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HORMONE-BEHAVIOR CORRELATES AMONG MALE AND FEMALE PSYCHOPATHIC PARTICIPANTS: RELATIONSHIP TO GRAY'S MODEL OF BEHAVIORAL INHIBITION AND ACTIVATION SYSTEMS

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Abstract

This study aimed to identify the role of hormonal substrates in relation to Gray's (1987) model of the behavioral inhibition (BIS) and activation (BAS) systems—constructs proposed to serve a role in the etiology and/or maintenance of psychopathy. Meta-analytic studies have identified a positive relationship between testosterone and aggressive, antisocial, deviant types of behaviors, whereas cortisol has been associated with stress and anxiety. Additionally, a few studies have found reduced cortisol among antisocial, aggressive men. Given that the BIS is associated with inhibited and anxious responding, and the BAS with reward-seeking, impulsive, and sometimes antisocial behaviors, this study proposed that hormones such as cortisol and testosterone, respectively, may significantly correlate with BIS and BAS-driven responses in differentiating psychopaths and nonpsychopaths—a finding that would have important implications for future intervention research and for understanding the nature of this disorder. No study to date has assessed the correlates of both hormones with an experimental measure of BIS and BAS-functioning among male and female psychopathic individuals as this study has done.

Utilizing the Iowa gambling task, a behavioral measure commonly used to elicit BIS/BAS activity in response to mixed punishment and reward contingencies, this study's findings both corroborated and extended previous findings. Both male and female psychopathic analogues as compared to nonpsychopathic analogues responded to the task by utilizing high-risk, disadvantageous decisions. These findings support the notion that psychopathic individuals exhibit low punishment and high reward sensitivity, consistent with Newman's (1987) *response modulation deficit model*. Although some of the predicted hormone-behavior relationships were not obtained, exploratory post-hoc analyses revealed unexpected moderating relationships

between gender and hormone levels in predicting differential BIS-BAS motivated behaviors. Testosterone (post-task only) moderated the relationship between gender and performance on the gambling task, such that males with the highest testosterone levels during the task made significantly better decisions than males with low testosterone levels. Females with low testosterone levels exhibited significantly better performance than those with high testosterone levels. Additionally, basal cortisol levels also moderated the relationship between gender and task performance. Specifically, females with low basal cortisol levels made significantly more advantageous decisions as compared to males with low basal cortisol levels, such that low cortisol levels optimized female performance, but dampered male performance. The implications of these moderator relationships and the consistencies and inconsistencies obtained in this study as compared to previous literature are enumerated.

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Introduction

The term psychopath has traditionally been used to describe an individual who is characterized by an unsocialized, impulsive, guiltless, selfish, and calloused demeanor, and who fails to learn to alter his/her behavior based on experience (Hare, 1998). Various etiological theories have been proposed to serve as explanations for the commission of antisocial and aggressive behaviors in which psychopaths engage. Psychopaths have been characterized as having deficits in modulating affective experiences, exemplified by their inability to inhibit behaviors that lead to punishment, and by their excessive reward-seeking behaviors. Such deficits have been attributed to an imbalance of two biologically based motivational systems known as the Behavioral Inhibition (BIS) and Behavioral Activation (BAS) systems, according to Gray's (1987) model. The BIS inhibits behavior in response to punishment, whereas the BAS activates behavior in response to reward.

Explanations for the etiology and maintenance of the behaviors and personality traits exhibited by psychopathic individuals have been a topic of much debate. One avenue of research that has received a significant amount of attention and has yielded a significant amount of debate is in studies examining differences in the autonomic responsivity of psychopathic as compared to nonpsychopathic controls across various paradigms. A common theme that has emerged across years of research is that psychopaths must have some biological and/or autonomic deficit that underlies their inability to inhibit egregious behaviors that most individuals can and do inhibit in society. Whereas findings in the early literature argued that psychopaths experience less physiological reactivity to fearful, painful and aversive consequences, and hence have a "low fear quotient," (Lykken, 1957), subsequent studies found flaws in this conclusion. Similar to the low-fear hypothesis, other studies have argued that psychopathic individuals have a deficit in

their behavioral inhibition system, the mechanism proposed to enable individuals to inhibit behaviors in response to punishment. An alternative formulation is that, it is not a deficit in the BIS, but rather an overzealousness toward reward and hence, a hyperactive behavioral activation system that is responsible for the behaviors of these sensation-seekers and risk-takers. Other researchers have argued that it is some combination of the aforementioned deficits that seems to more effectively account for the etiology and maintenance of psychopathic characteristics. The present study aimed to empirically examine differences between psychopathic and nonpsychopathic participants on behavioral and personality measures as operationalizations of BIS and BAS-motivated activity that are proposed to underlie the psychopaths' tendency to make poor decisions and to fail to learn from punishment. Additionally, this study also examined potential hormone correlates that may underlie differential affective or arousal states in both psychopathic analogue and nonpsychopathic analogue groups. The present study proposed that various hormones, namely cortisol and testosterone, may serve as neuro-endocrinological markers underlying psychopaths' learning deficits. This formulation stems from the role of cortisol in activating the body's hypothalamic-pituitary axis (HPA) in response to stress, and the role of testosterone in underlying aggression and antisocial behaviors.

Learning Theory & Precursors to Gray's Model for the Etiology of Psychopathy

Predating Gray's enumeration of the theoretical constructs known as the BIS and the BAS, which are presumed to underlie the psychopathic individual's deficits in fear-conditioning, Mowrer's (1960) two-factor theory of avoidance learning laid the groundwork for understanding basic human socialization processes. Classical and operant conditioning paradigms have been fundamental to the understanding of the human socialization process—namely behaviors that are

associated with reward or positive reinforcers are increased and behaviors that are associated with punishment or negative consequences are likely to decrease over time across repeated pairings. Given this general fundamental framework guiding human behavior and socialization processes, the literature on psychopaths has consistently pointed to psychopaths' inability to respond to or to learn from punishment (e.g., Fowles, 1980; Hare, 1970, 1987a), also described as poor passive avoidance or poor fear conditionability (Arnett, 1997). Because passive avoidance, that is, inhibiting behaviors that have led to punishment in the past, has been identified as key to the socialization process (Trasler, 1978), these deficits have been the focus of much of the literature aimed at explaining etiological models of psychopathy.

According to Mowrer's (1960) theory, avoidance learning involves both classical conditioning and operant conditioning processes. Similar to Pavlov's conditioning or associated learning model, punishment is presumed to elicit a negative state (i.e., pain, discomfort, distress) that becomes associated with the behavior that immediately preceded the onset of the punisher. This association is then integral to the facilitation of the learning that occurs through operant conditioning processes. When an individual engages in actions that previously elicited punishment, these actions serve as the conditioned stimuli, evoking the negative punishment state and engendering a conditioned emotional response (CER). Because the CER produces an uncomfortable negative state, the individual learns to inhibit the particular sequence of behaviors that brought on the negative state—also known as a "passive avoidance" of punishment by inhibiting behaviors that have previously elicited punishment. Because the individual develops fear and anxiety in regard to the potential of reexperiencing the negative state, behaviors leading to punishment are typically extinguished. Subsequently, because the individual experiences a sense of relief and a reduction in fear by inhibiting these behaviors, this relief serves to reinforce

the passive avoidance. From Mowrer's theory (1960), any deficit in this conditioning sequence can result in poor passive avoidance learning in psychopathic individuals.

Subsequent research stemming from Mowrer's theory has primarily focused on identifying the mechanism underlying psychopaths' poor passive avoidance. A bulk of this literature has focused on differences in autonomic responsivity, that is, skin conductance and heart rate variability across psychopathic and nonpsychopathic individuals in anticipation of punishment paradigms. The premise tested in these paradigms is whether psychopathic individuals experience lower levels of physiological (autonomic) reactivity that prevents the establishment of an association between conditioned and unconditioned stimuli, and hence, underlies their deficits in passive avoidance learning. The studies involving anticipatory arousal to punishment paradigms have been thoroughly reviewed by Arnett (1997). Hare's countdown paradigms have been the most widely used to assess these anticipatory arousal differences. In these paradigms, Hare and others have measured palmar skin conductance reactivity in response to participants observing a counter (or similar indicator) that moves slowly, i.e., from 10 to 1, and participants are told that they will receive an aversive stimulus, such as a shock at the end of the countdown (Lykken, 1995). Across various studies, using both shock and an aversively loud noise as the anticipated stimulus, in comparison to controls, psychopathic individuals have demonstrated attenuated electrodermal activity (EDA) as measured by palmar sweating measures (i.e., Hare, 1965; Hare, Frazelle, & Cox, 1978). Hare (1978) and others (Kilzieh & Cloninger, 1993; Lykken, 1957, 1995) have interpreted this finding of low-level electrodermal arousal obtained in such experiments as an index of a lack of fear or anxiety, or at least a slowed fear arousal mechanism, which may be responsible for poor learning in aversive situations (also coined poor passive avoidance learning) among psychopathic groups.

Role of Emotions in Motivating behaviors: The Somatic Marker Hypothesis

Internal bodily states of arousal, mediated in part through the actions of the autonomic nervous system, have been postulated to greatly impact emotional, motivational, and social behaviors (Damasio, Tranel & Damasio, 1991; Damasio, 1999; Heims, Crtichley, Dolan, Mathias, & Cipolotti, 2004). Grounded in the tenets of the James-Lange theory of emotion, which concluded that the experience of an emotion is the direct result of the brain's perception of bodily arousal states, Damasio's groundbreaking hypothesis (1996) also underscored the importance of bodily arousal states in guiding subsequent behaviors (Heims et al., 2004). Damasio's (1996) *Somatic Marker Hypothesis* proposes that somatic feedback to the brain influences higher cognitive processes, such as human decision-making. An awareness of one's internal bodily states is presumed to underlie particular feeling states that may lead to an emotional experience, may motivate behavior, and may guide an individual's social interactions (Damasio, 1999). When choosing between options that differ in relative risk, Damasio argues that a somatic marker (i.e., signals sent via the spinal cord, the vagus and other cranial nerves to the brainstem) produces feedback to the brain to influence cognitive appraisal, likely without conscious awareness. In relation to the current study's focus, the somatic marker hypothesis specifically proposes that a defect in the processing of emotions and feelings plays an important role in impaired decision-making ability (Bechara, Damasio, & Damasio, 2000). This hypothesis was initially developed to explain the types of deficits exhibited by patients with damage to the ventromedial prefrontal cortex (VM). Whereas their intellectual capacities remained intact, as demonstrated by their normal performance on tasks entailing learning and memory, language and attention, and on tasks of executive functioning; however they demonstrated impairments in their processing of emotions and in their social behaviors (Bechara et al., 2000). Structures in the

ventromedial prefrontal cortex provide individuals with the ability to learn an association between situations and associated bioregulatory states (including emotional states). Given the similarities in the personality traits of patients with ventromedial prefrontal cortex damage to patients identified as psychopathic, the term *acquired sociopathy* has been used to describe the condition of patients with VM damage (Bechara et al., 2000). Given the need to study the decision-making deficits exhibited by VM patients, an instrument called the Iowa Gambling Task was developed to measure their impairments in the laboratory (Bechara, Damasio, Damasio, & Anderson, 1994), and has subsequently yielded parallel patterns of disadvantageous decisions among psychopathic individuals, as will be discussed in a later section.

Echoing the main tenets of the Somatic Marker hypothesis, emotions (or synonymously affective states) have been described as complex functions that promote adaptation (Davidson, 2003) by other investigators. Complex decisions are proposed to be guided by affective cues, such that decisions are essentially a confluence of emotions and cognitive processes involved in motivating particular approach or withdrawal behaviors. According to Davidson (2003), two core dimensions along which affect is organized are the approach and the withdrawal systems. The prefrontal cortex and the amygdala have been identified as the key structures that govern the approach and withdrawal components of emotion (Miller & Cohen, 2001). The prefrontal cortex is presumed to maintain the representation of goals and the means to achieve those goals. In situations that are unclear, the PFC sends signals to other regions of the brain to facilitate the expression of task-appropriate responses in the face of competing demands. For example, the pursuit of an immediate reward may not be in the best interest of the individual given other sources of peripheral information; in such a case, the PFC is expected to produce a signal to other brain regions that guide behavior toward the acquisition of a more adaptive goal, which

may entail delay of immediate gratification or inhibition of a particular behavior. Affect-guided planning and anticipation that involves the experience of emotion associated with an expected outcome is the hallmark of adaptive, emotion-based decision making.

Neurostructural System Underlying the BIS/BAS: Gray's Model

To explain the mechanisms involved in the control of emotional behavior and their potential relationship to the development of anxiety and impulsivity, Gray (1987) proposed a three-component arousal model. This model includes the behavioral inhibition system (BIS), which is believed to control the inhibition of ongoing behavior, the increase in vigilance, and the increase in arousal which can be produced by stimuli associated with pain, punishment, failure, loss of reward, novelty or uncertainty (Gray & McNaughton, 2000) and hence, organizes unconditioned responses to aversive events. It has been suggested that this mechanism operates by releasing Gaba-ergic signals (Lewis, 1991) to inhibit behavior in response to punishment or to threatening situations. A second component of the arousal model is the behavioral activation system (BAS), which organizes unconditioned behaviors in response to rewarding stimuli (Gray, 1987; Fowles, 1980) and also initiates active avoidance behaviors, or behaviors aimed at escaping punishment. Finally, the nonspecific arousal or fight/flight system mediates the interactions between these two systems by organizing behavior in response to unconditioned punishment or rewards. The underlying premise is that individuals differ in the way that they react to aversive and rewarding stimuli; thus, individuals with a hyperactive BIS may be more prone to experience extreme anxiety to aversive stimuli, and hence this serves to inhibit them from engaging in threatening, deviant behaviors. Individuals with a hyperactive BAS may

engage in more pleasure-seeking, rewarded behaviors or predatory aggressive types of behaviors, as well as pursue behaviors that lead to an avoidance or escape from punishment (Lykken, 1995).

The behavioral inhibition system (BIS) has thus been associated with the activation of passive-avoidance behaviors. Passive avoidance involves learning to inhibit previously punished behaviors, and is learned by the association of negative affect such as anxiety and remorse with the commission of certain types of behaviors, as controlled by the BIS. Via conditioning, punishment has attached a fear response to a desired behavior, and hence that behavior is inhibited in the future. Active avoidance refers to learned behaviors that involve actively escaping from danger and/or behaviors that promote receiving some type of a reward, as controlled by the BAS. Stimuli associated with the escape from fear or the avoidance of expected punishment act like positive rewards or relief from aversive stimuli.

