ABSTRACT

Psychophysiological Evidence of Psychopathy as a Normal Range Personality Construct in a University Sample of Females

Nathaniel Erik Anderson, M.A.

Mentor: Matthew S. Stanford, Ph.D.

While there has been steady progress in identifying psychophysiological traits associated with psychopathy, most of the research has been carried out using incarcerated male participants. It is not presently clear whether recent reports of attenuated P3 amplitudes are representative of psychopathy per se, or a characteristic related to secondary traits such as antisocial behavior or externalizing vulnerability. This study examined both affective startle blink modulation and P3 amplitudes in a sample of female undergraduates grouped by scores on the Psychopathic Personality Inventory. Those who demonstrated high levels of psychopathic traits lacked significant affective modulation of the startle response. Furthermore, this group showed significantly larger P3 amplitudes than those with low levels of psychopathic traits. This data supports the generalizability of deficient startle potentiation to a non-incarcerated psychopathic female population, and supports theories suggesting that primary psychopaths may show increased P3 amplitudes as an indicator of certain information processing proficiencies.

Psychophysiological Evidence of Psychopathy as a Normal Range Personality Construct in a University Sample of Females

by

Nathaniel Erik Anderson, B.A.

A Thesis

Approved by the Department of Psychology and Neuroscience

Jaime L. Diaz-Granados, Ph.D, Chairperson

Submitted to the Graduate Faculty of
Baylor University in Partial Fulfillment of the
Requirements for the Degree
of
Master of Arts

Approved by the Thesis Committee

Matthew S. Stanford, Ph.D., Chairperson

Jim H. Patton, Ph.D.

James A. Marcum, Ph.D.

Accepted by the Graduate School
August 2009

J. Larry Lyon, Ph.D., Dean

Page bearing signatures is kept on file in the Graduate School.

TABLE OF CONTENTS

	Page
LIST OF TABLES	v
LIST OF FIGURES	vi
LIST OF ABBREVIATIONS	vii
ACKNOWLEDGMENTS	viii
DEDICATION	ix
CHAPTER 1: A Review of Relevant Psychopathy Literature	1
History and Conceptual Development	3
Early Conceptualization of Psychopathy	3
Modern Conceptualization of Psychopathy	4
Primary and Secondary Psychopathy	6
Sociopathy and Psychopathy	10
Non-Criminal Psychopaths	12
Diagnostic Issues of Psychopathy	13
Psychopathy and Antisocial Personality Disorder	14
Measuring Psychopathy: PCL-R and its Factor Structure	16
Measuring Psychopathy: Non-Criminal Populations	19
Gender Differences in Psychopathy	24
Neural Correlates of Psychopathy	31
Limbic Dysfunction	32
Neuroimaging Studies	34
Lesion Studies	36

Psychophysiology and the Startle Reflex 39
Event Related Potentials and Cognitive Correlates 44
CHAPTER 2: Experimental Methodology 52
Participants 53
Self-Report Measures 53
Revised Psychopathic Personality Inventory 53
Drug Abuse Screening Test 55
Alcohol Use Disorders Identification Test 55
Sensation Seeking Scale 55
Psychophysiological Assessment 56
Startle Paradigm 56
Event Related Potentials Paradigm 58
Data Analysis 60
CHAPTER 3: Results 63
Personality Factors and Substance Use 63
Analysis of Startle Physiology 65
Analysis of Event Related Potentials 68
CHAPTER 4: Discussion 73
Self-Report Interpretations 74
Startle Physiology Interpretations 75
Event Related Potentials Interpretations 76
Conclusion 82
REFERENCES 84

LIST OF TABLES

Page

TABLE 1: *Personality Measures and Self-Report Scores by Group Division* 63

TABLE 2: *Correlations between Self-Report Measures* 64

TABLE 3: *P3 Amplitudes at Midline Electrode Sites by Group Division* 69

LIST OF FIGURES

Page

FIGURE 1 66

Grouped comparisons of standardized blink magnitudes for each affective valence reveal a reduction in blink potentiation that is driven by the primary factor elements psychopathy, a represented by Fearless Dominance scores.

FIGURE 2 70

Amplitude comparisons at midline electrode sites reveal augmented P3s for those with high psychopathic traits on the auditory oddball task.

FIGURE 3 71

Comparisons of auditory target stimuli to non-target stimuli reveal that those with high psychopathic traits show significantly greater differentiation between targets and non-targets than those with low psychopathic traits.

LIST OF ABBREVIATIONS

AUDIT: Alcohol Use Disorders Identification Test

ASPD: Antisocial Personality Disorder

BIS: Behavioral Inhibition System

BAS: Behavioral Activation System

DAST: Drug Abuse Screening Test

DSM: Diagnostic and Statistical Manual of Mental Disorders

DSM-IV-TR: The most recent edition of the DSM

EEG: Electroencephalogram

EMG: Electromyogram

ERP: Event Related Potential

IAPS: International Affective Picture System

MMPI: Minnesota Multiphasic Personality Inventory

P3: P300 ERP component of an EEG recording

PCL: Hare's Psychopathy Checklist

PCL-R: Hare's Revised Psychopathy Checklist

PPI: Psychopathic Personality Inventory

PPI-R: Revised Psychopathic Personality Inventory

SSS: Zuckerman's Sensation Seeking Scale

ACKNOWLEDGMENTS

This thesis is the product of many minds and many hands which have helped bring an idea from its nascent stages to fruition. The completion of the research reported herein would not have been possible without the involvement of many friends, to whom I owe immense gratitude. First and foremost, I must recognize the professional direction and personal encouragement of my mentor, Dr. Matthew Stanford, whose distinguished scholarship has been a source of continued motivation. Your amicable guidance and perpetual example have truly been a blessing to me. Likewise, I would like to thank two unparalleled minds, Dr. Jim Patton and Dr. James Marcum for making my academic training both challenging and rewarding. You have taught me how to value knowledge for its own sake. Furthermore, I owe thanks to Dr. Keith Young, whose ongoing investigations and professional initiative has helped to broaden the scope of my own scholarship. Finally, I owe special appreciation to those who got their hands dirty with me. Thanks to Dr. Li Wan for her technical expertise and patience with my interminable questions, and to our selfless lab assistants, Kate Cockcroft and Joanna Price, for their many hours of work. Your efforts made this research both possible and enjoyable.

DEDICATION

To my father, the smartest man I know,

and my mother, for her prayers

CHAPTER ONE

A Review of Relevant Psychopathy Literature

The empirical study of psychopathic individuals has reached a new level of popularity, which perhaps reflects a growing interest in the general community and increased attention from all forms of media. Understanding the nature of psychopathy has become an increasingly urgent necessity as data have shown that psychopaths show an increased propensity for violent attacks and aggressive behavior (Hare & Jutai, 1983; Hare & Mcpherson, 1984; Porter, Birt, & Boer, 2001), that rates of criminal and violent recidivism are much higher for psychopaths than nonpsychopathic criminals (Porter et al., 2001; Rice, Harris, & Cormier, 1992; Seto & Barbaree, 1999), and that therapeutic intervention and rehabilitation strategies have often proven to be ineffective or even counterproductive (Garrido, Esteban, & Molero, 1995; Rice et al., 1992; Seto & Barbaree, 1999). Perhaps as a result of such data, psychopaths have been popularly characterized as particularly cold and remorseless individuals, well-suited for serial murders, violent crimes, and other atrocious acts. While it is important to note that such behavior often described in fictionalized accounts are not necessarily inconsistent with our current understanding of the psychopath, this is a sorely incomplete characterization, and may ultimately be an unfair representation of the true nature of a complex disorder with several varied manifestations.

To begin, it will be particularly important to the nature of this project for one to recognize psychopathy not as categorical taxon, but as a normal-range personality disorder existing on a continuum of severity, influenced by both specific biological

triggers and significant environmental inertia. That is to say, if it is true that psychopathy results from a set of genetically predicated physiological deficits which contribute to the development of a recognizable collection of personality traits and behaviors, as has been suggested by Blair (2001, 2006), Damasio (1994), Hare (1999), Lykken (1995; 2006), and many others, it stands to reason that environmental influences would affect both the type and severity of these behavioral manifestations. Also, with respect to the degree that we identify instances of extreme pathology with personality traits corresponding to a diagnosis of psychopathy, we should expect to see with an even greater frequency milder versions of these personality traits in individuals who exhibit more subtle manifestations of the same behavioral tendencies.

Evidence of these diverse manifestations and continuum of severity has been recognized in the assessment of community samples and other non-incarcerated populations with psychopathic traits, referred to as successful, adaptive, sub-clinical, or simply non-criminal psychopaths (e.g. Lilienfeld & Andrews, 1996; Vanman, Mejia, Dawson, Schell, & Raine, 2003; Widom, 1977). Some pressing questions remain conspicuous in this realm of research, however. Namely, can the same physiological correlates which have been repeatedly implicated in the more extreme cases of psychopathy, such as those measured in incarcerated samples, be identified in individuals with sub-clinical manifestations of these personality traits? Furthermore, will the same trends and correlations which have been identified primarily in populations of male inmates generalize to a population of females? It is the purpose of this study to provide some data toward answering those questions, and contribute to the body of evidence supporting complementary theories of the nature of psychopathy.

History and Conceptual Development

With the goal in mind of proceeding with a more informed notion of the possible manifestations of psychopathy, it is necessary first to examine the origin of the concept and proceed to examine several major distinctions that have been identified within the overall construct of psychopathy. For example, it will be important to recognize the distinction between primary psychopathy and secondary psychopathy (Karpman, 1948), along with the distinction between the sociopath and the psychopath (Lykken, 1957; 1995), as well as the non-criminal psychopath (Widom, 1977). Understanding these concepts is important to the broader recognition of psychopathy as a clinical disorder with a continuum of severity that is impacted both by one's environment and phenotypic deficits in emotional processing, yielding a spectrum of individuals between the healthy, well-socialized majority and the quite literally criminally insane.

Early Conceptualizations of Psychopathy

The documented clinical interest in psychopaths has a rich history which perhaps begins with Pinel (1806), who recognized a condition he described as *madness without delirium,* and characterized these individuals in much the same way we characterize psychopaths today. These individuals were not psychotic or hysterical, but displayed aberrant and destructive behavior without apparent deficits in intelligence or other debilitating functional impairments. A similar diagnosis was described by Pritchard (1835) for a disorder he termed *moral* insanity. The early 19th century marked the infancy of our understanding of abnormal psychology in that practitioners were only beginning to understand the organic relationship between the brain and individual differences in thoughts and behavior, but these clinical descriptions are not the only

source of information we have regarding historical accounts of psychopathy. One can also look to some striking descriptions of individuals fitting this profile in both historical records and fiction.

From descriptions of infamous political leaders such as Nero to the villains of Shakespeare's plays, we have access to some remarkable examples of individuals who could meet our modern conceptualization of psychopathy. The importance of this claim is only that it is reasonable to conclude that the disorder is not merely a product of our modern culture, or a sign of the swift degradation of our moral fabric as some social critics might suggest; but rather, that individuals with a character fitting this description have quite likely been present across cultures, throughout history. One researcher in particular went to great lengths to show that even modern primitive cultures such as Alaskan Eskimos and small tribes in Africa have equivalent descriptions and native-language labels for the type of person we would call a psychopath (Murphy, 1976), so it should not even be thought of as a distinction reserved for urban civilized society. Rather, it is likely a personality variant which depends on some developmental component common to humans across all cultural variations, and we may thus being to suspect it is some inherent variability in our human physiology.

Modern Conceptualization of Psychopathy

Our modern conceptualization of the disorder relies heavily on the classic set of case studies found in the authoritative text, *The Mask of Sanity* (Cleckley, 1941). The author's major contribution therein has been to unify a set of behavioral criteria, which most closely resembles common traits among those who exhibit this complex and often destructive personality. In this work, Cleckley delineates 16 core diagnostic elements

fundamental to the character of psychopathic individuals. To be thorough, Cleckley describes the prototypical psychopath as an individual capable of exhibiting superficial charm and normal to above-normal intelligence, but presenting with a lack of anxiety and a lack of guilt, while failing to show insight into the impact of their behavior on others. Furthermore, psychopaths are generally undependable, dishonest, show no genuine sense of remorse or shame, tend to engage in antisocial behavior, and exhibit a failure to learn from punishment. They are egocentric, display a poverty of emotional reactions, often display a variety of fantastic attitudes or behaviors, they may occasionally deliver insincere threats of suicide, fail to form lasting intimate relationships, often maintain an impersonal sex life, and finally they may appear to show a general disregard for the future and subsequently fail to plan ahead accordingly.

As Cleckley's conceptualization gained recognition, a growing number of investigators began using his descriptions to operationalize psychopathy in experimental settings. Researchers began using a variety of measurement tools and components of existing personality tests such as the MMPI which identified several of these individual traits in order to develop useful experimental groups. Some informal checklists of observable traits were also being employed (Hare, 1968). Widom (1977) resourcefully recruited subjects through newspaper ads in a countercultural newspaper and incorporated cleverly-worded aspects of Cleckley's criteria into the description of ideal volunteers. There was an undeniable interest in studying this population in a laboratory setting, but progress was slow due to lack of any standard methodology. In order to hone psychopathy's descriptive utility it would be necessary to develop a reliable and valid method for operationalizing the construct, and it had to be based on Cleckley's criteria.

A major candidate for the most significant contribution to this field since Cleckley would be that of Hare and his colleagues who, having used Cleckley's criteria as a rubric, developed the Psychopathy Checklist (PCL) and its major revision, the PCL-R (Hare, 1980; 1991). This diagnostic tool is meant to identify how many of these personality traits could be identified in an individual and with what degree of severity. The PCL and PCL-R are specifically designed for use with incarcerated individuals in an institutionalized setting and consist of behavioral items to be scored based on a personal interview and an extensive file review. Analyses have shown these tests to be reliable and valid methods of operationalizing Cleckley's traits (Harpur, Hakstian, & Hare, 1988; Hare et al. 1990), and as a result of their experimental utility, research in the field of psychopathy gained a great deal of momentum. The PCL-R and its derivatives are still the most prominent tools for assessing psychopathy in use today.

Primary and Secondary Psychopathy

Given the heterogeneous nature of Cleckley's diagnostic traits common to psychopathy, many found it difficult to accept this disorder as a unitary construct. It seemed that while there remained a consistent correlation between these traits, psychopathy may be of better use as an umbrella term to describe subtler, more precise collections of these personality features, or that the term psychopathy was applied to too broad a spectrum of behaviors. Several distinctions within the broader concept of psychopathy have been theoretically conceptualized and empirically validated by various authors, (for a good review of this body of evidence, see Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003), and by far the most pervasive and widely applied of these

conceptual divisions has been the differentiation between primary and secondary psychopathy.

Karpman was one of the earliest and most outspoken proponents of such a distinction. He laid the theoretical foundation for differentiating between what he called primary, or idiopathic pscychopathy and secondary, or symptomatic psychopathy (Karpman, 1948), the most significant qualities of each being defined by the nature of behavioral characteristics being either acquired through social learning environment, or of unknown origin, which we might now presume to be genetic in nature (Viding, Blair, Moffitt, & Plomin, 2005). This theoretical framework for the study of antisocial behavior was later mirrored by Lykken (1957; 1995). According to these theorists, primary psychopaths, who are biologically predisposed to psychopathy, are characterized by apparent fearlessness and lack of shame. They show poor anticipation of punishment and have reduced physiological responses to aversive stimuli. When engaged in criminal behavior they are more calm and calculating, and show a greater degree of instrumentality, methodically manipulating and deceiving people, and when necessary acting violently to gain what they want. Secondary psychopaths, on the other hand have acquired deviant behavioral habits through poor social conditioning, for instance, by incompetent parents (Lykken, 1995), and while they too are likely perpetrators of criminal acts, some vivid functional differences from primary psychopaths are recognized. Secondary psychopaths exhibit higher levels of trait anxiety and present with more intense emotional displays. They are more impulsive by nature, and their criminal activity tends to be more reactive and characterized by rash decisions, sometimes including unplanned outbursts of violence.

After the development of the PCL, initial factor analyses of this measurement tool seemed to confirm this two-factor conceptualization of the construct as items on the measure were shown to load into two discrete factors (Harpur et al., 1988; Hare et al., 1990), and authors were generally agreeable with this structure, since it was easily interpretable in light of noted conceptual models. For instance, Factor 1 elements of the PCL describe affective and interpersonal traits including narcissism, and correlate negatively with trait anxiety, whereas Factor 2 elements of the PCL describe primarily behavioral and lifestyle characteristics and is positively correlated with both anxiety and impulsivity (Hare, 1991). Factor 1 was labeled *emotional detachment*, whereas Factor 2 was labeled *antisocial behavior* (Patrick, Bradley, & Lang, 1993).

It should be noted here that the factor elements recognized in the PCL and other similar tests are not necessarily synonymous with the primary/secondary distinction made above; in fact, recent work suggests more complex factor solutions to the PCL and related tests, which appear more appropriate. These factor analyses deserve a more thorough treatment, and will be discussed in greater detail below. For now, it is enough to say that most researchers on the topic is still dominated by this two-factor conceptualization of psychopathy, and there appears to be an interpretable relationship between this recognized primary/secondary division and the behavioral correlates of the factor structures of tests used to operationalize the disorder.

The distinction between primary and secondary psychopathy has also been validated by the recognition of behavioral differences correlated to the two factors of Hare's diagnostic tool. For instance, in the assessment of violent acts two forms of aggression have been reliably distinguished: an impulsive, reactive variety and a

premeditated, instrumental variety (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999; Stanford et al. 2003). Data has indicated that primary and secondary psychopaths can be reliably distinguished by the nature of their crimes. Primary psychopaths commit predominantly instrumental violence, carefully planning these acts as a means to an end. In contrast, secondary psychopaths' violent acts are more impulsive in nature, typically ill-conceived, unplanned outbursts (Patrick & Zempolich, 1998; Cornell et al., 1996, Williamson, Hare, & Wong, 1987). Even more recently, major differences between these two subtypes were found in a sample of incarcerated psychopaths. Prisoners with secondary psychopathic attributes were found to have higher levels of trait anxiety and comparable levels of antisocial behavior when compared to primary psychopaths, but showed fewer primary psychopathic traits overall (Skeem, Johansson, Andershed, Kerr, & Louden, 2007).

Aside from the recognizable differences in the nature of their crimes and levels of trait anxiety, these two varieties of psychopathy can also be distinguished by their differential correlations with Gray's (1990) behavioral inhibition and behavioral activation constructs, as measured through Carver and White's (1994) BIS/BAS scale. Newman, MacCoon, Vaughn, and Sadeh (2005) showed that primary psychopathy correlated negatively with behavioral inhibition, an indication that these individuals have difficulty using aversive cues to inhibit behavior; on the other hand, secondary psychopathy was positively correlated with behavioral activation, an indicator of impulsivity. As a general principle in describing motivational patterns in psychopathic individuals, it is helpful to understand that they are driven by a strong sensitivity to reward, while being extraordinarily insensitive to punishment.

Sociopathy and Psychopathy

A related distinction that has risen out of definitional conflicts in the literature is the refinement of categorization between sociopaths and psychopaths. Lykken, one of the foremost contributers to the modern conception of psychopathy, described sociopathy as a disorder with similar behavioral outcomes as psychopathy, but with different origins—namely, the result of an impoverished upbringing with incompetent parents engaging in destructive rearing practices (Lykken, 1995; 2000). In this way, sociopathy may be thought of equivalently with secondary psychopathy. A sociopath may present with similar behavioral features as a primary psychopath, but these similarities should be considered as originating from a different cause than purely physiological deficit. For this reason, Lykken sometimes refers to sociopathy as "acquired psychopathy," or "neurotic sociopathy," in contrast to what he sometimes refers to as "primary sociopathy," which we can equate with primary psychopathy (Lykken, 1957). Hare (1999) has also reflected this distinction in his own writings on psychopathy.

