.46
Response	0.63	(2)	.01
Two-Way Interactions			
Gender*Agg	4.31	(1)*	.09
Electrode*Response[a]	0.75	(2.89)	.02
Gender*Electrode[a]	5.56	(1.46)*	.06
Gender*Response	0.64	(2)	.02
Aggression*Electrode[a]	0.34	(1.46)	.00
Aggression*Response	0.86	(2)	.02
Three-Way Interactions			
Gender*Agg*Electrode[a]	1.30	(1.46)	.02
Gender*Agg*Response	1.70	(2)	.04
Gender*Electrode*Resp[a]	0.47	(2.89)	.01
Agg*Electrode*Resp[a]	0.38	(2.89)	.01
Four-Way Interactions			
Gender*Agg*Elec*Resp[a]	0.19	(2.89)	.00

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Finally, a second 2-way interaction was observed between gender and electrode group, $F_{(1, 80)} = 4.31$, $p < .05$; however, men and women did not differ in P3 latency at any single site ($M_{Mf} = .52$, $sd = .07$; $M_{Ff} = .48$, $sd = .06$; $M_{Mc} = .48$, $sd = .08$; $M_{Fc} = .46$, $sd = .06$; $M_{Mp} = .39$, $sd = .07$; $M_{Fp} = .42$, $sd = .07$; $t_{FC(42)} = 1.75$; $t_{FP(42)} = .92$; $t_{CP(42)} = -1.35$; all ps > .05). P3 latencies in the aggression blocks were uncorrelated with mean reaction time, $r = .31$, $p = .13$. Means and sds for the aggression waveforms (N2 and P3) are located in Tables C5-C8 (Appendix C).

Post-Task Questionnaire

In order to rule out any effects differences in auditory or pain perception might have on the results, we compared aggressive and non-aggressive males and females on their auditory thresholds and pain thresholds assessed during the threshold procedure using two 2 (aggression) × 2 (gender) ANOVAs. Perception threshold and pain threshold for the auditory stimuli did not differ as a function of gender, aggression history, or their combination (all *p*s > .05).

In the post-task questionnaire, participants were asked how painful was their auditory pain threshold, how much tissue damage they thought the "high" noise blast would cause, and if they had not received the "high" noise blast (which none did) how painful they expected it to be. Participant rated these questions on a scale from 1 (*not at all*) to 8 (*very much*). Again participants did not differ in their answers to any of these questions as a function of gender, aggression group, or their interaction. On average, participants rated the painfulness of their auditory pain threshold a 3.56 out of 8 (*sd* = 2.22), the tissue damage due to the "high" noise blast as a 2.52 out of 8 (*sd* = 1.77), and the predicted painfulness of the high noise blast as a 5.06 out of 8 (*sd* = 2.37).

CHAPTER V

DISCUSSION

One goal of this study was to examine the effects of provocation on P3 in men and women with and without a marked life history of aggressive behavior. Another purpose of the study was to examine how the magnitude and latency of ERP components (N2, P3) previously linked to aggression and inhibition relate to the expression or inhibition of aggressive behavior under conditions of high and low provocation in real time, and how gender and aggression history affect these differences.

Categorization Errors

Participants were eight times more likely to make errors categorizing the provocation stimulus on the rarer oddball trials than on the frequent trials. It is interesting to note that aggressive participants committed significantly more errors identifying the "high" provocation oddball (i.e., when the opponent selected the "high" noise blast for them) than the "low" provocation oddball (10% error rate vs. 6%), whereas non-aggressive participants had similar error rates in both blocks (7% and 8% errors). It is unlikely that aggressive participants were less attentive during the high provocation block than the low, particularly as the blocks had been counterbalanced. A possible explanation is that when confronted with the potential for a noxious stimuli (noise blast), aggressive participants engaged in less processing related to categorizing and instead responded impulsively to the cue, thus making a greater number of errors. Indeed, the oddball task is essentially a type of continuous performance task such as those used to assess behavioral impulsivity and inhibition.

Behavioral Aggression

The original Taylor Reaction-Time Task was modified to allow efficient ERP recordings while still allowing the expression of aggressive behavior from participants. Provocation was manipulated to examine the effects of low provocation and high provocation on response choices, and we expected that participants would behave more aggressively following the high

provocation block, possibly as a function of life history of aggression. The high and low provocation blocks were counterbalanced and the order in which participants completed them did not affect the level of aggression displayed. Consistent with our prediction, participants were more aggressive in the block following high provocation, selecting more high noise blasts for the opponent and fewer low noise blasts, compared to the low provocation block. Overall, however, the use of the "high" noise blast was rare compared to the medium and low, even in the high provocation block. This suggests that participants viewed the high noise blast as quite noxious (which is consistent with the definition of aggression). Alternatively, the participants might have been concerned how the use of this selection would be perceived by the opponent or were conscious of social prohibitions against aggressive behavior, or some combination of these.

With regard to aggression history and provocation, aggressive participants used the aggressive response option ("high") more often than the low aggressive participants, but the difference was not statistically significant. However, high aggressive participants did use the high aggression option significantly more than non-aggressive participants in the low provocation block, suggesting they engaged in more unprovoked aggression, or alternatively, were provoked at a lower level than low aggressive participants. The former is more likely as the "opponent" did not use the high noise blast at all in the low provocation block, and therefore any use of the high noise blast by the participant following this block was indicative of greater aggressive behavior than the opponent. In sum, aggressive participants displayed more behavioral aggression in the low provocation block compared to non-aggressive participants, but the groups behaved more similarly when provocation level was increased.

Reaction Time

Response time was examined as a behavioral measure related to aggression and several interesting effects were found. The first was that participants were slower to press the correct button for oddball as compared to frequent stimuli. This finding is well-established and appears to be accounted for by stimulus evaluation *and* response selection times increasing as the probability

of the stimulus appearing decreases (Duncan-Johnson & Donchin, 1981). Research has also shown that electrophysiological activity related to preparation for a motor response is often evident well before the response is initiated and may even be apparent while stimulus categorization processes are still being carried out (Duncan-Johnson & Donchin, 1981), a phenomenon that seems to reflect motor priming. As 80% of the stimuli in the provocation block were the word "medium," participants likely quickly noticed that stimulus was the most frequent selection by the "opponent" and therefore may have been more primed even before the presentation of the stimulus to select that button.

A second finding was that participants were slower to respond to stimuli in the more provocative block. Previous research has shown that in choice paradigms such as this one, in which the participant must categorize the stimuli and select one or another response to indicate which class of stimuli it reflects, more intense stimuli are associated with slower reaction times (Jaśkowski, 2009). In this study intensity reflected the luminance of the visual stimuli presented on the screen. It has been proposed that increased arousal as a result of stimulus intensity interferes with the process of selecting the correct motor response (Van der Molen & Keuss, 1981). If this explanation is correct, it may extend to these findings such that more provocative stimuli generate greater arousal which then interferes with and slows the response selection phase of the trial. The interaction of stimulus probability and provocation level (whereby the high provocation oddball was associated with slower reaction time compared to the low provocation oddball, the high provocation frequent stimuli, and the low provocation frequent stimuli (in that order) likely reflects a combination of the above two effects (surprisingness and arousal levels).

With regard to between group differences in reaction time, aggressive females seemed particularly affected by the high provocation stimuli, responding significantly more slowly than both non-aggressive males and females to stimuli in the high provocation block. Arousal levels may have also played a role in this effect. It would be helpful, however, to have a better sense of

what is meant by arousal, for example, whether they might be experiencing more angry or anxious arousal.

As noted above, the processes related to stimulus evaluation and categorization have generally been found to be distinct from reaction time in most oddball experiments (Duncan-Johnson & Donchin, 1982). In this study, however, response times in the provocation blocks were highly correlated with P3 latency. This may be due to the fact that participants were instructed to respond with a button press to all stimuli, and therefore were already primed to press one of the buttons even during the stimulus evaluation period. In the classic oddball task, participants only respond to the rare target stimuli and the "default" response is therefore no response. Once the stimuli were categorized by participants in this study, the task characteristics may have provided a head start with regard to their motor responses compared to participants in other studies in which the classic oddball task was used.

In contrast to the provocation blocks, there were no significant effects or interactions involving group, response choice, or provocation level on reaction times in the aggression blocks. This may be due to the fact that participants were free to select any button on a given trial and did not have to categorize stimuli. It is probable that on some portion of the trials, participants decided on their response during the SOA, before the word "select" appeared on the screen. There was a trend toward responses in the aggression block following high provocation being slower. This suggests that participants may have engaged in more decision-making processes prior to making their selection in this block as compared to the low provocation block. It may also reflect greater response conflict, as they may have had mixed feelings as to how to respond to the opponent, who had just been aggressive toward them.