According to Gray (1982) the septo-hippocampal system (SHS) underlies the functioning of the hypothetical behavioral inhibition system (BIS) that mediates individuals' reactions to punishment, and is implicated in the explanation of anxious and impulsive presentations. It includes the hippocampus, the dentate gyrus, the entorhinal cortex, the subicular area, and the posterior cingulate cortex. The septohippocampal system and its control of theta (inhibitory GABAergic input) in the hippocampus have been identified as the most important neural component of the BIS (Mcnaughton & Gray, 2001). A major function of the SHS is to act as an information processing system to help initiate or guide behaviors based on input received from the environment, namely to detect goal conflicts, especially those involving approach-avoidance decisions. Lesion data from earlier studies (Gray 1970a; Gray & McNaughton, 1983) have established that the septo-hippocampal system is responsible for the functioning of the BIS (Gray & McNaughton, 2000). The adaptive functions of this system have been thought to include

interruption of ongoing actions that may result in aversive consequences, preparation for active coping strategies (i.e., fight or flight), and facilitation of learned associations acquired from environmental exposure so that learned consequences are readily available in the future to aid in inhibiting future maladaptive behaviors (Patterson, Kosson & Newman, 1987). According to Patterson et al. (1987), the SHS codes all types of anxiety, whereas the amygdala mediates the fear that underlies both active and passive avoidance tendencies and controls behavioral avoidance related to anxiety. Using their model, the BIS is perceived to be an information-processing system that receives extensive information from cortical areas, interacts with the amygdala-based defensive (fear) system and produces inhibition of behavior (McNaughton & Gray, 2001).

Gray and Smith (1969) developed a neuropsychological model of approach-avoidance learning, illustrated as Figure 1, which has been described as a model for conflict and discrimination learning (Gray, 1987). The model finds it origins in experiments in which the brains of rats were implanted with electrodes, and then the rats were allowed to press a bar which either caused a small electric current to flow in their brains or alternatively, turned off a current which the experimenter caused to flow in their brains. With certain placements of electrodes, the rat pressed a bar to stimulate its own brain electrically for hours, and with other placements, the rat terminated the bar pressing in order to prevent the occurrence of the electrical stimulation. Gray explained that these results lend support for the existence of two fundamental motivation systems in the brain, a "reward" and a "punishment" mechanism respectively. He elaborates on the principles of classical conditioning and their relationship to his model of conflict and discrimination learning, noting that stimuli that regularly precede the occurrence of a reward acquire the capacity to activate the reward mechanism. This reward mechanism (BAS) is

connected with the "motor" system to facilitate acquisition of innately rewarding stimuli, and hence the reward mechanism facilitates "approach" behaviors. Similarly, stimuli that regularly precede the occurrence of punishment, via classical conditioning, acquire the capacity to activate the punishment mechanism in the brain. The punishment mechanism (BIS) is also presumed to have a direct connection with the motor system, and serves to minimize its own inputs, that is, it serves to put a brake on behavior, or to facilitate passive avoidance behavior, in order to avoid the experience of aversive consequences. Hence, this "punishment" mechanism has been coined the behavioral inhibition system (BIS), as it is concerned with the suppression of responses that lead to punishment.

Besides the reward, punishment, and motor systems (see Figure 1), Gray describes a "decision mechanism" that serves to make decisions between approach and avoidance under conditions of conflict. Such a system serves to reduce the sending of conflicting messages directly to the motor system. The decision mechanism, according to Gray (1987), either shuts the switch en route from the reward mechanism to the behavioral command to approach or shuts the switch en route from the punishment mechanism to inhibit behaviors, but not both. The magnitude of the inputs to the arousal mechanism from both the reward and punishment mechanisms is presumed to determine the subsequent motor behavior to approach or to avoid the stimulus.

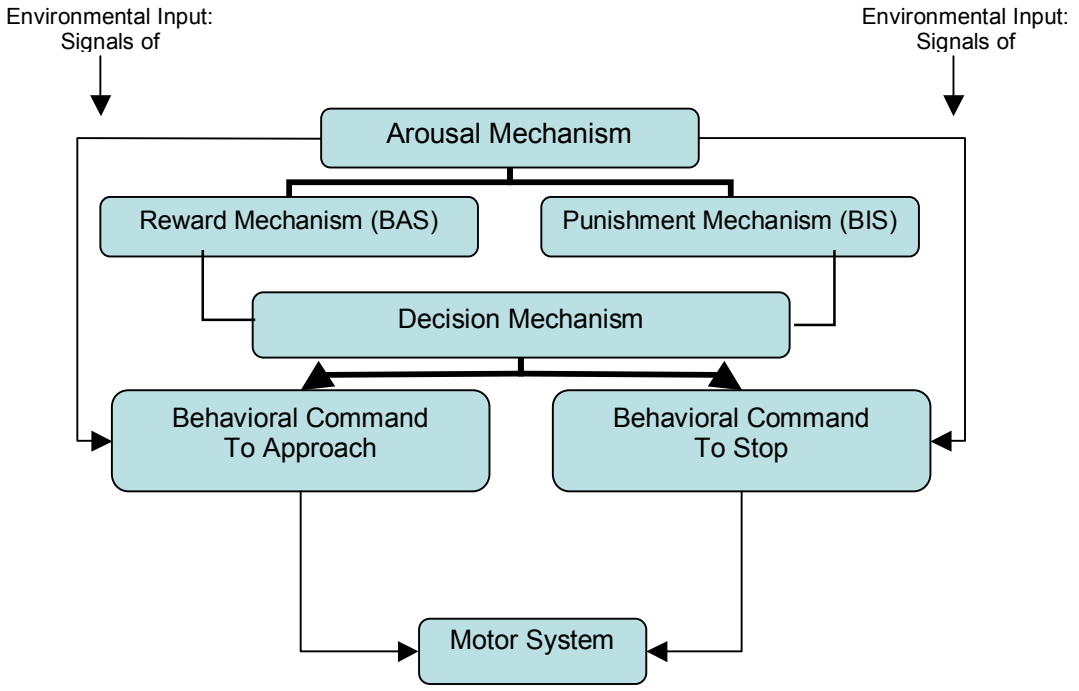


Figure 1. Gray & Smith's (1969) neuropsychological model of approach-avoidance learning adapted from *The Psychology of Fear and Stress* by J.A. Gray, 1987, New York: Cambridge University Press.

Applications of Gray's Model to the Etiology of Psychopathy

Whereas Gray's work primarily focused on anxiety and to a lesser extent on impulsivity using the septal nuclei of animal models to elaborate his theories, other researchers (e.g., Gorenstein & Newman, 1980; Newman, 1987; Arnett, Smith & Newman, 1997; Arnett & Newman, 2000) have relied on Gray and Smith's (1969) model as a springboard to further understand potential dysfunctional systems underlying psychopathic personality and behaviors. The focus of Gray's work was on the sensitivity to signals for punishment and for reward as the psychological processes underlying personality traits such as anxiety and impulsivity. However, Newman's model (1987) focuses more on *disinhibition* and the amount of *reflectivity* engaged in by individuals following punishment. Much of Newman and colleagues' laboratory research has focused on a number of behavioral syndromes characterized by impulsive, disinhibited behavior,

referred to as the "syndromes of disinhibition" (Gorenstein & Newman, 1980). These include psychopathological disorders such as psychopathy and hyperactivity as well as nonpathological forms of impulsivity such as extraversion. Newman (1987) posits that these various *disinhibition syndromes* share a psychological diathesis that may be better understood relative to Gray's (1987) animal model involving dysfunction of the septal nuclei.

Although their line of research on impulsivity shares many commonalities and in essence complements Gray's work on anxiety, there is one notable difference in the focus of the research between Newman's and Gray's work. According to Newman's (1987) conceptualization, impulsivity entails rapid action and absence of adequate forethought in addition to poor judgment, because impulsive responding fails to take into account the range of potential consequences for a given action or a more efficient means of achieving the same goal. Implicit in this conceptualization of impulsive behavior is the notion that pausing to reflect facilitates acquiring information that would be helpful in guiding subsequent behavior, that is, processing information that guides an altered response to a given situation. The failure to make use of situational cues to modulate instinctive responding is key to the understanding of impulsive behavior, and provides the impetus for Newman's investigations into disinhibited participants' ability to use discriminative stimuli to modulate responding on various go/no-go discrimination tasks to examine passive avoidance behaviors. Hence, whereas Gray (1987) primarily focuses on sensitivity to signals for reward and punishment, Newman's (1987) primary focus is on the disinhibition and degree of reflectivity following punishment cues (Newman, 1987).

In one investigation (Newman, 1987), participants won tokens worth monetary rewards for pressing a button during the display of a "good number" and lost tokens during the display of "bad numbers." Errors of commission (responding to bad numbers) were operationalized as

passive avoidance errors, because they represent a failure to inhibit responses that result in punishment. The assumption underlying the task is that if impulsive participants are deficient in learning to use environmental cues to regulate approach behavior, then they should commit more passive avoidance errors compared to controls. The results of this study and from several of their previous investigations support this prediction with extraverts compared to introverts (Newman, Widom, & Nathan, 1985), and with incarcerated psychopaths compared to nonpsychopaths (Newman & Kosson, 1986), in that disinhibited participants made significantly more commission errors. In a study that further divided juvenile delinquent psychopathic offenders along the dimension of anxiety level, similar results were obtained in that primary (low-anxious) psychopaths made significantly more commission errors than the secondary (high-anxious) psychopaths and the nonpsychopathic controls (Newman et al., 1985).

In another study, Newman and Kosson (1986) demonstrated evidence of what the authors coin as "response perseveration" among the psychopathic participants. That is, whereas controls (nonpsychopathic offenders) adjusted their responses accordingly on a card-playing task as the probability of punishment increased across the successive trials, psychopathic offenders failed to alter their dominant response set for reward, reflecting potential deficits in the balance between BIS and BAS-motivated activity. In addition, in their immediate feedback condition, the majority of the psychopaths (9 of 12) never quit or suspended their approach behaviors (playing all 100 cards) despite losing money on 19 of the last 20 trials. In summary, psychopaths' response persistence compared to the controls that adjusted their responses accordingly to avoid further monetary loss was maladaptive, resulting in their earning significantly less money than the controls.

The combined results of these studies provide consistent evidence that disinhibited participants are less likely to inhibit punished responses while they are in pursuit of reward (approach behaviors). According to their previously elucidated "septal model" (Gorenstein & Newman, 1980), the impulsive responding of disinhibited participants reflects a response modulation deficit related to their inability to alter a dominant response set, that has been termed "response perseveration." Response perseveration refers to the tendency to continue a response set for reward despite punishment or changes in environmental contingencies that reduce the adaptiveness of continued responding (Newman, Patterson, & Kosson, 1987). Hence, one interpretation of these data suggests that once disinhibited individuals have focused on behaviors aimed at obtaining reward, they are less likely to interrupt their response set to consider cues for punishment that signal the need for behavioral inhibition.

To test alternative interpretations, they (Newman, 1987) investigated the performance of disinhibited and control participants on the same go/no go discrimination task using punishment only or reward only contingencies. When they eliminated the competing or conflicting contingencies of reward and punishment, they failed to observe a significant difference in the performance of disinhibited participants in comparison to their counterparts. These data seem to refute the findings of other investigators that suggest that disinhibited individuals (notably psychopaths) are insensitive to punishment (i.e., Lykken, 1957) and are characterized by a weak BIS, such that their behavioral and physiological responses to punishment cues are insufficient to interrupt approach behaviors (Fowles, 1980). Such findings would also seem to be inconsistent with the notion that psychopathic individuals are hypersensitive to reward cues (strong BAS model) that elicit a bias toward approach behaviors (Arnett, Smith, & Newman, 1997; Newman et al., 1985; 1986). Across Newman's and his colleagues' studies as presented, the group

differences across psychopathic and nonpsychopathic participants in response modulation are salient only in situations of competing approach-avoidance contingencies, lending support to their *response modulation model* (Newman et al., 1987; Patterson, Kosson, & Newman, 1987).

Information-processing deficits have been postulated to underlie psychopaths' behavioral and affective symptoms, specifically that psychopaths are deficient in response modulation (Bernstein, Newman, Wallace, & Luh, 2000). Response modulation has been defined as a brief and automatic shift of attention from effortful organization and implementation of goal-directed behaviors to process peripheral information. This model posits that psychopaths are deficient in processing and utilizing contextual cues that interfere with their dominant or goal-directed behavior to effectively regulate their behaviors (Bernstein et al., 2000). Psychopathic individuals are presumed to have difficulties in shifting attention away from a goal or reward-motivated behavior to process other important incoming information, such as attending to negative consequences of their behaviors or emotional cues that could lead to better choices. Using this model, Newman et al. (1987) assert that psychopathic individuals are equally responsive to rewards and punishments in most situations. However, they have difficulty inhibiting a dominant response set for reward when cues for punishment are present, because they are less adept at interrupting or modulating BAS-driven activity, which may partly be due to their failure to pause and process aversive feedback (Arnett et al., 1997).

Based on findings (Newman et al., 1987; Patterson & Newman, 1993; Arnett et al., 1997) gained from their systematic research with psychopathic individuals, the current study makes predictions that are consistent with their *response modulation model.* A related variant of Newman et al.'s (1987) *response modulation model* is the *motivational imbalance model* (Fowles, 1980; Gray, 1987; Arnett, 1997), in which a combination of reduced punishment

sensitivity plus heightened reward dependency are posited as key aspects in understanding the etiology and maintenance of psychopathy. Consistent with the motivational imbalance model, analogue psychopathic participants were found to make more disadvantageous decisions on the Iowa Gambling task (Bechara, Damasio, Damasio, & Anderson, 1994), marked by low punishment sensitivity and high reward dependency as compared to nonpsychopathic participants (Van Honk, Hermans, Putman, Montagne, & Schutter, 2002).

The Iowa Gambling Task

In the current study, analogue psychopathic and nonpsychopathic participants were asked to engage in the Iowa gambling task (IGT; Bechara et al., 1994). This neuropsychological tool has been effectively used to simulate punishment-reward contingencies in a real-life manner (Van Honk et al., 2004). The Iowa Gambling Task instructs players to try to gain as much money as possible by drawing cards from four different decks, with the stated goal of the game being to win as much and to avoid losing as much money as possible. The task is designed so that decisions to choose from decks are influenced by reward and punishment schedules inherent in the task. Two of the decks are disadvantageous, producing immediate large rewards, but these are (after a pre-punishment phase of about 10-15 cards) accompanied by significant money loss due to more extreme punishments. The other two decks are advantageous, in that rewards are modest but more consistent and punishment remains low (Bechara, Damasio, Damasio, & Lee, 1999).

Although suitable normative information has yet to be published, the Iowa Gambling Task (IGT) has been described as a well-established measure of the role of emotion in decision-making in that it demonstrates the extent to which learning based on emotion is useful in dealing

with complex problem-solving situations (Evans, Kemish, & Turnbull, 2004). Despite the lack of available psychometric data attesting to the validity and reliability of the task, the IGT has been used in over 100 studies to date since its development in 1994 (Bechara, Damasio, Damasio & Anderson, 1994) across a variety of neurological and psychiatric populations. Performance differences on the IGT have been found to be robust across various studies when investigators have used a manual versus a computerized administration of the task (Bechara, Damasio, & Damasio, 2000a), have compared the use of real versus fake monetary rewards (Bowman & Turnbull, 2003), and most recently when performance on a manual, a computerized version with time delay between card selections, and on a computerized version without a time delay was compared (Bowman et al., 2005).