In differentiating between the biological underpinnings of psychopathy and the environmental influences, we must simply recognize that a large component to the personality characteristics and behavioral manifestations of the disorder is the result of poor socialization brought on by slow or absent affective learning. It is perhaps this affective learning component of development that has the most direct influence on development of psychopathic and sociopathic traits. As Lykken (2006) describes it, when a child is born, they are born with a particular genotype which falls somewhere on a normal distribution from *easy to socialize* to *difficult to socialize*. Also, they are born with parents who have varying degrees of competence in imparting socialization learning.

It is ultimately the success or failure of this socialization process which dictates the degree of antisocial tendencies.

For example, one child may simply be born with deficient mental components for social learning, and despite a relatively appropriate level of discipline and parental guidance, never builds the appropriate associations between behavior and consequence, and never fully comes to appreciate emotive responses in others. A second child may be equipped with the proper neurological tools to acquire proper socialization, but in an impoverished environment is not exposed to scenarios which would teach them these associations. A third child born with defective neurological components, and born into an impoverished environment would be at extreme risk for developing seriously destructive interpersonal habits. We can see, then, the unique but interacting contributions that both one's genes and one's environment contribute.

Having now laid the conceptual foundation, and in order to proceed unequivocally for the remainder of this paper, I will refer to psychopathy as a disorder distinct from sociopathy as defined by Lykken, and defined by two discrete factors, which correspond respectively to the primary core affective traits and only secondarily to deviant behavioral patterns and criminality. It should also be noted that it is the core affective/interpersonal component of psychopathy that is of primary interest in the progression of this study, whereas the secondary features of antisocial behavior will be discussed as a potential confound and source of variability in existing empirical reports.

Non-Criminal Psychopaths

It should be clear by this point that while criminality is a common correlate of psychopathy, it is not a fundamental or necessary component of it. All in all, there is strong evidence to conclude that antisocial behavior is only a possible symptom psychopathy, but neither necessary nor sufficient for its diagnosis. Rather it is the emotional deficiency and resulting personality traits which are designated as the primary features of psychopathy. Available evidence may even suggest that the prominence of social deviance may result from a unique milieu of influential factors. Considering the distinctions between primary and secondary psychopathic traits and between psychopathy and sociopathy, it should not be surprising that there is an identifiable population of individuals who possess the core personality features of psychopathy, but who either refrain from serious antisocial behavior, or at least have been able to avoid incarceration (Lilienfeld & Andrews, 1996). Variables such as socioeconomic status, education, or even a simple aptitude for avoiding conviction have produced the subpopulation of psychopathic individuals referred to as successful, adaptive, or non-criminal. Indeed, these individuals appear to exhibit differentiable qualities from psychopathic criminals. For instance, it has been demonstrated that non-criminal/successful psychopaths exhibit better performance on measures of executive function such as the Wisconsin Card Sorting Test (Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001).

Within Cleckley's case studies are examples of individuals who exhibited the core, fundamental attributes of psychopathy but maintained an arms length from serious legal infarctions (Cleckley, 1941). Though there has been consistent interest in studying this population for decades (e.g. Widom, 1977), it has mostly been overshadowed by the

study of criminal psychopaths. More recently, however, a trend has developed to this end, and public attention has grown as we have become increasingly aware of incidents of white collar crime and political atrocities at home and around the globe. This brand of psychopath still shows a callous demeanor, arrogant and egocentric interpersonal style, and can display a striking disregard for the impact of their actions on others as they con and manipulate others for personal gain. Some have even argued that these core attributes are adaptive and even beneficial within certain contexts (Lykken, 1995; Smith 1999). Whether it is seen as an asset or a deficit, it will be a valuable thing to learn more about how we use emotion and empathy in practical, everyday decision making. To this end, steps are currently being made to determine if the same patterns of empirical results gathered in institutional settings generalize to community samples of non-incarcerated subjects who possess similar affective traits as criminal psychopaths.

Diagnostic Issues of Psychopathy

Much of the relevant progress that has been made in the study of psychopathy has resulted from attempts to solve various controversies surrounding the conceptualization, etiology, diagnosis, and operationalization of the disorder. Professionals in the realms of clinical psychology, criminal justice, sociology, and neuroscience all have their own particular interest in and impetus for the development of a standardized, universal definition of the disorder, each with its own implications on its application. So far, this goal has been an elusive one; however, recent progress has been promising.

Psychopathy and Antisocial Personality Disorder

A primary issue that must be addressed before looking at more specific issues relevant to the disorder is the controversial absence of psychopathy as an exclusive disorder in the current version of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, DSM-IV-TR (APA, 2000). For all practical diagnostic purposes, the psychopath must currently be diagnosed under antisocial personality disorder (ASPD), or conduct disorder for those under the age of 18. Psychopathic Personality Disorder can be found in the first two versions of the manual (APA 1952; 1968), and was removed in the third version (APA, 1980), in favor of the newly included ASPD. Many authors have gone to great lengths to express their views on the inadequacy of the DSM-IV-TR designation of ASPD to account for psychopathy (Cunningham & Reidy, 1998; Hare, 1999; Hare, Hart, & Harpur, 1991). The evident criticism of this diagnostic conceptualization is that it places a primary emphasis on criminal behavior, which may only be a secondary consequence of psychopathy, and it ignores the issue of an affective disruption and the subsequent personality traits, which most experts consider as the disorder's primary characteristics (Blackburn, 2007).

To appreciate the nature of this criticism, it is important to remember that our modern concept of psychopathy, including descriptions in the earlier versions of the DSM, have relied heavily on Cleckley's characterization of the disorder in *The Mask of Sanity*. It is notable that antisocial behavior is only one element among the 16 core personality traits Cleckley outlined as common to psychopathy. A great portion of these elements describe personality features distinct from outright criminal behavior or social deviance. Antisocial behavior is a common consequence of psychopathy; however,

psychopaths do not necessarily have ASPD, and ASPD can be caused by other things besides psychopathy, as has been suggested in the case of sociopaths (Lykken, 2000), who despite the lack of a biologically-based functional deficit may simply have endured an impoverished rearing environment and/or incompetent parents.

Consider the differential diagnoses in a typical criminal population. The actual incidence of ASPD in the prison community has been measured at between 50% and 80% (reviewed by Widiger & Corbit, 1995) with most estimates being much closer to the 80% mark. The variability among these measurements is not surprising given the disappointing interrater reliability of an ASPD diagnosis of approximately 50% (reviewed by Cunningham & Reidy, 1998). However, if we test for psychopathy in a prison sample with Hare's PCL-R, we discover an occurrence of between 15% and 30% (Hare, 1991; 2003), which clearly demonstrates the necessity for a distinction. Simply put, in a prison setting, almost all psychopaths have ASPD, but relatively few individuals with ASPD will qualify as psychopaths. We can see, then, that the DSM-IV diagnosis of antisocial personality disorder may be a deficient tool in accounting for psychopathy.

The functional difference between these two populations is that the psychopath theoretically lacks a degree of affective processing which influences every decision he or she makes, rather than exhibiting a simple disregard for the law. Evidence for this will be outlined in greater detail below; but for now, I make the simple claim that beyond the conceptual differences between ASPD and psychopathy, there is also a practical utility in differentiating the two. It has been demonstrated that criminal psychopaths (diagnosed with the PCL) commit more crimes (Porter et al., 2001) that are more violent than nonpsychopaths (Hare & McPherson, 1984), and in predicting violent recidivism, PCL-R

assessed psychopaths commit nearly four times the number of post-release violent crimes as nonpsychopaths (Harris, Rice, & Cormier, 1991). Furthermore, it has been demonstrated that psychopathic offenders are much less responsive to institutional therapy and intervention programs and in some cases therapeutic strategies have only shown to increase levels of violent recidivism in psychopaths (Rice et al., 1992). Though we understand that criminal behavior is not a necessary or sufficient indicator of psychopathy, we must recognize that the psychopathic criminal offender is a far more dangerous breed of criminal than one who meets the simple criteria of ASPD.

Measuring Psychopathy: PCL-R and its factor structure

As research in the field has progressed, several tools have been developed with the specific intent of operationalizing and measuring psychopathy within specific populations. The development of such measures is always accompanied by an assessment of the test's reliability, validity, and factor structure. The factor structure of the test is an important component in the assessment of its overall validity, as it is usually compared to the theoretically conceived concept of the construct as a whole. Factor analysis can be an artful endeavor with various experts arguing for alternative numbers and arrangements of a test's factor components, but it should be kept in mind that the factor structure of a particular test is different from the conceptual structure of the disorder. It may support the test's overall validity, however, if the factor structure coincides with the construct's theoretical structure.

As mentioned above, the most common tool for defining psychopathy in scientific literature is the PCL-R, developed by Hare (1991) after the revision of an earlier version (Hare, 1980). Its development and validation as a standardized tool for

assessment of psychopathy was a remarkable catalyst in studying this disorder empirically. The tool was developed specifically for the assessment of incarcerated or forensically institutionalized subjects. It consists of an extensive interview and file-review and is scored on a 40 point scale. A score of 30 or higher is generally considered psychopathic, whereas a score of 20 or below is nonpsychopathic—21 to 29 are borderline, and may require additional interpretation on the part of the examiner. Mean PCL-R scores of prison inmates are about 23, and in forensic patients about 20, with standard deviations being approximately 8 in both populations (Hare, 2003).

A useful organizational pattern emerged through the PCL's validation process. Researchers first recognized Hare's Checklist as having a two-factor structure (Harpur et al., 1988), and this structure was duplicated in the updated version, the PCL-R (Hare et al., 1990). Furthermore, it was apparent that these two factors had distinct intercorrelations with other personality measures, and that Factor 1 was more representative of the classic clinical description of psychopathic personality (Harpur, Hare, & Hakstian, 1989). Factor 1 elements of the test corresponded to items measuring affective and interpersonal traits, whereas Factor 2 elements corresponded to behavioral and lifestyle patterns. This structure was met with great favor initially, as it lent itself to an easy generalization to distinctions made regarding primary and secondary attributes of psychopathy. While this structure is still the most widely referenced in the literature, recent analyses have been more critical of the two-factor model of the PCL-R.

Cooke and Michie (2001) convincingly argued that the PCL structure was actually served better by a three factor solution. For this three factor model, the superordinate construct of psychopathy is broken down into an interpersonal factor, an

affective factor, and a behavioral factor and notably excludes explicit references to antisocial behavior per se. We can see that in this assessment, the core theoretical components of the superordinate factor of psychopathy have not changed dramatically with this model, but rather have been recategorized in such a manner as to better account for the statistical variance between response items. The three factor model essentially divides the interpersonal/affective components of the two factor model into an interpersonal factor and an affective factor. Items accounting for behavioral components were also limited, at the exclusionary expense of antisocial traits, which Cooke and Michie argued did not fit the model neatly in their analysis.

Hare (2003) eventually retorted with data supporting a four factor solution, accounting for an interpersonal factor, an affective factor, a lifestyle factor, and a separate antisocial factor. Again, with this model, the core components of the overarching construct remain, but the author has differentiated the behavioral factor into components which emphasize separately the impulsive lifestyle and deviant, rule-breaking tendencies seen in psychopaths.

Subsequent analyses of the PCL-R, and its derivatives in different samples, have confirmed that both the three factor model and the four factor model provide a good fit. In a large sample of over 1500 sex offenders, Weaver, Meyer, Van Nort, and Tristan (2006) found that a three factor solution best fit the PCL-R data. Williams, Paulhus, and Hare (2007) favored a four factor solution in a college sample, using a self-report version of the test. Neumann, Kosson, Forth, and Hare (2006) also found that a four factor solution fit best when using a version of the PCL designed for youth. Two other studies found that both the three and four factor solutions provided a good fit under confirmatory

factor analysis (Salekin, Branhen, Zalot, Leistico, & Neumann, 2006; Vitacco, Rogers, Neumann, Harrison, & Vincent, 2005).

When assessing the utility of these models, it is necessary to keep in mind the nature of factor analysis and its purpose. It is a statistical method for data reduction, which is purely dependent upon the test being examined and the items included in that test. Factor analysis, by its very nature is one step removed from accessing true personality features, in that it assesses the correlations and variance between individual test items for a given measure, which have been devised and compiled by the test-maker. If, however, we examine and interpret these analyses with regard to theoretical frameworks built on naturalistic observation and clinical assessments, it is a very effective tool for deciphering which traits differ independently within larger superordinate constructs such as psychopathy.

For the purposes of this study, I am primarily concerned with differentiating between the two major components of psychopathy defined by the affective and behavioral distinctions, as I believe this division is the most theoretically elegant. It is also the most practical model for the purposes of this study in terms of the measures available, which will become clearer in the following discussion.

Measuring Psychopathy: Non-Criminal Populations

A limitation of Hare's Pscychopathy Checklist is that it presupposes incarceration in its diagnostic technique. Questions in the interview rely heavily on evaluation of behavior in the prison environment, and a component of the assessment relies on an extensive file review. This is a limitation, of course, because not all psychopaths are criminals; and even among those who are consistent law-breakers, not all of them have

criminal records. The growing interest in studying psychopathy and psychopathic traits in community and university samples has cultivated a need for some alternative measurement tools.

Several measures have been developed for the purpose of identifying psychopathic personality traits in community samples, and each of these has faced strict critiques regarding their validity. Methodologically, they fall into two categories: interview-based evaluations and self-report measures. Each methodology has its costs and benefits. Most notably, those scored by an interviewer are constrained by the subjective judgments of the test administrator, they usually presuppose some form of professional training, and they are generally less economical. Self-report measures, on the other hand, rely on the accuracy of the subjects' own responses, and often presuppose some level of insight into ones own behaviors and motivations. In the case of psychopaths, both the honesty of their responses and their capacity for self-evaluation is subject to criticism. The preferred measures of psychopathy in non-forensic samples have taken steps to account for these challenges, such as the inclusion of subscales designed to identify suspicious trends in responses, and have demonstrated varying degrees of success.

Two of the most prominent non-forensic, interview-based methods have been developed by Hare and his colleagues, and were designed to mimic the performance of the PCL-R. The Screening Version of the Revised Psychopathy Checklist, PCL-R: SV (Hart, Hare, & Forth, 1994), was intended as a more concise and easily administrable version of the PCL-R, which could be used to assess both forensic and non-forensic subject pools. In early tests it showed promising reliability and convergent validity with

measures of ASPD and self-reported criminal activity; however, factor analysis failed to show the desired two-factor structure (Forth, Brown, Hart, & Hare 1996). Hare also developed the Pscychopathy Scan, or just P-Scan, which was designed as a rough assessment tool for administration in non-clinical setting by those with limited knowledge of psychopathy assessment (Hare and Herve, 1999). In subsequent analyses, reliability was strong, but its validity has been called into question, as it scored very modest correlations with alternative measures of psychopathy (Elwood, Poythress, & Douglas, 2004).

Among the available self-report measures of psychopathy for use in non-forensic settings, three have stood out as some of the best validated and most frequently used tools in this style of assessment. Hare again has been involved in this realm of investigation, having developed the Self Report Psychopathy Scale and its revision, the SRP-II (Hare, Harpur, & Hemphill, 1989). The SRP-II correlated more closely with the PCL-R than the earlier, original version, the use of which was sparse. In a recent evaluation of its factor structure Williams and colleagues (2007) showed that it mimicked the most recent four-factor model of the PCL-R. However, an earlier assessment by Williams and Paulhus (2004) had shown a two-factor structure, for which the second factor did not fit any clear conceptual factor of the psychopathy construct. This led the authors to doubt its convergent validity with the PCL-R.

Another useful measure has been the Levenson Self Report of Psychopathy (Levenson, Kiehl, & Fitzpatrick, 1995), sometimes referred to as Levenson's Primary and Secondary Psychopathy Scales. The authors employed a theoretical methodology for distinguishing between primary and secondary psychopathy features. Confirmatory

factor analysis by Lynam, Whiteside, and Jones (1999) validated a two-factor structure for this measure, consistent with primary and secondary psychopathy features. Criticisms of this measure, included in Lynam and colleagues' analysis, focused on the discriminant validity between primary and secondary psychopathy measures, as they both correlated positively with alternative measures of antisocial behavior. In another recent evaluation by Brinkley, Schmitt, Smith, & Newman (2001), investigators showed poor concordance between the PCL-R and Levenson's Self Report, which led them to suggest further refinement of the instrument.

The third evaluative tool for identifying psychopathic traits based on self-report, will be discussed in greater detail here, as it is the tool to be employed in the current study. The Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996) and its recent revision (PPI-R; Lilienfeld & Widows, 2005) is a self-report, diagnostic test which was specifically designed for evaluating psychopathic traits in non-criminal settings. Quite specifically, it was originally implemented within a large university student sample, and yielded favorable results for its utility in identifying psychopathic personality traits within this population, with high reliability.

Every published study specifically examining the factor structure of the PPI has confirmed a two-factor model (e.g. Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Patrick, Edens, Poythress, Lilienfeld, & Benning, 2006; Uzieblo, Verschuere, & Crombez, 2007). The PPI shows a high level of construct validity in its correspondence with relevant constructs on the PAI (Edens, Poythress, & Watkins, 2001). It has also been shown as a valid measurement in forensic samples with a high correlation to PCL-R scores on inmates (Poythress, Edens, & Lilienfeld, 1998). On the whole, assessments of

its reliability and construct validity have made it the most attractive measurement tool of its kind currently available.

The PPI-R includes items measuring eight subscales, which load into the two overall factors. Three subfactors, social potency, stress immunity, and fearlessness, comprise the components that load into *Fearless Dominance* (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005), which corresponds to the interpersonal/affective traits of PCL-R Factor 1, and is referred to as PPI-I. Four subfactors, impulsive nonconformity, Machiavellian egocentricity, carefree nonplanfulness, and blame externalization, make up PPI-II, which is termed *Impulsive Antisociality* by Benning and colleagues (2005), or *Self Centered Impulsivity* by Lilienfeld and Widows (2005), and corresponds to the PCL-R Factor 2. Incidentally, the leftover subfactor "coldheartedness" did not favor application to either of the two main factors (Benning et al., 2003), and is rarely a feature of physiological or behavioral analyses of psychopathy.

Some familiar behavioral differences are also reported along with this factor structure. PPI-I correlates negatively with trait anxiety and positively with venturesomeness, while PPI-II correlates positively with impulsivity, substance abuse, and antisocial behavior (Benning et al. 2003; Uzieblo et al., 2007). These differences correspond to the behavioral distinctions found between primary and secondary psychopaths reported initially by Lykken (1957) and Hare (1968), and more recently by Newman and colleagues (2005) as well as Skeem and colleagues (2007).

In their initial validation of the measure, Lilienfeld and Andrews (1996) reported internal consistencies with Chronbach's alphas between .90 and .93, with most of its subscales yielding Chronbach's alphas between .80 and .90. Additionally, they reported

a test-retest reliability of r = .95 for total score, with subscales ranging between r = .82 to .94. Convergent validity was also reported which included several theoretically relevant scales such as MMPI-2 antisocial personality scale (r = .56 and .58 in two samples), the CPI socialization scale (r = -.59), and elements of the MPQ including harm avoidance (r = -.55). Furthermore, the PPI has been particularly useful because of its corroboration with the PCL-R even within samples of incarcerated individuals. Poythress and colleagues (1998), reported a correlation of r = .54 between the PCL-R and the PPI total scores, r = .54 between Factor 1 elements of each, and r = .40 between Factor 2 elements of each. In short, it seems that the gold standard for operationalizing psychopathy within a prison population has been Hare's Psychopathy Checklist, and if our goal is achieving a strong corroboration with the PCL-R within community samples, using a self-report methodology, the PPI is the best tool we have available.