Mood

Participants' self-ratings of mood over the course of the task were not reliably affected by provocation level, aggression history, or gender and thus would appear to have no effect on the present findings. One possibility is that the block design was not amenable to eliciting strong

emotions. A more similar design to the classic TRT would be likely to exhibit higher levels of emotional arousal. This is a variable that warrants continued attention as previous researchers have found that variations in state and trait mood affect cognitive processing and behavior on tasks such as the emotional Stroop. It appears from other results in this study, however, that the emotional or affective aspects of the task did exert an effect on ERPs, even though they did not result in significant effects on subjective reports of mood.

Provocation P3

P3 has traditionally been used to understand processes related to stimulus identification, discrimination, and categorization; however, the types of stimuli used have largely been affectively neutral. In this study, the oddball stimuli were manipulated to reflect varying levels of provocation, making the task more relevant to understanding social information processes than the classical oddball task. In our study, we found the classic oddball effect of larger amplitude to rare stimuli, though it is difficult to ascertain from this alone whether, in the context of this particular task, this effect is due to the rare stimuli being perceived as more unexpected, salient, or relevant.

A non-significant trend (p = .06) was observed for non-aggressive participants producing higher P3 amplitudes for the high provocation oddball than for the low provocation oddball, whereas aggressive participants showed similar amplitudes to both. Aggressive participants also showed smaller P3s than non-aggressive participants in response to the high provocation oddball, as predicted. As the high and low provocation oddball stimuli had equal probabilities (20%), these findings suggest that non-aggressive participants perceived the high provocation stimulus as either more salient or more unexpected than the low provocation stimulus, whereas aggressive participants did not. In fact, as can be seen in Figure 8, aggressive participants processed the two oddball stimuli virtually identically, regardless of whether it reflected that the opponent had set the “low” or “high” noise blast. This finding could be taken to mean that relative to less aggressive individuals, aggressive individuals are impaired in their ability to distinguish high and

low provocative cues. Notably, the stimuli used in this study (the words “low,” “medium,” and “high”) were unambiguous, as their meanings with regard to noise blast had been explained in detail prior to the task starting. If P3 amplitude in this case reflects the surprisingness of the stimuli, it could suggest that aggressive individuals are more prone to expect (i.e., are less surprised by) aggressive social cues than are non-aggressive individuals. On the other hand, if P3 amplitude reflects the salience of the stimuli, it could indicate that aggressive individuals are less aroused by provocative stimuli, and may therefore be less likely to avoid it in a naturalistic setting. These two explanations are not mutually exclusive.

Provocation exerted its effect on P3 amplitude at frontal sites, with high provocation oddball stimuli generating larger P3 amplitudes than low provocation stimuli. This is consistent with the observations of Surguy and Bond (2006) who found that aggressive stimuli were processed primarily at frontal sites. The effects of provocation on P3 amplitude were most apparent in non-aggressive females, who showed the larger P3 amplitudes overall in the high provocation block compared to the low provocation block (this was evident at frontal sites only), whereas other groups did not. This finding suggested that non-aggressive females may have been more anxiously aroused during the high provocation relative to the low provocation block than any other group, a finding that is consistent with non-aggressive women being higher in traits such as harm avoidance (Struber, Luck, & Roth, 2008). That this effect was observed independently of stimulus probability suggests that arousal levels did play a role in P3 amplitudes in this study.

Our findings replicate previous studies of ERPs and social information processing related to aggression. Control subjects evidenced larger P3s in response to threatening stimuli, while participants who are more aggressive (and presumably more impulsive) did not. These findings extend the previous research in that the stimuli were delivered in the context of an actual aggressive exchange, making the stimuli more self-relevant and ecologically valid.

P3 latency was examined as a putative index of the speed with which stimulus evaluation and categorization takes place. In most studies, speed of information processing (P3 latency) and reaction time are unrelated, but, as noted above, in this study they were highly correlated, suggesting that for affectively charged situations and situations in which individuals are primed to make a response (such as a fight or flight response), speed of processing may have an effect on observed behavior.

Rarer stimuli were processed more slowly in this study as in previous studies (Duncan-Johnson & Donchin, 1982). Additionally, non-aggressive participants showed slower processing of the high provocation oddball stimuli than the low provocation and all other stimuli, suggesting that they may have been slower to direct cognitive resources to evaluating the provocative stimuli, as at least one previous study has found (De Pascalis et al., 2004). Differences in the extent to which the groups were able to distinguish the two stimuli (low versus high noise blasts set by the opponent) or were expecting the opponent's choices cannot be ruled out. For example, P3 amplitude and latency may reflect participants' processing or evaluation of the opponent's motives. A similar effect has been observed in emotional Stroop tasks, wherein stimuli that evoke an anxious reaction (i.e., interference) are associated with delayed reaction time. Alternatively, non-aggressive participants may have been more surprised by the opponent's selection of the high noise blast and may therefore have been slower to categorize the stimulus. Finally, arousal may account for some or all of the delay in processing of the opponent's selection of the "high" noise blast.

Aggressive women were slower to categorize high provocation stimuli than any other group except for non-aggressive men. Previous researchers have found aggressive and impulsive individuals to display later P3 peaks (e.g., De Pascalis et al., 2004). As physical aggression is less common in women, the high aggression female group may have represented the most extreme group in this study. This might explain why this group but not the others displayed significantly

later P3 latencies. The fact that the latency difference was for high provocation stimuli suggests that these stimuli were more salient for aggressive females compared to the other stimuli.

Aggression N2

Consistent with most prior studies, the N2 component was observed most prominently at frontal electrode sites. The finding that aggressive males showed larger N2 amplitudes at frontal sites when selecting the "high" response as compared to the "medium" response is not straightforward. At first glance, this result is more consistent with the explanation that N2 represents the activation of a behavioral response (the response being aggression). Indeed, examination of the N2 amplitude means (Table D5) shows that the largest (most negative) mean N2 amplitudes were found in aggressive men and women when they selected the medium and high responses. That the low response would generate a similar N2 amplitude to the high response suggests that for aggressive men, the medium response option may have served as the default, with any departure from that response generating greater response conflict. This finding stands in contrast to previous studies that have shown larger N2 amplitudes on successful stop trials in a Go/No-Go task. In this case, the component was generated in the context of a response option that was actually carried out. Our results are also inconsistent with the finding that adults and children with ADHD display smaller N2 amplitudes. In short, there are mixed explanations in the literature as to what cognitive processes are reflected by the N2 component. Furthermore, no study to date has examined N2 in the context of an emotionally-valenced task or a free response task (past N2s have been cued by "go" and "stop" signals). Clearly, there is much more to learn about the processes reflected by N2 and how they relate to inhibition in real-world behavior.

N2 was found to peak earliest at parietal sites, followed by central and finally frontal sites, the opposite pattern from N2 amplitude. This finding highlights the difficulty in drawing conclusions about the neural generators of ERPs based on scalp recordings. Aggressive participants were observed to have later N2 peaks at parietal sites than non-aggressive participants. Most explanations of the N2 component refer to its involvement in either the

expression or inhibition of behavior, or the conflict between two competing response options. It is conceivable then that the latency of these processes might be related to whether or when aggressive behavior is expressed or inhibited (similar to the race model of inhibition). However, it should be noted the N2 latency and reaction times were uncorrelated in this study.

Aggression P3

The observation that P3s are elicited during Go/No-Go tasks and may reflect inhibitory processes led us to include this component in analyses related to aggressive responding on the task. However, the only significant effect involving P3 amplitude in the aggression blocks involved electrode group, with P3s being largest at parietal sites followed by central and frontal sites (this finding is consistent with other No-Go P3s). The classic No-Go P3 is observed for successfully inhibited stop trials. It has been found to be sensitive to advance cueing, such that when a stop trial is signaled in advance, the P3 is not observed. Presumably, this is because the would-be response is never initiated and therefore does not have to be inhibited once it has started. It appears that aspects of the oddball/TRT-task were not amenable to eliciting the No-Go P3 component. The free response aspect of the aggression blocks may have something to do with the failure to elicit this component. It would seem rare that during these blocks someone would initiate a response, only to "take it back" a brief interval later, especially given that there were many trials in these blocks. Furthermore, participants had an interval of approximately four seconds between each aggression trial during which to decide on their response.

P3 latency was examined in the aggression blocks as a potential marker of the timing of inhibitory cognitive processes. In previous studies, individuals with disinhibitory psychopathology have been found to have later P3 latencies. Later P3 latencies in combination with earlier reaction time have been associated with failed stops on Go/No-Go tasks. P3 latency was observed to be shortest at parietal sites, followed by central sites, and finally frontal sites. A 2-way interaction between aggression history and gender revealed that non-aggressive men had longer P3 latencies that either aggressive men or non-aggressive women. It is difficult to explain

this finding in light of past research. It seems most likely that this effect represents a somewhat different process or group of processes in this particular task compared to previous studies. Furthermore, P3 latencies in the aggression blocks were unrelated to reaction times.