Its widespread use has been attributed to its utility in assessing an aspect of executive function, namely emotion-based learning (Damasio, 1994) that had previously been difficult to assess (Bowman, Evans, & Turnbull, 2005). The IGT has been plauded for its ability to capture a deficit in patients with lesions to the ventromesial frontal lobes that clinical neuropsychologists have been identifying but have not had a measure by which to quantify the extent of this population's deficits (Evans et al., 2004). These brain injured patients exhibit deficits in making effective life choices despite good performance on other measures of neuropsychological functioning, including measures of executive function (Damasio, 1996). Initially developed as an instrument to further study the decision-making impairments of patients with ventromedial lesions, the IGT has been used to corroborate the tenets of the Somatic Marker Hypothesis.

Several prominent researchers agree that the IGT is a good measure for assessing the role of emotion in complex decision-making, sometimes referred to as emotion-based learning (Turnbull, Evans, Bunce, Carzolio & O'Connor, 2004; Bechara, Damasio, & Damasio, 2000).

Whereas neurological patients with ventromesial frontal lobe lesions have shown normal intelligence and relatively normal performance on executive tasks (e.g., Bechara et al., 2000), they have pronounced difficulties in social relationships and exercise poor judgment in making decisions. Their poor decision-making skills have been attributed to an inability to use emotion-learning systems, which provide information about the outcomes of particular decisions and their possible emotional consequences (Bechara et al., 2000). Patients with VM lesions have often been described as having 'acquired sociopathy' following their orbitofrontal damage, and have pronounced hyporeactive autonomic responses in the recognition of emotions (Blair & Cipolotti, 2000). Likewise, studies with psychopathic individuals have demonstrated similar findings in that psychopathic individuals fail to attend to emotional cues signaling punishment, fear, pain etc., and have exhibited blunted autonomic arousal to sad and fearful emotional expressions in particular (i.e., Blair, Jones, Clark, & Smith, 1997). Consistent with predictions made by the Somatic Marker Hypothesis, results from psychophysiological measures have revealed that VM patients fail to generate the anticipatory skin conductance responses that normals develop before selecting cards from the disadvantageous decks on the gambling task, and hence, may lack the experience of a somatic signal that is useful in guiding behaviors (Bechara, 2000). Given the shared characteristics between psychopathic individuals and VM patients in their presumed emotional deficits, and subsequently in their proclivity to make more risky decisions, the Iowa Gambling task has become a useful instrument in research comparing psychopathic and control participants. In the present study, both male and female psychopathic participants were predicted to exhibit a responding pattern consistent with the tenets of the motivational imbalance model, that is, a tendency to perseverate on decks that yield high rewards, while failing to

process the cues for even higher monetary losses that are paired with the high-yield decks ("bad" decks).

Psychopathy and Anxiety

Although the hypothesis of a reduced capacity for fear and anxiety (Lykken, 1957) has pervaded the literature on explaining psychopaths' disregard for others and their lack of empathy, subsequent empirical evidence for an inverse relationship between psychopathy and self-reported anxiety has been equivocal (Hale, Goldstein, Abramowitz, Calamari, & Kosson, 2004). Whereas attenuated skin conductance reactivity has been a fairly robust finding distinguishing psychopathic from nonpsychopathic individuals across experiments assessing anticipatory anxiety, studies examining the hypothesis that psychopaths may have lower baseline levels of arousal and fearfulness (lower resting skin conductance levels) have yielded inconclusive findings (Siever, 1998; Hare & Cox, 1978) to date. Recent studies have failed to find a significant relationship between psychopathy as measured by the Psychopathy Checklist Revised (PCL-R; Hare, 1991) and numerous measures of self-report anxiety and fear scales in large samples of Caucasian and African-American incarcerated men (Schmitt & Newman, 1999; Ghebrial & Arnett, 2003 manuscript submitted for publication). One criticism of Schmitt and Newman's (1999) study is that they utilized trait measures of anxiety that may fail to capture the state anxiety central to the construct of the BIS in Gray's model. Ghebrial and Arnett (2003) found that the BIS items from Carver and White's (1994) BIS/BAS scales also failed to differentiate psychopathic from nonpsychopathic offenders. Whereas in both of the aforementioned studies, self-report measures of fear, anxiety, and BIS-related activity failed to yield significant differences in the endorsement of these constructs, it may be that psychopathic

individuals are less willing to admit to or are not aware of these traits, and hence, fail to self-report them. Since BIS-related constructs are presumed to reflect trait anxiety and to underlie avoidance learning (Patterson & Newman, 1993), psychopaths' deficits in avoidance learning (i.e. Hare, 1978) may not reflect inherent trait characteristics, but rather may emerge under certain circumstances as empirical evidence has shown (Newman & Kosson, 1986).

Whereas studies examining the relationship between overall PCL-R (Hare, 1991) psychopathy and measures of anxiety have yielded equivocal results, recent studies have obtained more consistent relationships between psychopathy and anxiety when psychopathy scores are parsed into factor scores (e.g., Hale et al., 2004; Patrick, 1994;). Factor analyses of the PCL-R have identified two distinct, but correlated factors (Harpur, Hare, & Hakistan, 1989), with more recent accounts suggesting that the PCL-R may be better represented by three factors (Cooke and Michie, 2001). Factor 1 assesses the emotional-interpersonal characteristics (i.e., lack of empathy and remorse; charm; grandiosity; manipulativeness, etc.) often identified as the Primary psychopathy traits, whereas Factor 2 assesses the antisocial lifestyle or chronic social deviance characteristics (i.e., early behavior problems, delinquency, aggressiveness, impulsiveness, sensation-seeking, etc.) often identified as Secondary psychopathy traits (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003).

To distinguish between primary and secondary or Factor 1 and Factor 2 psychopathy, investigators have traditionally used self-report measures of neurotic anxiety, such as the Welsh Anxiety Scale (WAS; Welsh, 1956) to subdivide psychopathic individuals (Newman, MacCoon, Vaughn, & Sadeh, 2005). The practice of using anxiety to parse the heterogeneity of psychopathy has received support from a substantial literature base (e.g., Newman, Widom, & Nathan, 1985; Schmitt & Newman, 1999; Arnett, Smith, & Newman, 1997) that has

demonstrated that high and low-anxious psychopathic individuals exhibit differential performance deficits across studies (Newman et al., 2005). Trait anxiety scores have been found to correlate negatively with Factor 1 and positively with Factor 2, yielding an overall null relationship with PCL-R total scores (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999). Patrick (1994) reported that self-report measures of fear and emotional distress were negatively related to PCL-R Factor 1 scores after controlling for PCL-R Factor 2, and positively related to Factor 2 after controlling for Factor 1. Other reasons for differentiating psychopathic groups along the dimension of anxiety have been enumerated by Arnett, Smith, and Newman (1997). One reason is that the absence of neurotic features has been considered a cardinal feature of psychopathy (e.g., Cleckley, 1976; Fowles, 1980). Thus, low-anxiety psychopathic individuals as delineated by WAS scores are more likely to be true or primary psychopathic individuals. Also, a pattern has emerged across past literature of finding poorer passive-avoidance learning or BIS-motivated deficits among low-anxiety psychopathic individuals.

Given the established method and reasons for assessing differences among high and low anxious psychopathic individuals and controls, in addition to very recent further empirical support for the use of anxiety as measured by the WAS to distinguish primary and secondary psychopathy (Newman et al., 2005), the current investigation also assessed the moderating role of anxiety using WAS median-split scores. This study sought to specifically assess differences in the choices made by psychopathic and nonpsychopathic participants, using a gambling task to assess BIS and BAS-BIS motivated approach-avoidance behavior when immediate feedback regarding monetary losses and gains is provided. Whereas some decks on the Iowa Gambling task offer large monetary gains, they simultaneously involve even higher losses. Given the findings that psychopaths are less likely to alter a dominant response set for reward, it was

expected that they would make more selections from the disadvantageous decks, despite the significant monetary losses. An examination of psychopathy and psychopathy score as a function of WAS anxiety scores are assessed in predicting BIS/BAS activity. Additionally, this study examined the role of another biological substrate, namely hormonal correlates, to examine any potential differences in the functioning of the hypothalamic-pituitary axis (HPA) among psychopathic and nonpsychopathic individuals that may serve a role in facilitating differential behaviors guided by BIS/BAS activity.

The Neuro-Endocrine System: A Mechanism for Activating Hormone-Behavior Relationships

The organizing mechanisms of behavior are found in the brain, the rest of the nervous system, and in the network of hormonal pathways known as the endocrine system. According to Gray (1987), these systems function so closely together that they are best referred to in an inter-dependent way as the "neuro-endocrine system." The nervous system is a complex network of interconnected nerve fibers that is comprised of the central and peripheral nervous systems Chrousos & Gold, 1992; Habib, Gold, & Chrousos, 2001); the former consisting of the brain and spinal cord, and the latter of the rest of the nerves in the body that transmit information to the brain and spinal cord (Taylor, 1999). Sensory nerve fibers provide input to the brain and spinal cord from sensory receptors; whereas motor nerve fibers provide output from the brain or spinal cord to muscles and organs resulting in voluntary and involuntary movement.

The peripheral nervous system consists of two parts, the somatic (or voluntary) nervous system that connects nerve fibers to muscles and provides the brain with feedback regarding voluntary movement, and the autonomic, or involuntary nervous system. The autonomic nervous system (ANS) is the portion of the nervous system that connects the central nervous

system with all the internal organs over which individuals do not have direct control, and is the most relevant to this study. The ANS has two branches, the sympathetic nervous system, which mobilizes the body for action in response to threat or stressor cues, whereas the parasympathetic nervous system is responsible for restoring the body's equilibrium and store of resources. The hypothalamus controls and coordinates the activities of the autonomic nervous system and the endocrine system; hence, this part of the brain is key in the regulation of various forms of emotional behavior. For the purpose of this study, the functioning of the ANS was the primary focus, and specifically the response of the sympathetic nervous system, and its role in facilitating behaviors vis`a vis an interdependent relationship with the endocrine system.

The endocrine system, which is comprised of ductless glands that secrete hormones into the blood, complements the nervous system in controlling bodily activities (Taylor, 1999). The endocrine and nervous systems are inter-dependent structures, working to stimulate and inhibit each other's activities. The endocrine system is regulated by the hypothalamus and the pituitary gland. The lines of communication between the endocrine glands and the central nervous system are mediated by the anterior pituitary gland. Neurohormones are secreted by the hypothalamus, a part of the brain situated immediately the anterior pituitary gland. This gland is then stimulated to secrete into the bloodstream particular types of hormones, based on the neurohormones or releasing factors relayed from the hypothalamus. The pituitary produces hormones that affect other glands and promote the production of other hormones. Hormones are specialized biochemicals that are able to regulate various functions of the bodily organs that they reach by traveling through the bloodstream. Whereas the anterior pituitary lobe secretes various hormones, some of which are responsible for growth, development, etc, adrenocorticotropic hormone (ACTH) is the hormone responsible for controlling the secretions of the adrenal glands

and stimulating the release of the hormones known as steroids that are the focus of the present study.

Major components of the endocrine system are the adrenal glands, two small glands located on top of each of the kidneys. Each adrenal gland consists of an adrenal medulla and an adrenal cortex. The adrenal medulla is responsible for the secretion of catecholamines, namely epinephrine and norepinephrine, used to modulate such bodily functions as heart rate, blood pressure, blood flow to muscles, and breathing. The adrenal cortex is stimulated by the adrenocorticotropic hormone (ACTH) from the anterior pituitary lobe to secrete corticosteroids, including androgens, estrogens, and cortisol, which aid in mobilizing the body's proteins and fats, energy resources, and other regulatory functions. The adrenal glands are critically involved in mobilizing bodily reactions to stress. In response to stress, the release of corticosteroids and catecholamines activates the sympathetic branch of the nervous system to increase arousal, to mobilize the body's resources, for example by increasing breathing rate and heart rate, facilitating pupil dilation, and diverting blood to the muscle tissues to activate the fight/flight response (refer to Figure 2 below).

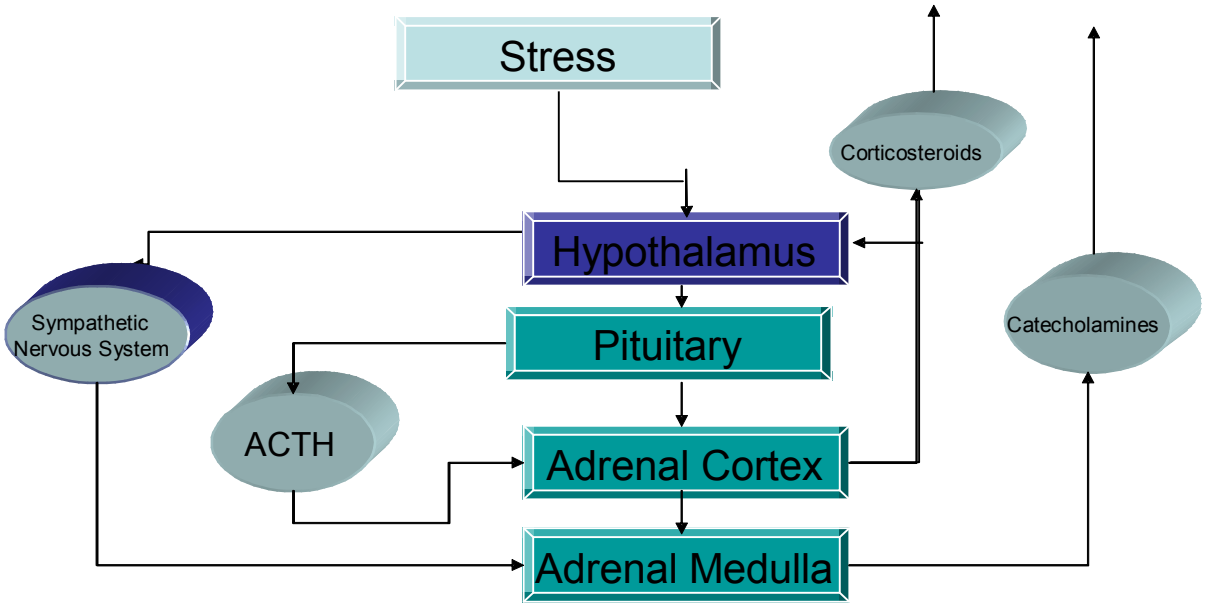


Figure 2. The hypothalamic-pituitary-adrenocortical axis (HPA) & the sympathetic adrenomedullary (SAM) system's response to stress. Adapted from Taylor, S.E. (1999). Health Psychology, 4th ed. What is stress?

Two interrelated systems are involved in responding to stressful cues from the environment, the sympathetic adrenomedullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis (Taylor, 1999). When an event is perceived as harmful or threatening, it is recognized as such by the cerebral cortex, which in turn sets off a chain of reactions mediated by these appraisals. Information from the cortex is transmitted to the hypothalamus, the brain's relay system, which initiates one of the earliest responses to stress, namely sympathetic nervous system arousal, or the body's fight-or-flight response as first described by Cannon (1932). Sympathetic arousal stimulates the adrenal medulla glands, which in turn, secrete the catecholamines, epinephrine and norepinephrine, activating the body's resources to respond to the stressor (i.e., increased blood pressure and heart rate, etc.). In addition to the activation of the sympathetic nervous system, the HPA axis is also activated. The effect of stress on the HPA

was initially elucidated by Selye (1956) in his general adaptation syndrome, which involves the nonspecific physiological reaction that occurs in response to stress, and the three stages of alarm, resistance, and exhaustion. The hypothalamus releases corticotrophin-releasing factor (CRF), which activates the pituitary gland to secrete adrenocorticotropic hormone (ACTH), which in turn activates the adrenal cortex to release corticosteroids, mainly cortisol in humans (Tsigos & Chrousos, 2002). In particular, cortisol acts to conserve some of the body's energy sources and helps the body return to homeostasis following stress.