Gender Differences in Psychopathy

Despite the fact that early conceptualizations of psychopathy, including Cleckley's original published case studies, included both males and females, there is a remarkable and growing controversy over the issue of gender differences within the disorder. This issue must be addressed here as this study will be carried out on a subject pool comprised of female undergraduates. In examining gender differences in psychopathy researchers must be careful to define what exactly they are looking for differences between: differential base rates of the overall construct between genders, some defined behavioral expression of the construct between genders, or the operational definition of the construct itself between genders. When not handled carefully, these issues can be easily confused and the logic behind some studies appears circular (i.e. the

construct of psychopathy is different between genders because females who meet a given operational definition of psychopathy have different traits than males). It is meaningless to argue that the core features of psychopathy are different in females, as that would be recharacterizing the disorder which is defined by that very set of core features. It is more productive to argue that, given how psychopathy is defined, there appear to be differential base rates in males and females, or that each gender displays a different set of dominant behaviors secondary to those core traits.

Relatively few studies have specifically examined differences in the construct and/or behavioral expression of psychopathy in men and women, though two particularly good reviews of the available data on the topic include Cale and Lilienfeld (2002) and Forouzan and Cooke (2005). A major factor contributing to the debate has been an overrepresentation of males in the empirical research, which leads to questions regarding the generalizability of those findings to a female population. Most of the empirical research related to psychopathy has been carried out in exclusively male populations. This has been true when addressing the construct validity of the measurement tools commonly used in studying psychopathy (e.g. Edens et al., 2001; Hart, Forth, & Hare, 1991; Hart & Hare 1989; Poythress et al., 1998), when assessing the factor structure of those tests (e.g. Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Cooke & Michie, 2001; Hare et al., 1990; Harpur et al., 1988; Weaver et al., 2006) when investigating behavioral implications of the disorder (e.g. Porter et al., 2001; Rice et al., 1992; Seto & Barbaree, 1999; Williamson et al., 1987), and also when examining its physiological correlates (e.g. Hare, 1968; Kiehl et al., 2001; Patrick et al., 1993; Patrick, Cuthbert, & Lang, 1994; Williamson, Harpur, & Hare, 1991). A few studies have targeted females

exclusively (e.g. Kennealy, Hicks, & Patrick, 2007; Salekin, Rogers, & Sewell, 1997; Salekin, Rogers, Ustad, & Sewell, 1998; Sutton, Vitale, & Newman, 2002); however, these reports are primarily focused on assessment issues, such as the higher base rates of psychopathy and ASPD in males, construct validity of measurement tools, and potentially distinct behavioral manifestations of psychopathy between genders.

Some researchers may have used male subjects primarily to avoid gender as a confounding variable, but an equally attractive reason to use exclusively male subjects is that when operationalized by any number of psychopathy measures, males have been consistently found more likely to score higher on these measures. For example, Hare (1991) has reported an incidence of psychopathy between 15% and 30% in incarcerated males, while Salekin and colleagues (1997) reported an incidence of 15% in a large sample of incarcerated females. Whether this means that psychopathy is more prevalent in male populations, or whether we need to consider different sets of indicators of psychopathy for males and females has yet to be determined.

Another trend to consider is the differential distributions of psychopathy scores between genders on various measures (PCL, PPI etc.). Not only is it true that a greater percentage of males will typically elevate on their respective psychopathy scores, but generally speaking, the average male scores higher on measures of psychopathy than the average female. Both the PCL and the PPI have different sets of normalized scores for males and females (Hare, 2003; Lilienfeld & Widows, 2005), though these published norms show comparable ranges of distributions between genders.

Based on data such as these, it is generally accepted that females show both lower base rates of psychopathy, and lower overall scores on psychopathy measures. Some

authors have also noted recognizable differences in the secondary antisocial behavioral characteristics of psychopathic females. For example, while incarcerated psychopathic males' behavior is characterized by conning, cheating, and violent acts, incarcerated psychopathic females' behavior is more characterized by promiscuity, flirtatious manipulation, and self-harm (Forouzan & Cooke, 2005). Similar conclusions can be drawn from a study conducted on a non-incarcerated sample, which showed that among those who met the criteria for ASPD, males were significantly more likely to commit crimes, whereas females were more likely to suffer relationship problems and exhibit lying (Mulder, Wells, Joyce, & Bushnell, 1994).

If we apply this knowledge to what we know about the two-factors associated with psychopathy, it is reasonable to conclude that females who exhibit the core affective deficits definitive of primary psychopathy likely exhibit a different behavioral expressions secondary to these traits than what we typically see in men. That is to say, psychopathic males are more likely to engage in behavior typical of criminal offenders, which may explain the higher base rates of psychopathy in male prison samples compared to female prison samples. Additional support for this conclusion can be drawn from a study in which investigators were unable to find significant differences on PPI scores between an incarcerated sample of females and a non-incarcerated undergraduate sample of females (Chapman, Gremore, & Farmer, 2003). We can interpret this in support of a distinction between the constructs of psychopathy and antisocial personality disorder; that is, a subject pool of female prisoners would not necessarily include psychopathic individuals, especially if it were true that psychopathic females are less likely than psychopathic males to meet DSM-IV-TR criteria for ASPD.

Many experts in fact have supported the idea that criteria for ASPD are gender biased, showing data that suggest men are more than twice as likely as women to receive an ASPD diagnosis, amongst substance abusers (Hesselbrock, Meyer, & Keener, 1985; Flyn, Craddock, Luckey, Hubbard, & Dunteman, 1996). In an undergraduate sample used for assessment of psychopathic traits in non-criminals, Forth and colleagues (1996) found that approximately 21% of males met criteria for ASPD, whereas less than 2% of females met these criteria. These differences could arise either because females are indeed less likely to suffer from ASPD, or because testing procedures and criteria for diagnosis are themselves gender-biased.

If this bias is indeed true, we must then ask how better to account for the secondary characteristics of psychopathy in females. It has been suggested that while psychopathy is likely to be expressed as ASPD in males, it is more likely to be expressed as histrionic personality disorder, or borderline personality disorder in females (Hamburger, Lilienfeld, & Hogben, 1996). It seems then, that a reliance on ASPD as the foremost indicator of psychopathy may not only be insufficient to account for its primary affective deficits, but it may also be gender biased, ignoring other DSM, axis II cluster B personality disorders as potential developmental consequences of psychopathy. If our primary concern is with these behavioral consequences, ASPD, histrionic personality disorder, borderline personality disorder, and narcissistic personality disorder all may be considered robust indicators of the core affective deficit. Blackburn (2007) supports this conclusion with data showing that the core personality traits of psychopathy are actually closer to narcissistic personality disorder and histrionic personality disorder. Ultimately, as noted above, the current study is more concerned with the primary affective traits

which define the psychopathy construct. The only present consequence of these trends would be a potential difficulty in identifying useful experimental groups, if the primary facets of psychopathy were an insufficient qualifier amongst females, which is not expected to be a significant obstacle with the use of the PPI-R.

It seems clear that the construct of psychopathy was originally characterized by Cleckley as a core set of personality traits, independent of gender. It is also apparent that this was done without any behavioral prerequisites, such as ASPD. Insofar as we examine psychopathy by means of these core personality traits (primary factors) without as much regard for behavioral expressions of criminal behavior (secondary factors), our evaluations should be sound. We run into difficulty, however, when the tool used to operationalize psychopathy relies too heavily on behavioral characteristics (e.g. ASPD diagnosis) or presumes certain behavioral indicators by implication of incarceration (e.g. PCL-R evaluation). Being aware of these potential obstacles, it remains clear that the PPI is a good tool for the purposes of this study in that it virtually ignores behavioral consequences and attempts instead to identify the core affective deficiencies and personality traits, which characterize the disorder.

Summary

We have seen that direct behavioral patterns may be too widely varied and too developmentally dependent to be considered reliable indicators of the core emotional deficits intrinsic to the disorder of psychopathy. Still, measures such as the PCL-R and the PPI-R reliably distinguish between groups of individuals with recognizable personality differences, which significantly correlate with distinct behavioral patterns within certain cohorts such as criminal offenders. These behavioral differences are part

of what make the study of psychopathy worthwhile and interesting. So, when we take on the task of examining psychopathy in a non-criminal setting, we are left with at least two questions. What consequential indicators are available to determine the legitimacy of group differences in psychopathy scores among non-offenders? Also, what value is there in determining these differences in a non-incarcerated sample?

The first question is really the impetus behind this investigation. Apart from behavioral indicators, it is still possible to reliably differentiate this population based on alternative measures, which speak to the organic nature of this disorder, namely physiological differences. These differences are some of the most reliable across all psychopathic subpopulations. A good example of relying on differentiable physiological characteristics to guide a theoretically based model of psychopathy is given by Vanman and colleagues (2003), who were able to reliably distinguish between the two factors of psychopathy based on the psychophysiological characteristics of their sample. Specifically, these investigators used the modification of startle blink magnitude with affective stimuli, a technique which will be discussed extensively below. If subjects are grouped by means of variability in their startle blink modulation, testing items fell into two distinct categories, clearly representative of the primary and secondary factor elements of psychopathy.

The second question from above simply asks for some justification of the importance of this line of investigation, and this justification comes in two parts. Clearly, we have seen that a population of non-criminal psychopaths does not necessarily suggest a population of law-abiding psychopaths; it only identifies a component in their behavioral development which has allowed them to avoid incarceration. We have also

seen that regardless of how adaptive the psychopathic individual is, the behavioral expression of these traits may put them at odds with friends and family, romantic partners, business partners and employers, if not the law per se. The often destructive nature of this pathology, even outside a criminal sample, legitimizes the value of studying this population. The use of a non-incarcerated sample may even be a more effective means of studying the most fundamental components of this disorder, as it is virtually impossible to control for ASPD in a prison sample. Apart from all this, the study of psychopathic traits in a university sample adds credence to the view of psychopathology as existing on a continuum of individual differences. If evidence can be gained of similar physiological distinctions among those with psychopathic traits but without significant antisocial characteristics, we can more confidently describe psychopathy in terms those biological antecedents, when assessing their varied consequences. Understanding these biological signatures is important to the development of the hypotheses of this project, and will now be discussed in greater detail.

Neural Correlates

Almost all forms of mental illness can be described, to some degree, in biological terms; that is, one can describe the physiological correlates that correspond to mental events. This does not mean that the root cause of every mental illness is biological in nature, only that there is a physical manifestation of the disorder that can be measured at some level. There is strong evidence to suggest, however, that psychopathy is a mental illness of the particular sort that has a primarily biological origin. That is to say, it can be described as a personality disorder resulting from functional abnormalities in the brain, presumably with a genetic origin (Blair, 2006; Viding, Blair, Moffitt, & Plomin, 2005).

These functional abnormalities which result can be demonstrated in several formats to be described below, and their sufficiency and primacy in producing psychopathic symptoms is quite convincing.

Limbic Dysfunction

A particular set of the primary diagnostic criteria for psychopathy may stick out as a beacon to any discriminating neuroscientist. Namely, the characteristic lack of anxiety and apparent fearlessness, the lack of guilt, lack of remorse or shame, failure to learn from punishment, and poverty of emotional reactions all raise flags for potential limbic dysfunction. More specifically, one could make an educated guess and implicate the amygdala, as we are aware of its involvement in emotional processing and affective learning, response to punishment, conditioned fear, and initiation of emotional states (Davis, 1997; Kapp, Whalen, Supple, & Pascoe, 1992; LeDoux, 1992; LeDoux, Iwata, Cicchetti, & Reis, 1988). The prefrontal cortex is another obvious choice because of its demonstrable role in behavioral inhibition and moral decision-making (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Bechara, Damasio, Tranel, & Damasio, 1997). In fact, there is a growing body of research which suggests these speculations are exactly true; that is, the primary physiological deficits found in psychopaths are found in the amygdala and prefrontal cortex.

The evidence for this can be organized into three categories: neuroimaging studies, neuropsychology and related lesion studies, and various forms of psychophysiological research. A great deal of research has been completed, which elucidates some of the underlying physiological deficits apparent in the psychopathic population. Unfortunately, the majority of this research has been done on criminal

psychopaths in an institutionalized setting, which leaves open for interpretation the generalizability of the results to a non-incarcerated sample. Of course it is expected that the deficits apparent in a population with a more severe manifestation of the disorder will be more difficult to identify among those closer to the middle of the distribution. But if we are to take seriously the notion that primary psychopathy is a biologically-based disorder, existing on a continuum of severity, we must still expect appreciable variability on psychophysiological measures in a non-incarcerated sample exhibiting discernable personality differences.

The other significant issue at hand when considering the biological basis of psychopathy is in differentiating the disorder from its relatives. Above, some effort has been made to differentiate between primary psychopathy and the more general antisocial personality disorder. However, it is clear that a great deal more research exists, which has focused on identifying correlates of ASPD, and other more specific behavioral manifestations such as impulsivity, physical violence, and aggressive behavior, which may or may not be the product of a psychopathic genotype. While these peripheral concepts are relevant, I want to be clear about my intentions to focus on a strictly-defined construct of psychopathy proper, as defined by validated measurement tools based on Cleckley's concept, such as the PCL-R and PPI-R, and when possible I prefer to focus more specifically on elements of factor-one, primary psychopathy, so reports regarding related behavioral constructs have been largely excluded. As a related concern, studies ostensibly specific to psychopathy have often been carried out using incarcerated subjects using the PCL-R, but have made no explicit attempt to control for ASPD. This is a

disconcerting methodological issue with serious interpretive consequences which will be discussed in greater detail below.

Neuroimaging Studies

A few researchers have made recent strides toward identifying specific areas of the brain that are apparently impacted in psychopaths using both structural and functional imaging techniques. It has been apparent that cognitive tasks involving affective appraisals and aversive cues elicit recognizable differences, and a review of the literature makes it clear that the two anatomical regions most often implicated in the disorder are the amygdala and areas of the prefrontal cortex. Still, it is important to interpret these findings with respect to how psychopathy is operationally defined in the study, what kind of sample is being studied, and if any attempts have been made to control for differences between psychopathy and ASPD.

For instance, Yang, Raine, Narr, Lencz, and Toga (2006) recently reported reduced structural volumes of the amygdala in psychopaths versus controls, and showed a significant negative correlation between amygdala size and the severity of psychopathic symptoms. Rilling and colleagues (2007) demonstrated that psychopaths have a reduced level of amygdala activity while engaging in a prisoner's-dilemma task, which requires decisions regarding social cooperation and an evaluation of one's own well being over that of another's. Gordon, Baird, and End (2004) implicated the amygdala and prefrontal cortex as showing reduced activity in psychopaths during a recognition task involving facial affect. Kiehl and colleagues have reported that criminal psychopaths show reduced activity in limbic regions including the amygdala and anterior cingulate cortex while evaluating emotional stimuli and while performing an affective memory task

(Kiehl, 2000; Kiehl et al., 2001). Likewise, psychopaths have shown reduced amygdala and medial prefrontal cortex activity during emotional decision-making tasks (Schwerdtner, Sommer, Weber, & Müller, 2004), and reduced activity in the amygdala and orbitofrontal cortex during aversive stimulus learning (Birbaumer et al., 2005).

In these studies which have specifically identified individuals with some defined level of psychopathy (such as PCL-R scores) as the target population, it is clear that amygdala function is the most consistently identifiable difference, followed by areas of the prefrontal cortex—specifically the orbitofrontal and medial prefrontal cortex. Again, most of these studies, and others not mentioned here, were carried out in prison populations and had not made explicit attempts to control for ASPD. From the information available about amygdala function and its connections with the prefrontal cortex, it is reasonable to conclude that the affective deficits primary to psychopathy are more closely related to amygdala functionality, whereas behavioral symptoms of ASPD are likely closely linked to dysfunction in prefrontal cortical regions (Blaire, 2004), which has a mitigating influence on the amygdala.

A larger number of studies exist, which focus specifically on antisocial personality disorder, and here we see a trend congruent with this hypothesis. Reviews of the available functional imaging and related physiological studies on violent, aggressive subjects (e.g. Davidson, Putnam, & Larson, 2000; Raine & Buchsbaum, 1996) stress the importance of prefrontal cortex in behavioral regulation, noting the apparent inhibitory effects of orbitofrontal cortex on the amygdala. It seems, then, that if any ambiguity exists in the literature regarding the respective contributions of these regions, it may only be a problem related to the variable occurrence of ASPD within a population of

psychopaths. Indeed, when accounting for the differences between primary and secondary symptoms of psychopathy, it has been demonstrated that executive dysfunction is related to secondary psychopathy, but not primary (Ross, Benning, & Adams, 2007). Psychopaths who have developed more severe antisocial tendencies should theoretically show reduced prefrontal behavioral inhibition, whereas psychopaths who exhibit primarily a functional impairment in emotional responsivity may present with behavior that can be accounted for sufficiently by deficient amygdala activity.

Lesion Studies

Another valuable source of information on the neuroanatomical identification of functional deficits of psychopaths comes from lesion studies. Consider, for instance, individuals with specific damage to areas of the prefrontal cortex, the part of our brain responsible for most executive functioning, planning ahead, and behavioral inhibition (for a review see Duncan & Owen, 2000). It is interesting to consider the similarities in behavior we observe in patients with frontal lobe dysfunction and in antisocial psychopaths, which include lack of impulse control, poor planning and judgment, increased levels of aggression, difficulty abiding by social norms, and temperaments ranging from mildly belligerent to outright malevolent. Brower and Price (2001) provide a valuable review of associations of focal brain injuries and subsequent behavioral consequences, specifically violent and criminal tendencies.

Several case studies have been reported indicating such personality changes following traumatic brain injury to certain areas of the prefrontal cortex. Almost invariably, the case studies involve a relatively successful, well-behaved, friendly individual suffering the brain injury and recovering, but with severe changes in character.

It seems as if these individuals take on behavioral tendencies following the injury that are not characteristic of psychopathy per se, but more characteristic of ASPD. Intelligence tests and most other neuropsychological examinations on these patients produce normal to superior marks; however, the individual takes on a set of maladaptive social behaviors and attitudes. Often they are described as raucous, sexually promiscuous, and offensive in public; they have difficulties remaining employed, let alone maintaining their long-standing relationships with friends, spouses, and other family members. These are sad stories, to be sure, but ones we can learn a great deal from.

The most historically notable of these case studies is that of Phineas Gage. This account, originally published in 1868 (reproduced as Harlow, 1993), described the personality transformation of the railroad worker who survived after having an iron rod pass through his head, damaging the prefrontal cortex. The skull from the now deceased patient is a bona fide historical relic, and has been passed around and studied extensively by interested scientists. Special attention has been paid to the specific brain regions likely damaged, given the entrance and exit wounds and skull geometry, and the ventromedial and orbitofrontal cortex appear to be the principal areas of injury (Damasio, 1994; Damasio, Grabowski, Frank, Galaburda, & Damasio, 2005). Another more recent example of a focal brain injury resulting in ASPD is described by Meyers, Berman, Scheibel, and Hayman (1992), with the patient having suffered damage to the same area as a result of the surgical resection of a tumor. A third example is given by Cato, Delis, Abildskov, and Bigler (2004), showing similar behavioral results. The evidence from these case studies is highly suggestive that the lack of behavioral inhibition characteristic of antisocial psychopaths could be related to these prefrontal anatomical elements. To be

precise, by dividing the prefrontal cortex into its functionally significant parts: the dorsolateral, orbital (ventral), and medial components, a careful examination of these case studies indicates the areas responsible for these personality changes are usually the orbital and medial divisions, but not the dorsolateral.

As suggested earlier, the archetypical characteristics resulting from such an injury might more appropriately fit under antisocial personality disorder, or specifically to the factor-two elements of psychopathy. Still, it is common in the literature to see both the terms *acquired psychopathy* and *acquired antisocial personality disorder* when referring to the personality changes and neurocognitive impairments following frontal lobe injury. The acquired form refers to personality characteristics acquired after traumatic brain injury as opposed to the developmental form, which would presumably arise from the phenotypic expression of some genetic recipe. The similarities and differences between the two are often compared in order to learn more about the compromised neural systems in the developmental form (e.g. Mitchell, Anvy, & Blair, 2006). Briefly stated, we can be sure that the orbital and medial prefrontal cortex is involved in limbic system control and behavioral inhibition, and it is likely that dysfunction in this anatomical region would contribute to the tendencies toward antisocial behaviors we see in psychopaths.