Aggression History

One of the primary goals of this study was to examine potential differences between aggressive and non-aggressive individuals in their psychophysiological responses to provocative stimuli and to engaging in aggressive behavior. The most notable difference between the two groups was in how they processed threat-related stimuli. Previous studies have shown that aggressive individuals display reduced P3 amplitudes to neutral stimuli. This study also showed a very strong trend of aggressive individuals displaying a deficit in processing highly provocative stimuli. Indeed, they showed the same psychophysiological reaction to provocative stimuli as they did to neutral stimuli, whereas non-aggressive participants responded with increased processing of the threatening material. This finding points to possible abnormalities in the arousal neurocircuitry systems of aggressive individuals, or it could indicate that they are less surprised by provoking events, perhaps because they have engaged in a greater number of aggressive encounters in the past. Non-aggressive participants, on the other hand, took *longer* to process the provocative stimuli, possibly because they were more anxious aroused or surprised by them. Other findings involving aggression tended to occur in conjunction with gender, which is consistent with research that men and women differ biologically in how they respond to and engage in aggression. In a real-world aggressive encounter, this difference in processing might express itself via a greater number of interpretation errors or greater difficulty in distinguishing ambiguous provocation cues.

Gender

A number of findings emerged showing differences between men and women in their psychophysiological responses to threat and aggression, although these effects were often moderated by aggression history. Aggressive women in particular displayed a number of

differences from the other groups. Indeed, they represent the most extreme group in this study, as physical aggression by women is relatively uncommon. Aggressive women showed slower reaction times when identifying provocation cues and longer processing times for provocative stimuli than other groups. Non-aggressive women were particularly sensitive (as evidenced by P3 amplitude) to the high level of provocation by the opponent.

Limitations

There are several limitations in this study which bear mentioning. First, although efforts were made to recruit both aggressive and non-aggressive participants, the use of a college student and community population for recruitment limits the generalizability of the findings to generally healthy adults. Caution must be taken in generalizing the findings to more severely violent samples. It would be informative to conduct a similar study in a forensic population with a more significant history of violent physical aggression.

Second, a block design was used to examine provocation- and aggression-related ERPs separately. However, in a real-world aggressive encounter, provocation and response unfold and escalate in a very rapid temporal sequence. We therefore do not know what effects the "back-and-forth" nature of provocation and aggression would have on ERPs over and above the effects we have observed in this study. Fatigue, wavering attention, and the effects of repetitive responding are also factors to consider when using many trials in a blocked design. In a follow-up study it would be beneficial to use a design more closely resembling the original Taylor Reaction-Time Task, including a more rapid exchange of provocation and aggression between the participant and fictitious opponent. This type of design might yield larger effects of interest on the dependent variables as it would more closely approximate a real-life aggressive encounter and generate higher levels of affective arousal. If this were the case it might also be possible to use fewer trials in the task and in turn be less subject to the effects of fatigue.

Finally, due to the unknown effects of measuring ERPs while administering shocks, noise blasts were used as the physical provocation in this study. The use of noise blasts, however, is

less than ideal as they are perceived as less aversive within the range of decibels that can safely be administered to participants. While subjectively experienced as more aversive, shocks have less potential to result in actual physical damage compared noise blasts. As a result, the physical provocation in this study may have been less salient compared to the classic TRT.

Future Directions

The use of the ERP technique to examine how ERP components related to the perception of provocation and the expression of aggression is a new area and there are many ways in which this research could expand. Future research might employ a similar task to this one with more subtle or ambiguous stimuli, requiring participants to categorize the stimuli as threatening or non-threatening under time pressure. This would more closely resemble a real-life aggressive encounter and would also allow the researcher to examine the performance and ERPs of aggressive versus non-aggressive participants under conditions of greater cognitive demand and ambiguity.

Future research using this task might focus on earlier sensory and attention-related ERP components, particularly as some studies have found differences between aggressive and non-aggressive individuals in the early stages of cognitive processing. Future studies might also combine the oddball/TRT task with neuroimaging techniques to gain a better understanding of the structural and functional neurobiological factors that are activated during the task. Neutral or aggression-themed ERP tasks might also be used to examine whether anger and aggression-related interventions (CBT, psychopharmacology, etc.) have concomitant effects on relevant psychophysiological and cognitive processes.

Conclusions

Overall it was found that aggressive participants committed more errors in identifying provocative stimuli, showed less processing of provocative stimuli, and took less time to categorize provocative stimuli compared to non-aggressive participants, who made fewer categorization errors, and showed more extensive and slower processing of provocative stimuli.

The results are consistent with Dodge and Crick's suggestion that aggressive individuals show differences from healthy control in social information processing, and further suggest that some of these effects may be partially mediated by biological factors (Crick & Dodge, 1994). Although we focused on later cognitive processes ("top-down" processes) the results do not rule out the possibility that earlier components reflecting sensory processing or orienting of attention may also play a role in the processing of and responding to provoking events. P3 amplitude and latency have been found to be sensitive to a number of factors including the expectedness, probability, salience, and relevance of the stimuli and the arousal level of the participant. This task was not designed to distinguish which of these factors were at work here and we can only make educated guesses about our findings. However, the notion that non-aggressive participants found the high provocation stimuli more surprising is consistent with the effects on both P3 amplitude and latency, although there was some evidence that arousal levels may also have played a role in some of the findings.

Aggressive and non-aggressive men and women also showed differences in ERP components that have previously been linked to inhibitory and response conflict processes. Our results may not map on to previous studies entirely due to the strong affective and interpersonal aspects of this task. More research is needed to understand how emotions and socially relevant information are reflected in ERPs. It is apparent, however, that ERP is a useful technique for understanding cognitive processes, and one that is growing in popularity for answering questions related of social information processing and social behavior such as aggression.

APPENDIX A

N450 AND NSW: RESULTS AND BRIEF DISCUSSION

Results

Two additional components were examined in this study, N450 and NSW, but due to the small number of studies on them, their analyses were not considered primary. Results of these analyses and discussion are presented below.

Aggression N450

In order to examine the factors influencing the N450 component of the ERP, two separate 2 (aggression) × 2 (gender) × 3 (electrode), × 3 (response) mixed model ANOVAs were conducted for mean N450 amplitude and mean N450 latency in the aggression blocks. Mean amplitude represents the average ERP amplitude between 400 and 520 ms post-stimulus onset. Latency represents the time between stimulus onset and peak amplitude during the 400 to 520 ms post-stimulus window.

Mean N450 amplitude. *F* statistics for the mixed model ANOVA are displayed in Table B1 (Appendix B). A main effect of electrode was observed for N450 mean. Largest (most negative) amplitudes were observed at frontal (M = 1.83, sd = 2.73), followed by parietal (M = 4.81, sd = 2.65), and finally at central (M = 5.02, sd = 3.01) electrode sites. Frontal N450 amplitude was significantly lower compared to central ($t_{(43)}$ = -11.86, $p < .001$) and parietal ($t_{(43)}$ = -8.03, $p < .001$) sites. Central and parietal sites were not significantly different ($t_{(43)}$ = .96, $p > .05$).

Analysis of higher order interactions revealed a significant interaction between gender, aggression group, response, and electrode site, $F_{(2.01, 80.51)} = 3.49$, $p < .05$. Compared to non-aggressive females, non-aggressive males showed much larger N450 at frontal sites (relative to central sites; $t_{(20)}$ = -3.21, $p < .001$) when selecting the high aggressive response option.

Mean N450 latency. *F* statistics for the mixed model ANOVA are displayed in Table B2 (Appendix B). N450 latency varied significantly among the different electrode groups, $F_{(1.35, 54.14)} = 11.74$, $p < .001$. The N450 component peaked earliest at central sites ($M = .43$, $sd = .03$), followed by frontal sites ($M = .43$, $sd = .03$), and finally parietal sites ($M = .45$, $sd = .03$). N450 peaked significantly later at parietal sites compared to both frontal ($t_{(43)} = -2.99$, $p < .01$) and central sites ($t_{(43)} = -4.74$, $p < .001$). There were no other significant main effects or interactions for mean N450 latency. N450 was moderately and significantly correlated with reaction time, $r = -.42$, $p < .05$.

Aggression NSW

In order to examine the factors influencing the NSW component of the ERP, two separate 2 (aggression) × 2 (gender) × 3 (electrode), × 3 (response) mixed model ANOVAs were conducted for mean NSW amplitude and NSW latency in the aggression blocks. Mean amplitude represents the average ERP amplitude between 600 and 1000 ms post-stimulus onset. Latency represents the time between stimulus onset and peak amplitude during the 600 to 1000 ms post-stimulus window.