Although the short-term mobilization of the body's resources to respond to stress was originally intended to prepare humans to fight or flee, rarely do daily stressful occurrences elicit an actual need for fight or flight, and hence, the elevation of circulating catecholamines does not seem to serve its original evolutionary purpose. A number of studies have demonstrated that over the long term, excessive discharge of epinephrine and norepinephrine is believed to lead to a suppression of cellular immune functions and to produce neurochemical imbalances that may contribute to the development of psychiatric disorders (e.g., Charmandari, Tsigos, & Chrousos, 2005; Habib et al., 2001; Chrousas & Gold, 1992). Corticosteroids, such as cortisol, have been shown to have immunosuppressive effects such as decreased lymphocyte responsivity (Chrousos, 1995). Prolonged HPA activation and cortisol secretion has also been related to the destruction of neurons in the hippocampus, leading to problems in concentration and memory, which may be characteristic of such psychiatric diagnoses as anxiety and depression (Taylor, 1999). Other studies have demonstrated that hormones can affect behavior by a direct action on the brain. For example, injections of ACTH (adrenocorticotropic hormone) have been shown to increase the persistence of avoidance behavior (e.g., Frankenhaeuser, 1975). Also output from the adrenal cortex (i.e., secretion of corticosterone) has been shown to be highly sensitive to

purely psychological variables, such as the expectation of reward or the frustration experienced when an expected reward is discontinued. Given the body's physiological response to stress and the impact that hormones have been shown to have on subsequent behavior, this study aimed to underscore the role of hormones such as cortisol and testosterone in differentiating the behaviors of nonpsychopathic and psychopathic individuals, and also to examine the relationships between anxiety and these hormones in driving BIS and/or BAS motivated behaviors.

Psychopathy and Hormone Relationships

Hormones have been implicated in the expression of particular types of behaviors. Raine's (2002) review of the literature concerning biosocial studies of antisocial and violent behavior recommends that further studies directly assess the interactions across biological and environmental variables. In particular, Raine asserts that researchers should attempt to develop a better understanding of the role hormones may play in the etiology of violence and antisocial behavior, something that is likely to better inform future intervention and prevention efforts. Given the dearth of available neuro-endocrinological literature regarding psychopathy, an examination of hormones such as cortisol (Van Honk et al., 2003) and testosterone (Van Honk, Schutter, Hermans, Putman, Tuiten, & Koppeschaar, 2004) may inform the literature on the etiology and maintenance factors of psychopathy, and hence, that is the focus of the current study. Since cortisol has been associated with the arousal associated with anxious presentations and testosterone with more dominant, aggressive behaviors, another key feature of the present study is its examination of cortisol and testosterone levels as potential endocrinological substrates that may underlie the psychopathic individuals' motivational imbalance for poor or disadvantageous decisions in approach-avoidance contingencies.

Testosterone and Psychopathy/Antisociality

Testosterone has often been linked with the expression of antisocial, psychopathic and aggressive types of behaviors whereas cortisol has often been associated with stress responses, including the expression of anxious symptomatology (e.g., Dabbs & Hopper, 1990). Earlier studies have shown high salivary, serum, and CSF testosterone levels in adult men to be significantly related to chronic aggressive behavior (Ehrenkranz, Bliss, & Sheard, 1974), violent crime (Dabbs, Frady, Carr, & Besch, 1987), and antisocial personality disorder (Dabbs & Morris, 1990). According to an extensive meta-analytic review of testosterone and dominance in men, Mazur and Booth (1998) conclude that high levels of endogenous testosterone encourage behavior intended to dominate. They note that dominant behaviors can be both aggressive with an intent to inflict harm, and nonaggressive in nature. Whereas much dominant behavior is nonaggressive, some dominant behavior is antisocial in nature, expressed as a rebellious act against established laws. In some studies with men, measurement of testosterone at a single point in time and reflecting basal testosterone level, has been predictive of dominant or antisocial behaviors. Although they distinguish between studies examining dominance and those examining aggression, for the purpose of the current study, only the relevant findings for the relationship between testosterone and more aggressive and antisocial types of behaviors are discussed.

Archer's (1991) review reports the findings across five different studies of male prisoners that have obtained a positive association between testosterone and aggression, a few of which will be reported as follows. In one study (Kreuz & Rose, 1972), although no differences were found when prisoners convicted of "violent" and "non-violent" crimes were compared, prisoners with a history of violent crime during adolescence showed higher testosterone levels than those

without such a history, suggesting that testosterone may have played a role in predisposing individuals to recurrent aggressive acts during adolescence. Similarly, prisoners with a history of chronic violent behavior had higher testosterone blood levels than a group of non-violent prisoners (Ehrenkranz, Bliss & Sheard, 1974). In another study (Rada, Laws, Kellner, Stivastava & Peake, 1983), rapists, operationalized as men who used physical aggression towards women to engage in coercive sexual acts, were found to have significantly higher testosterone levels than both a non-criminal male control group, and a group of child molesters. In a study that measured free (unbound) testosterone in men via saliva samples, higher testosterone levels were obtained among men ranked by their peers to be highly aggressive as compared to peer rankings of men with low aggressiveness levels (Lindman, Jarvinen & Vidjeskog, 1987). Most studies have been correlational, often comparing low and high aggressive groups. Whereas the aforementioned studies all seem to suggest that testosterone leads to violent/aggressive behavior, given the correlational nature of all of these findings, it is also possible that engaging in violent/aggressive behavior leads to higher levels of testosterone. Another group of studies has focused on the reverse possibility associated with the correlational design, that is, studying the effects of how behavior can modify hormonal behaviors. Most of these studies have examined athletes' hormone levels following a competition. Specifically, testosterone levels have been found to increase following offensive behavior, such that a positive relationship was found between number of attacks displayed during a judo competition and the players' rise in testosterone levels following these aggressive behaviors (Salvador, Suay, Martinez-Sanchis, Simon, & Brain, 1999). From the various approaches used to examine the links between aggressive behavior and testosterone, a basic assumption emerges that hormones and behavior have reciprocal influences (Salvador et al., 1999).

Although a bulk of the research has been correlational in nature, precluding the ability to determine the directional relationship between testosterone and aggressive behaviors, a recent study has directly examined the impact that testosterone has on behavior by administering testosterone to participants. Utilizing a controlled experimental design (Van Honk et al., 2004), it was possible for the authors to conclude that testosterone had direct effects on the behaviors of humans in punishment-reward contingencies. This study provided the first direct evidence for the effects of testosterone on altering behavioral choices on the Iowa Gambling task from less risky to more risky decisions. Twelve healthy young women received either a single dose of testosterone (.5 mg of testosterone with cyclodextrines) or a placebo in a randomly assigned, crossover and counterbalanced design. Participants were asked to complete the Iowa Gambling Task (IGT) on two subsequent days, pre and post-administration of either the testosterone dose or the placebo.

In accordance with Van Honk et al.'s (2004) hypothesis that clinical and subclinical psychopaths are likely to exhibit a risky, disadvantageous pattern of decisions, marked by an insensitivity to punishment while there is an opportunity for large monetary rewards, the female participants who were administered testosterone made more disadvantageous choices as compared to those females who were administered the placebo. The results of this study are especially interesting and unique in that the participants were not selected on the basis of psychopathy, hence providing evidence for a significant relationship between the functioning of the endocrine system (via testosterone) and its effects on motivation and behaviors. Based on the findings from their controlled experimental design, the authors concluded that decisions on the Iowa Gambling task are influenced by bioregulatory responses to a single administration of testosterone. Whereas testosterone was found to have a significant effect on unconsciously

generated physiological and emotional responses on the gambling task, no effects on consciously reported mood states on the Profile of Mood States (POMS) were obtained. The authors explain that the role of steroid hormones in motivation and emotion is mainly associated with rudimentary physiological responses and unconsciously motivated behavioral tendencies that may be outside of the realm of one's awareness. Gonadal steroid hormones, like testosterone, for example, are said to act by binding to subcortical neuronal networks in the brain, such as the amygdala, which is a key brain structure in the neural circuits involving punishment and reward (Van Honk et al., 2004; Bechara et al., 1999).

Psychopathy-Testosterone Correlates in Female Samples

Although most of the research on testosterone and aggression has been conducted with male forensic samples, the few studies that have been conducted with females have elicited parallel trends (Bjork et al, 2001; Archer, 1991). In women, androgens are secreted by both the ovaries and the adrenal cortex. One study (Ehlers, Rickler & Hovey, 1980) found that young women who had a history of violence exhibited significantly higher plasma testosterone levels than the non-violent control group of young women patients coming from the same neurobehavioral clinic. In another study (Dabbs, Ruback, Frady, Hopper & Sgoutas, 1988), saliva samples were collected from female prison inmates who were divided into groups based on the nature of their crimes. Across the groups, women convicted of unprovoked acts of violence exhibited the highest levels of salivary testosterone as compared to the other groups (e.g., provoked violence, theft, drug offenses) (Archer, 1991). Higher levels of testosterone had a positive relationship with increased levels of aggressive dominance and violent behaviors among 87 female inmates in a maximum security state prison (Dabbs & Hargrove, 1997), as

similarly found in previous studies with men. Their findings suggest a model in which lower testosterone levels are associated with less violent crime and less aggressive dominance in prison, with a significant relationship between testosterone and aggressive dominance in prison and in the violent criminal acts.

Additionally, it has been speculated that testosterone may correlate with subtypes of aggressive behavior via the mechanism of deficits in higher-order cognitive processes that deter the inhibition of behaviors (Bjork, Moeller, Dougherty, & Swan, 2001). In a sample consisting of 27 psychiatrically healthy women, commission errors on two versions of a continuous performance test (CPT—immediate and delayed memory tasks) were positively related to plasma total testosterone concentration levels. Given the significant relationship between plasma levels of testosterone and errors of commission on the continuous motor task in this study, these authors suggested that testosterone may play a role in aggression via its influence on impaired impulse control. They concluded that endogenous testosterone levels may relate to the ability to inhibit motor behavior under conditions in which selective attention and working memory are mutually required to perform accurately and rapidly to the task's demands.

Cortisol, Anxiety and Psychopathy

In the movement toward understanding potential physiological bases underlying human behavior, there has been an increasing focus on studying the hormones of the endocrine system (Dabbs & Hopper, 1990). Within the endocrine system, cortisol is responsible for mobilizing energy needed to deal with emergency situations, whereas testosterone has been associated with facilitating sexual activity and aggression. Heightened cortisol concentrations have specifically been related to internalizing disorders and symptoms, namely in anxiety and depression (e.g.,

Dabbs, Jurkovic, and Frady, 1991; Mason, Giller, & Kosten, 1988; Rubin, 1981). Basal cortisol measures provide an index of the resting activity of the HPA system. Reactions to laboratory stressors, however, provide a useful index to assess individual differences across the HPA system's reactivity to stressful situations, and hence, the present study's examination of baseline and post-task collections of saliva samples to assess participants' reactivity across hormone levels following a gambling task. Given the finding that cortisol has been associated with anxiety and arousal, Dabbs et al. (1991) suggest that cortisol might serve to inhibit (for example, through anxiety) violent behaviors. Hence, psychopaths' hallmark trait of sensation-seeking may be due to a biological deficit in the functioning of cortisol, which makes them seek out behaviors that would produce an intolerable level of arousal and anxiety in nonpsychopathic individuals.

It has been noted that the neuro-endocrinological basis of psychopathy, which has been described as a disorder characterized by a lack of fear, is relatively unknown (Van Honk et al., 2003). Fear and anxiety have been found to be adaptive responses in that these emotional states have instructional attributes—helping individuals to differentiate danger from safety cues in their environment, and activating responses accordingly. When fear reaches an extreme level, and the fear response is activated when the environmental cues do not suggest danger or threat, this fear frequently leads to psychopathology, such as anxiety disorders which are often associated with distorted cognitive processing and maladaptive behavioral and autonomic-system responses (Korte, 2001). Fear has also been conceptualized to play a key role in the motivational system that guides human behavior, through reaching a balance between sensitivity for punishment and activating responses toward rewards and reinforcers in one's environment that guide subsequent behaviors (Van Honk et al., 2003). Whereas a heightened punishment sensitivity and decreased reward dependency has been observed among anxious patients (Arnett, 1997), Van Honk et al.

(2003) note that the psychopath occupies the other extreme, in that his insensitivity for punishment along with his dependency for reward results in risk-taking behaviors and anti-social acts. Support was obtained for their (Van Honk et al., 2003) hypothesis that low levels of cortisol may underlie behaviors reflecting an *imbalance* in punishment sensitivity and reward dependency, as observed on the Iowa Gambling task.

Undergirding Van Honk et al.'s (2003) aforementioned hypothesis is their explanation for the neuroendocrinological role of cortisol. Cortisol is identified as a neuroendocrine hormone that serves a crucial role in the regulation of the body's neural fear circuits (Korte, 2001). Elevated levels of cortisol secreted by the adrenal glands act on the amygdala to facilitate corticotrophin releasing hormone (CRH), which is responsible for activating fear states associated with the inhibition of behaviors. Whereas a hyperactive hypothalamic-pituitary-adrenal (HPA) axis, resulting in high levels of cortisol has been observed among anxious and depressed patients (Schulkin, Gold, & McEwen, 1998), a hypoactive HPA axis, characterized by deficiently low levels of cortisol, is hypothesized to underlie psychopathic and aggressive tendencies (Van Honk et al., 2003). Van Honk et al. (2003) elaborate that low levels of cortisol may result in fearlessness, which could play a role in the etiology and/or maintenance of psychopathy. Such a formulation is consistent with the *motivational imbalance* model (previously discussed) that emphasizes reduced punishment sensitivity and heightened reward dependency as a potential explanation for psychopathic behaviors.

To examine these hypotheses, Van Honk et al. (2003) investigated relationships between basal cortisol and IOWA gambling task performance with an analogue sample of 30 males and females characterized by high and low scores on psychopathic personality inventories. Although self-reported psychopathy was unrelated to cortisol in that particular investigation, cortisol was

inversely related to choices of risky decks, indicating that participants with the lowest cortisol levels exhibited the most disadvantageous pattern of decision-making on the task, marked by the greatest monetary losses. The nonsignificant relationship between psychopathy and cortisol levels in that study may be a result of the investigators' choice to use scores obtained on Carver and White's BIS and BAS (1994) scales to classify participants into low and high psychopathy groups, a practice that has not been validated in earlier studies. In a more recent study by some of the same investigators (Montagne, Van Honk, Kessels, Frigerio et al., 2005), in which the BIS/BAS scales were again used as a criterion measure, they attribute some of their null findings to their use of the BIS/BAS scales to screen participants stating that it only takes into account the fearlessness component of psychopathy (via punishment insensitivity/reward-seeking behaviors), and not the other facets of psychopathy. Their earlier findings (Van Honk et al., 2002), did however provide support for a pattern of low punishment sensitivity and high reward dependency among participants with low levels of cortisol, also potentially indicative of a hypoactive HPA axis.