It is worth noting that lesions of the amygdala have various and unique adverse effects; however, specific damage to this area deep in the brain of humans is indeed rare and case studies are few. In any case, such an injury should not be expected to produce behavior consistent with primary psychopathy, because many of these traits are of a more developmental nature, resulting from deficient amygdala response throughout stages of moral and social development. Interpersonal style takes time to develop so we could not

expect to see an acute onset of primary psychopathic traits following amygdala lesions. We can however rely on direct indicators of physiological dysfunction of the sort that we recognize in psychopaths with advanced primary symptoms, and this indeed has been reported following specific damage to the amygdala (Angrilli et al., 1996); namely, this report indicated a reduction in startle potentiation, one of the most replicated demonstrations of physiological dysfunction in primary psychopaths.

Psychophysiology and the Startle Reflex

If our goal is to search for observable indicators of amygdala dysfunction, we are fortunate to engage in these studies at a time when much of the necessary ground work has been completed and replicated many times over. It is actually quite simple to exploit the reduced responsivity of the amygdala which has been apparent in functional imaging studies on psychopathic populations; in fact, the functional imaging data has been much more recent and really just confirmed what we already expected from the results of studies which manipulate emotional and motivational responsivity. One of the most useful experimental methods for measuring this is startle reflex modulation (Bradley, Cuthbert, & Lang, 1999).

This type of manipulation can be done by delivering stimuli, which would ordinarily produce a rapid increase in physiological arousal due to their threatening or aversive nature, and which in turn allow the recording of subsequent physiological changes stereotypical of a stress response. A common paradigm for investigating this phenomenon involves the delivery of a short, sudden burst of white noise loud enough to activate a reflexive startle response. In mammals, this acoustic insult activates a "threat circuit" which elicits a set of species-specific stereotypical behaviors. In humans this

produces blinking of the eyes, tightening of most skeletal muscles, and a sympathetic autonomic response. One can measure the magnitude of the startle response by recording an EMG at the *orbicularis oculi* muscle, responsible for the eye blink. The startle response is a reflexive, hard-wired, autonomically influenced behavior, common among many animal species. The physiology of the circuit responsible for the startle reflex has been described extensively (Davis, 1989; Gregg & Siegel, 2001; LeDoux et al., 1988), and is inextricably linked with a primary response from the amygdala. The amygdala in turn relays messages to the locus coeruleus for norepinephrine release, increasing arousal and vigilance, and sends projections to the hypothalamus for corticotropin response and peripheral sympathetic responses.

The amygdala also shares important connections with the prefrontal cortex, which sustains a kind of inhibitory control on the entire circuit. Like most reflexes, acoustic startle can be inhibited or facilitated by conscious effort or by unconscious influences such as distraction, appetitive motivation, and emotional priming. In a shocking demonstration of the importance of cortical function in the regulation of this circuit, it has been shown that if one removes the outer cortex of a cat (or dog, or another mammal) thus removing this inhibitory control over the threat circuit, the animal exhibits what is called sham rage, described by Bard (1928) as a vicious and intense expression of rage that is elicited by even minor disturbances or annoyances. In humans, this lack of inhibitory control might be more akin to the impulsive expression of reactive aggression and secondary antisocial traits (Patrick & Zempolich, 1998).

Experimentally, if we are inclined to expose the functionality of the amygdala, we can measure the degree to which the startle response can be modulated by relevant cues,

to which the amygdala would have an evaluative response. One would expect psychopaths to show deficits in such modulation, as it has been demonstrated that they suffer deficits in autonomic responsivity to aversive stimuli (Hare & Quinn, 1971). Modulation of the startle reflex can be done in a number of ways; one of the most common is having the subject view emotionally arousing pictures and delivering the startling noise at unexpected intervals during picture-viewing. In normal subjects, viewing negatively arousing or fear-inducing pictures such as disembodied limbs, burn-victims, or personally threatening scenes produces a potentiated startle response compared to that while viewing neutral pictures of household objects or abstract geometric designs. The viewing of positively arousing photographs such as erotic images or nature scenes will reduce the magnitude of the startle response (Lang, Bradley, & Cuthbert, 1990).

There is, however, a recognizable peculiarity in the physiology of psychopaths in that they do not reliably show a potentiated startle response to negative affective stimuli (Levenston, Patrick, Bradley, & Lang, 2000; Patrick et al., 1993; Patrick, et al., 1994). Some relevant studies have even suggest that the psychopaths' responses to standardized aversive stimuli are more similar to their response to the positive stimuli; that is, they show a reduction in blink magnitude for both positive and negative stimuli compared to neutral stimuli (Patrick et al., 1993). Authors interpret these results with special recognition of the functional role the amygdala has on these physiological responses and how they relate to our fear response.

This lack of fear-potentiated startle has turned out to be one of the most replicated psychophysiological findings in psychopaths. These findings have been replicated in

various populations of identifiable psychopaths, both incarcerated (Patrick, 1994; Patrick, et al., 1993) and non-incarcerated (Benning, Patrick, & Iacono 2005; Justus & Finn, 2007; Vanman et al., 2003), and in at least one sample of incarcerated females (Sutton et al., 2002). These findings fit with an even larger body of research on psychopathic populations which has shown abnormalities in general autonomic responses to fear-inducing images, specifically apparent in cardiac response, galvanic skin response, and facial muscle EMGs (Patrick, et al., 1994). Essentially, these measured responses do not show reliable autonomic influence resulting from aversive stimuli. Interestingly, these authors also found that these abnormalities are limited to psychopaths who exhibit the affective, factor 1 symptoms. This finding agrees with other similar results indicating that the biological underpinnings and psychophysiological cues characteristic of psychopathy appear to be more indicative of the primary, factor-one elements of the disorder (Hare, 1968; Skeem, et al., 2007). These abnormalities in autonomic response have also been shown to exist in psychopaths simply during anticipation of aversive stimuli (Hare, 1982; Hare, Frazelle, & Cox, 1978). Finally, in returning to our discussion of focal brain damage above, it has been shown that specific lesions of the amygdala block the effect of fear-potentiated startle in rats (Kim & Davis, 1993). This finding was later replicated in a human case with a rare, localized amygdala lesion (Angrilli et al., 1996).

Startle modulation, then, seems to be a particularly useful tool for identifying those with functional deficits in the amygdala, and is therefore a good means of determining whether or not subcategories of psychopaths share the same physiological abnormalities. It seems likely from what has been reviewed so far that individuals who exhibit the core emotional features of psychopathy will share these physiological

characteristics regardless of predilection for antisocial behavior, which would more accurately be described as a secondary feature of psychopathy related to prefrontal cortex function. Expecting similar recordable psychophysiological measurements from an identified non-criminal population with psychopathic traits as identified by a reliable reporting measure such as the PPI-R would be consistent with the theoretical conceptualization of the disorder.

Three particularly noteworthy studies have shown promising support for the claims above. Vanman and colleagues (2003) showed that psychopaths gathered from a non-incarcerated, community sample showed similar characteristics of startle modulation as have been shown in incarcerated samples, as long as they scored high on factor 1 elements of psychopathy. In this study; however, they used a version of Hare's psychopathy checklist to identify psychopaths in the community, which is unconventional but has occasionally been done under the suggestion by Kosson, Steuerwald, Forth, and Kirkhart (1997) that it maintains its validity in this sample. Benning, et al. (2005) found similar results in a study of male twins, who had been identified by means of their scores on theoretically relevant items from the Multidimensional Personality Questionnaire (MPQ). Again, here, lack of startle potentiation under negative affective primes was related to scores on the interpersonal/affective elements of psychopathy. Justus & Finn (2007) examined this effect and investigated the additional effects of gender and startle-probe-latency on the phenomenon, while at the same time being the only study to use a version of the PPI to group subjects for analysis of the startle data. When these non-incarcerated subjects were scored as a single group, with data collapsed over startle-probe-latencies, no significant

effects were apparent; however, significant effects were apparent with more precise grouping methods. For early startle probes (2 seconds post picture onset), only males scoring high on factor 1 elements showed the characteristic lack of startle potentiation, whereas high and low scoring female subjects showed no significant difference. For late probes (4.5 seconds post picture onset), significant differences were apparent without a significant interaction with gender. Both males and females with high total scores, and high factor 1 scores showed reduced startle potentiation, whereas factor 2 scores did not predict a significant effect.

Interpretation of these data support the notion that measurable psychophysiological features of psychopathy are identifiable in a population of well-socialized individuals, who have remained successfully adaptive in the community or at least have refrained from significant antisocial, criminal tendencies. This suggests that a community sample of non-criminals may be an even better source for studying the primary aspects of psychopathy than an institutionalized sample. Within this sample we can see a clear differentiation between the functional elements at work in primary psychopaths without perhaps the confounding variable of severe antisocial behavior, which might be presumed in incarcerated samples. Clearly though, more replication is needed, especially to interpret potential differences that may exist in a female population, as these results have remained less consistent, and the relative paucity of data which include females has led to some rather overzealous interpretations.

Event Related Potentials and Cognitive Correlates

Further physiological support for a functional deficit in emotional processing in a psychopathic population can be found by examining massed cellular activity in real time

during cognitive tasks. This can be done by recording an electroencephalogram (EEG) and averaging brain wave components elicited by repeated trials of simple, categorical cognitive tasks which are referred to as event-related potentials (ERPs). ERPs have been used for decades both diagnostically and experimentally in diverse populations, and their value to the study of psychopaths is considerable, though only in its infancy. It should be noted that much replication and validation of experimental methods still needs to take place in this realm, which is why I have included ERPs in the current study.

To review the work that has been done using ERPs to study psychopathy per se, one only needs to look at a handful of studies, which have used a variety of tasks to examine cortical activity in rather non-specific ways. The most notable interpretive assessments of the data seem to suggest some form of differentiable semantic processing strategies among psychopaths when they are faced with semantic tasks or categorization tasks featuring affective lexical stimuli (Howard & McCullagh, 2007; Kiehl, Hare, McDonald, & Brink, 1999; Williamson et al., 1991; Jutai, Hare, & Connolly, 1987), though the exact nature of these differences will require more data to be accurately interpreted. These interpretations are typically the result of rather non-specific ERP component evaluation, and expressed as reduced component differentiation between target and non-target stimuli in psychopaths as compared to nonpsychopaths. All of these studies were conducted on an institutionalized criminal sample. The data is so sparse and varied with respect to design and interpretation, that it is primarily clear that more work needs to be done in this arena before any reliable trends will appear.

Another avenue of ERP investigation that has begun to emerge in the psychopathy literature is in examining a more specific ERP component—one of the most robust

identifiable components of task-relevant EEG waves, the P300 (or simply P3). In ERP studies, the P3 is a positive fluctuation in the EEG wave at around 300 milliseconds post-target-stimulus, and has generally been interpreted as an indication of cognitive resources being allocated to attentional processes, working memory, and orienting/categorization of a target stimulus (for reviews see Polich, 2007; Soltani & Knight, 2000). The most significant volume of recent research in applying this ERP component directly to the study of psychopathy has been carried out by Kiehl and his colleagues, and still in only a handful of studies. One thing is clear from a thorough investigation into P3s and psychopathy: data in this line of research is both sparse and contradictory.

Keihl and colleagues have reported significantly smaller P3s in psychopaths sampled from criminal institutions (Kiehl, Bates, Laurens, Hare, & Liddle, 2006; Kiehl, Hare, Liddle, & McDonald, 1999; Kiehl, Hare, McDonald, & Brink, 1999; Kiehl, Smith, Hare, & Liddle, 2000). Jutai et al. (1987) found no significant differences in the P3s of criminal psychopath. Raine & Venables (1987; 1988) have found larger P3s in criminal psychopaths. A much earlier study by Syndulko and colleagues (1975) found no significant differences in P3 amplitude between controls and a group of sociopaths as defined by psychiatric interview and MMPI scale scores. Upon first examination it is not entirely clear what differences in P3 characteristics one might expect from a population of psychopaths. One possibility, however, is that these investigators are encountering a similar problem to what we have seen with other psychophysiological correlates of psychopathy, namely the differentiable trends influenced by the heterogeneous nature of psychopathy. A significant confounding variable may be the presence of characteristics such as antisocial behavior and impulsivity, as left uncontrolled in these studies using

incarcerated subjects. It may be true that the P3 is indicative of cognitive process, which is differentially affected by some latent variable which also influences the course of development psychopathy takes. If we look again at behavioral indicators as a means of distinguishing between subclasses of psychopaths, this seems like a likely explanation for the variability evident in these studies.

There is no ambiguity in the available literature about psychophysiological correlates of antisocial behavior. A large body of evidence suggests that criminality and violent behavior are strongly associated with smaller P3 amplitudes (e.g. Bauer, O'Connor, & Hesselbrock, 1994; Bernat, Hall, Steffen, & Patrick, 2007; Costa et al., 2000; O'Connor, Bauer, Tasman, & Hesselbrock, 1994). However, these studies are almost exclusively based on a DSM diagnosis of ASPD, which we know is not a sufficient designator of psychopathy, and may require further qualification to distinguish between behaviors typical of primary and secondary psychopaths. A few researchers, though, have been careful to differentiate between these characteristics and those which we now know are indicative of psychopathy's primary features. For instance, P3s in violent criminals are indeed found to have reduced amplitudes in subjects whose violence can be characterized as impulsive aggression, but this reduction in amplitude has been conspicuously absent in those exhibiting primarily premeditated forms of aggression (e.g. Barratt, Stanford, Kent, & Felthouse, 1997; Houston, Stanford, Villemarette-Pittman, Conklin, & Helfritz, 2003; Stanford, Houston, Villemarette-Pittman, & Greve, 2003), suggesting that secondary psychopaths may show reduced P3 amplitude, while primary psychopaths may not.

Another behavioral indicator of psychopathy has to do with appetitive and inhibitory mechanisms in the brain (Gray, 1990), which can be described in terms of the Carver and White (1994) BIS/BAS scale. It has been hypothesized that while psychopaths show poor passive avoidance learning, primary psychopaths may actually have higher than normal appetitive reward systems, which make them particularly bad at avoiding aversive stimuli under conditions of reward-oriented inhibition (Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985). This could be reflected as higher behavioral activation sensitivity (higher BAS scores). At least one recent study investigating the relationship between BIS/BAS scores and ERP components showed a positive correlation between P3 amplitude and BAS scores (Nijs, Franken, & Smulders, 2007). This may initially seem to contradict data specific to P3 amplitude and impulsivity, but with a more careful interpretation, may fit well with some ERP data collected from psychopaths.

Most notably, Raine (1989) has formed a hypothesis to interpret findings that psychopaths show increased P3 amplitudes. Raine reported consistent findings in three separate samples of psychopaths, reported in two publications (Raine & Venables, 1987; 1988). Furthermore, these investigations were reportedly inspired by Syndulko and colleagues' (1975) study, which despite failing to show statistically significant differences in P3 amplitude for psychopaths, still showed larger amplitudes by half a standard deviation. Raine suspected that lack of significance here may have only been the result of a small sample size. Ultimately, the author's interpretation of this data suggests that under certain conditions psychopaths exhibit recognizable information processing proficiencies for particular forms of salient stimuli. This agrees with Jutai and

Hare's (1983) suggestion that psychopaths show an enhanced ability to attend to events of particular interest. This may generalize to an enhancement of appetitive response.

As noted earlier, more data is needed before we can say whether psychopaths have larger or smaller P3's, or whether this is even a reliable differentiating characteristic; but if any progress is to be made, it will at least be necessary to attempt to control for these known developmental variations within the disorder. Again, it seems that a population of university students with presumably only the mildest antisocial tendencies, yet who exhibit certain relevant personality traits might be an ideal one to sample from in order to gain some much needed insight into this question of P3 characteristics. Within this restricted population, it is at least reasonable to expect that P3 amplitudes might be larger for those with primary psychopathic traits, than for those without psychopathic personality features.

Summary

Ultimately, it seems that most of the relevant personality characteristics and behavioral tendencies of psychopathic individuals can be explained by dysfunctions in two regions, the amygdala and prefrontal cortex. Several integrative neurocognitive models of psychopathy from leading researchers agree on the primary importance of these areas to the disorder (Blair, Mitchell, & Blair, 2005; Kiehl, 2006; Raine & Yang, 2006). According to such models, a dysfunctional amygdala impairs basic emotional responses and therefore weakens aversive learning conditions. At the same time, a compromised prefrontal cortex provides inadequate behavioral inhibition and mismanagement of relevant environmental cues responsible for social learning. Neuroimaging and psychophysiological data support the presence of these functional

impairments and their relative contributions to the construct of psychopathy. The sources of these dysfunctions could theoretically range from the genetic expression of an inherited trait, to an impoverished environment with incompetent parents, to an unfortunate, traumatic brain injury. Though as we have seen, damage to just one of the areas implicated in psychopathy produces partially similar, but incomplete behavioral manifestations compared to the full-fledged disorder.

Variability in relative functionality in these regions contributes to a variety of related manifestations of psychopathy, which may be directly related to the severity of local impairment. In this regard, we have seen that psychopaths present with a range of social adaptiveness: some blending relatively well into a functioning society and likely able to avoid incarceration and some with more prominent antisocial behavior who are likely to be at odds with the justice system most of their lives. This speaks well for the conceptualization of psychopathy as a personality construct with a normal continuum of expression, rather than an all-or-none diagnosis. Realizing this and understanding the measurable components which contribute the most to valid risk-assessments will better equip us to make further efforts toward practical interventions. Perhaps the most promising avenue of intervention would come from the identification of a genetic marker indicating a level of phenotypic risk, which could then make interventions possible at a much earlier age, and ultimately make these efforts toward socialization more fruitful.

The Current Study

While there has been steady progress in identifying psychophysiological traits associated with psychopathy, most of the work has been done in an institutionalized setting with subjects from a prison community. Furthermore, most of these studies have been

performed on exclusively male samples, and investigations which have included females have resulted in less consistent findings, calling into question the generalizability of recognized psychophysiological trends to the population of female psychopaths. It is the goal of this study to examine those same psychophysiological traits in a non-criminal, university sample of females with a similar psychopathic personality profile as identified by the PPI-R. This study examines three main hypotheses. First, it is hypothesized that correlations between self-report measures will demonstrate the typical trends indicating behavioral differences between those with high psychopathic traits and those with low psychopathic traits; namely, high scorers will report significantly more drug and alcohol use, and will score higher on sensation seeking measures. Second, it is hypothesized that those participants scoring high for psychopathic traits will exhibit reduced overall affective modulation of the startle blink reflex; that is, effect sizes for blink magnitude differences will be substantially smaller for those with high psychopathic traits. Third, it is hypothesized that those scoring high for psychopathic traits will show significantly larger P3 amplitudes than those with low psychopathic traits, using a standard P3 generating task. These findings would provide support to the body of work which validates the use of the PPI-R for identifying psychopathy in community samples. Furthermore, they would provide additional support for the concept of primary psychopathy as a normal-range personality construct, identifiable in a non-criminal population, presenting with physiologically measurable deficits in emotional responses. Finally, these results would add support to theories that propose psychopathic personality traits are indicative of observable psychophysiological qualities indicating differences in allocation of attentional and/or cognitive resources.

CHAPTER TWO

Experimental Methodology

Participants were first required to complete a number of self-report measures in an online format, consisting of the Psychopathic Personality Inventory (PPI), the Drug Abuse Screening Test (DAST-20), the Alcohol Use Disorders Identification Test (AUDIT), and the Zuckerman Sensation Seeking Scale (SSS). After completing the online tests, participants were required to complete the second phase of the study, during which psychophysiological data was collected in the laboratory. The psychophysiological recordings included both startle and ERP paradigms, and utilized an affective picture-viewing task and a P3-generating oddball task, respectively.