Mean NSW amplitude. Several main effects and interactions were observed on mean NSW amplitude (see Appendix B, Table B3). A main effect of electrode was observed for mean NSW amplitude, $F_{(1.29, 51.66)} = 36.08$, $p < .001$. NSW was smallest at central sites ($M = 4.03$, $sd = 2.70$), followed by parietal sites ($M = 3.65$, $sd = 2.55$), and largest (most negative) at frontal sites ($M = 2.10$, $sd = 2.66$). These differences were all significant, $t_{FC(43)} = -7.75$, $p < .001$; $t_{FP(43)} = -4.43$, $p < .001$; $t_{CP(43)} = 2.21$, $p < .05$.

This main effect was qualified by a 3-way interaction between gender, aggression group, and electrode site ($F_{(1.29, 51.66)} = 7.15$, $p < .01$); however, between subjects' simple effects were not significant.

A 2-way interaction between electrode site and response was observed, $F_{(2.62, 90.47)} = 5.65$, $p < .001$. Follow-up repeated measures ANOVAs for response at each electrode site revealed a significant effect of response at central sites, $F_{(2, 86)} = 5.13$, $p < .01$, but not frontal or parietal sites. "High" response choices were associated with significantly smaller NSW amplitude at central sites compared to "medium" responses ($M_{HM} = 3.54$, $sd = 2.64$; $M_{HH} = 4.49$, $sd = 3.19$; $t_{(43)} = -2.99$, $p < .01$).

A 4-way interaction qualifies this effect (gender × aggression group × electrode × response; $F_{(2.26, 90.47)} = 3.46$, $p < .05$). A follow-up reduced 2-way ANOVA (gender × aggression group at response × electrode) was significant, $F_{(1, 40)} = 4.84$, $p < .05$. Aggressive females showed significantly larger NSW amplitude at parietal sites for when choosing the "high" response ($M = 4.73$, $sd = 2.44$) compared to non-aggressive females ($M = 3.99$, $sd = 3.07$; $t_{(19)} = 2.21$, $p < .05$) and to aggressive males ($M = 2.17$, $sd = 2.81$; $t_{(20)} = -2.25$, $p < .05$).

Mean NSW latency. No significant main effects or interactions emerged for NSW latency (Table B4, Appendix B). Trends ($p < .10$) were evident for electrode group, an interaction between gender, aggression, and response, and an interaction between gender, aggression, response, and electrode group. No follow-up analyses were conducted. NSW latency was uncorrelated with reaction time, $r = .16$, $p = .44$. Means and sds for the aggression waveforms (N450 and NSW) are located in Tables C9-C12 (Appendix C).

Discussion

Aggression N450

The N450 component was included in this study as a measure of response conflict. N450 was observed to be largest at central sites, followed by parietal sites and finally at central sites. A complex interaction emerged involving N450 such that in non-aggressive males, N450 was significantly larger at frontal as compared to central sites than was the case for non-aggressive females, during the selection of the high response. It is not surprising that selecting the high response would generate response conflict in non-aggressive participants, as these people are

more likely to hold prohibitions against behaving aggressively. The interactions involving gender and electrode site are less clear. Research suggests that the expression of aggressive behavior is driven by the interacting functions of the amygdala (which processes emotional and threat-related information) and the frontal lobes, which are home to the executive functions. A large negative deflection over frontal parts of the brain could indicate increased activation of the frontal lobes. Furthermore, aggressive men and women have been shown to have distinct patterns of activation in the connections between the frontal lobes and amygdala in the presence of provocation. Clearly, however, more information is needed to understand the physiological and cognitive processes this complex interaction represents. The N450 component was found to peak earliest at central sites, followed by frontal sites, and finally at parietal sites.

N450 was the only component in the aggression blocks to correlate with reaction time. It is likely that the range of this component (400 to 520 ms) significantly overlapped with the motor movements that took place as participants pressed the reaction time button (button presses took place on average around 300 ms post-stimulus in the aggression block). Motor contamination is less likely to be a factor for the nearby components, P3 and NSW, as they had longer epochs. It is also important to note that the presence of motor effects in N450 amplitude, if this was the case, does not mean that other cognitive processes were not also reflected in the component.

Aggression NSW

Finally, the negative slow was (NSW) was examined in the aggression blocks because of its links in previous studies to processes related to inhibition. NSW has been shown to be present for successfully inhibited stop signals, and to be larger for trials in which greater response conflict is present. NSW is typically observed later in the epoch (between 600 and 1,000 ms), after the response has taken place. Thus, it has been proposed to represent the resolution of inhibitory or response conflict processes. In short, NSW has been interpreted as an index of the extent to which inhibitory resources were recruited earlier in the trial. The NSW was largest at central sites, followed by parietal sites, and finally at frontal sites. A 2-way interaction revealed that NSW was

larger at central sites after selecting the "medium" versus the "high" response. This finding is consistent with the notion that NSW reflects inhibitory control processes, as more inhibitory control would have been necessary to select a medium than a high aggressive response, particularly if the two responses were competing. With regard to the lack of difference between the high and low responses, it may be that for trials in which the participant chose the low response, there was less activation related to wanting to retaliate and that therefore fewer inhibitory resources needed to be recruited to inhibit the potential competing response. In other words, in the absence of a strong competing response option (response conflict), fewer inhibitory resources may have been recruited.

It was also observed that aggressive females demonstrated larger NSW on trials in which they chose the "high" response than did non-aggressive females and non-aggressive males. This finding is somewhat counter-intuitive. Previous research suggests that NSW is sensitive to both the strength of the activation of competing responses and the extent of recruitment of inhibitory resources. We did not examine any components in the aggression blocks that would indicate the level of activation associated with a given response, however, it is possible that aggressive females experienced considerable response *activation* during trials in which they ultimately administered an aggressive response, and they may have additionally experienced high level of inhibitory processing (NSW), resulting in a high level of response conflict. Non-aggressive participants, on the other hand, may have shown less activation (i.e., desire to behave aggressively) and as a result recruited fewer inhibitory resources. In the past, NSW has been reported for *successful* stop trials in a Go/No-Go task. In the case of this interaction effect, whatever attempt there might have been to inhibit an aggressive response failed as participants proceeded to select the "high" noise blast option. It is conceivable, however, that inhibitory resources could have been recruited and yet failed to override the competing activated response. As we did not measure response activation we have no way of testing this hypothesis. An

alternative explanation is that NSW in this task reflects different processes than in other previous tasks.

No significant effects were found involving NSW latency. Latency was also uncorrelated with reaction time.

APPENDIX B

N450 AND NSW TABLES

Table B1

Main and Interaction Effects on Aggression N450 Mean Amplitude (400-520 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.00	(1)	.00
Aggression	0.00	(1)	.00
Electrode[a]	74.78	(1.41)***	.64
Response[a]	0.29	(1.58)	.01
Two-Way Interactions			
Gender*Agg[a]	2.57	(1)	.06
Electrode*Response[a]	0.12	(2.01)	.00
Gender*Electrode[a]	0.27	(1.41)	.00
Gender*Response[a]	0.32	(1.58)	.01
Aggression*Electrode[a]	0.61	(1.41)	.01
Aggression*Response[a]	0.10	(1.58)	.00
Three-Way Interactions			
Gender*Agg*Electrode[a]	1.71	(1.41)	.01
Gender*Agg*Response[a]	0.20	(1.58)	.00
Gender*Electrode*Resp[a]	1.23	(2.01)	.03
Agg*Electrode*Resp[a]	0.21	(2.01)	.00
Four-Way Interactions			
Gender*Agg*Elec*Resp[a]	3.49	(2.01)*	.08

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Table B2

Main and Interaction Effects on Aggression N450 Latency (400-520 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.35	(1)	.01
Aggression	0.03	(1)	.00
Electrode[a]	11.74	(1.35)***	.22
Response	0.17	(2)	.00
Two-Way Interactions			
Gender*Agg	0.65	(1)	.02
Electrode*Response[a]	0.84	(2.91)	.02
Gender*Electrode[a]	0.56	(1.35)	.01
Gender*Response	0.86	(2)	.02
Aggression*Electrode[a]	0.33	(1.35)	.01
Aggression*Response	1.37	(2)	.03
Three-Way Interactions			
Gender*Agg*Electrode[a]	0.01	(1.35)	.00
Gender*Agg*Response	0.87	(2)	.02
Gender*Electrode*Resp[a]	0.79	(2.91)	.02
Agg*Electrode*Resp[a]	1.60	(2.91)	.04
Four-Way Interactions			
Gender*Agg*Elec*Resp[a]	0.30	(2.91)	.00