Echoing the results obtained by Van Honk et al. (2003) of a negative relationship between cortisol levels and risky behaviors often associated with psychopathy, several other studies have obtained a relationship between cortisol and aggressive behavior, or antisocial personality disorder. Among these findings, low cortisol levels were found in adult men diagnosed with antisocial personality disorder (Virkkunen, 1985). Aggressive, antisocial participants had a significantly lower excretion of urinary free cortisol and lower CSF adrenocorticotropin hormone (ACTH) than controls in their study. In a study in which 38 male participants (aged 7-12 yrs) with conduct disorder (CD) were assessed for cortisol levels, a similar finding emerged (McBurnett, Lahey, Rathouz, & Loeber, 2000). A single saliva sample

was gathered from each child during the clinical visits. Cortisol concentrations were directly linked with aggression, in that salivary cortisol concentration was inversely associated with several measures of aggression and disruptive behaviors. In another study (Maes, Van West, De Vos, Westenberg, Hunsel, Hendriks, Cosyns, & Sharpe, 2001), cortisol levels were compared between pedophiles and normal volunteer control participants. These authors make the argument that, to the extent that pedophiles use various degrees of coercion and physical force in their sexually aggressive acts, this group can be likened to aggressive, antisocial participants examined in previous studies (Virkkunen, 1985). In accordance with the negative correlation obtained between antisocial individuals and cortisol in other studies, Maes et al. (2001) also found that baseline serum cortisol levels were significantly lower in pedophiles as compared to controls. They argue that as far as pedophilia is related to aggression, their findings extend those of previous studies that have demonstrated an inverse relationship between HPA-axis activity and aggression.

As the aforementioned review indicates, several studies have yielded a significant inverse relationship between cortisol levels as activated by the HPA axis and antisocial, aggressive, or psychopathic presentations. However, other studies assessing the relationship between cortisol and aggressive behaviors have yielded inconsistent findings, partially attributed to the various mediums from which cortisol has been extracted (saliva, serum, and urine). This diversity in methodological approaches and cortisol sample collection strategies may account for the inconsistent findings across studies (Scerbo & Kolko, 1994). Another reason for the inconsistencies may be due to comorbid anxiety levels found among psychopathic samples (McBurnett, Kumar, Kumar, Perez, Lahey, & Shaw, 2000). McBurnett et al.'s (2000) study entailed a sample of thirty inpatient children, aged 9-15, from a general inpatient psychiatric

program and from a program for sex-offending boys. Twenty-five of the 30 youth met criteria for conduct disorder. In their examination of cortisol, aggression, and anxious symptoms, anxiety significantly moderated the relationship between aggression and cortisol levels. That is, high anxiety with high aggression scores were significantly associated with high cortisol levels. In contrast, high aggression scores with low anxiety scores were associated with lower cortisol levels. Hence, the findings for a significant interaction between anxiety, aggression and cortisol obtained in this study may shed some light on some of the inconsistent findings for the relationship between cortisol and aggression obtained across previous studies. The importance of examining the moderating role for anxiety among psychopathic groups was also previously underscored (Arnett et al., 1997; Newman et al., 1985), with some authors recommending that subsequent studies differentiate between high and low-anxious psychopaths given the drastic differences that these groups may exhibit in skin conductance responses, passive avoidance learning, and sensitivity to reward.

Further review of other studies that have failed to obtain an inverse relationship between cortisol and aggression has yielded other methodological problems that may be accounting for the null results as follows. In one study in which the investigators failed to find a difference in cortisol levels between their identified *aggressive* and *nonaggressive* groups (Moeller, Allen, Cherek, Dougherty, Lane & Swann, 1998), their findings may be a result of several methodological and statistical problems. In addition to a potential problem of insufficient power to detect any between-group differences, with only eleven participants in their *aggressive* and twenty participants in their *non-aggressive* group, their classification of participants into these two respective groups also seemed to be problematic. Although participants in the *aggressive* group had, on average, higher Buss Durkee Hostility Inventory scores, the differences in scores

between groups failed to reach significance. Additionally, four participants in the group classified as *non-aggressive* met criteria for Antisocial Personality Disorder, whereas only two participants in the *aggressive* group met these criteria. These findings suggest that the null cortisol results may be due to a failure to establish valid groups, and that the groups seemed to be marked by more construct overlap than actual differences. Additionally, the authors did not seem to assess any potential comorbid anxiety across participants that may have confounded their results, as several studies have found that anxiety may moderate the relationship between aggression and cortisol (McBurnett et al., 2000) and between psychopathy and subsequent behaviors (Arnett et al., 1997).

In another study in which the inverse cortisol relationship was not obtained the authors had a reasonable explanation for their null findings. Gerra et al. (1997) aimed to assess how 30 healthy male participants varying in degrees of aggressiveness (although all still within the range of normality) would react to an aggressiveness-inducing lab task with different neuroendocrine responses. As predicted, male participants with higher basal testosterone levels engaged in more aggressive acts; however, these men also exhibited higher cortisol levels. Their findings reflecting higher cortisol responses in participants who engaged in more aggressive acts contrasts with patterns obtained across previous studies (e.g., Scerbo & Kolko, 1994; Virkkunen, 1985). They report that these contrasting findings are not surprising, given that their sample consisted of "healthy" men, free from any psychological/personality disorders (as measured by the MMPI & interviews). Additionally, the levels of aggressiveness observed, although operationalized as "high" and "low" aggressiveness groups, fell in the range of "normal" behavior. They suspect that because of the stressful nature of their aggressive task, it likely activated the HPA-axis, which may have accounted for the rise in cortisol levels across all of their participants, especially

given that none of their participants met any criteria for any type of deviant or psychopathic classification.

In line with a bulk of the previous research, the present study predicted that psychopathic analogues may have diminished cortisol-mediated arousal, and hence in turn, decreased BIS sensitivity, something that may make them seek arousing situations through their deviant acts. The reverse was predicted for individuals identified as nonpsychopathic analogues, in that they may have higher cortisol levels, and hence increased levels of uncomfortable arousal that facilitates an increased BIS sensitivity, and subsequent inhibitions to engage in behaviors that elicit aversive consequences. This inhibition may then lead to a tendency to engage in more cautious, less risky decisions on the Iowa Gambling task, as compared to individuals identified as psychopathic analogues in this study.

Cortisol & Testosterone Interactions in Psychopathy: A Proposed Moderator Relationship

Given the variability of empirical findings in the testosterone-violence relationship, it has been argued that moderating variables should be assessed to establish the conditions under which testosterone and violent behavior are most strongly related. One possible moderator is cortisol (Dabbs et al., 1991). In Dabbs et al.'s study, in which saliva samples were obtained from a sample of 113 late adolescent male offenders in a state prison, a significant interaction effect was found, in which cortisol served as a moderator in the relationship between testosterone and the severity/violence level of the offenders' crimes. Specifically, the testosterone-violence severity relationship was strongest at low cortisol levels. This finding is consistent with their speculation that cortisol may moderate the behavioral effects of testosterone. In another study, Scerbo & Kolko (1994) aimed to assess the relationship of testosterone and cortisol (via saliva assays) to

aggressive, hyperactive, and internalizing behaviors among 40 clinically-referred antisocial children. They found significant positive relationships between staff-rated aggression and testosterone and between cortisol and parent-rated internalizing behavior. However, this study failed to demonstrate a moderator role for cortisol in the testosterone-aggression relationship. The lack of a moderator relationship in the latter study (Scerbo & Kolko, 1994) as compared to the former study (Dabbs et al., 1991) may be a function of the population being assessed by Scerbo and Kolko. They utilized a sample of children identified as "disruptive" across a range of behaviors including inattention, overactivity, and aggressive behaviors. Given their small sample of disruptive children, it may be that their sample size had insufficient power to yield significant moderator relationships.

Previous studies have elucidated a modest, but direct relationship between testosterone and aggressive, antisocial, psychopathic presentations. Although the findings have been mixed, an inverse relationship has been obtained for the relationship between cortisol and aggressive presentations. A more common finding has been that of a direct relationship between cortisol and internalizing symptoms such as anxiety. A less frequently tested relationship has been that of cortisol as a moderator between testosterone and psychopathic behaviors. Utilizing a variety of methodologies and types of tasks as previously enumerated, psychopathic and nonpsychopathic participants have been compared on passive-avoidance and active-avoidance learning strategies in various conditions involving punishment only, reward only, and mixed-incentive or competing punishment-reward paradigms. Most recent studies conclude that the learning deficits found among psychopathic participants are most salient in conditions of competing reward-punishment contingencies, such as those presented in the Iowa Gambling task. The findings most consistently support Newman's (1987) response modulation hypothesis in that

psychopathic individuals are less likely to inhibit behaviors leading to punishment when there is a competing signal for reward. Whereas Van Honk's studies have assessed the relationship between baseline cortisol and psychopathic performance on the Iowa Gambling task (2003), and the impact of testosterone administrations on women's performance on the gambling task (2004), respectively, the present study sought to extend these findings.

By screening from a sample of 1100 female and male participants utilizing two validated measures of psychopathy with a student population, this study classified participants into psychopathic and nonpsychopathic groups based on scores in the upper and lower quartiles, obtaining a larger sample than most previous studies by identifying 120 participants. Using two validated measures for analogue psychopathy in this study may increase the validity of classifying psychopathic and nonpsychopathic groups as compared to previous studies (Van Honk, Hermans, Putman, Montagne, and Schutter, 2002; Van Honk et al., 2003) that selected participants based on scores from Carver and White's BIS and BAS scales (1994). This latter strategy may be problematic given that Carver and White's BIS scales failed to differentiate psychopathic from nonpsychopathic offenders in a previous study (Ghebrial and Arnett, 2003, unpublished manuscript), and at any rate, such scales are indirect measures of psychopathy, at best. The present study utilizes both behavioral and personality-based constructs as an operationalization of BIS-motivated and BAS-motivated behaviors and traits among both male and female psychopathic and nonpsychopathic groups. This study also assesses levels of cortisol, testosterone, and both hormones in concert at baseline and following the administration of the Iowa Gambling task, assessing the role of these hormones as potential underlying biological markers for Gray's model of BIS and BAS-motivated behaviors in the same study. Because saliva samples were collected at baseline and following the task, an assessment of both

between-group basal differences between analogue psychopathic and nonpsychopathic male and female participants, and an assessment of participants' change or reactivity in hormone levels following the task are examined. No study to date has assessed the correlates of both hormones to an experimental measure of BIS and BAS-functioning among adult male and female psychopathic analogues as this study has done.

This study predicts that if cortisol is highly correlated with internalizing behaviors and anxiety, it may moderate the relationship between testosterone and aggressive or psychopathic types of behaviors. Specifically, individuals with high cortisol levels may exhibit a *dampened* testosterone-aggression relationship, potentially via the mechanism of heightened BIS sensitivity, and hence have a decreased likelihood of acting out on their antisocial impulses. The present study seeks to further develop a potential biopsychosocial model for understanding how cortisol and testosterone, alone and in concert, may underlie individual differences in BIS and BAS arousal, something that may in turn explain individual differences in psychopathic characteristics and behaviors. As previously stated, the current study aims to assess the relationship between the BIS and BAS motivational systems and cortisol and testosterone, using an experimental manipulation task and measuring pre and post hormone levels and reactivity among an adult analogue sample of male and female psychopathic individuals as compared to a control group of nonpsychopathic individuals.

Hypotheses:

Psychopathy & the Motivational Imbalance Model:

1. Given that the BIS has been conceptualized as the mechanism responsible for inhibiting behavior in response to punishment, whereas the BAS activates behavior in response to

reward, this study conceptualized behaviors on the Iowa Gambling task as operationalizations of BAS, reward-seeking activity, and BIS, inhibition-based activity. Psychopathic analogue participants were predicted to be less likely to suspend their approach behaviors in the face of punishment (loss of money) as compared to nonpsychopathic participants. This prediction was assessed by determining if a greater number of cards were played from the disadvantageous decks (decks A and B) by the psychopathic as compared to the nonpsychopathic analogues on the Iowa gambling task. Specifically, the overall number of "good deck" selections minus the number of "bad deck" selections made by participants provided the data necessary to assess this hypothesis.

2. The opposite prediction was made for the nonpsychopathic group, in that they were predicted to make more selections from the advantageous decks (decks C and D) as compared to the nonpsychopathic group, reflecting their ability to utilize punishment cues in an effective way to alter their behaviors. Again, the overall number of "good deck" selections minus the number of "bad deck" selections made by participants provided the data necessary to assess this hypothesis.

Self-reported BIS and BAS Activity:

3. The behavioral manifestation of BIS activity on the gambling task (as measured by more selections from *advantageous decks*) was predicted to be positively related to a personality-based measure of self-reported BIS activity (Carver and White's (1994) scale).
4. The behavioral manifestation of BAS activity on the gambling task (as measured by more selections from *disadvantageous decks*) was predicted to be positively related to a personality-based measure of self-reported BAS activity (Carver and White's (1994) scale).

Anxiety Correlates:

5. BIS activity was predicted to be positively correlated with measures of anxiety, whereas BAS activity was predicted to be negatively correlated with measures of anxiety.
6. Since the literature has revealed mixed findings in the relationships between anxiety and psychopathy, a few exploratory analyses were examined. Primary or Factor 1 psychopathy was hypothesized to be negatively related to measures of anxiety while secondary or Factor 2 psychopathy was hypothesized to be positively related to measures of anxiety. Any differences obtained among high and low anxious psychopathic and nonpsychopathic participants were expected to be in the same direction.

Hormone Relationships:

7. Cortisol was predicted to be positively related to self-reported BIS activity (Carver & White's (1994) scale) and to more advantageous decisions (selections from decks C and D) on the Iowa gambling task.
8. Cortisol was predicted to be positively correlated with self-reported endorsement of higher anxiety levels as measured by the State-Trait Anxiety Inventory and the Welsh Anxiety scale.
9. Relatedly, self-reported anxiety was predicted to be negatively related to testosterone levels.
10. Psychopathic analogue participants were predicted to have lower levels of cortisol as measured both at baseline and in the second saliva collection following peak arousal levels on the Iowa Gambling task, as compared to nonpsychopathic analogue participants. Additionally, given that psychopathic analogues are presumed to be less impacted by stressful, high-risk situations and more focused on the rewarding elements of the IGT, it was predicted that the psychopathic group would experience a smaller increase in cortisol levels than the elevation in cortisol levels among nonpsychopathic participants in response to the

task. That is, the change score in cortisol level from baseline to post-task reactivity levels among psychopathic analogues was expected to be smaller than the cortisol change score among nonpsychopathic analogues.