In order to achieve an a priori estimate of the number of subjects needed to achieve appropriate statistical power, several published investigations were considered as well as some existing pilot data. Effect sizes were computed from their corresponding reported *F* values and *t* values. The two analyses of primary concern in this study involved startle potentiation and ERP analysis. Startle potentiation carried out on non-incarcerated samples by Justus and Finn (2007) and Vanman et al. (2003) produced approximate Cohen's *f* values of 0.27 (medium effect) and 0.56 (large effect) respectively. ERP analysis by Raine and Venables (1987) yielded an approximate Cohen's *d* value of 0.70 (medium effect), and Raine and Venables (1988) produced approximate Cohen's *f* values of 0.27 (medium effect). Pilot data in our lab has yielded medium effect sizes for startle ($f = 0.37$) and ERP analysis ($f = 0.28$). Based on these data, medium effect sizes are expected for both startle and ERP analyses. In order to

achieve a minimum power of 0.80, estimates suggest that a sample of 60 total subjects will suffice.

Participants

Participants included a total of 76 female undergraduate psychology students at Baylor University, participating for partial course credit. Recruitment was carried out via the Human Participants in Research (HPR) website, describing all currently active projects at Baylor University in need of research participants. Volunteers were excluded from participation if self-reporting major neurological disorder, head injury, auditory deficits, or uncorrected visual deficits. Equipment failure prevented recording of startle data for nine participants. Also, startle data was ignored if the participant lacked any reliable blink response to the startle probe (n = 10), yielding 57 participants with useable startle data. Two participants were excluded from ERP analysis as outliers (2.5 SD), and two were excluded based on unidentifiable P3s, yielding a total of 72 participants with useable ERP data. After scheduling a laboratory session, participants were instructed to refrain from caffeine and nicotine for at least 2 hours prior to their arrival. All laboratory sessions were scheduled between 12:00 pm and 5:00 pm, with each session lasting approximately two hours.

Self-Report Measures

The Revised Psychopathic Personality Inventory (PPI-R)

Psychopathic traits were assessed using the Revised Psychopathic Personality Inventory (PPI-R; Lilienfeld & Widows, 2005). The PPI-R is a self-report test designed for use in identifying psychopathic personality traits in a non-criminal population, but

which has been validated for use in both institutionalized and community samples (Patrick, Edens, Poythress, Lilienfeld, & Benning, 2006). An earlier version exists (PPI; Lilienfeld & Andrews, 1996), which had been similarly validated (Poythress et al., 1998); however, this most recent revision has removed a few poorly worded questions from the original. Sandler (2007) has validated the congruency of the PPI-R in an online, computer-based format, versus the standard paper form.

The test consists of 154 items requiring responses on a 4 point, Likert-type scale, with responses ranging from false to true. It yields a total score representing global psychopathy, and grades on 8 separate factor scores. *Machiavellian Egocentricity* represents a willingness to exploit others for personal benefit, *Social Potency* measures interpersonal fortitude, *Fearlessness* is a measure of willingness to take physical risks with low anticipatory anxiety, *Coldheartedness* represents an absence of empathy, *Impulsive Nonconformity* measures one's disregard for tradition, *Blame Externalization* scores a tendency to divert responsibility, *Carefree Nonplanfulness* measures an apparent disregard for the future, and *Stress Immunity* represents low anxiety response in ordinarily anxiety-provoking situations. Seven of these elements have been found to feed into two primary factors (Benning et al., 2003): an interpersonal/affective component dubbed *Fearless Dominance* and a component of social deviance dubbed *Self Centered Impulsivity*. The subfactor *Coldheartedness* stands on its own. Because it was developed and originally implemented in a university sample, antisociality items do not necessarily suggest criminal behavior as unequivocally as the PCL-R items, but rather suggest personality traits consistent with rule-breaking and a subversive attitude toward authority. A non-incarcerated sample, then, can still be expected to show a normal distribution of

scores on this impulsive/antisocial component, as it is not synonymous with DSM-IV-TR criteria for ASPD.

Drug Abuse Screening Test (DAST)

The DAST (Skinner, 1982) is a 20 item questionnaire requiring "yes" or "no" responses from the subject pertaining to drug-use behaviors. It is a brief, but well-validated tool used to screen for drug-abuse problems, while also providing a score useful for quantitative representation of the extent of such problems.

The Alcohol Use Disorders Identification Test (AUDIT)

The AUDIT (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) consists of 10 questions about the subjects' alcohol use, which are scored on a level of 1 to 4. Total scores are used as an indicator relating to severity of alcohol use and the likelihood of alcohol abuse.

The Sensation Seeking Scale (SSS)

The SSS, form-V (Zuckerman, 1977), consists of 40, forced choice items in the form of two discriminating sentences the subject must choose between, which would best represent their personality or behavior. It has been extensively used in a wide variety of populations to measure four distinct elements of behavior: thrill-adventure seeking, experience seeking, disinhibition, and boredom susceptibility (Zuckerman, Eysenck, & Eysenck, 1978).

Psychophysiological Assessment

There were two components to the psychophysiological assessment of these participants. First, participants underwent preparations for and complete a standard affective picture-viewing eye-blink startle paradigm. At the completion of the startle task, participants received a short break while EMG electrodes were removed and preparations were made (scalp, skin, etc.) for the EEG recording. Participants then completed a standard auditory oddball paradigm followed by a standard visual oddball paradigm while EEG data was recorded for offline analysis of ERPs.

Startle Paradigm

The affective picture-viewing task utilized pictures from the International Affective Picture System (IAPS; Lang, Ohman, & Vaitl, 1988), a set of images with standardized ratings of affective valence and arousal level (Lang & Greenwald, 1988). The picture presentation consisted of 60 IAPS images with equal numbers of positive, negative and neutral images. The IAPS images[1] were chosen from a pool such that arousal levels were comparable between negative and positive valence categories. The majority of these pictures were previously used in the study by Larson, Ruffalo, Nietert, and Davidson (2005), which verified test-retest reliability for emotion-modulated startle at varying picture presentation times and probe latencies. All subjective ratings of valence and arousal levels for these pictures are reported in Lang & Greenwald (1988). Neutral pictures minimize arousal levels, and do not favor positive or negative emotional valence. These pictures consisted mostly of household items such as a lamp or a chair.

[1] Positive: 8080, 5629, 7270, 8501, 8190, 8490, 5910, 8030, 8370, 5460, 8500, 8034, 8180, 1710, 5621, 8502, 7502, 5626, 5470, 5700. Neutral: 7490, 7020, 2880, 5740, 7004, 7185, 5510, 7090, 7000, 7233, 5531, 7235, 7025, 7100, 7217, 7491, 7950, 7010, 7002, 6150. Negative: 3010, 9252, 6230, 9570, 2730, 3100, 3110, 6260, 6570, 6510, 6250, 3530, 6370, 6350, 3400, 3102, 3120, 3060, 6560, 9410.

Care was used to eliminate gender-specific pictures in the positive valence category, such as erotic imagery. The positive images, therefore, consisted primarily of adventure scenes and nature scenes. Pictures in the negative valence category consisted primarily of human injury, mutilation, and some pictures of personal attacks, such as a mugging.

Participants were seated in a padded chair in a dimly-lit, radio frequency anechoic chamber (Raymond EMC Enclosures Ltd. Ottawa, Ontario, Canada), designed to minimize superfluous electromagnetic waves, and prevent interference with recordings. They were seated at a desk directly in front of a computer monitor, and fitted with a pair of noise-canceling headphones, through which the startle probes were delivered. Participants were instructed to keep their full attention on the screen during the entire picture presentation and were informed that they would occasionally hear bursts of static through the headphones, which were to be ignored. Images were presented in a single block of 60 images in a fixed-per-subject, pseudo-randomized order. The presentation was designed such that no two images of the same valence were repeated serially—that is, two negative stimuli would not occur next to each other in the presentation. Pictures remained on the screen for six seconds, followed by a two second interstimulus interval, during which a white plus sign was displayed in the center of the monitor, as a point of focus. Fifty percent of the pictures were paired with the startle probe: a 50 ms, 100 dB white noise burst, with immediate rise. Startle probes were assigned randomly to images, and again were fixed-per-subject in a manner such that each valence category was paired with an equal number of startle probes. In total there were 20 images from each of three valences (positive, negative, and neutral), ten of which were paired with a startle probe, which occurred randomly between three and five seconds after the onset of the visual

stimulus. Also, in the tradition of similar paradigms, to reduce predictability, six interstimulus intervals are paired with a startle probe—though responses to these six probes were ignored in the analysis. The startle paradigm was designed and delivered using Superlab 4.0 software (Cedrus Corporation, San Pedro, CA).

Magnitude of the startle response was measured by electromyogram recordings from the orbicularis oculi muscle of the subject's right eye. A pair of Ag-AgCl electrodes (Biopac Systems Inc., Goleta, CA) was placed 1 cm below the lower lid of the right eye, one directly below the pupil and a second 1 cm to the right of that electrode. A third electrode was placed in the center of the forehead as a ground. Skin was prepared with isopropyl alcohol and a mildly abrasive gel (NuPrep) to improve surface conduction; Signa gel brand saline gel was used as a conducting medium, and impedances were kept below 5 kΩ. Raw EMG signals were collected using BioPac MP150 data acquisition hardware with a 60 Hz notch filter, amplified to 5000 Hz. EMG data was filtered with a bandpass at 10-500 Hz, rectified and integrated with a time constant of 10 ms. Data was recorded with AcqKnowledge 3.9 software (Biopac Systems Inc.). Blink magnitudes were defined as a smoothed EMG signal, recorded as baseline to peak differences for each startle probe. The baseline was defined as the mean orbicularis oculi EMG reading during the 25 milliseconds prior to onset of the noise; the peak was defined as the maximum EMG amplitude between 8 and 200 milliseconds after the onset of the noise.

Event Related Potentials Paradigm

Standard auditory and visual oddball paradigms were employed as a means to elicit a reliable P3 ERP component in the EEG signal. The oddball paradigms were designed and administered via Stim^2 software and auditory stimuli were delivered via

Stim Audio System P/N 1105 (both by Compumedics Neuroscan, Charlotte, NC). The auditory paradigm consisted of 200 trials with 40 low-probability and 160 high-probability stimuli; that is, the "oddball" stimulus occurred on 20% of the trials. These stimuli consisted of a high-probability low frequency (500 Hz) beeping tone, and a low-probability high-frequency (1000 Hz) beeping tone. Recordings were conducted in the same anechoic chamber described above. Participants were asked to react to the stimuli by depressing one of two response buttons, under the guise of a "reaction-time test." This task was then repeated for every participant as a computerized visual task using the letter "A" as the high-probability stimulus and the letter "B" as the low-probability stimulus. These letters appeared in yellow, 50 point font against a black background on the computer monitor.

EEG data was recorded from scalp sites using a fitted, elastic cap (Electro-Cap International, Inc. Eaton, OH) consisting of 64 tin electrodes arranged in the international 10-20 system, with standard and intermediate positions. Electrode sites were first cleansed with isopropyl alcohol and prepared with a mildly abrasive gel (Nuprep) to improve scalp conduction. Impedences of electrodes were kept below 5 kΩ. Scalp electrodes were referenced to two electrodes affixed at the mastoids. Additionally, four electrodes placed around the subjects eyes were used to record blinks and eye movements, which were removed using an offline ocular artifact reduction technique in order to prevent contamination of pertinent epochs. EEG data was recorded continuously at a sampling rate of 1,000 samples per second and amplified by SYNAMPS^2 amplifiers (Compumedics Neuroscan, Charlotte, NC). Offline analysis consisted of a bandpass filter set at 0.1 Hz to 35 Hz, removal of artifacts, rereferencing to the mastoids,

implementing a correction to the baseline, and averaging trials within subjects, all using Scan 4.3 software by Neuroscan. P3 peak amplitudes and latency were determined relative to baseline, 100 milliseconds prior to stimulus onset. Epochs were established as data existing between 100 ms prior to stimulus onset to 900 ms post stimulus onset. The P3 component of the ERP was defined as the highest positive peak amplitude from baseline occurring between 250 and 450 ms after stimulus onset. Latency of the P3 was defined as the number of milliseconds between the target stimulus onset and the P3 peak.

Data Analysis

Analyses were carried out with the goal of exhibiting differences between high and low scorers on the PPI-R. Groups were determined by a median split, with group membership assigned based on percentile ranks from Lilienfeld and Widows (2005) published norms for females age 18 – 24, as these values were based on much larger samples. The primary group division of interest was the total PPI-R score, measuring overall psychopathic traits. Further analysis was carried with group assignments based on published norms for the two major factor elements of the PPI-R, Fearless Dominance, and Self Centered Impulsivity, which may indicate traits specific to primary and secondary characteristics of the psychopathy construct. For analysis of the startle data, blink magnitudes were standardized as has been traditional in this line of research, because of high individual variability between subjects, which can obscure potentiation effects within subjects, upon averaging across subjects.

The peculiar nature of this type of analysis is worth mentioning here. To demonstrate that psychopaths do not show the typical potentiated startle while viewing aversive pictures, it is necessary to argue that a statistical difference in average

standardized blink amplitudes does not exist between scores grouped by affective valence. This kind of argument is antithetical to traditional hypothesis testing, as we are aware that a statistical difference between groups will always exist given a large enough number of observations—a fact that has brought traditional hypothesis testing under severe criticism by those favoring interpretation of effect sizes as a measure of practical significance (Jones & Tukey, 2000; Kirk, 1996; Thompson, 1998). It has been the tradition in this line of research, however, either to establish significant interaction effects between group divisions and main effects of blink magnitude in a repeated measures analysis, or simply to demonstrate that paired samples t-tests comparing blink magnitudes between valence conditions show significant or non-significant differences.

In the current study, interaction comparisons were considered as a trend indicator; however, the paired samples *t*-test was the primary means of assessing the raw effect sizes of startle potentiation. This is favorable in light of the primary goal in implementing the startle paradigm as a means of demonstrating a well-replicated psychophysiological trend, startle potentiation, in this particular sample before examining a more controversial one, P3 amplitude. The reduced power associated with determining significant interactions relative to main effects (in this case, overall differences across valences) in a repeated measures analysis makes it a less attractive choice for demonstrating this trend in a normal-range sample. Effect sizes were evaluated in addition to t-tests' p-values in order to provide a more practical comparison of the degree to which the affective valences of the images modulate overall blink magnitudes between the groups.

For analysis of ERP data, P3 peaks were identified for target stimuli and non-target stimuli and averaged within subjects, per electrode, for each of the oddball tasks, and these averaged amplitudes under each condition were used as the dependent variable. Overall P3 amplitudes were compared between groups using ANOVA with the electrode sites as a repeated measure and group identity as a between subjects factor. Again, effect sizes were evaluated to determine the practical significance of differences observed in P3 amplitude. Additionally, a comparison was made to determine if group differences existed on P3 amplitude differences between target and non-target stimuli. Difference scores were calculated between peak amplitudes for targets and non-targets within subjects, and the average group difference scores were evaluated using an independent samples *t*-test. This may provide evidence to evaluate whether those with high levels of psychopathic traits differentiate between targets and non-targets in a physiologically distinct manner.

There are currently no existing published studies which report analyses of both startle modulation and P3 amplitudes from the same sample. It seems that this combination of measures on a sample of relatively well socialized, high-functioning individuals has the potential to effectively establish a set of core psychophysiological characteristics representative of psychopathy's primary features, recognizable in the absence of severe antisocial tendencies. These results will help substantiate the neuropsychological basis for psychopathy amongst non-criminals, and will guide future research by encouraging the careful interpretation of data from forensic samples with high incidence of antisocial behavior.

CHAPTER THREE

Results

Personality Factors and Substance Use

Table 1 provides descriptive statistics for all the self-report data, separated into pertinent group divisions. Those with high psychopathic traits generally scored higher on the sensation seeking scale. Likewise, these individuals reported more drug and alcohol use; however, DAST and AUDIT scores were very low for both groups, ultimately limiting the variability between groups.

Table 1

Mean (SD) Personality Measures and Self-Report Scores by Group Division

Grouping	PPI	FD	SCI	SSS	DAST	AUDIT
All	269.9 (30.9)	112.1 (19.9)	129.9 (18.1)	16.3(6.4)	0.74 (1.7)	2.6 (3.8)
High PPI	301.9 (25.2)	127.1 (17.5)	145.4 (18.3)	21.4 (6.5)	1.2 (2.5)	3.8 (5.0)
Low PPI	250.4 (17.6)	101.7 (14.4)	121.9 (13.4)	12.1 (4.6)	0.4 (0.7)	1.7 (2.7)
High FD	289.4 (31.4)	127.9 (13.3)	133.9 (22.0)	20.5 (6.4)	1.0 (2.2)	3.3 (4.7)
Low FD	251.8 (21.5)	95.6 (9.4)	128.1 (15.7)	11.8 (5.1)	0.4 (0.7)	1.6 (2.5)
High SCI	292.8 (32.0)	114.4 (20.7)	149.0 (13.4)	20.2 (7.7)	1.2 (2.5)	3.3 (5.2)
Low SCI	255.0 (23.1)	109.5 (19.5)	118.8 (11.1)	14.2 (5.9)	0.4 (0.9)	2.1 (2.3)

Note. High and Low groups for each division are based on a median split using published norms from the PPI-R handbook (Lilienfeld & Widows, 2005). PPI = PPI-R total score, FD = Fearless Dominance factor score, SCI = Self Centered Impulsivity factor score, SSS = Sensation Seeking Scale total score, DAST = Drug Abuse Screening Test total score, AUDIT = Alcohol Use Disorders Identification Test total score.

Table 2 provides correlations between these measures. As expected, PPI-R total scores and factor scores on Fearless Dominance and Self Centered Impulsivity all strongly correlated with Sensation Seeking total scores (all *p*-values < .001). Likewise, PPI-R total scores and Self Centered Impulsivity strongly correlated with drug and alcohol use as measured by the DAST and AUDIT (all *p*-values < .001), and Fearless Dominance correlated only slightly less with DAST ($p < .01$) and AUDIT ($p < .02$) scores. Amongst all the self-report data, the only two items that were not significantly correlated were Fearless Dominance scores with Self Centered Impulsivity scores ($p > .10$), supporting the independence of these two factors within the present sample.

Table 2

Correlations between Self-Report Measures

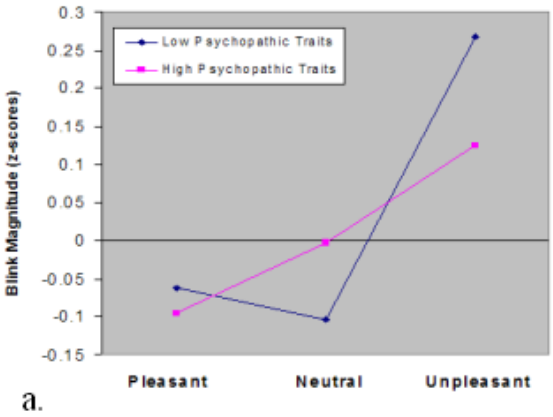
Grouping	AUDIT	DAST	SSS	SCI	FD	PPI Total
PPI Total	.474**	.484**	.670**	.744**	.753**	
FD	.275*	.299**	.594**	.160		
SCI	.388**	.410**	.465**			
SSS	.517**	.436**				
DAST	.687**					
AUDIT						

Note. PPI = PPI-R total score, FD = Fearless Dominance factor score, SCI = Self Centered Impulsivity factor score, SSS = Sensation Seeking Scale total score, DAST = Drug Abuse Screening Test total score, AUDIT = Alcohol Use Disorders Identification Test total score.
*$p < .05$, two-tailed test. **$p < .01$, two–tailed test.