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Table B3

Main and Interaction Effects on Aggression NSW Mean Amplitude (600-1000 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	.03	(1)	.00
Aggression	1.78	(1)	.04
Electrode[a]	36.08	(1.29)***	.43
Response	2.73	(2) †	.06
Two-Way Interactions			
Gender*Agg	.40	(1)	.01
Electrode*Response [a]	5.65	(2.26)**	.11
Gender*Electrode [a]	.44	(1.29)	.01
Gender*Response	.77	(2)	.02
Aggression*Electrode [a]	.97	(1.29)	.01
Aggression*Response	.74	(2)	.02
Three-Way Interactions			
Gender*Agg*Electrode [a]	7.15	(1.29)**	.08
Gender*Agg*Response	1.13	(2)	.02
Gender*Electrode*Resp [a]	1.32	(2.26)	.03
Agg*Electrode*Resp [a]	.65	(2.26)	.01
Four-Way Interactions			
Gender*Agg*Elec*Resp [a]	3.46	(2.26)*	.07

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

Table B4

Main and Interaction Effects on Aggression NSW Latency (600-1000 ms), Under High Provocation

	F	*(df)*	η^2
Main Effects			
Gender	0.23	(1)	.01
Aggression	0.36	(1)	.01
Electrode[a]	2.86	(1.39) †	.07
Response	0.27	(2)	.01
Two-Way Interactions			
Gender*Agg	(1.40)	(1)	.03
Electrode*Response[a]	0.95	(2.83)	.02
Gender*Electrode[a]	0.51	(1.39)	.01
Gender*Response	1.29	(2)	.03
Aggression*Electrode[a]	0.24	(1.39)	.01
Aggression*Response	2.07	(2)	.05
Three-Way Interactions			
Gender*Agg*Electrode[a]	0.27	(1.39)	.01
Gender*Agg*Response	2.59	(2) †	.06
Gender*Electrode*Resp[a]	0.82	(2.83)	.02
Agg*Electrode*Resp[a]	1.24	(2.83)	.03
Four-Way Interactions			
Gender*Agg*Elec*Resp[a]	2.19	(2.83) †	.05

*$p < .05$, **$p < .01$, ***$p < .001$, † < .10 (trend); [a] Greenhouse-Geisser corrected.

APPENDIX C

MEANS AND STANDARD DEVIATIONS

Table C1

Mean Reaction Times for Provocation and Aggression Blocks

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	#Obs	M	sd	#Obs	M	sd	#Obs	M	sd	#Obs
						Low Provocation						
Oddball	.46	.05	11	.51	.06	15	.50	.06	12	.52	.06	10
Frequent	.41	.06	11	.45	.06	15	.43	.04	12	.44	.06	10
						High Provocation						
Oddball	.53	.07	11	.53	.05	15	.52	.11	12	.62	.08	10
Frequent	.45	.05	11	.44	.06	15	.44	.10	12	.48	.06	10
						Aggression (following Low Provocation)						
Low	.30	.06	11	.28	.04	15	.27	.04	11	.30	.05	9
Medium	.30	.06	10	.29	.05	13	.28	.04	12	.38	.26	10
High	.30	.04	4	.30	.05	8	.31	.11	11	.30	.03	5
						Aggression (following High Provocation)						
Low	.31	.05	11	.29	.05	15	.27	.03	12	.30	.06	10
Medium	.31	.07	10	.32	.09	14	.28	.04	12	.32	.05	10
High	.31	.06	9	.31	.06	13	.29	.04	12	.30	.05	10

Table C2

Mean Mood Ratings at Five Time Points

	Non-Aggressive				Aggressive			
	Male		Female		Male		Female	
	M	sd	M	sd	M	sd	M	sd
	Low Provocation First							
	(n=8)		(n=6)		(n=5)		(n=7)	
Nervous								
Time 1	29.13	26.32	26.50	27.97	37.80	24.22	19.00	25.82
Time 2	10.75	10.05	15.33	25.08	15.40	10.26	18.71	25.29
Time 3	4.50	4.24	18.67	26.19	7.40	8.85	14.86	17.20
Time 4	2.63	1.60	13.17	21.09	6.40	6.58	13.14	16.95
Time 5	3.13	2.59	6.67	9.20	7.20	9.96	3.86	3.98
Angry								
Time 1	4.38	4.75	6.17	9.85	3.60	4.39	24.57	34.16
Time 2	6.50	12.18	10.33	18.55	2.00	1.22	10.57	12.49
Time 3	8.63	16.94	14.67	26.71	5.20	8.90	4.14	4.06
Time 4	6.75	11.35	10.50	19.53	3.60	3.58	16.00	20.22
Time 5	5.88	11.23	8.67	15.56	3.20	2.17	5.57	6.83
	High Provocation First							
	(n=3)		(n=7)		(n=9)		(n=3)	
Nervous								
Time 1	46.00	27.87	40.11	27.35	25.86	18.61	49.67	22.23
Time 2	26.67	23.03	18.89	16.53	11.71	13.88	31.67	23.50
Time 3	10.00	12.17	10.56	12.05	4.86	9.35	19.33	13.32
Time 4[a]	4.67	4.73	13.00	8.72	6.83	13.82	8.00	1.00
Time 5	1.00	1.00	13.33	12.65	7.14	15.41	9.67	5.77
Angry								
Time 1	4.67	2.08	5.44	5.77	4.57	6.90	14.33	16.29
Time 2	1.33	0.58	8.33	8.29	4.43	6.19	21.00	26.15
Time 3	1.67	1.15	10.78	19.19	3.14	5.27	22.67	23.03
Time 4	2.67	3.06	19.11	27.68	7.67	10.09	14.33	14.74
Time 5	18.67	27.15	9.89	12.13	9.00	15.63	11.00	10.58

Note: [a] Non-aggressive female (n=6) due to missing data points.

Table C3

Mean Amplitudes for Provocation P3

	Non-Aggressive				Aggressive			
	Male (n=11)		Female (n=15)		Male (n=12)		Female (n=10)	
	M	sd	M	sd	M	sd	M	sd
Low Provocation Oddball								
Frontal	4.53	4.15	4.15	7.36	5.53	3.54	1.47	4.84
Central	9.47	3.86	9.70	5.54	9.82	4.22	6.83	4.89
Parietal	12.06	3.99	12.63	3.74	11.62	3.46	10.33	5.16
Low Provocation Frequent								
Frontal	2.91	4.19	2.39	4.11	3.19	2.59	1.30	3.05
Central	6.97	3.89	6.36	3.36	6.66	3.17	4.95	3.76
Parietal	7.94	3.45	7.52	1.87	7.28	2.62	5.97	2.80
High Provocation Oddball								
Frontal	5.81	6.39	6.86	5.77	5.62	4.02	2.16	5.13
Central	10.56	6.20	10.91	4.56	8.89	3.98	7.62	5.32
Parietal	13.18	6.28	12.76	3.85	10.41	3.97	10.74	6.07
High Provocation Frequent								
Frontal	2.44	3.30	3.26	4.05	2.85	2.94	1.64	3.31
Central	6.42	3.70	6.84	3.34	6.25	3.57	5.62	3.92
Parietal	7.58	3.73	7.88	2.51	7.01	2.96	6.75	3.48

Table C4

Mean Latencies for Provocation P3

	Non-Aggressive				Aggressive			
	Male (n=11)		Female (n=15)		Male (n=12)		Female (n=10)	
	M	sd	M	sd	M	sd	M	sd
Low Provocation Oddball								
Frontal	0.42	0.07	0.42	0.06	0.44	0.08	0.45	0.09
Central	0.42	0.07	0.45	0.06	0.47	0.08	0.47	0.07
Parietal	0.42	0.06	0.44	0.06	0.43	0.08	0.45	0.05
Low Provocation Frequent								
Frontal	0.42	0.07	0.42	0.06	0.43	0.06	0.45	0.08
Central	0.42	0.08	0.43	0.06	0.42	0.08	0.45	0.08
Parietal	0.38	0.06	0.39	0.07	0.37	0.04	0.39	0.05
High Provocation Oddball								
Frontal	0.45	0.08	0.43	0.04	0.43	0.05	0.45	0.05
Central	0.46	0.08	0.45	0.05	0.44	0.06	0.47	0.04
Parietal	0.46	0.05	0.45	0.05	0.42	0.07	0.48	0.05
High Provocation Frequent								
Frontal	0.42	0.08	0.41	0.05	0.43	0.05	0.49	0.07
Central	0.44	0.08	0.42	0.06	0.43	0.07	0.47	0.07
Parietal	0.38	0.05	0.38	0.05	0.37	0.06	0.43	0.06