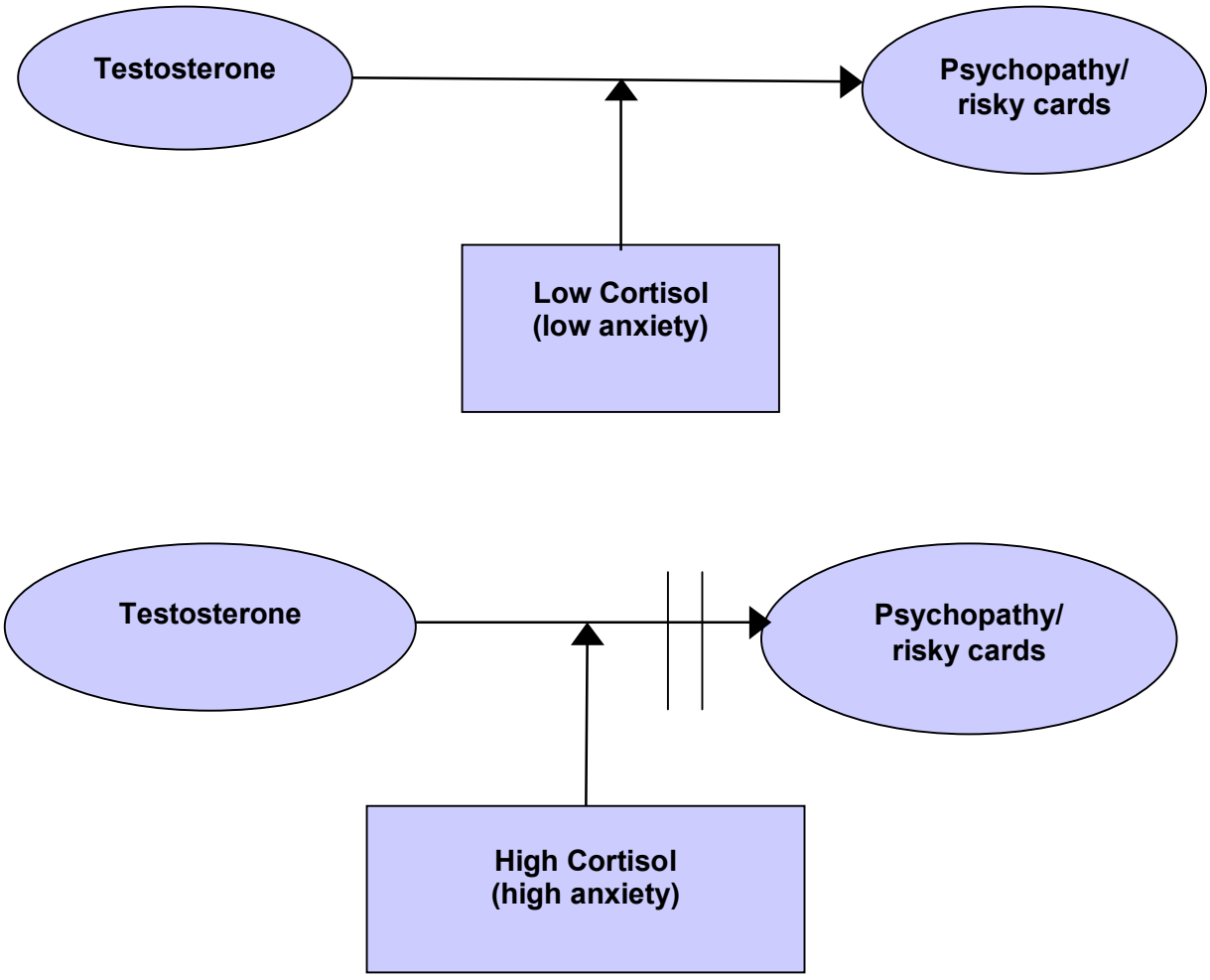
11. Testosterone was predicted to be positively related to self-reported BAS activity (Carver & White's (1994) scale) and to more risky behaviors on the gambling task, as measured by a higher number of selections from the disadvantageous decks (A and B) on the Iowa Gambling task.
12. Psychopathic analogues were predicted to exhibit higher levels of testosterone both at baseline and in the second saliva collection following peak arousal levels on the Iowa Gambling task, as compared to nonpsychopathic analogues. Additionally, given the findings from previous literature suggesting that psychopathic individuals may have a stronger proclivity toward reward and are therefore more likely to focus on the rewarding elements of the IGT, it was predicted that psychopathic analogue groups may experience a greater elevation in testosterone levels as compared to nonpsychopathic analogue groups from baseline to post-task levels. That is the change score in testosterone level from baseline to post-task levels was expected to be higher among psychopathic analogues than among nonpsychopathic analogues.
13. Given the robust findings for gender differences in the literature, it was predicted that female participants would likely have lower levels of testosterone and self-reported aggression as compared to men, but the relationships between psychopathy, hormones and behaviors were expected to remain salient despite these gender differences.

Moderator Relationships: Hormones, Risky Behaviors, Anxiety & Psychopathic Status

14. Finally, as an extension of previous research, a few moderator relationships were predicted. It was hypothesized that cortisol would moderate the relationship between testosterone and psychopathic behaviors. In particular, the relationship between testosterone and risky (psychopathic) behavior, as measured by card selections from disadvantageous decks, was expected to weaken when cortisol and/or self-reported BIS levels were high. To clarify, whereas higher testosterone levels would likely be associated with higher risk behaviors on the card task, the aforementioned suggests that cortisol levels may serve as a moderator, such that participants with both high testosterone and high cortisol levels may be less likely to engage in high-risk behaviors. This relationship is similar to that found in previous studies with the moderating role of anxiety on skin conductance responses of psychopathic participants in a paradigm with aversive consequences. Hence, similar moderator predictions were made for participants who endorse high levels of self-reported anxiety, as anxiety, much like cortisol, is also presumed to serve the same role in underlying BIS activity. That is high anxiety scores may serve to reduce the likelihood of engaging in risky behaviors. To assess the moderating role of anxiety, these analyses were conducted by dividing participants across psychopathy status and using median-split scores on the Welsh Anxiety score, a commonly used instrument for assessing anxiety among psychopathic groups. This procedure led to the formation of four groups, psychopathic and nonpsychopathic analogues classified into high and low anxiety groups. These analyses were exploratory given some previous findings in which psychopathic participants were unlikely to self-report anxiety symptoms.

15. Relatedly, when self-reported BAS levels are high and cortisol levels are low, it was predicted that the relationship between testosterone and psychopathic, risky behaviors would be strengthened. Analogously, in this scenario, it was predicted that self-reported anxiety levels, would likely be low. (see Figures 3 and 4 below).

Integration of Hormones, Psychopathy and Gray's BIS/BAS Model for Proposed Predictions



Figures 3 and 4: Predicted relationships among hormones, psychopathy and BIS/BAS activity.

Method

Participants

The sample consisted of 113 undergraduate students (54 males, 59 females) enrolled in an Introductory Psychology course at the Pennsylvania State University following guidelines of the departmental subject pool procedures. Out of the original 114 participants, one participant's data had to be excluded because scantron responses did not match up with the items from the questionnaires. It is likely that this individual skipped items, which led to subsequent mistakes in correctly lining up subsequent responses on the scantron. Of the 113 participants included in the final sample, 60 were identified as the psychopathic analogues and 53 individuals were identified as the nonpsychopathic analogues. A large sample was recruited because of the relatively low base rates of psychopathy among male and female college students, and because of the power needed to detect differences in hormone reactivity across participants. Because base rates of psychopathy within a college student population range from about 2-10%, eligible participants were selected from a pool of approximately 1100 students who completed screening measures. Participants received $5 for their participation for approximately 1 to 1.5 hours, and also had the opportunity to earn 1.5 hours of course credit if they provided a third saliva sample that they were asked to collect at home and return to the lab. Participants were informed about the broad focus of the experiment, that their responses were confidential, and that their participation was voluntary.

Participants were asked to provide brief demographic information including their age, gender, and ethnic background. The sample consisted of 48% male and 52% female students. The majority of the sample was between the ages of 18 – 20 (78%), followed by ages 21 – 23 (18.4%), and an equal percentage were between the ages of 24 – 26 (1.8%) and 27 – 32 (1.8%),

respectively. The racial composition of the sample was representative of the overall population demographics of this university located in Central Pennsylvania with the majority of the participants self-identified as Caucasian. Specifically, 84% Caucasian, 6% African-American, 2.6% Hispanic, 3.6% Asian, and 1.2% classified themselves by the Other category.

To obtain useful saliva samples for hormone assays, based on consultation with Dr. Granger, a list of exclusionary criteria were used to screen participants for inclusion into the study. Screening was conducted via a brief telephone interview conducted by trained experimenters. Participants were excluded from participation in the study based on the following reasons: for use of anabolic steroids or other prescription medications or supplements that are known to cause changes in testosterone and/or cortisol levels such as Flonase for allergies, hydrocortisone creams, DHEA supplements, andristerone, or for frequent use of inhalers. Additionally, individuals with any form of cancer including testicular cancer or any chronic endocrine disorder such as Addison's Disorder or Cushing's Syndrome were also excluded. Participants were not allowed to participate if they were sick with a fever exceeding 102, were hung-over, were experiencing higher stress levels than that of a typical average day, or if they had smoked cigarettes in the 30 minutes prior to sampling time. Participants presenting with any of the latter set of criteria were allowed to reschedule for another time.

Measures

The Psychopathic Personality Inventory

The Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996), in its original form, is a 187-item measure designed to assess psychopathic traits in noncriminal

populations. Due to time constraints, in consultation with the author of the PPI (Lilienfeld), it was recommended that the current investigation use the 56-item PPI-short version for this study. The PPI short form has been demonstrated to be highly correlated (r = .90) with the full form, and yielded an internal consistency coefficient of .85 for the full 56-item scale in this study. A benefit of the PPI is that, although it is highly correlated with indices of Antisocial Personality Disorder, it possesses substantial variance related to personality traits not shared with other measures of antisocial behavior. Items assess characteristics traditionally associated with psychopathy including: manipulativeness, superficial charm, persuasiveness, callousness, lack of guilt, risk-taking behaviors, disregard for social norms, absence of forethought, and a lack of anxiety. The PPI was developed to assess the multi-faceted construct of psychopathy (Benning et al., 2003). Initial validation studies suggested that it includes items reflecting both facets of psychopathy contained in the PCL-R, the gold standard measure of assessing psychopathy among offender populations (Lilienfeld & Andrews, 1996). In another investigation (Poythress, Edens, & Lilienfeld, 1998), PPI total scores were found to correlate substantially and significantly with both Factor 1 (r = .54) and Factor 2 (r = .40) of the PCL-R.

The PPI has proven to be quite useful for research on the etiology of psychopathy, and hence relevant to the present study, which seeks to elucidate the mechanisms underlying psychopathic traits and behaviors. This instrument was included to further examine the construct of psychopathy within a college student sample on the actual day of the experiment, as a means of ascertaining that the participants' responses on the screening measures were not subject to random responding, given that the screening measures were part of a large packet of questionnaires used in the department's subject pool. Scores on this scale should be highly correlated with the scores obtained from the previously administered psychopathy scales and

hence provide a secondary check on the validity of the grouping variable, "psychopath" versus "nonpsychopath" analogue operationalizations discerned from the two psychopathy measures used previously for screening. Responses to this questionnaire, administered on the task day, were not used to classify participants into groups, but may capture unique variance related to the construct of psychopathy not assessed by the screening measures, that can be used for subsequent post-hoc analyses.

Although for the purpose of this study, the total score was of primary interest, primary and secondary psychopathy factors were also examined. Previous studies have noted the importance of differentiating psychopathic groups based on level of anxiety (Arnett, Smith & Newman, 1997), and anxiety has been shown to be negatively related to Factor 1 psychopathy and positively related to Factor 2 psychopathy. A PPI primary (social potency, coldheartedness, fearlessness, impulsive nonconformity, and stress immunity) and a PPI secondary psychopathy score (machiavellian egocentricity, blame externalization, and carefree nonplanfulness) can be determined, which have yielded internal consistency coefficients of .86 and .82 respectively (Lilienfeld & Hess, 2001). The primary and secondary PPI factors yielded comparable internal consistency coefficients of .83 and .87 in this study respectively. Given that self-report anxiety measures were of interest, this study also examined the relationship between Primary and Secondary psychopathy features with anxiety measures and with cortisol levels, given that cortisol has been associated with anxiety in previous studies. Exploratory, post-hoc analyses of these relationships were conducted, namely aimed at assessing any differential relationships between psychopathy factor scores, anxiety, cortisol and BIS activity. Based on previous findings, it was expected that secondary psychopathy would be positively correlated with measures of anxiety, and subsequently cortisol level and BIS measures.

The Aggression Questionnaire

The Aggression Questionnaire (Buss & Perry, 1992) is a 29-item measure of Physical Aggression, Verbal Aggression, Anger, and Hostility that primarily assesses behavioral aspects of aggression. This measure has yielded an overall alpha coefficient of .89 in previous studies (Buss & Perry, 1992; Ghebrial & Hall, 2001, unpublished), and of .91 in the current study. This questionnaire was selected since much of the previous literature with testosterone has examined the relationship between testosterone and measures of aggression, and very few have specifically examined its relationship with psychopathy. Additionally, recent accounts (Skeem, Polythress, Edens, Lilienfeld, & Cale, 2003) have discussed the overlap between Factor 2 or secondary psychopathy traits and aggression, so a measure specifically targeted at breaking down various forms of aggression may provide a more detailed account of the specific aggressive behaviors that psychopathic individuals may or may not endorse.

According to Blackburn (1998), the callous violation of the rights of others that is an inherent trait of psychopathy, is likely to entail psychological or physical acts of aggression. Aggression is one of the defining diagnostic traits of Antisocial Personality disorder. Because Factor 2 psychopathy represents the unsocialized behaviors assessed by APD (Blackburn, 1998), assessing aggressive behaviors as measured by the Buss Aggression Questionnaire may inform this study's findings by providing a finer-grained analysis of specific behaviors underlying psychopathic versus nonpsychopathic presentations. For example, in addition to assessing the relationships between hormones and behaviors on the gambling task across psychopathic and nonpsychopathic groups, assessing correlations with the Buss Aggression Questionnaire to capture unique variance not assessed by the psychopathy questionnaires was also expected to be

informative. Also, since the psychopathy questionnaires focus more on assessing personality characteristics that may be endorsed by both men and women in this study, this study was also interested in assessing gender differences on a more behavioral measure of aggression and its relationship with hormone levels, as previous studies have illustrated robust gender differences in aggression across men and women.

BIS/BAS Scales

The BIS/BAS Scales (Carver & White, 1994) consist of a total of 20 items with four subscales, designed as self-report measures to assess the sensitivity of the two general motivational systems originally theorized by Gray (1982, 1987b) to underlie behavior and affect, namely the behavioral inhibition (BIS) and the behavioral activation (BAS) systems. The BIS, as postulated by Gray, is sensitive to signals of punishment, and serves to inhibit behavior that may lead to negative or painful outcomes, such as the experience of fear or anxiety. The BAS is sensitive to signals of reward and escape from punishment and serves to facilitate the experience of positive feelings such as happiness (Carver & White, 1994). The BIS scale consists of 7 items that assess affective reactions to the anticipation of punishment. The BAS measure consists of 3 brief, interrelated subscales. The Reward Responsiveness scale consists of 5 items that assess positive responses to the anticipation of or following the occurrence of reward. The Drive scale consists of 4 items that assess a determined pursuit of desired goals. Finally, the Fun Seeking scale consists of 4 items that reflect both a desire and a willingness to partake in rewarding events on the spur of the moment.