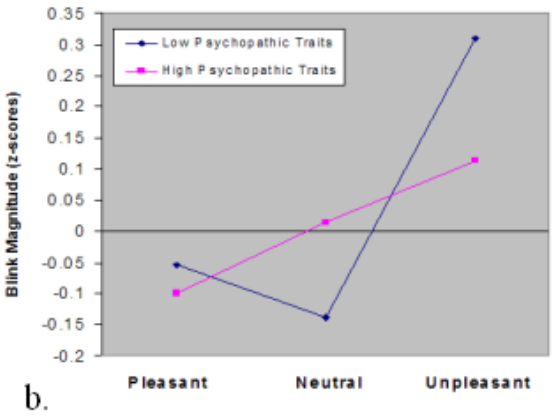
Analysis of Startle Physiology

Differential startle modulation patterns by emotional valence category were assessed for each of three group divisions, based on PPI-R total score, and the two subfactors, Fearless Dominance and Self Centered Impulsivity. The average standardized eye blink magnitudes for each valence were compared for each group division using the three picture valences as a repeated measure within groups. For all repeated measures analyses, Greenhouse-Geisser corrections are made, and consequent degrees of freedom are reported here. When dividing groups by PPI-R total score, there was a marginally significant picture valence x group interaction, F (1.89, 104.11) = 1.965, p = .148, and an apparent quadratic interaction, F (1, 55) = 3.51, p = .066, when assessing within groups contrasts, suggesting differential potentiation effects between groups.

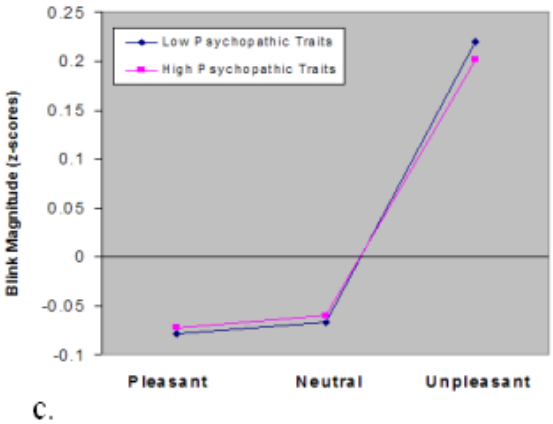
This potential interaction effect appears to be driven by scores on the emotional/interpersonal factor of the PPI-R, Fearless Dominance. When subjects were divided into groups for high and low Fearless Dominance scores, there was, again, a significant picture valence x group interaction F (1.90, 104.23) = 4.462, p = .015, and an apparent quadratic interaction F (1, 55) = 8.467, p = .005, when assessing within groups contrasts. However, when subjects were divided into groups for high and low Self Centered Impulsivity there was no apparent picture valence x group interaction F (1.89, 103.70) = .029, p = .966. Likewise the quadratic interaction disappeared F (1, 55) = .018, p = .893, when assessing within groups contrasts, and it is visually apparent that these individuals show the same linear pattern of blink potentiation. These results are illustrated in Figure 1.



a.



b.



c.

Figure 1. Grouped comparisons of standardized blink magnitudes for each affective valence reveal a reduction in blink potentiation that is driven by the primary factor elements psychopathy, as represented by Fearless Dominance scores. Participants grouped by a. Total PPI-R score b. Fearless Dominance c. Self Centered Impulsivity scores.

A visual assessment of the plotted average standardized eye blink magnitudes for each psychopathy group reveals an apparently large difference between neutral and negative valences, representing the effect of startle potentiation for negative valence images. When dividing subjects by total PPI-R score, high scorers lacked a significant difference between blink magnitudes during aversive pictures compared to neutral pictures, t (21) = 1.27, p = .218, and may therefore be described as lacking the effect of potentiated startle. Low scorers on the other hand, showed the typical potentiated effect with larger blink magnitudes during aversive pictures, t (34) = 4.51, $p < .001$. A more appropriate comparison of these differences can be made using effect sizes. For high scorers, Cohen's d = 0.41, while for low scorers Cohen's d = 1.31. Therefore, the effect of startle blink potentiation was more than three times larger for those with low levels of psychopathic traits, compared to those exhibiting high levels of psychopathic traits

Again, these differences in startle potentiation between psychopathy groups can be narrowed further and appear to be driven by the emotional/interpersonal Fearless Dominance factor of the PPI-R. When dividing subjects by Fearless Dominance scores, high scorers, again, failed to show a significant potentiation effect, t (27) = 1.31, p = .202, d = 0.37. Low Fearless Dominance scorers, on the other hand, showed significant startle potentiation, t (28) = 4.71, $p < .001$, d = 1.48. Comparisons of effect sizes reveal the effect of blink potentiation was approximately four times larger for those with low Fearless Dominance scores. However, when dividing subjects by the antisocial component of PPI-R, Self Centered Impulsivity, this effect disappears. Both high scorers, t (22) = 3.07, $p < 0.01$, and low scorers, t (33) = 3.08, $p < 0.01$, on this factor

show significant potentiation, with very similar effect sizes, $d = 1.05$ and $d = 0.89$ respectively.

Analysis of Event Related Potentials

For comparative purposes, nine electrodes (F4, FZ, F3, C4, CZ, C3, P4, PZ, P3) were chosen as a representation of the electrode array. Table 3 provides means and standard deviations of P3 amplitudes at midline electrode sites for various groups of participants. Descriptive statistics suggest a clear trend for larger P3 amplitudes for those with high psychopathic traits. Dividing participants into two groups based on total PPI-R score, and using the nine selected electrode sites as a repeated measure within groups, those with high psychopathic traits showed significantly larger P3 amplitudes for target stimuli during the auditory oddball task than those with low psychopathic traits, F (1, 70) = 4.01, $p = .049$, $\eta^2 = .054$. This effect seems to be exaggerated in the parietal region and along the midline, as isolating the three selected parietal electrodes P4, PZ, and P3 revealed a slightly stronger effect, F (1, 70) = 4.83, $p = .031$, $\eta^2 = .065$. Likewise the effect is similar when isolating midline electrode sites FZ, CZ, and PZ, F (1, 70) = 4.55, $p = .036$, $\eta^2 = .061$.

Figure 2 illustrates these P3 amplitude differences at the midline electrode sites for the auditory oddball task. No significant differences were immediately apparent between groups divided by either Fearless Dominance scores or Self Centered Impulsivity scores.

Table 3

Mean (SD) P3 Amplitudes (μV) at Midline Electrode sites by Group Division

Grouping	FZ	CZ	PZ
All Participants	14.3 (6.7)	18.6 (7.5)	18.6 (7.0)
High PPI	16.3 (7.4)	20.5 (8.3)	20.8 (6.9)
Low PPI	13.0 (5.7)	17.4 (6.8)	17.2 (6.7)
High FD	14.9 (7.2)	19.2 (7.7)	19.3 (7.6)
Low FD	13.7 (6.1)	17.9 (7.3)	17.7 (6.2)
High SCI	15.6 (7.1)	19.4 (7.3)	19.5 (6.2)
Low SCI	13.6 (6.4)	18.2 (7.7)	18.8 (7.4)

Note. High and Low groups for each division are based on a median split using published norms from the PPI-R handbook (Lilienfeld & Widows, 2005). PPI = PPI-R total score, FD = Fearless Dominance factor score, SCI = Self Centered Impulsivity factor score.

When assessing P3 amplitude differences between groups for the visual oddball task, the trend was very similar but not as strong as that for auditory targets. Using the nine selected electrode sites, those with high total PPI-R scores showed marginally larger P3 amplitudes for target stimuli than those with low PPI-R scores, F (1, 70) = 2.62, p = .110, η^2 = .036. Again, this trend was slightly exaggerated at parietal sites F (1, 70) = 3.83, p = .054, η^2 = .052, and was similarly exaggerated when isolating the midline, F (1, 70) = 3.56, p = .063, η^2 = .048. Just as with the auditory oddball task, no differences were apparent when dividing groups by Fearless Dominance scores or Self Centered Impulsivity scores.

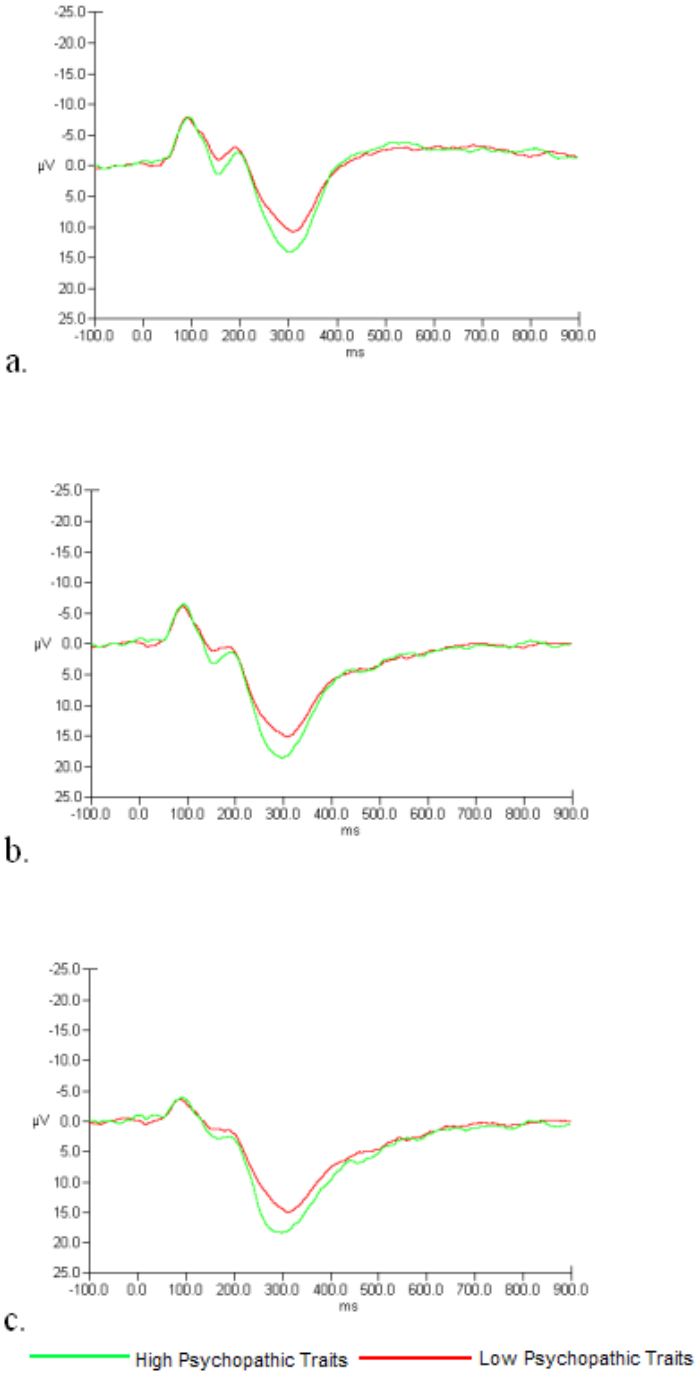


Figure 2. Amplitude comparisons at midline electrode sites reveal augmented P3s for those with high psychopathic traits on the auditory oddball task. a. FZ b. CZ c. PZ.

Another method of comparison applied in previous studies (e.g. Kiehl, Hare, Liddle, et al., 1999, Kiehl, Hare, McDonald et al., 1999) is to compare relative differences in P3 amplitude between target and non-target stimuli for each group. Difference scores were calculated within subjects for each of the nine electrode sites and these difference scores were evaluated between groups with electrode sites used as a repeated measure. When dividing participants into two groups based on total PPI-R score, high scorers showed significantly larger differences in P3 amplitude between target and non-target stimuli, F (1, 70) = 4.62, p = .035, η^2 = .061. This effect remained recognizable at parietal sites alone, F (1, 70) = 2.87, p = .094, η^2 = .039, and when isolating the midline sites, F (1, 70) = 4.58, p = .036, η^2 = .061. Figure 3 illustrates the differences between target and non-target stimuli for high and low scorers on the PPI-R total score.

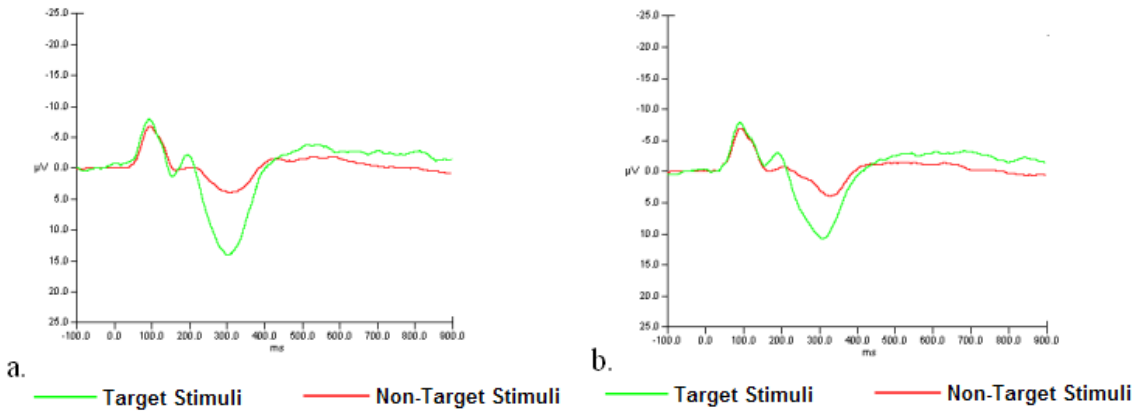


Figure 3. Comparisons of auditory target stimuli to non-target stimuli reveal that those with high psychopathic traits (a.) show significantly greater differentiation between targets and non-targets than those with low psychopathic traits (b.).

While no significant differences were apparent while dividing participants by Fearless Dominance or Self Centered Impulsivity, Fearless Dominance appeared to have a stronger influence on this trend as its effect size, $\eta^2 = .020$, was much larger than that for Self Centered Impulsivity, $\eta^2 = .001$. Bivariate correlations were found to be positive for both Fearless Dominance and Self Centered Impulsivity with P3 amplitude. At electrode PZ for instance, Fearless Dominance correlated positively ($r = .211$, $p = .075$), as did Self Centered Impulsivity ($r = .159$, $p = .182$). When using both subfactors in a regression model to predict an average P3 amplitude calculated from the 9 representative electrodes, standardized coefficients for Fearless Dominance (Standardized Beta = .170, $p = .139$) remained slightly more prominent than that for Self Centered Impulsivity (Standardized Beta = .092, $p = .444$).

CHAPTER FOUR

Discussion

The startle modulation data from this sample of female university students support the large body of available literature using psychopaths from incarcerated samples (e.g. Patrick et al., 1993) and from community samples (e.g. Vanman et al., 2003). Participants from the current sample who scored high for psychopathic traits lacked potentiated startle, and this trend was clearly driven by the emotional/interpersonal facets of psychopathy rather than antisocial traits. This demonstrates a consistent trend in psychophysiology among those with psychopathic traits, from a normal-range sample, who lack severe antisocial tendencies. Furthermore, this outcome supports the preservation of this effect in a female sample, which has been a moderately elusive finding (e.g. Justus & Finn, 2007). This portion of the study serves to add some needed clarity to this realm of research in non-incarcerated and female samples. It also serves to demonstrate a practical and reliable physiological distinction between the groups being compared prior to subsequent ERP analysis, which has been in even greater need of added clarity.

The ERP data from this sample support previous reports by Raine and Venables (1987; 1988) which had suggested that P3 amplitude is augmented in psychopaths. The current sample showed significantly larger P3 amplitudes in subjects scoring high for psychopathic traits, and showed significantly larger differences between P3 amplitudes for targets vs. non-target stimuli in subjects scoring high for psychopathic traits. While this outcome runs contrary to some recent reports of reduced P3 amplitudes in

psychopaths (e.g. Kiehl et al., 2000; 2006), a major difference between these studies was the sampling pool, which in the current study represents normal-range, non-incarcerated females rather than incarcerated male psychopaths.

At the time of this investigation, this appears to be the only report which measures both startle physiology and ERP components within the same sample, and relates those data to psychopathic personality traits, showing significant differences between groups on both psychophysiological measures. Based on the present finding of reduced startle potentiation in those with high psychopathic traits, it can be argued confidently that the defined experimental groups indeed represent subjects with divergent sets of physiological responses, despite being in a normal-range, non-incarcerated sample. In light of this distinction, the augmented P3 amplitudes should be regarded as a relevant effect in need of careful interpretation.

Self-Report Interpretations

The relationships between self-report measures in the current sample agree with existing reports of correlations between measures of psychopathy and sensation seeking (Zuckerman, 2002), and substance use (Hemphill, Hart, & Hare, 1994; Smith & Newman, 1990). Those reporting higher levels of psychopathic traits also report more alcohol and drug use and also report more sensation seeking behavior. The present data confirms that this normal-range sample of female university students demonstrates significant behavioral differences related to reported differences in psychopathic traits. Additionally, it can be presumed from the relatively low scores for both drug and alcohol use among those in the current sample (see Saunders et al., 1993; Skinner, 1982) that

psychophysiological outcomes should not be interpreted as an effect related to substance abuse disorders.

Startle Interpretations

It is clear from the analysis that participants with high psychopathic traits exhibited the typical reduced startle potentiation relative to those with low psychopathic traits, and reduced overall affective modulation of the startle response. This portion of the study succeeded, therefore, in establishing groups from a normal range sample with demonstrably different psychophysiological features related to the identifiable psychopathic personality traits. It can therefore be argued with more confidence that the observable trends in ERP measurements are indeed a relevant effect related to these same personality measures.

There is, however, a potentially atypical trend evident in the current data. From previous studies one would expect to see a generally linear trend with positive affective pictures producing, on average, the lowest blink magnitudes in normal subjects (*cf.* Patrick, Bradley, & Lang, 1993). In contrast, it appears that those participants reporting low psychopathic traits showed slightly higher average blink amplitudes for positive images, than for neutral images. Though these differences were not significant, even showing similar blink amplitudes for positive and neutral images is a deviation from previous reports. It may be relevant, however, that the majority of studies which have reported these linear trends utilized IAPS pictures with predominantly erotic content for the positive valence. In the current study, erotic image choices were avoided, because of the female sample and unknown differential modulation effects between gender and erotic imagery. The kinds of pictures used in the current study under the positive valence

were primarily adventure scenes and awe-inspiring nature scenes, which may represent a significant departure from standard protocol in startle modulation paradigms. While interpretation here is unavoidably speculative, it may be true that these images were producing a state of anxiety in the current sample, or that they were not sufficiently positive in valence for these participants. At the very least, it must simply be recognized that these images did not have as significant an attenuating effect on the startle-reflex circuits as has been reported using erotic imagery. While it is possible that this effect was idiosyncratic to the current sample, it is a noteworthy deviation deserving attention in future research, especially while assessing normal-range females, and when non-erotic images are chosen for the paradigm. Most importantly, however, this effect is not directly relevant to the main effect of interest, that is, the clear lack of negative-valence startle potentiation in psychopaths, which was confirmed in the current sample.

ERP Interpretations

The outcome of the ERP segment of the current study will require more careful interpretation, since this has been a more prominent issue of contention in the existing literature. ERP data in the current study are consistent with data reported by Raine and Venables (1987; 1988) indicating augmented P3s in psychopaths, and may add support to a trend reported by Carlson et al. (2009), in which increased P3 amplitudes were related to Factor 1 elements of the PPI. In the current sample, those with high psychopathic traits showed larger P3 amplitudes than those with low psychopathic traits, as measured by the PPI-R total score. Those with high psychopathic traits also showed greater amplitude differences between target and non-target stimuli, demonstrating a stronger effect of target discrimination. These effects were limited to groups based on PPI-R total

scores, as there were no significant differences when grouped solely by either factor score; however, effect sizes, bivariate correlations, and multiple regression analysis all indicated the predictive contribution of Fearless Dominance scores was greater than the contribution of Self Centered Impulsivity scores. While components of the regression model indicated a noteworthy trend in this direction, the respective slope weights did not reach levels of significance with alpha at .05. The unfortunate limitation of such a regression analysis is that one is forced to compute a unique model predicting amplitudes at each electrode site or use a composite average for the amplitude, which reduces the power achieved by ANOVA to detect differences between groups utilizing amplitudes at multiple electrodes simultaneously. For this reason, the results from ANOVA are preferred, although the relevant trends recognized using regression analysis should not be ignored.