Table C5

Mean Amplitudes for Aggression N2

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	1.98	2.94	11	2.21	2.67	15	1.22	2.79	12	1.59	2.29	10
Central	4.00	2.68	11	4.33	2.85	15	3.59	2.94	12	4.21	2.20	10
Parietal	5.46	2.24	11	5.47	2.79	15	5.31	2.08	12	5.19	2.33	10
						Low Provocation "Medium"						
Frontal	2.05	2.87	10	2.11	3.38	13	0.58	2.28	12	0.73	1.72	10
Central	4.15	2.83	10	4.12	2.72	13	2.93	2.66	12	3.55	2.05	10
Parietal	5.87	2.37	10	5.26	2.68	13	4.94	1.70	12	4.77	2.43	10
						Low Provocation "High"						
Frontal	0.92	2.13	4	2.28	4.86	8	-1.72	4.47	11	0.43	3.24	5
Central	2.03	1.96	4	3.68	4.94	8	1.36	2.96	11	4.07	3.25	5
Parietal	4.63	1.30	4	4.99	4.61	8	4.86	3.13	11	7.51	4.99	5
						High Provocation "Low"						
Frontal	2.43	2.43	11	1.79	2.96	15	1.33	2.59	12	1.96	2.20	10
Central	4.36	2.45	11	3.67	2.30	15	3.61	3.08	12	4.29	1.92	10
Parietal	5.88	2.47	11	5.16	2.58	15	5.38	2.72	12	5.24	2.34	10
						High Provocation "Medium"						
Frontal	2.62	2.83	10	2.83	4.36	14	1.39	2.78	12	0.81	2.24	10
Central	4.66	2.85	10	4.39	2.78	14	3.69	3.08	12	3.39	2.30	10
Parietal	6.08	2.16	10	5.39	2.15	14	5.78	2.89	12	4.54	2.70	10
						High Provocation "High"						
Frontal	2.01	2.82	9	2.24	3.82	13	0.76	3.82	12	0.29	1.67	10
Central	4.55	2.49	9	3.95	2.62	13	2.66	3.23	12	3.32	1.75	10
Parietal	6.70	2.70	9	5.19	2.17	13	4.70	2.94	12	4.58	2.49	10

Table C6

Mean Latencies for Aggression N2

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	0.27	0.02	11	0.25	0.04	15	0.26	0.04	12	0.26	0.04	10
Central	0.24	0.05	11	0.23	0.05	15	0.23	0.06	12	0.23	0.05	10
Parietal	0.19	0.04	11	0.19	0.02	15	0.21	0.04	12	0.22	0.04	10
						Low Provocation "Medium"						
Frontal	0.26	0.03	10	0.26	0.05	13	0.25	0.04	12	0.24	0.04	10
Central	0.23	0.04	10	0.23	0.06	13	0.22	0.05	12	0.23	0.05	10
Parietal	0.19	0.04	10	0.18	0.02	13	0.20	0.03	12	0.21	0.05	10
						Low Provocation "High"						
Frontal	0.26	0.01	4	0.25	0.03	8	0.24	0.04	11	0.25	0.04	5
Central	0.22	0.04	4	0.24	0.03	8	0.22	0.04	11	0.25	0.05	5
Parietal	0.19	0.02	4	0.19	0.05	8	0.20	0.03	11	0.22	0.05	5
						High Provocation "Low"						
Frontal	0.27	0.02	11	0.25	0.04	15	0.25	0.03	12	0.27	0.03	10
Central	0.24	0.04	11	0.22	0.04	15	0.21	0.04	12	0.22	0.04	10
Parietal	0.20	0.03	11	0.19	0.03	15	0.20	0.03	12	0.21	0.04	10
						High Provocation "Medium"						
Frontal	0.26	0.04	10	0.25	0.04	14	0.25	0.03	12	0.26	0.03	10
Central	0.23	0.03	10	0.23	0.04	14	0.23	0.05	12	0.24	0.04	10
Parietal	0.18	0.02	10	0.19	0.02	14	0.21	0.03	12	0.22	0.04	10
						High Provocation "High"						
Frontal	0.26	0.04	9	0.25	0.04	13	0.25	0.04	12	0.26	0.03	10
Central	0.24	0.04	9	0.23	0.04	13	0.22	0.04	12	0.23	0.03	10
Parietal	0.20	0.02	9	0.19	0.03	13	0.20	0.02	12	0.20	0.05	10

Table C7

Mean Amplitudes for Aggression P3

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	0.94	2.27	11	1.50	3.28	15	2.08	2.58	12	1.11	2.65	10
Central	4.47	2.08	11	4.35	2.94	15	4.69	2.65	12	3.88	2.67	10
Parietal	4.47	2.16	11	4.55	1.63	15	4.42	2.60	12	3.96	2.22	10
						Low Provocation "Medium"						
Frontal	1.11	2.20	10	1.65	3.95	13	1.28	2.14	12	0.90	3.72	10
Central	4.72	2.79	10	4.34	3.03	13	3.82	2.18	12	3.37	2.83	10
Parietal	4.91	2.61	10	4.56	1.94	13	3.85	1.83	12	3.93	2.54	10
						Low Provocation "High"						
Frontal	1.69	2.18	4	1.25	5.43	8	1.75	5.33	11	1.65	3.49	5
Central	3.62	1.45	4	4.52	5.58	8	5.32	3.46	11	5.19	3.80	5
Parietal	3.76	0.61	4	5.66	4.87	8	6.03	3.59	11	6.00	2.84	5
						High Provocation "Low"						
Frontal	1.46	2.81	11	0.83	3.12	15	1.57	1.65	12	2.20	2.13	10
Central	5.22	3.16	11	3.84	2.82	15	4.47	2.35	12	5.13	2.41	10
Parietal	5.23	3.23	11	4.38	2.06	15	4.32	2.47	12	4.92	2.25	10
						High Provocation "Medium"						
Frontal	1.25	2.98	10	1.62	4.20	14	1.85	1.93	12	1.31	2.39	10
Central	5.25	3.00	10	3.82	2.98	14	4.30	2.21	12	4.59	2.43	10
Parietal	5.11	2.12	10	4.26	2.30	14	4.32	2.26	12	4.55	2.55	10
						High Provocation "High"						
Frontal	1.47	3.46	9	1.77	4.41	13	1.26	2.88	12	1.32	2.61	10
Central	5.98	3.94	9	3.97	4.06	13	3.87	3.24	12	4.84	2.80	10
Parietal	6.40	3.88	9	4.21	2.82	13	3.97	3.01	12	4.86	2.88	10

Table C8

Mean Latencies for Aggression P3

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	0.49	0.08	11	0.49	0.08	15	0.50	0.10	12	0.50	0.08	10
Central	0.44	0.08	11	0.47	0.08	15	0.47	0.11	12	0.47	0.08	10
Parietal	0.39	0.08	11	0.36	0.06	15	0.36	0.06	12	0.44	0.09	10
						Low Provocation "Medium"						
Frontal	0.51	0.07	10	0.48	0.07	13	0.52	0.07	12	0.48	0.07	10
Central	0.50	0.08	10	0.46	0.08	13	0.46	0.09	12	0.46	0.08	10
Parietal	0.38	0.08	10	0.38	0.07	13	0.39	0.08	12	0.43	0.09	10
						Low Provocation "High"						
Frontal	0.51	0.05	4	0.52	0.06	8	0.51	0.08	11	0.53	0.02	5
Central	0.51	0.05	4	0.49	0.07	8	0.47	0.08	11	0.46	0.09	5
Parietal	0.45	0.11	4	0.44	0.09	8	0.40	0.06	11	0.42	0.10	5
						High Provocation "Low"						
Frontal	0.55	0.04	11	0.49	0.09	15	0.50	0.09	12	0.48	0.06	10
Central	0.50	0.09	11	0.47	0.09	15	0.43	0.10	12	0.46	0.06	10
Parietal	0.43	0.11	11	0.40	0.09	15	0.37	0.08	12	0.45	0.07	10
						High Provocation "Medium"						
Frontal	0.53	0.05	10	0.46	0.11	14	0.51	0.09	12	0.50	0.06	10
Central	0.51	0.05	10	0.43	0.10	14	0.44	0.10	12	0.47	0.07	10
Parietal	0.41	0.09	10	0.40	0.08	14	0.36	0.07	12	0.43	0.08	10
						High Provocation "High"						
Frontal	0.51	0.08	9	0.50	0.06	13	0.50	0.08	12	0.48	0.08	10
Central	0.50	0.07	9	0.46	0.08	13	0.45	0.10	12	0.46	0.09	10
Parietal	0.41	0.10	9	0.41	0.08	13	0.37	0.07	12	0.44	0.07	10