Respondents are asked to indicate the degree to which they agree or disagree with statements using a 4-point Likert-type scale, with 1 indicating strong agreement and 4 indicating

strong disagreement (Carver & White). A previous study (Ghebrial & Arnett, 2003, unpublished) obtained indices of reliability that were comparable to those originally obtained by Carver and White, with the exception of a lower alpha for the Reward Responsiveness scale. The BAS subscales yielded the following alpha values: Reward Responsiveness, .58, Drive scale, .73, and Fun-Seeking, .71, compared with values of .73, .76, and .66, respectively from Carver and White's study. Although Carver & White did not calculate an alpha for the total BAS scale in their initial validation study, the alpha coefficient for the total BAS scale was calculated in previous studies, yielding an alpha of .81 (in Ghebrial & Arnett, 2003) and .86 in another study assessing the BIS/BAS scales among college undergraduates (Gable, Reis, & Elliot, 2000). In previous studies, the BIS scale yielded an alpha coefficient of .75 (Ghebrial & Arnett) and .74 (Carver & White). Similarly, in this study, the BIS scale yielded a Cronbach's alpha of .72, whereas the BAS scale yielded a Cronbach's alpha coefficient of .84.

Whereas the Iowa Gambling task has been used to simulate punishment-reward contingencies in a real-life manner (Van Honk et al., 2003, 2004), which this study furthers as a behavioral indicator of BIS/BAS activity, the assessment of BIS and BAS activity using Carver & White's (1994) scale serves as a direct measure of these constructs tapping the endorsement of personality traits consistent with Gray's hypothetical enumeration of the BIS & BAS. A significant correlation between scores on the self-report BIS/BAS scales and with performance on the Iowa Gambling task can serve to bolster this study's operationalization of these integral constructs in furthering our knowledge regarding etiological explanations of psychopathy. For the purpose of this study, endorsement of higher self-reported BIS traits was expected to be positively correlated with a tendency to select from the less risky, advantageous decks on the Iowa gambling task. Likewise, endorsement of higher self-reported BAS traits was expected to

be positively correlated with a tendency to select from the more risky, disadvantageous decks that yield seemingly higher gains with a penalty of very high monetary losses.

State-Trait Anxiety Inventory

The State-Trait Anxiety Inventory, Form X (STAI; Spielberger, Gorsuch & Lushene, 1968) consists of 40 self-report items measuring state and trait levels of anxiety. For the first 20 items comprising the state scale, respondents are asked to indicate the degree to which each statement reflects their current state, that is, *how they feel right now at this moment*, using a four-point Likert-type scale (not at all, somewhat, moderately so, and very much so). For the next 20 items comprising the trait scale, respondents are asked to indicate to what extent the statement reflects *how they generally feel,* also on a four-point Likert-type scale (almost never, sometimes, often, and almost always). The 20 items of each scale are summed to generate total state and trait scores, respectively. The internal consistency for both scales has been reported to range from alphas of .86 to .95 across studies (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). This questionnaire was selected as one way of measuring participant's reported anxiety levels both situationally ("state") on the day of the study, as well as a more general "trait" characteristic outside of the lab setting. An assessment of anxiety will serve to inform some of the predicted relationships between high anxiety levels and behavioral and personality measures of high BIS activity. Given that cortisol has been found to be significantly related to anxiety, both anxiety and cortisol may serve as different markers for measuring BIS activity in this study. Hence, as predicted for cortisol, an endorsement of self-reported anxiety may also moderate the relationship between testosterone and risky behaviors, such that high self-reported anxiety may reduce the relationship between testosterone and risky behaviors among psychopathic

individuals. It has also been predicted that high anxiety (and/or high BIS activity) may be negatively correlated with total psychopathy scores, positively correlated with Factor 2 psychopathy scores (for exploratory analyses), and significantly related to less risky decisions (higher monetary gains) on the Iowa Gambling task.

Welsh Anxiety Scale

The Welsh Anxiety Scale (WAS; Welsh, 1956) is a self-report anxiety scale also used to assess anxiety and negative affectivity in the current study. The 39 true-false items comprising this scale have been derived from the MMPI personality inventory to assess five anxiety-related symptoms including: decreased mental efficiency, negative emotional tone, interpersonal over-sensitivity, schizoid mentation, and pessimism and loss of energy. The WAS has often been used to supplement the Psychopathy Checklist Revised in order to differentiate psychopathic offenders with high and low levels of anxiety (i.e., Schmitt & Newman, 1999a; Arnett, Smith & Newman, 1997) and has been the most widely used instrument to assess anxiety levels in psychopathy studies (Hale, Goldstein, Abramowitz, Calamari, & Kosson, 2004; Newman & Kosson, 1986). Analogous to the inclusion of the State-Trait anxiety inventory, the Welsh scale was used as a supplementary measure of anxiety to further classify psychopathic and nonpsychopathic participants into low and high anxiety groups based on median-split scores. As previously mentioned, given that high cortisol levels have been found to be related to anxiety, endorsement of anxiety items may, like cortisol, underlie BIS sensitivity and serve to moderate the relationship between testosterone and psychopathy. Individuals endorsing high Welsh anxiety were predicted to gain more money on the Iowa Gambling task, to exhibit higher behavioral and personality characteristics consistent with high BIS-activity, and to be more

likely to exhibit either secondary psychopathic characteristics, or to be classified in the nonspsychopathic group in this study.

Social Desirability Scale

The use of anonymous self-report measures, which involve minimal contact with the experimenters, was expected to mitigate the effects of social desirability in participant responses. However, social desirability was formally assessed. Because a cardinal feature of psychopathy is deception, the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960) was administered to assess response style (as in Oliver & Toner, 1990). The measure consists of 33 true-false questions reflecting culturally approved behaviors that are relatively unlikely to occur, hence testing a participant's attempt to endorse socially desirable responses. It has been used to assess a participant's motive to avoid negative evaluation or to engage in some type of deception in their responses. The scale has yielded a coefficient alpha of 0.75 (Ballard, 1993) in previous studies. If participants' responses on the Marlowe-Crowne are significantly correlated with their responses on the items assessing psychopathic and aggressive behaviors, this would indicate that their responses were influenced by a desire to appear socially desirable. To account for any effects that may be biased by socially desirable responses, social desirability was treated as a covariate within the ANOVA analyses. In summary, responses to the Marlowe-Crowne were assessed as a covariate to ensure that significant differences between groups were a result of the proposed variables, and not as a result of a response bias for selection of more socially desirable responses.

Iowa Gambling Task

The Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994) is a computerized gambling task that was used as a behavioral task sought to activate BIS and BAS motivated behavior because of its implicit design that allows participants to select from four decks of cards, each of which is associated with a specific degree of reward or punishment. Players are instructed to gain as much as money as possible by drawing 100 selections from a choice of four decks, while starting with a loan. Decisions to choose from decks are motivated by reward and punishment schedules inherent in the task. Two of the decks are disadvantageous, producing immediate large rewards but these are (after a pre-punishment phase of about 10-15 cards) accompanied by significant money loss due to extreme punishments. The other two decks are advantageous; reward is modest but more consistent. The task takes 20 minutes to administer, and yields a value for the net monetary loss or gain for each participant. As previously mentioned, the Iowa Gambling task, a behavioral index of BIS/BAS activity, served as the primary outcome variable assessing the main hypotheses of this study. Specifically, psychopathic analogues (due to the motivational imbalance model) were predicted to be more likely to engage in disadvantageous decisions on a task that mimics "real-world" decisions, as compared to nonpsychopathic analogues. Specifically, for the purpose of this study, BIS activity was operationalized as a tendency to select more cards from the "good" decks (C and D) with more consistent rewards and low monetary losses. BAS activity was consequently operationalized as a tendency to select more cards from the "bad" decks (A and B) that offer higher rewards, but even more prominent monetary losses, yielding a higher net loss in the game.

Neuroendocrine Measures: Saliva Sample Collection and Hormone Assays

As discussed earlier and further elaborated upon in the procedures section below, a noninvasive procedure that involves a collection of participants' saliva at three different times was used to directly assess levels of the hormones cortisol and testosterone. These hormones were predicted to underlie the *motivational imbalance model* that has been found with clinical and subclinical samples of psychopathic individuals in previous studies (e.g., Van Honk et al., 2003, 2004). When scheduled by telephone, participants were given specific instructions regarding factors that would impact the saliva collection. Participants were also reminded of the pre-lab "do's and don'ts" regarding saliva collections in writing via an email on the day before their scheduled appointment. Participants were asked to try to drink plenty of water on the day before and at least one glass of water one hour before their scheduled saliva collection time, in order to stay adequately hydrated. Participants were also asked to refrain from drinking water within the 10 minutes prior to saliva collection, so that the collection would not primarily consist of water. They were asked to avoid ingesting a large meal or drinking punch or lemonade in the hour prior to saliva collection. They were asked to avoid chewing gum and to avoid brushing their teeth within the hour prior to saliva collection. Finally, participants had been asked to avoid consuming alcohol to the point of intoxication (beyond the legal limit) on the night preceding their scheduled appointment time.

In an attempt to control for the natural circadian rhythm cycles for cortisol and testosterone levels in which hormones levels are highest in the morning hours with large decreases in levels (Susman, Dorn, Inoff-Germain, & Chrousaos, 1997), participants were only scheduled for sample collection within a proscribed window of time between the hours of 11:30 to 5 daily. Because this study was interested in assessing intraindividual reactivity in hormone

levels in response to the experimental manipulation task (the Iowa Gambling task), the experiment sessions were restricted to the afternoon hours, in which hormone levels become more stable. Any pronounced change in hormone levels could then be expected to be a result of reactions to the task, and not due to the normally declining levels from the elevated AM levels.

Samples were collected following the procedures outlined in Granger, Schwartz, Booth and Arentz (1999). The experimenters instructed participants on how to do the self-collection steps. The experimenter first checked to make sure that the appropriate label was on the vial and handed a short plastic straw (1/3 of a regular size straw) and a 2 ml vial with the appropriate label to the participant and said,

> *"Now we'd like to collect your saliva sample. Please take a few moments to imagine that you are eating your most favorite food.* (Experimenter should ask participant to identify a favorite food). *It is helpful if you close your eyes and imagine that you are smelling the item* (can refer to identified food). *Please move your jaw as if you are chewing the item. You should notice an increase in saliva. Please allow the saliva to accumulate in your mouth, and try not to swallow the excess saliva."* [This procedure used to stimulate saliva flow should only take 15-20 seconds. If after 20-30 seconds, she/he doesn't notice that saliva is accumulating, then she/he can gently chew on the end of one of the short sections of plastic drinking straw]. Experimenter tells the participant, "*As you notice the saliva building up, please spit it through the short plastic straw into the plastic vial that I have given you. You should hold the vial with the left hand, the straw with the right hand, and spit the saliva directly through the straw into the vial. Please continue to imagine that you are chewing your favorite food, and as saliva pools in your mouth, you can periodically spit that material through the short plastic straw into the vial. Please continue to keep collecting the saliva in your mouth and spitting it in the same way* (pool then spit, pool then spit) *until the vial is about 50% full.*"

Following receipt of the expectorated saliva samples into the vials, individually labeled vials were tightly sealed, placed into a Ziploc bag, and stored in the freezer compartment in the lab refrigerator for a maximum period of 3 days. As in previous studies (e.g., Bateup, Booth, Shirtcliff, & Granger, 2002), samples were subsequently transported in a cooler to the Penn State Behavioral Endocrinology Laboratory and stored frozen at -40°C until assay. As described by Bateup et al. (2002), all saliva samples of interest in the study were centrifuged at 3000 rpm for

10 minutes to remove mucins. The clear samples were then pipetted into testing tubes and subjected to testing to assess any potential problems in the pH level (namely, checking for low pH). Any samples assessed as outside the acceptable pH range of 4-9 were then diluted in phosphate buffered saline (PBS) to correct for pH levels prior to testing as recommended in earlier studies (e.g., Schwartz, Granger, Gunnar, & Laird, 1998). Also, as previously recommended for assay quality control purposes, samples were assayed in duplicate. Those samples that yielded duplicate values varying by more than 5% were then subject to repeated testing. The average values determined from the duplicate tests were used in the analyses.

Tests of normality revealed that the hormone data were positively skewed. Because the distributions of baseline and post-task cortisol and testosterone levels were positively skewed, logarithmic transformation as performed in other studies (Kivligan et al., 2005; Vedhara et al., 2003) was conducted to establish approximately normal distributions prior to subsequent analyses. These transformations yielded data with characteristics of more normal distributions. Whereas log-transformed values for the cortisol and testosterone samples were used across the statistical analyses, non-transformed data are reported in the means tables to provide more meaningful measures of baseline and post-task hormone levels.

Cortisol Assays (µg/dl)

Following the precise procedures utilized in previous studies (e.g., Bateup et al., 2002; Kivligan, Granger, & Booth, 2005), samples were assayed for salivary cortisol using a highly sensitive enzyme immunoassay at Salimetrics, a corporation owned by Dr. D. Granger located in University Park, PA. The test requires 25 µl of saliva for singlet determinations and has a lower limit of sensitivity of 0.007 µg/dl, a range of sensitivity from 0.007 to 1.8µg/dL, and average

intra- and interassay coefficients of variation of less than 10 and 15%, respectively. The standard curve was highly reproducible (mean r = .999). Method accuracies, determined by spike recovery, and linearity, determined by serial dilution, were 105% and 95%.

Testosterone Assays (pg/mL)

Saliva samples were assayed for testosterone using an enzyme immunoassay kit for total salivary testosterone based on similar procedures described in previous studies (e.g., Kivligan et al., 2005; Granger et al., 1999; Shirtcliff, Granger, & Likos, 2002; Bateup et al., 2002). The assay utilizes 50 μL of saliva for singlet determinations with a sensitivity or detection range of 3.70 to 360 picograms per milliliter (pg/mL). Average intra- and interassay CV's were less than 10 and 15% respectively. The standard curve was highly reproducible (mean r = .992). Method accuracies, determined by spike recovery, and linearity, determined by serial dilution, were 99.2% and 92.8%, respectively.

Procedures

Approximately 1100 participants completed screening questionnaires assessing psychopathic types of behaviors. From this large sample, the goal was to identify 60 male and 60 female participants as psychopathic and nonpsychopathic analogues, who were willing to continue participation in the study. Those who expressed their willingness to participate were asked to come to the lab to complete additional measures assessing self-report BIS/BAS constructs, anxiety and aggression. Participants were also asked to engage in a behavioral approach-avoidance task, the Iowa Gambling Task (Bechara, Damasio, & Damasio, 1994). This task consists of variable punishment (loss of money) and reward (gain of money) conditions.