The major issue in need of interpretation here is why various studies continue to find contrasting outcomes with regard to P3 amplitude and its relationship with measures of psychopathy. Raine originally interpreted his findings of augmented P3s in light of evidence that psychopaths show certain information processing proficiencies especially related to attending to explicit task requirements (see Raine, 1989). At least one model has described and demonstrated proficiencies in motivated attention as a feature of psychopathy (Newman, Schmitt, & Voss, 1997), and the authors linked this feature to reduced attention-related errors in a distraction task. Likewise, Salekin, Neumann, Leistico, & Zalot (2004) reported positive correlations between psychopathic personality traits and certain measures of intelligence in adolescents. Sellbom and Verona (2007) reported an inverse relationship between executive cognitive functioning and PPI

indicators of social deviance, while the affective/interpersonal components of primary psychopathy were associated with increased executive functioning performance. There is considerable evidence, then, supporting certain information processing proficiencies related to psychopathy's primary features, which might contribute to the occurrence of increased P3 amplitudes relative to nonpsychopaths.

This conclusion may appear to contradict some recent studies which have reported attenuated P3 amplitude in psychopathic populations; however, as noted repeatedly above, most studies reporting this reduced amplitude have been carried out using incarcerated samples (Kiehl et al., 2006; Kiehl, Hare, Liddle, & McDonald, 1999; Kiehl, Hare, McDonald, & Brink, 1999; Kiehl et al., 2000). Similarly, one recent study using a non-incarcerated sample, reported attenuated P3 amplitudes, but attributed the source of the effect exclusively to psychopathy's secondary, antisocial features and noted marked increases in P3 amplitude related to the primary factor elements (Carlson et al., 2009). It seems reasonable, then, that all available reports of attenuated P3 amplitudes in psychopaths might be specifically related to secondary features of the construct such as antisocial behavior, which would really be no surprise given the preponderance of evidence for this in existing literature not specifically related to psychopathy.

Aside from the well-documented associations between violence, impulsivity, substance abuse, and antisocial behavior with reduced P3 amplitudes, as discussed above, Murray and Janelle (2007) have reported reductions of P3 amplitude associated with trait levels of anxiety and subsequent inefficiencies in information processing, a hallmark distinction between primary and secondary psychopaths. There has also been a recent trend in psychopathy research acknowledging the relationship between P3 reductions and

general Externalizing Vulnerability, defined as the common factor underlying symptoms of substance dependence, conduct disorder, and antisocial behavior (e.g. Patrick, 2008; Patrick et al., 2006). Furthermore, many of these same behavioral characteristics have been associated with focal traumatic brain injury and related dysfunction in various regions of the prefrontal cortex (Brower & Price, 2001). These are the kinds of injuries associated with "acquired antisocial personality disorder," described above. Even in the cases of such acquired deficits, it has been demonstrated that those with frontal lobe lesions exhibit attenuated P3 amplitudes (Knight, 1984; Knight 1990; Knight, Grabowecky, & Scabini, 1995). It seems, then, that while the relationship between P3 amplitude and symptoms of disinhibition and externalizing behavior is clearly a negative correlation, there is both theoretical and empirical data to support the incidence of P3 augmentation in psychopaths who demonstrate resistance to these behavioral control issues, or externalizing vulnerability.

The recent report by Carlson and colleagues (2009) was the first to examine the relationship between P3 amplitudes and specific psychopathic personality traits as assessed by the PPI. Having noted trends for reduced P3 amplitudes associated with Self Centered Impulsivity, Carlson et al. also recognized a significant relationship between Fearless Dominance scores and increased P3 amplitudes when both factors were considered simultaneously in a regression model. The authors attributed this to a possible suppression effect between elements of externalizing vulnerability and trait fearlessness; that is to say, while psychopathy's primary and secondary features both contribute positively and directly to measurements of the overall psychopathy construct, these distinct factor elements may have opposing effects on other correlates, such as

psychophysiological measures. This suppression effect between the two major factor elements contributing to psychopathy has indeed been supported elsewhere (Hicks & Patrick, 2006; Sellbom & Verona, 2007), and its effects are visible in a variety of measures.

As discussed earlier, variability in a variety of psychophysiological recordings show sensitivity to levels of the two major factor elements of psychopathy (Skeem et al., 2007) including anxiety response (Lykken, 1957), anticipatory response to punishment (Hare, 1982), startle blink physiology (Patrick, 1994), and simple autonomic responses to aversive pictures (Patrick et al., 1994). In light of this evidence, it seems vital that any interpretation of P3 amplitude differences related to psychopathy account for the possibility of divergent influences from primary and secondary traits.

When assessing psychopaths from an incarcerated sample, and using nonpsychopathic control subjects who are also incarcerated, it is likely that between groups levels of secondary psychopathic traits will be similar. The relative ratios of secondary traits to primary traits will be idiosyncratically dependent upon the specific sample taken, which may help explain the divergence of existing reports from incarcerated samples. This is the approach taken by Carlson and colleagues (2009) while interpreting the conflicting outcomes of studies by Raine and Venables (1988) and Kiehl et al. (1999, 2000), noting the likelihood of elevated externalizing vulnerability in all subjects from these samples. The outcomes of these studies help emphasize the value nonforensic samples in this line of research. The use of non-incarcerated, normal-range samples, with more moderate levels of psychopathic traits under both factors, would reduce the suppression effect between these contributing factors, making

psychophysiological measurements more sensitive to individual factors which may be elevated in a given sample.

The results of the current study demonstrated augmented P3 amplitudes in those scoring higher for psychopathic traits, which is consistent with the idiosyncratic characteristics of this sample. An examination of the scores on the PPI-R reveal that the median total score for this sample (266) is actually at the 40th percentile of published norms for females in this age group. Similarly, the median Self Centered Impulsivity score (130) for the current sample is at the 40th percentile of the published norms. Fearless Dominance scores, on the other hand are relatively elevated, with a sample median (113) at the 60th percentile of published norms. Participants in the current sample who have high PPI-R total scores are more likely to elevate on Fearless Dominance than they are to elevate on Self Centered Impulsivity, showing higher levels of primary psychopathic traits than secondary traits. For the high scoring group on total PPI-R score, the average Fearless Dominance score among participants was 127, corresponding to the 84th percentile of Fearless Dominance scores among females of this age group. The average Self Centered Impulsivity score for this same group is only 145, corresponding to the 68th percentile of these published norms.

Furthermore, the current sample is composed exclusively of females, and the outcomes apparent here emphasize the relevance of the earlier discussion regarding the possibility of an idiosyncratic manifestation of psychopathy between genders. It was previously mentioned that a body of research exists which has suggested that antisocial traits are a less characteristic manifestation of psychopathy in females than they are in male psychopaths (Hesselbrock et al., 1985; Flyn et al., 1996; Forth et al., 1996).

Likewise, it has been suggested that female psychopaths are more likely to exhibit personality traits related to borderline and histrionic personality disorders, rather than antisocial behavior (Hamburger et al., 1996). It is at least reasonable, then, to suspect that high scorers for psychopathic traits in the current sample of normal-range, non-incarcerated females are less likely to exhibit the same variety of externalizing vulnerability prevalent in samples of incarcerated male psychopaths, which have occasionally resulted in reports of reduced P3 amplitudes. If this is true, it would go a long ways in helping to explain both the current outcome of augmented P3s, and the variability in outcomes of such previous studies.

Conclusion

Given the evidence we have for other dissociable psychophysiological effects related to the heterogeneous nature of psychopathy, the possibility of this pattern extending to electrocortical responses should not be ignored. Doing so, would result in the limitation of experimental predictions based on a single observable effect associated with a single psychopathy factor, and the available data doesn't point to a story as simple as that. It seems likely that the divergent findings evident among relevant studies, including the current one, may be the result of interfering electrocortical effects related to an interaction between psychopathy's primary features and those related to antisocial behavior, namely externalizing vulnerability and disinhibition. It would therefore be prudent to employ methods to control for antisocial behavior and related second-order factors of psychopathy when assessing its psychophysiological correlates, including ERPs.

The current sample of females represents a relatively well socialized and adaptive group, engaged in secondary education and in good standing at the university, and does not exhibit the significant antisocial tendencies apparent in a prison sample. Furthermore, their scoring characteristics on the PPI-R indicate elevated Fearless Dominance scores, and relatively low Self Centered Impulsivity scores. Since the current sample demonstrates the physiological distinctions of reduced startle potentiation, it is an extraordinarily valuable group for assessing P3 amplitude with relatively low suppression effects between primary and secondary factors, which would commonly be much higher in a prison sample. In the current sample, the augmented P3 amplitudes evident in those with elevated psychopathic traits are likely the result of elevated primary psychopathic characteristics with limited suppression effects. This outcome should serve to guide future research on this topic, as it has provided additional evidence for divergent trends between primary and secondary factor elements of psychopathy, between incarcerated and non-incarcerated samples of psychopaths, and possibly between males and females with psychopathic traits.

REFERENCES

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: APA.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: APA.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: APA.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: APA.

Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature neuroscience, 2*(11), 1032-1037.

Angrilli, A., Mauri, A., Palomba, D., Flor, H., Birbaumer, N., Sartori, G., et al. (1996). Startle reflex and emotion modulation impairment after a right amygdala lesion. *Brain: A Journal of Neurology, 119*(6), 1991-2000.

Bard, P. (1928). A diencephalic mechanism for the expression of rage with special reference to the sympathetic nervous system. *American Journal of Physiology, 84*, 490-515.

Barratt, E. S., Stanford, M. S., Dowdy, L., Liebman, M. J., & Kent, T. A. (1999). Impulsive and premeditated aggression: A factor analysis of self-reported acts. *Psychiatry Research, 86*(2), 163-173.

Barratt, E. S., Stanford, M. S., Kent, T. A., & Felthous, A. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. *Biological Psychiatry, 41*(10), 1045-1061.

Bauer, L. O., O'Connor, S., & Hesselbrock, V. M. (1994). Frontal P300 decrements in antisocial personality disorder. *Alcoholism: Clinical and Experimental Research, 18*(6), 1300-1305.

Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science, 275*(5304), 1293-1294.

Benning, S. D., Patrick, C. J., Blonigen, D. M., Hicks, B. M., & Iacono, W. G. (2005). Estimating facets of psychopathy from normal personality traits: A step toward community-epidemiological investigations. *Assessment, 12*, 3–18.

Benning, S. D., Patrick, C. J., Hicks, B. M., Blonigen, D. M., & Krueger, R. F. (2003). Factor structure of the psychopathic personality inventory: Validity and implications for clinical assessment. *Psychological assessment, 15*(3), 340-350.

Benning, S. D., Patrick, C. J., & Iacono, W. G. (2005). Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. *Psychophysiology, 42*(6), 753-762.

Bernat, E. M., Hall, J. R., Steffen, B. V., & Patrick, C. J. (2007). Violent offending predicts P300 amplitude. *International Journal of Psychophysiology, 66*(2), 161-167.

Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., et al. (2005). Deficient fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Archives of General Psychiatry, 62*(7), 799-805.

Blackburn, R. (2007). Personality disorder and antisocial deviance: Comments on the debate on the structure of the psychopathy checklist-revised. *Journal of Personality Disorders, 21*(2), 142-159.

Blair, R. J. R. (2004). The roles of orbital frontal cortex in the modulation of antisocial behavior. *Brain and cognition, 55*(1), 198-208.

Blair, R. J. R. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery & Psychiatry, 71*(6), 727-731.

Blair, R. J. R., (2006). The emergence of psychopathy: Implications for the neuropsychological approach to developmental disorders. *Cognition, 101*, 414 – 442.

Blair, R. J. R., Mitchell, D., & Blair, K. (2005). *The psychopath: Emotion and the brain*. Malden, MA: Blackwell.

Bradley, M.M., Cuthbert, B. N., & Lang, P. J., (1999). Affect and the startle reflex. In M. E. Dawson, A. M. Schell, & A. H. Böhmelt (Eds.), *Startle modification: implications for neuroscience, cognitive science, and clinical science* (pp. 157-183). Cambridge: Cambridge University Press.

Brinkley, C. A., Schmitt, W. A., Smith, S. S., & Newman, J. P. (2001). Construct validation of a self-report psychopathy scale: Does levenson's self-report psychopathy scale measure the same constructs as hare's psychopathy checklist-revised? *Personality and Individual Differences, 31*(7), 1021-1038.

Brower, M. C., & Price, B. H. (2001). Neuropsychiatry of frontal lobe dysfunction in violent and criminal behavior: a critical review. *Journal of Neurology, Neurosurgery, & Psychiatry, 71*(6), 720-726.

Cale, E. M., & Lilienfeld, S. O. (2002). Sex differences in psychopathy and antisocial personality disorder a review and integration. *Clinical Psychology Review, 22*, 1179-1207.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67*(2), 319-333.

Cato, M. A., Delis, D. C., Abildskov, T. J., & Bigler, E. (2004). Assessing the elusive cognitive deficits associated with ventromedial prefrontal damage: A case of a modern-day phineas gage. *Journal of the International Neuropsychological Society, 10*(3), 453-465.

Chapman, A. L., Gremore, T. M., & Farmer, R. (2003). Psychometric analysis of psychopathic personality inventory (PPI) with female inmates. *Jounal of Personality Assessment, 80*(2), 164-172.

Cleckley, H. (1941). *The mask of sanity: An attempt to reinterpret the so-called psychopathic personality*. Oxford, England: Mosby.

Cooke, D. J. & Michie, C. (2001). Refining the construct of psychopathy: Towards a hierarchical model. *Psychological Assessment, 13*(2), 171-188.

Cornell, D. G., Warren, J., Hawk, G., Stafford, E., Oram, G., & Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. *Journal of consulting and clinical psychology, 64*(4), 783-790.

Costa, L., Bauer, L., Kuperman, S., Porjesz, B., O'Connor, S., Hesselbrock, V., et al. (2000). Frontal P300 decrements: Alcohol dependence, and antisocial personality disorder. *Biological Psychiatry, 47*(12), 1064-1071.

Cunningham, M. D., & Reidy, T. J. (1998). Antisocial personality disorder and psychopathy: Diagnostic dilemmas in classifying patterns of antisocial behavior in sentencing evaluations. *Behavioral sciences & the law, 16*(3), 333-351.

Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York, NY: Avon.

Damasio, H., Grabowski, T., Frank, R., Galaburda, A. M., & Damasio, A. R. (2005). The return of phineas gage: Clues about the brain from the skull of a famous patient. In J. T. Cacioppo, & G. G. Berntson (Eds.), *Social neuroscience: Key readings.* (pp. 21-28). New York, NY, US: Psychology Press.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation--a possible prelude to violence. *Science, 289*(5479), 591-594.

Davis, M. (1989). Neural systems involved in fear-potentiated startle. *Annals of the New York Academy of Sciences, 563*, 165-183.

Davis, M. (1997). Neurobiology of fear responses: The role of the amygdala. *Journal of Neuropsychiatry & Clinical Neurosciences, 9*(3), 382-402.

Duncan, J., & Owen, A. M. (2000). Common regions of the human frontal lobe recruited by diverse cognitive demands. *Trends in Neuroscience*, 23, 475-483.

Edens, J. F., Poythress, N. G., & Watkins, M. M. (2001). Further validation of the Psychopathic Personality Inventory among offenders: Personality and behavioral correlates. *Journal of Personality Disorders*, *15*(5), 403-415.

Elwood, C. E., Poythress, N. G., & Douglas, K. S. (2004). Evaluation of the hare P-SCAN in a non-clinical population. *Personality and Individual Differences, 36*(4), 833-843.

Flynn, P. M., Craddock, S. G., Luckey, J. W., Hubbard, R. L., & Dunteman, G. H. (1996). Comorbidity of antisocial personality and mood disorders among psychoactive substance-dependent treatment clients. *Journal of Personality Disorders, 10*(1), 56-67.

Forouzan, E., & Cooke, D. J. (2005). Figuring out la femme fatale: Conceptual and assessment issues concerning psychopathy in females. *Behavioral Sciences and the Law*, *23*, 765-778.

Forth, A. E., Brown, S. B., Hart, S. D., & Hare R. D. (1996). The assessment of psychopathy in male and female noncriminals: Reliability and validity. *Personality and Individual Differences*, *20*(5), 531-543.

Garrido, V., Esteban, C., & Molero, C. (1995). The effectiveness in the treatment of psychopathy: a meta-analysis. *Issues in Criminological and Legal Psychology*, 24, 57-59.

Gordon, H. L., Baird, A. A., & End, A. (2004). Functional Differences Among Those High and Low on a Trait Measure of Psychopathy. *Biological Psychiatry*, 56, 516-521.

Gray, J. A. (1990). Brain systems that mediate both emotion and cognition. *Cognition & Emotion, 4*(3), 269-288.

Gregg, T. R., & Siegel, A. (2001). Brain structures and neurotransmitters regulating aggression in cats: Implications for human aggression. *Progress in neuro-psychopharmacology & biological psychiatry, 25*(1), 91-140.

Hamburger, M. E., Lilienfeld, S. O., & Hogben, M. A. (1996). Psychopathy, gender, and gender roles: Implications for antisocial and histrionic personality disorders. *Journal of Personality Disorders, 10*(1), 41-55.

Hare, R. D. (1968). Psychopathy, autonomic functioning, and the orienting response. *Journal of abnormal psychology, 73*(3), 1-24.

Hare, R. D. (1980). A research scale for the assessment of psychopathy in criminal populations. *Personality and Individual Differences, 1*(2), 111-119.

Hare, R. D. (1982). Psychopathy and physiological activity during anticipation of an aversive stimulus in a distraction paradigm. *Psychophysiology, 19*(3), 266-271.

Hare, R. D. (1991). *The hare psychopathy checklist--revised*. Toronto, ON, Canada: Multi-Health Systems.

Hare, R. D. (1999). *Without conscience: The disturbing world of the psychopaths among us*. New York, NY, US: Guilford Press.

Hare, R. D. (2003). *Manual for the Hare Psychopathy Checklist, 2nd edition revised*. Toronto, ON. Multihealth Systems.

Hare, R. D., Harpur, T. J., Hakstian, A. R., Forth, A. E., Hart, S. D., & Newman, J. P. (1990). The revised psychopathy checklist: Reliability and factor structure. *Psychological Assessment, 2*(3), 338-341.

Hare, R. D., Harpur, T. J., & Hemphill, J. D. (1989). *Scoring pamphlet for the self-report psychopathy scale: SRP-II*. Unpublished manuscript, Simon Frasure University, Vancouver, British Columbia, Canada.

Hare, R. D., Hart, S. D., & Harpur, T. J. (1991). Psychopathy and the DSM-IV criteria for antisocial personality disorder. *Journal of abnormal psychology, 100*(3), 391-398.

Hare, R. D., & Herve H. F. (1999). *Hare P-Scan: Research version*. New York: Multi-Health Systems.

Hare, R. D., Frazelle, J., & Cox, D. N. (1978). Psychopathy and physiological responses to threat of an aversive stimulus. *Psychophysiology, 15*(2), 165-172.

Hare, R. D., & Jutai, J. (1983). Criminal history of the male psychopath: Some preliminary data. In Van Dusen, K. T., & S. A. Mednick (Eds.), *Prospective studies of crime and delinquency* (pp. 225-225-236). Boston, MA: Kluwer-Nijhoff.

Hare, R. D., & McPherson, L. M. (1984). Violent and aggressive behavior by criminal psychopaths. *International journal of law and psychiatry, 7*(1), 35-50.

Harlow, J. M. (1993). Classic text no. 14: Recovery from the passage of an iron bar through the head. *History of Psychiatry, 4*, 271-281.

Hare, R. D., & Quinn, M. J. (1971). Psychopathy and autonomic conditioning. *Journal of Abnormal Psychology, 77*(3), 223-235.

Harpur, T. J., Hakstian, A. R., & Hare, R. D. (1988). Factor structure of the psychopathy checklist. *Journal of consulting and clinical psychology, 56*(5), 741-747.

Harpur, T. J., Hare, R. D., & Hakstian, A. R. (1989). Two-factor conceptualization of psychopathy: Construct validity and assessment implications. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1*, 6-17.

Harris, G. T., Rice, M. E., & Cormier, C. A. (1991). Psychopathy and violent recidivism. *Law and Human Behavior, 15*(6), 625-637.