Table C9

Mean Amplitudes for Aggression N450

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	1.00	2.77	11	1.94	3.50	15	1.97	2.36	12	1.90	3.18	10
Central	4.61	2.32	11	4.76	3.21	15	4.73	2.43	12	4.78	3.12	10
Parietal	4.23	2.22	11	4.65	2.07	15	4.29	2.52	12	4.52	2.46	10
						Low Provocation "Medium"						
Frontal	1.09	2.13	10	2.14	4.07	13	1.24	1.96	12	2.14	5.60	10
Central	4.81	2.35	10	4.77	3.21	13	3.98	2.04	12	4.39	3.60	10
Parietal	4.67	2.30	10	4.65	2.24	13	3.87	2.11	12	4.37	2.74	10
						Low Provocation "High"						
Frontal	2.51	1.24	4	2.19	6.31	8	2.30	5.71	11	2.08	4.06	5
Central	4.50	0.90	4	5.44	6.33	8	6.03	3.79	11	5.85	3.37	5
Parietal	4.09	1.19	4	6.50	5.58	8	6.33	4.31	11	6.51	2.45	5
						High Provocation "Low"						
Frontal	1.71	2.90	11	0.80	3.24	15	1.42	1.72	12	2.94	2.63	10
Central	5.51	3.03	11	3.97	3.06	15	4.48	2.58	12	6.11	2.82	10
Parietal	5.12	3.08	11	4.38	2.54	15	4.07	2.54	12	5.48	2.45	10
						High Provocation "Medium"						
Frontal	1.55	3.21	10	1.66	3.90	14	1.84	2.12	12	2.14	2.69	10
Central	5.69	3.03	10	3.93	3.26	14	4.43	2.30	12	5.60	2.79	10
Parietal	5.10	1.86	10	4.23	2.95	14	4.17	2.37	12	5.18	3.04	10
						High Provocation "High"						
Frontal	1.52	3.24	9	2.19	4.43	13	1.61	2.94	12	2.34	3.87	10
Central	6.18	3.62	9	4.30	4.42	13	4.30	3.43	12	6.00	3.45	10
Parietal	6.12	3.61	9	4.34	3.32	13	4.00	3.21	12	5.60	3.04	10

Table C10

Mean Latencies for Aggression N450

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	0.44	0.03	11	0.43	0.04	15	0.43	0.03	12	0.41	0.01	10
Central	0.44	0.04	11	0.44	0.04	15	0.43	0.04	12	0.42	0.03	10
Parietal	0.46	0.04	11	0.45	0.04	15	0.46	0.04	12	0.44	0.04	10
						Low Provocation "Medium"						
Frontal	0.44	0.03	10	0.42	0.04	13	0.43	0.04	12	0.41	0.02	10
Central	0.43	0.02	10	0.42	0.03	13	0.44	0.04	12	0.43	0.04	10
Parietal	0.46	0.04	10	0.44	0.04	13	0.46	0.04	12	0.44	0.05	10
						Low Provocation "High"						
Frontal	0.45	0.03	4	0.46	0.03	8	0.45	0.04	11	0.43	0.03	5
Central	0.44	0.04	4	0.45	0.04	8	0.44	0.03	11	0.43	0.03	5
Parietal	0.42	0.03	4	0.46	0.04	8	0.45	0.03	11	0.45	0.02	5
						High Provocation "Low"						
Frontal	0.43	0.03	11	0.44	0.04	15	0.43	0.04	12	0.43	0.03	10
Central	0.43	0.03	11	0.43	0.03	15	0.44	0.04	12	0.43	0.03	10
Parietal	0.44	0.04	11	0.45	0.04	15	0.46	0.04	12	0.45	0.05	10
						High Provocation "Medium"						
Frontal	0.43	0.03	10	0.44	0.04	14	0.43	0.04	12	0.43	0.03	10
Central	0.43	0.03	10	0.44	0.03	14	0.42	0.04	12	0.43	0.04	10
Parietal	0.46	0.04	10	0.45	0.03	14	0.45	0.05	12	0.45	0.05	10
						High Provocation "High"						
Frontal	0.42	0.02	9	0.45	0.04	13	0.44	0.04	12	0.44	0.04	10
Central	0.42	0.02	9	0.44	0.04	13	0.42	0.04	12	0.43	0.05	10
Parietal	0.44	0.04	9	0.45	0.04	13	0.45	0.04	12	0.44	0.04	10

Table C11

Mean Amplitudes for Aggression NSW

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	0.29	2.45	11	2.11	2.92	15	2.37	3.26	12	1.31	2.04	10
Central	2.72	2.60	11	3.56	1.98	15	3.50	3.21	12	2.51	2.71	10
Parietal	2.63	2.41	11	3.26	1.43	15	2.74	2.86	12	2.62	3.09	10
						Low Provocation "Medium"						
Frontal	1.22	2.85	10	3.20	3.30	13	2.49	2.07	12	1.46	4.10	10
Central	3.48	3.08	10	4.42	2.08	13	3.26	2.35	12	2.76	2.67	10
Parietal	3.05	2.55	10	3.97	1.71	13	2.84	2.57	12	3.50	2.91	10
						Low Provocation "High"						
Frontal	0.98	2.08	4	2.49	6.47	8	3.49	3.37	11	-0.64	4.58	5
Central	1.94	1.88	4	4.68	4.80	8	4.92	3.71	11	2.31	3.38	5
Parietal	1.26	1.11	4	3.99	4.70	8	4.48	3.35	11	3.54	2.28	5
						High Provocation "Low"						
Frontal	2.05	4.14	11	2.62	2.65	15	2.32	2.09	12	1.68	3.32	10
Central	4.76	4.68	11	4.02	2.30	15	3.32	2.09	12	3.56	1.95	10
Parietal	4.47	4.72	11	3.74	2.23	15	2.44	2.28	12	3.56	1.42	10
						High Provocation "Medium"						
Frontal	1.71	3.70	10	2.13	2.73	14	2.52	1.96	12	0.85	2.95	10
Central	4.38	3.44	10	3.23	2.98	14	3.20	2.45	12	3.09	1.47	10
Parietal	3.57	2.19	10	3.21	2.48	14	2.72	2.12	12	3.29	1.42	10
						High Provocation "High"						
Frontal	1.74	4.01	9	2.85	2.62	13	1.71	2.92	12	1.56	2.29	10
Central	5.80	4.84	9	4.80	2.71	13	3.25	2.81	12	4.41	2.08	10
Parietal	5.89	4.87	9	3.99	3.07	13	2.17	2.81	12	4.73	2.44	10

Table C12

Mean Latencies for Aggression NSW

	Non-Aggressive						Aggressive					
	Male			Female			Male			Female		
	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs	M	sd	# Obs
						Low Provocation "Low"						
Frontal	0.90	0.06	11	0.83	0.10	15	0.81	0.10	12	0.78	0.12	10
Central	0.91	0.07	11	0.87	0.09	15	0.84	0.12	12	0.84	0.10	10
Parietal	0.89	0.11	11	0.85	0.11	15	0.78	0.11	12	0.86	0.12	10
						Low Provocation "Medium"						
Frontal	0.93	0.05	10	0.82	0.12	13	0.83	0.12	12	0.81	0.13	10
Central	0.91	0.07	10	0.85	0.09	13	0.82	0.10	12	0.86	0.12	10
Parietal	0.87	0.07	10	0.80	0.14	13	0.75	0.10	12	0.89	0.12	10
						Low Provocation "High"						
Frontal	0.85	0.14	4	0.90	0.13	8	0.78	0.10	11	0.81	0.08	5
Central	0.86	0.09	4	0.86	0.10	8	0.84	0.12	11	0.77	0.10	5
Parietal	0.86	0.07	4	0.81	0.12	8	0.82	0.10	11	0.70	0.11	5
						High Provocation "Low"						
Frontal	0.88	0.11	11	0.83	0.13	15	0.87	0.11	12	0.82	0.11	10
Central	0.87	0.11	11	0.85	0.11	15	0.89	0.12	12	0.90	0.10	10
Parietal	0.87	0.10	11	0.83	0.13	15	0.79	0.12	12	0.85	0.12	10
						High Provocation "Medium"						
Frontal	0.88	0.12	10	0.81	0.15	14	0.82	0.13	12	0.87	0.08	10
Central	0.87	0.08	10	0.84	0.12	14	0.84	0.12	12	0.91	0.09	10
Parietal	0.84	0.11	10	0.80	0.13	14	0.79	0.11	12	0.90	0.09	10
						High Provocation "High"						
Frontal	0.90	0.08	9	0.85	0.14	13	0.84	0.13	12	0.81	0.06	10
Central	0.90	0.07	9	0.86	0.14	13	0.83	0.14	12	0.82	0.10	10
Parietal	0.87	0.10	9	0.85	0.15	13	0.86	0.09	12	0.78	0.09	10

APPENDIX D

EVERYTHING ELSE

Table D1

Reaction Time Means, Standard Deviations, and Differences (T-tests) for Provocation Block Stimuli

	LL_o [b]	LM_f	HH_o	HM_f
	M = 0.50	*M* = 0.43	*M* = 0.55	*M* = 0.45
	sd = 0.06	*sd* = 0.05	*sd* = 0.08	*sd* = 0.07
		t [a]		
LL_o	--			
LM_f	12.53***	--		
HH_o	-3.80***	-9.60***	--	
HM_f	4.62***	-2.42*	10.92***	--

*$p < .05$; **$p < .01$; ***$p < .001$, † < .10 (trend); [a] $df = 46$. [b] See List of Abbreviations for definition of terms.