Hart, S. D., Forth, A. E., & Hare, R. D. (1991). The MCMI-II and psychopathy. *Journal of Personality Disorders, 5*(4), 318-327.

Hart, S. D., & Hare, R. D. (1989). Discriminant validity of the psychopathy checklist in a forensic psychiatric population. *Psychological Assessment, 1*(3), 211-218.

Hart, S. D., Hare, R. D., & Forth, A. E. (1994). Psychopathy as a risk marker for violence: Development and validation of a screening version of the revised psychopathy checklist. In J. Monahan, & H. J. Steadman (Eds.), *Violence and mental disorder: Developments in risk assessment.* (pp. 81-98). Chicago, IL, US: University of Chicago Press.

Hemphill, J. F., Hart, S. D., & Hare, R. D. (1994). Psychopathy and substance use. *Journal of Personality Disorders, 8*, 169-180.

Hesselbrock, M. N., Meyer, R. E., & Keener, J. J. (1985). Psychopathology in hospitalized alcoholics. *Archives of General Psychiatry, 42*(11), 1050-1055.

Hicks, B. M., Patrick, C. J. (2006). Psychopathy and negative emotionality: Analyses of suppressor effects reveal distinct relations with emotional distress, fearfulness, and anger-hostility. *Journal of Abnormal Psychology, 115*(2), 276-287.

Houston, R. J., Stanford, M. S., Villemarette-Pittman, N. R., Conklin, S. M. and Helfritz, L. E. (2003). Neurobiological correlates and clinical implications of aggressive subtypes. *Journal of Forensic Neuropsychology, 3*, 67-87.

Howard, R., & McCullagh, P. (2007). Neuroaffective processing in criminal psychopaths: Brain event-related potentials reveal task-specific anomalies. *Journal of Personality Disorders, 21*(3), 322-339.

Ishikawa, S. S., Raine, A., Lencz, T., Bihrle, S., & Lacasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of abnormal psychology, 110*(3), 423-432.

Jones, L. V., & Tukey, J. W. (2000). A sensible formulation of the significance test. *Psychological Methods, 5*(4), 411-414.

Justus, A. N., & Finn, P. R. (2007). Startle modulation in non-incarcerated men and women with psychopathic traits. *Personality and Individual Differences, 43*(8), 2057-2071.

Jutai, J. W., & Hare, R. D. (1983). Psychopathy and selective attention during performance of a complex perceptual-motor task. *Psychophysiology, 20*(2), 146-151.

Jutai, J. W., Hare, R. D., & Connolly, J. F. (1987). Psychopathy and event-related brain potentials (ERPs) associated with attention to speech stimuli. *Personality and Individual Differences, 8*(2), 175-184.

Kapp, B. S., Whalen, P. J., Supple, W. F., & Pascoe, J. P. (1992). Amygdaloid contributions to conditioned arousal and sensory information processing. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction.* (pp. 229-254). New York, NY, US: Wiley-Liss.

Karpman, B. (1948). The myth of the psychopathic personality. *American Journal of Psychiatry, 104*, 523-534.

Kennealy, P. J., Hicks, B. M., & Patrick, C. J. (2007). Validity of factors of the psychopathy checklist revised in female prisoners: Discriminant relations with antisocial behavior, substance abuse, and personality. *Assessment, 14*(4), 323-340.

Kiehl, K. A. (2000). *A neuroimaging investigation of affective, cognitive, and language functions in psychopathy* (Doctoral dissertation, University of British Columbia, Canada). *Dissertation Abstracts International: Section B: The Sciences and Engineering, 61*(5-B), 2766. US: ProQuest Information & Learning.

Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: Evidence for paralimbic system dysfunction. *Psychiatry research, 142*(2), 107-128.

Kiehl, K. A., Bates, A. T., Laurens, K. R., Hare, R. D., & Liddle, P. F. (2006). Brain potentials implicate temporal lobe abnormalities in criminal psychopaths. *Journal of abnormal psychology, 115*(3), 443-453.

Kiehl, K. A., Hare, R. D., Liddle, P. F., & McDonald, J. J. (1999). Reduced P300 responses in criminal psychopaths during a visual oddball task. *Biological Psychiatry, 45*(11), 1498-1507.

Kiehl, K. A., Hare, R. D., McDonald, J. J., & Brink, J. (1999). Semantic and affective processing in psychopaths: An event-related potential (ERP) study. *Psychophysiology, 36*(6), 765-774.

Kiehl, K. A., Laurens, K. R., Bates, A. T., & Liddle, P. F. (2006). Psychopathy and semantic processing: An examination of the N400. *Personality and Individual Differences, 40*(2), 293-304.

Kiehl, K. A., Smith, A. M., Hare, R. D., & Liddle, P. F. (2000). An event-related potential investigation of response inhibition in schizophrenia and psychopathy. *Biological Psychiatry, 48*(3), 210-221.

Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., et al. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological psychiatry, 50*(9), 677-684.

Kim, M., & Davis, M. (1993). Electrolytic lesions of the amygdala block acquisition and expression of fear-potentiated startle even with extensive training but do not prevent reacquisition. *Behavioral neuroscience, 107*(4), 580-595.

Kirk, R. E. (1996). Practical significance: A concept whose time has come. *Educational and Psychological Measurement, 56*(5), 746-759.

Kosson, D. S., Steuerwald, B. L., Forth, A. E., & Kirkhart, K. J. (1997). A new method for assessing the interpersonal behavior of psychopathic individuals: Preliminary validation studies. *Psychological Assessment, 9*(2), 89-101.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review, 97*(3), 377-395.

Lang, P. J., & Greenwald, M. K. (1988). *The international affective picture system standardization procedure and initial group results for affective judgments: Technical reports 1A & 1B*. Gainsville: Center for Research in Psychophysiology, University of Florida.

Lang, P. J., Ohman, A., & Vaitl, D. (1988). The international affective picture system [Photographic slides]. Gainesville: Center for Research in Psychophysiology, University of Florida.

Larson, C. L., Ruffalo, D., Nietert, J. Y., & Davidson, R. J. (2005). Stability of emotion-modulated startle during short and long picture presentation. *Psychophysiology, 42*, 604-610.

LeDoux, J. E. (1992). Emotion and the amygdala. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction.* (pp. 339-351). New York, NY, US: Wiley-Liss.

LeDoux, J. E., Iwata, J., Cicchetti, P., & Reis, D. J. (1988). Different projections of the central amygdaloid nucleus mediate autonomic and behavioral correlates of conditioned fear. *Journal of Neuroscience, 8*(7), 2517-2529.

Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in a noninstitutionalized population. *Journal of Personality and Social Psychology, 68*(1), 151-158.

Levenston, G. K., Patrick, C. J., Bradley, M. M., & Lang, P. J. (2000). The psychopath as observer: Emotion and attention in picture processing. *Journal of abnormal psychology, 109*(3), 373-385.

Lilienfeld, S. O. & Andrews, B. P. (1996). Development and preliminary validation of a self-report measure of psychopathic personality traits in noncriminal populations. *Journal of Personality Assessment,* 66(3), 488-524.

Lilienfeld, S. O. & Widows, M. (2005). Professional Manual for the Psychopathic Personality Inventory-Revised (PPI-R). Lutz, FL: Psychological Assessment.

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *The Journal of Abnormal and Social Psychology, 55*(1), 6-10.

Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.

Lykken, D. T. (2000). The causes and costs of crime and a controversial cure. *Journal of personality, 68*(3), 559-605.

Lykken, D. T. (2006). Psychopathic personality: The scope of the problem. In C. J. Patrick (Ed.), *Handbook of the psychopathy.* (pp. 3-13). New York, NY, US: Guilford Press.

Lynam, D. R., Whiteside, S., & Jones, S. (1999). Self-reported psychopathy: A validation study. *Journal of Personality Assessment, 73*(1), 110-132.

Meyers, C. A., Berman, S. A., Scheibel, R. S., & Hayman, A. (1992). Case report: Acquired antisocial personality disorder associated with unilateral left orbital frontal lobe damage. *Journal of Psychiatry & Neuroscience, 17*(3), 121-125.

Mitchell, D. G. V., Avny, S. B., & Blair, R. J. R. (2006). Divergent patterns of aggressive and neurocognitive characteristics in acquired versus developmental psychopathy. *Neurocase, 12*(3), 164-178.

Mulder, R. T., Wells, J. E., Joyce, P. R., & Bushnell, J. A. (1994). Antisocial women. *Journal of Personality Disorders, 8*(4), 279-287.

Murphy, J. M. (1976). Psychiatric labeling in cross-cultural perspective. *Science, 191*(4231), 1019-1028.

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology, 95*(3), 252-256.

Newman, J. P., Kosson, D. S., Forth, A. E., & Hare, R. D. (2006). Factor structure of the Hare psychopathy checklist: youth version (PCL: YV) in incarcerated adolescents. Psychological Assessment, 18(2), 142-154.

Newman, J. P., MacCoon, D. G., Vaughn, L. J., & Sadeh, N. (2005). Validating a distinction between primary and secondary psychopathy with measures of Gray's BIS and BAS constructs. *Journal of Abnormal Psychology, 114*(2), 319-323.

Newman, J. P., Schmitt, W. A., & Voss, W. D. (1997). The impact of motivationally neutral cues on psychopathic individuals: Assessing the generality of the response modulation hypothesis. *Journal of Abnormal Psychology, 106*(4), 563-575.

Newman, J. P., Widom, C. S., & Nathan, S. (1985). Passive avoidance in syndromes of disinhibition: Psychopathy and extraversion. *Journal of Personality and Social Psychology, 48*(5), 1316-1327.

Nijs, I. M. T., Franken, I. H. A., & Smulders, F. T. Y. (2007). BIS/BAS sensitivity and the p300 event-related brain potential. *Journal of Psychophysiology, 21*(2), 83-90.

O'Connor, S., Bauer, L., Tasman, A., & Hesselbrock, V. (1994). Reduced P3 amplitudes are associated with both a family history of alcoholism and antisocial personality disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 18*(8), 1307-1321.

Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology, 31*(4), 319-330.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath: Startle reflex modulation. *Journal of abnormal psychology, 102*(1), 82-92.

Patrick, C. J., Cuthbert, B. N., & Lang, P. J. (1994). Emotion in the criminal psychopath: Fear image processing. *Journal of abnormal psychology, 103*(3), 523-534.

Patrick, C. J., Edens, J. F., Poythress, N. G., Lilienfeld, S. O., & Benning, S. D. (2006). Construct validity of psychopathic personality inventory two-factor model with offenders. *Psychological Assessment*, 18(2), 204-208.

Patrick, C. J., & Zempolich, K. A. (1998). Emotion and aggression in the psychopathic personality. *Aggression and Violent Behavior, 3*(4), 303-338.

Pinel, P. (1806). Section IV: Mental derangement distributed into different species: Second species of mental derangement. In P. Pinel (Ed.), *A treatise on insanity.* (pp. 150-156). London, United Kingdom: Messers Cadell & Davies, Strand.

Polich, J. (2007). Updating p300: An integrative theory of P3a and P3b. *Clinical Neurophysiology, 118*(10), 2128-2148.

Porter, S., Birt, A., & Boer, D. P. (2001). Investigation of the criminal and conditional release profiles of canadian federal offenders as a function of psychopathy and age. *Law and human behavior, 25*(6), 647-661.

Poythress, N. G., Edens, J. F., & Lilienfeld, S. O. (1998). Criterion-related validity of the psychopathic personality inventory in a prison sample. *Psychological assessment, 10*(4), 426-430.

Prichard, J. C. (1835). *A treatise on insanity and other disorders affecting the mind.* London, England: Sherwood, Gilbert, and Piper.

Raine, A. (1989). Evoked potential models of psychopathy: A critical evaluation. *International Journal of Psychophysiology, 8*, 29-34.

Raine, A., & Buchsbaum, M. S. (1996). Violence, brain imaging, and neuropsychology. In D. M. Stoff, & R. B. Cairns (Eds.), *Aggression and violence: Genetic, neurobiological, and biosocial perspectives.* (pp. 195-217). Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers.

Raine, A., & Venables, P. H. (1987). Contingent negative variation, P3 evoked potentials, and antisocial behavior. *Psychophysiology, 24*(2), 191-199.

Raine, A., & Venables, P. H. (1988). Enhanced P3 evoked potentials and longer P3 recovery times in psychopaths. *Psychophysiology, 25*(1), 30-38.

Raine, A., & Yang, Y. (2006). The neuroanatomical bases of psychopathy: A review of brain imaging findings. In C. J. Patrick (Ed.), *Handbook of the psychopathy.* (pp. 278-295). New York, NY, US: Guilford Press.

Rice, M. E., Harris, G. T., & Cormier, C. A. (1992). An evaluation of a maximum security therapeutic community for psychopaths and other mentally disordered offenders. *Law and human behavior, 16*(4), 399-412.

Rilling, J. K., Glenn, A. L., Jairam, M. R., Pagnoni, G., Goldsmith, D. R., Elfenbein, H. A., et al. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological psychiatry, 61*(11), 1260-1271.

Ross, S. R., Benning, S. D., & Adams, Z. (2007). Symptoms of executive dysfunction are endemic to secondary psychopathy: An examination in criminal offenders and noninstitutionalized young adults. *Journal of Personality Disorders, 21*(4), 384-399.

Salekin, R. T., Brannen, D. N., Zalot, A. A., Leistico, A., & Neumann, C. S. (2006). Factor structure of psychopathy in youth: Testing the applicability of the new four-factor model. *Criminal Justice and Behavior, 33*(2), 135-175.

Salekin, R. T., Rogers, R., & Sewell, K. W. (1997). Construct validity of psychopathy in a female offender sample: A multitrait, multimethod evaluation. *Journal of Abnormal Psychology, 106*(4), 576-585.

Salekin, R. T., Rogers, R., Ustad, K. L., & Sewell, K. W. (1998). Psychopathy and recidivism among female inmates. *Law and Human Behavior, 22*(1), 109-128.

Sandler, J. C. (2007). Computer equivalency of the psychopathic personality inventory revised in a nonincarcerated population. *Criminal Justice and Behavior, 34*(3), 399-410.

Saunders, J.B., Aasland, O.G., Babor, T.F., de la Fuente, J.R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption. II. *Addiction*, 88, 791-804.

Schwerdtner, J., Sommer, M., Weber, T., & Müller, J. (2004). fMRI an der forensischen klinik am bezirksklinikum regensburg - neuroanatomische grundlagen der emotionsverarbeitung. *Psychiatrische Praxis, 31*, S66-s67.

Sellbom, M., & Verona, E. (2007). Neuropsychological correlates of psychopathic traits in a non-incarcerated sample. *Journal of Research in Personality, 41*(2), 276-294.

Seto, M. C., & Barbaree, H. E. (1999). Psychopathy, treatment behavior, and sex offender recidivism. *Journal of Interpersonal Violence, 14*(12), 1235-1248.

Skeem, J., Johansson, P., Andershed, H., Kerr, M., & Louden, J. E. (2007). Two subtypes of psychopathic violent offenders that parallel primary and secondary variants. *Journal of abnormal psychology, 116*(2), 395-409.

Skeem, J. L., Poythress, N., Edens, J. F., Lilienfeld, S. O., & Cale, E. M. (2003). Psychopathic personality or personalities? exploring potential variants of psychopathy and their implications for risk assessment. *Aggression and Violent Behavior, 8*(5), 513-546.

Skinner, H. (1982). The drug abuse screening test, *Addictive Behaviors, 7*(4), 363-371.

Smith, R. J. (1999). Psychopathic behavior and issues of treatment. *New Ideas in Psychology, 17*, 165-176.

Smith, S. S. & Newman, J. P. (1990). Alcohol and drug abuse/dependence disorders in psychopathic and nonpsychopathic criminal offenders. *Journal of Abnormal Psychology, 99*, 430-439.

Soltani, M., & Knight, R. T. (2000). Neural origins of the P300. *Critical Reviews in Neurobiology, 14*(3), 199-224.

Stanford, M. S., Houston, R. J., Mathias, C. W., Villemarette-Pittman, N. R., Helfritz, L. E., & Conklin, S. M. (2003). Characterizing aggressive behavior. *Assessment, 10*(2), 183-190.

Stanford, M. S., Houston, R. J., Villemarette-Pittman, N. R., & Greve, K. W. (2003). Premeditated aggression: Clinical assessment and cognitive psychophysiology. *Personality and Individual Differences, 34*, 773-781.

Sutton, S. K., Vitale, J. E., & Newman, J. P. (2002). Emotion among women with psychopathy during picture perception. *Journal of Abnormal Psychology, 111*(4), 610-619.

Syndulko, K., Parker, D. A., Jens, R., Maltzman, I., & Ziskind, E. (1975). Psychophysiology of sociopathy: Electrocortical measures. *Biological Psychology, 3*(3), 185-200.

Thompson, B. (1998). In praise of brilliance: Where the praise really belongs. *American Psychologist, 53*(7), 799-800.

Uzieblo, K., Verschuere, B., Crombez, G. (2007). The psychopathic personality inventory: Construct validity of the two-factor structure. *Personality and Individual Differences*, 43, 657-667.

Vanman, E. J., Mejia, V. Y., Dawson, M. E., Schell, A. M., & Raine, A. (2003). Modification of the startle reflex in a community sample: Do one or two dimensions of psychopathy underlie emotional processing? *Personality and Individual Differences, 35*(8), 2007-2021.

Viding, E., Blair, R. J. R., Moffitt, T. E., & Plomin, R. (2005). Evidence for substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry, 46*(6), 592-597.

Vitacco, M. J., Rogers, R., Neumann, C. S., Harrison, K. S., & Vincent, G. (2005). A comparison of factor models on the PCL-R with mentally disordered offenders: The development of a four-factor model. *Criminal Justice and Behavior, 32*(5), 526-545.

Weaver, C. M., Meyer, R. G., Van Nort, J. J., & Tristan, L. (2006). Two-, three-, and four-factor PCL-R models in applied sex offender risk assessments. *Assessment, 13*(2), 208-216.

Widiger, T. A. & Corbit, E. M. (1995). Antisocial Personality Disorder. In J. W. Livesley (Ed.), *The DSM-IV Personality Disorders*. (pp. 103-126). New York, NY, US: Guilford Press.

Widom, C. S. (1977). A methodology for studying noninstitutionalized psychopaths. *Journal of consulting and clinical psychology, 45*(4), 674-683.

Williams, K. M., Paulhus, D. L., & Hare, R. D. (2007). Capturing the four-factor structure of psychopathy in college students via self-report. Journal of Personality Assessment, 88(2), 205-219.

Williams, K. M., & Paulhus, D. L. (2004). Factor structure of the self-report psychopathy scale (SRP-II) in non-forensic samples. *Personality and Individual Differences, 37*(4), 765-778.

Williamson, S., Hare, R. D., & Wong, S. (1987). Violence: Criminal psychopaths and their victims. *Canadian Journal of Behavioural Science Revue canadienne des Sciences du comportement, 19*(4), 454-462.

Williamson, S., Harpur, T. J., & Hare, R. D. (1991). Abnormal processing of affective words by psychopaths. *Psychophysiology, 28*(3), 260-273.

Yang, Y., Raine, A., Narr, K. L., Lencz, T., & Toga, A. W. (2006, October). Amygdala volume reduction in psychopaths. Poster presented at the annual meeting of the *Society for Research in Psychopathology*, San Diego, CA.

Zuckerman, M. (1977). Preliminary manual with scoring keys and norms for form V of the sensation seeking scale. Newark: University of Delaware.

Zuckerman, M. (2002). Personality and psychopathy: Shared behavioral and biological traits. In J. Glicksohn (Ed.), *Neurobiology of criminal behavior*. (pp. 27-49). Dordrecht, Netherlands: Kluwer Academic Publishers.

Zuckerman, M., Eysenck, S., & Eysenck, H. J. (1978). Sensation seeking in England and America: cross-cultural, age, and sex comparisons. *Journal of Counseling and Clinical Psychology, 46*, 139-149.