APPENDIX E

PARTICIPANT INSTRUCTIONS

(Read over the intercom)

Instructions for Setting Tone Threshold:

"Okay Subjects A and B, I'm going to open the microphone so that we can all hear each other. Please listen carefully."

"To start off I will play each of you a series of short tones increasing the loudness of each one. When the tone is first presented, it will be below your hearing threshold and you will NOT hear it. As the loudness increases: first, you will become aware of it; second, the tones will become progressively louder; and third, the loudness of the tones will become uncomfortable or very unpleasant. I want you to tell me two things: one, tell me when you first hear the tone and two, tell me when you don't want it to increase anymore, that is, when it is DEFINITELY uncomfortable or unpleasant (just say "that's enough")."

"Okay Subject A (in Lab 228), let's begin with you. Tell me when you first hear the tone. Just say "I hear it."

…

"Okay, Subject A, now tell me when you don't want the tone to increase anymore, that is, when the volume becomes very uncomfortable or unpleasant. I will stop the procedure when you tell me the tone is VERY uncomfortable or unpleasant—that is, when you can't take anymore increase. PLEASE wait to stop until the tone is VERY uncomfortable or unpleasant (just say "that's enough")."

…

“Okay Subject A, we’ll stop there”.

“Okay Subject B, now it’s your turn. Tell me when you first hear the tone.”

…

“Okay, now tell me when you don’t want anymore, that is, when the tone becomes VERY uncomfortable or unpleasant. I will stop the upper threshold procedure when you tell me the tone is very uncomfortable or unpleasant. PLEASE wait to stop until the tone is very uncomfortable or unpleasant.”

…

“Okay Subject B we’ll stop there”.

Instructions for the Reaction Time Task:

"Okay Subjects A and B, we’ll do the task now. The purpose of this task is to determine the relationship between EEG (or brainwaves) and the speed with which you can press a reaction time button—the buttons on the button box. Although the two of you are seated in separate rooms, we will be recording your responses to see who has the faster reaction time.”

“While the study is going on, please keep your fingers resting on the three keys that you will be using; you will not need to press the fourth key at any time. Whenever you see instructions on the computer screen, read them carefully. When you are finished reading the instructions, press the “START” button to move on to the next page. As soon as both of you have finished reading the instructions, the experiment will start. During the computer task you will complete four blocks of trials. There will be a break between each block. There will also be a break halfway through each block. While the experiment is going on, you must keep your eyes focused on the fixation point in the center of the screen. Too much eye movement or body movement can interfere with the

data collection and may require us to end the experiment early for both of you. During the breaks you can relax and move about as long as it does not cause pulling on the cap. One of the lab personnel will check in with you during each break. While the experiment is running, please respond to all stimuli that appear on the computer screen. If you forget to respond to or miss one of the stimuli, you will hear a tone reminding you to pay close attention. Again, missing too many responses may require us to end the task early for both of you."

"In the first block of trials, one of you will be selecting noise tones of various levels of loudness for the other person to receive, and the other person will be viewing those choices on the computer screen and will acknowledge them by pressing the corresponding button. We are going to flip a coin to determine who does what first.

[coin flip]

"Okay Subject B, you're going to be selecting the noise blasts first. Subject A you will be acknowledging Subject B's selection by pressing a button when you see their choice on the screen. The two of you will switch tasks 3 times during the study."

Subject B, in this first block, as soon as you see the word "SELECT" on the computer screen, press the LOW, MEDIUM, or HIGH button on the button press box as fast as possible. These correspond to tones that will be either LOW, MEDIUM, or HIGH loudness. Be sure to respond as quickly as possible.

The LOW button on your response panel will produce a tone that is about one quarter as loud as the loudest tone Subject A could tolerate. This tone is much below their discomfort threshold and not at all unpleasant.

The MEDIUM button on your response panel will produce a tone that is about three quarters as loud as the loudest tone Subject A said they could tolerate. The MEDIUM tone loudness should be pretty loud for the other person, but it is definitely tolerable. The HIGH button on your response panel will produce a tone that is about twice as loud as the loudest tone the other person said they could tolerate. This sound is EXTREMELY LOUD AND POTENTIALLY PAINFUL for your opponent. It is twice as loud as the sound they judged very uncomfortable or unpleasant during the threshold procedure. The HIGH tone may cause headache, dizziness and buzzing in the ears that will resolve within a few hours, but will have no permanent effects. At the end of the 2nd and 4th blocks, the computer will select some of the selected tones to administer to the other person.

"Subject B, you will have only a short amount of time after you see the word "SELECT" to make your choice, so be sure to respond as quickly as possible while still being accurate. If you forget to press a button you will hear a tone reminding you to play close attention to the task. Please note that the tones will be delivered AFTER the task. So, Subject A, you will NOT hear any tones while Subject B is making their choice. Instead you will hear them in the middle of the experiment and at the end of the experiment. The computer will randomly select some of the tones your opponent set for you and deliver them to you.

"While Subject B is selecting tones for Subject A to receive, Subject A will be seeing Subject B's tone choices on their computer screen. Subject A, you will see the word LOW, MEDIUM, or HIGH on the computer screen, indicating what tone Subject B just selected for you. Your job is to acknowledge Subject B's selection by pressing the LOW, MEDIUM, or HIGH button corresponding to their choice. Press the correct button as soon as you see their choice; you will have a limited amount of time to respond. Respond as quickly as you can without making a

mistake. If you forget to press a button when you should you will hear a tone reminding you to pay close attention to the task."

"After the first block of trials, you will switch tasks and the person who selected the noise tones will now have noise tones selected for them, and vice versa. Each of the four blocks will have instructions on the screen at the beginning. This entire process will be repeated once again for blocks three and four. At the start of each new block, read the instructions on the computer screen so that you know which block you are doing. Then press the START button to advance to the next screen. Let the experimenter know if you have any questions about what you are to be doing. The experiment will not start until both of you have read and understand the instructions."

"To summarize: Read the instructions at the start of each block for instructions for that particular block. The experiment will start once both of you have finished reading the instructions. During the task respond as quickly and as accurately as possible to the messages that appear on the computer screen. Let the experimenter know if you have a question about what you are supposed to be doing and he or she will pause the experiment. While the task is going on, keep your eyes on the fixation point in the center of the screen. Try to minimize your blinking and do not move your body while the experiment is going on. Do not lean forward during the task. Sit with your back against the back of the chair and your feet under the chair. Most importantly, try to avoid blinking after the word appears on the screen. You will have several breaks during the experiment. Remember that although you will be selecting noise tones for each other, neither of you will actually hear the tones until after the 2nd and 4th blocks. Does anyone have any questions about the instructions? Okay, I'm going to go ahead and start the experiment."

APPENDIX F

HUMAN SUBJECTS REVIEW COMMITTEE APPROVAL

THE UNIVERSITY OF SOUTHERN MISSISSIPPI

Institutional Review Board

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HUMAN SUBJECTS PROTECTION REVIEW COMMITTEE
NOTICE OF COMMITTEE ACTION

The project has been reviewed by The University of Southern Mississippi Human Subjects Protection Review Committee in accordance with Federal Drug Administration regulations (21 CFR 26, 111), Department of Health and Human Services (45 CFR Part 46), and university guidelines to ensure adherence to the following criteria:

- The risks to subjects are minimized.
- The risks to subjects are reasonable in relation to the anticipated benefits.
- The selection of subjects is equitable.
- Informed consent is adequate and appropriately documented.
- Where appropriate, the research plan makes adequate provisions for monitoring the data collected to ensure the safety of the subjects.
- Where appropriate, there are adequate provisions to protect the privacy of subjects and to maintain the confidentiality of all data.
- Appropriate additional safeguards have been included to protect vulnerable subjects.
- Any unanticipated, serious, or continuing problems encountered regarding risks to subjects must be reported immediately, but not later than 10 days following the event. This should be reported to the IRB Office via the "Adverse Effect Report Form".
- If approved, the maximum period of approval is limited to twelve months. Projects that exceed this period must submit an application for renewal or continuation.

PROTOCOL NUMBER: **C29051501**
PROJECT TITLE: **An Electrophysiological Investigation of the Cognitive Processes Underlying Provoked Aggression in Humans**
PROPOSED PROJECT DATES: **06/01/09 to 08/31/2011**
PROJECT TYPE: **Previously Approved Project**
PRINCIPAL INVESTIGATORS: **Jennifer Fanning, M.A.**
COLLEGE/DIVISION: **College of Education & Psychology**
DEPARTMENT: **Psychology**
FUNDING AGENCY: **N/A**
HSPRC COMMITTEE ACTION: **Expedited Review Approval**
PERIOD OF APPROVAL: **06/22/2010 to 06/21/2011**

Lawrence A. Hosman

Lawrence A. Hosman, Ph.D.
HSPRC Chair

7-20-2010
Date

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