To assess hormone levels, a non-invasive procedure was conducted for collecting saliva. Saliva samples were collected and assayed for levels of cortisol and testosterone at baseline prior to the administration of the behavioral task, and following peak arousal on the Iowa Gambling Task. Hence, saliva was collected on two occasions on the day of the experiment, by passive drool. This involved asking participants to spit through a straw into a small plastic vial as previously described. During each collection, approximately 2 mL of saliva was requested. The first measure served as a baseline measure, which was collected immediately following the informed consent form. To capture peak arousal levels of hormone reactivity, the second measure was obtained 20 minutes after the behavioral task (gambling task). Participants were also supplied with a saliva take-home kit, consisting of a plastic vial, a straw, and instructions for the collection and storage of a third saliva sample. They were asked to provide one saliva sample at the exact same time of day as the baseline measure on the following day of the experiment and to drop off this sample the next day. The at-home sample served as a comparison measure to the baseline collected on the day of the experiment. This procedure has been recommended to assess whether the baseline collected on the day of the experiment truly represents a baseline normal state, or whether the baseline may have been impacted by the nervousness/anxiety associated with participating in an experiment.

Prescreening Phase I

The first phase of this study involved the initial administration of two psychopathy questionnaires used as pre-screening devices in the Psychology department's mass screening subject pool. Of the approximately 1,676 subject pool participants in the Spring Semester of 2004, an estimated 1100 students received packets that included both or at least one of the

screening measures used in this study. Out of these 1100, the screening procedures yielded data from 706 participants who completed either one or both of the screening measures. Of these 706 total participants, 636 students completed both screening questionnaires, whereas 70 of the participants completed only one of the screening Q's (53 Levenson Psychopathy Scale only, 17 Self-Report Psychopathy Scale only).

From this large pool, a list of participants was identified based on scoring in the upper and lower quartiles (25%) on the screening questionnaires. Although it was preferable to select participants who scored in the upper and lower quartiles on both screening questionnaires, a subset of the subject pool participants only completed one of the two screening questionnaires. An effort was then made to initially contact all individuals who were identified as either a "psychopathic analogue" or a "non-psychopathic analogue" on both questionnaires. However, when all individuals based on quartile scores from this pool were called and invited and that list was completely utilized, individuals with scores on only one of the questionnaires were subsequently invited to participate in the study. Based on quartile scores, 353 individuals qualified to participate in this study. A total number of 327 individuals were contacted (via email and phone contact) and invited to participate in waves across the semester, starting with the highest and lowest in the range of quartile scorers until the desired sample size was obtained. From the participants who had completed both questionnaires, further analyses revealed that all participants who were classified as "psychopath" or "nonpsychopath" on one questionnaire, were also classified in the same group when examining their scores on the second questionnaire, lending validity to the classification of participants into groups based on responses to just one of the screening questionnaires. Over the course of the phone pre-screening interview process, 3 participants who were interested in participating were excluded based on the selection criteria

outlined for the hormone analyses (1 excluded for daily use of inhaler; 1 excluded because reported a melanoma; 1 for endocrine/thyroid condition). Out of the final sample of 114 participants in the study, only 3 participants were selected based on having scores on only one out of the two screening questionnaires.

Two measures were selected to screen for high and low psychopathy characteristics within this college population. The Primary and Secondary Psychopathy Scale (Levenson, Kiehl, & Fitzpatrick, 1995) was selected as a brief 26-item measure that assesses a continuous or dimensional degree of psychopathic traits found in college samples. It distinguishes interpersonal styles and philosophies that capture features of primary and secondary psychopaths detectable among university students who are unlikely to meet clinical diagnostic criteria (Levenson et al., 1995). This measure contains no questions pertaining directly to criminal activity, and hence may be less subject to elicit defensive and/or socially desirable responding patterns. Although they found low base-rates, Levenson et al. (1995) found sufficient endorsement on primary psychopathy items in their university sample to permit interpretation. Twenty-three percent of the male and 6% of the female respondents endorsed 8 or more of the 16 primary psychopathic items. Internal consistency coefficients have been reported to be 0.82 for primary psychopathy and 0.63 for secondary psychopathy, respectively (Levenson et al., 1995). For the purpose of identifying "psychopathic" and "nonpsychopathic" participants for this study, participants were classified based on their total sum score across both primary and secondary factors, similar to how participants are typically classified based on their sum score on the Psychopathy Checklist Revised (PCL-R; Hare, 1991) across most studies.

The second measure used for screening purposes was the Self-Report Psychopathy Scale—Version III (SRP-III; Hare, Paulhus, & Hemphill, 2002), a 40-item self-report scale

derived from Hare's (1991) Psychopathy Checklist Revised, the gold standard measure for assessing the construct of psychopathy. The last 10 items of this scale directly assess involvement in illegal/criminal activity in the previous 5 years. The SRP-III requires respondents to rate the extent to which they agree or disagree with a set of statements using a five point Likert-type scale. The first version of the scale (SRP; Hare, 1985) was constructed empirically by identifying items that discriminated between low and high psychopathy groups as assessed by the PCL. Much like the PCL, the SRP also consists of two factors, one aimed at assessing the core personality features of psychopathy or Factor 1 (e.g., callousness, grandiosity) and the second assessing antisocial behaviors, and thrill-seeking behaviors associated with psychopathy, or Factor 2 (Lilienfeld & Hess, 2001). In their undergraduate sample (Williams, Paulhus, & Hare, 2004, submitted manuscript), the internal consistency (Cronbach's alpha) of the overall SRP-III was reported to be .91. A study assessing the validity of the scale and its link to similar personality and behavior measures provided evidence that the SRP-III is a valid measure of psychopathy in a normal population (Williams et al.). The SRP-III was significantly correlated with the Levenson Self-Report Psychopathy Scale ($r = .63$, $p < .01$), with the Psychopathic Personality Inventory ($r = .33$, $p < .01$), and with other personality and behavioural measures of delinquency and antisocial behaviours (Williams et al.). Once again, as with the previous screening measure, participants were classified into respective "psychopath analogue" and "nonpsychopath analogue" groups based on their total score across Factor 1 and Factor 2 subscales. Participants were selected and contacted based on total scores falling in the upper and lower quartiles across the distribution. Given that the SRP-III has been described as a scale that provides a reliable, valid and comprehensive measure of subclinical psychopathy in non-forensic

samples (Williams et al.) its usage as a screening measure in a non-forensic sample was well-suited to the focus of the current study.

Pearson correlations between the two screening questionnaires yielded significantly positive correlations between the Levenson Primary and Secondary Psychopathy scale (LPSP) and the Self-Report Psychopathy (SRP-III) Scale measures, r (110) = .66, p = .00. Positive correlations were also obtained between each of the screening questionnaires with the Psychopathic Personality Inventory (PPI-short) administered on the day of the study. The PPI-short administered on the day of the study, partially as a validity check of the psychopathic and nonpsychopathic group classifications, was positively correlated with the SRP-III, r (105) = .60, p = .00, and with the LPSP scales, r (104) = .40, p = .00, respectively.

Subsequent analyses were conducted to assess any potential differences between individuals who participated in the study and individuals who qualified based on their scores from the screening measures but declined the invitation to participate. The only available data on the non-participants consisted of demographic information (gender and ethnicity) and scores on the screening questionnaires. Although, as a group, participants and non-participants did not differ in gender, ethnicity or in their endorsement of items on the Levenson Primary and Secondary Psychopathy scales, the non-participant group endorsed fewer items on the Self-Report Psychopathy scale than individuals who participated in the study as assessed by a T-test analysis, t (169) = -3.11, p = .00 ($M_{\text{nonparticipants}}$ = 60.10, $M_{\text{participants}}$ = 66.13).

Pre-screening Phase II

Eligible participants based on scoring in the upper and lower quartiles were then initially contacted via a mass-email invitation. Those who responded to the email, stating their interest in

participating then received a phone call from one of the research assistants. This phone call briefly introduced the study and entailed a list of screening questions to determine eligibility to participate in the study, which is included herein as Appendix A. Eligible participants were then given a scheduled appointment to come to the lab to participate in the study. They were also given a set of instructions regarding preparation for providing saliva samples. For example, among other things, they were asked to drink a glass of water within about one hour before their appointment time, to refrain from smoking for two hours prior, and to avoid ingesting a full meal for approximately two hours prior to their scheduled appointment time.

Pre-screening Phase III

On the day of the experiment, participants came to their scheduled appointment in the lab with an experimenter who was blind as to the group membership of the participants (psychopathic or nonpsychopathic analogue group). Participants were greeted and asked to be seated. The experimenter proceeded to ask further pre-screening questions to assess continued eligibility for participation in the study on that day. The questions included an assessment of the participant's mood, use of alcohol, drugs or cigarettes, illness, stress level, consumption of food, and use of an asthma inhaler—all of which could influence the hormone content in their saliva. These questions are included as Appendix A. If participants were still eligible to participate in the study on that day based on their responses to the aforementioned questions, the experimenter then administered the final pre-screening measure, which is an abridged version of the Daily Inventory of Stressful Events (DISE; Almeida, Wethington, & Kessler, 2002).

The DISE is a semi-structured interview consisting of four components, including a) a list of seven stem questions that pertain to occurrences of stressful events across various life domains

that have occurred in the previous 24 hours, b) a series of open-ended probe questions to elicit a description of the stressful event, c) a question regarding the perceived severity of the experienced stressor, and d) a list of structured questions aimed at assessing the impact of the stressor on various avenues of the individual's life. For the purposes of screening for stressful events that may have occurred within the immediate past in the participant's life that may inadvertently affect the participant's cortisol levels, the experimenter only administered part "A" and "C" from above, namely conducting a brief semi-structured type of interview, asking the participant the seven stem questions, and following up (if relevant) with a question assessing the participant's appraisal of the degree or severity of the stressor, ranging from not at all to very stressful on a four-point scale. The modified DISE used in this study is included herein as Appendix B. Participants were only asked to reschedule if they reported a stressful event and rated the event as *very stressful*. This concluded the pre-screening phase of the study.

Lab Tasks & Procedures

For participants who were still eligible, the study commenced by having them read and sign the Informed Consent form. Subsequently, participants were asked to give their first, baseline saliva sample. Following the first saliva sample collection, participants were then escorted to a computer in the lab, and were read aloud instructions as to how to play a card game (Iowa Gambling Task). The Iowa Gambling task takes about 20 minutes to complete. Following the task, participants were then administered the questionnaire packets, and were told that they would be interrupted in 20 minutes to provide the second saliva sample. If they had not finished the questionnaires, they continued to complete them following the second saliva sample collection. The tester remained in the testing room throughout the experiment. Following the

second saliva collection and the completion of the questionnaires, the experimenter handed participants the at-home saliva collection kit, and gave them instructions regarding proper storage and return of the samples. Participants were then awarded their compensation of $5 and were told that they would only receive course credit when they returned their final saliva sample.

Results

Means and standard deviations for all of the germane variables including psychopathy, anxiety, BIS, BAS, gambling task performance and hormone measures are classified by gender and psychopathy groupings in Table 1. Zero-order correlations among the aforementioned variables are also provided in a correlations matrix table (Table 2). Alpha coefficients obtained across the various scales used in the study are also provided (Table 3). Initial analyses using Pearson correlations yielded many of the predicted relationships between psychopathy, risky behaviors (as operationalized by performance on the gambling task), and behavioral inhibition and activation measures, whereas some of the predicted relationships between psychopathy, anxiety, and hormone measures failed to yield the predicted associations. Each of the predicted hypothesized relationships and the subsequent analyses are presented in detail as follows.

Psychopathy & the Motivational Imbalance Model: Response Modulation Deficits

Hypotheses 1 and 2 (gambling task: risky behaviors): As predicted by our first two hypotheses, psychopathic participants were less likely to suspend their approach behaviors in response to punishment (loss of money) by making more selections from risky decks, whereas nonpsychopathic participants made more selections from the more conservative, advantageous decks. A 2 x 2 Analysis of Variance (psychopath group, gender) with the sum of good minus bad selections on the gambling task as the dependent variable yielded a significant main effect for group, such that the psychopathic group made significantly more card selections from the disadvantageous decks (decks A and B) on the Iowa gambling task, whereas the nonpsychopathic group made significantly more selections from the advantageous decks, $F(1, 111) = 8.39$, $p < .01$ ($p = .005$). Since the nonpsychopathic group exhibited a tendency to

provide significantly more socially desirable responses than psychopathic participants, t (111) = 4.89, p = .00 ($M_{nonpsychopath}$ = 53.00, $M_{psychopath}$ = 48.57), analyses were also conducted entering social desirability as a covariate. When the effects of social desirability where entered as a covariate, the same 2 x 2 ANOVA (gender, group) still yielded the same predicted relationship with a significant main effect for group, F (1, 111) = 7.05, p < .01 (p = .009).

Self-reported BIS and BAS Activity

Hypothesis 3: Contrary to prediction, the behavioral manifestation of BIS activity on the gambling task (as measured by more selections from *advantageous decks*) was not significantly (positively) related to a personality-based measure of self-reported BIS activity (Carver and White's (1994) scale), r (113) = -.05, p = *ns.*

Hypothesis 4: Also, contrary to prediction the behavioral manifestation of BAS activity on the gambling task (as measured by more selections from *disadvantageous decks*) was not positively related to a personality-based measure of self-reported BAS activity (Carver and White's (1994) scale), r (113) = .08, p = *ns.*

Anxiety Correlates

Hypothesis 5: As predicted, self-report BIS activity was positively correlated with all measures of anxiety used in this study, including state anxiety, r (114) = .32, p = .00 trait anxiety, r (90) = .51, p = .00, and WAS anxiety, r (113) = .39, p = .00, respectively. Contrary to prediction, self-report BAS activity was not significantly negatively correlated with any of the measures of anxiety.

Hypothesis 6: Since the literature has revealed mixed findings in the relationships between anxiety and psychopathy, these are exploratory analyses. Individuals who scored higher on psychopathy were expected to endorse lower anxiety levels than those classified as nonpsychopathic, or there may be no significant relationship between anxiety and overall psychopathy. Specifically, Factor 1 psychopathy was expected to be negatively related to measures of anxiety while secondary or Factor 2 psychopathy was expected to be positively related to measures of anxiety. Any differences obtained among high and low anxious psychopathic and nonpsychopathic participants were expected to be in the same direction.

Contrary to predictions, individuals in the psychopathic group endorsed significantly higher levels of self-reported trait anxiety (STAI-T), $t(88) = 2.09$, $p < .05$, and a similar marginal trend emerged in the same direction for self-reported state anxiety (STAI-S), $t(112) = 1.96$, $p = .056$. Also, contrary to prediction, results from a one-way ANOVA indicated that the psychopathic group endorsed significantly higher Welsh anxiety levels as compared to the nonpsychopathic group, $F(1, 111) = 3.81$, $p = .05$. Since unexpected relationships emerged for a positive relationship between psychopathy and anxiety, further analyses were conducted to assess the relationship between anxiety and the two factors of psychopathy as assessed by the Psychopathic Personalilty Inventory Short form (PPI; Lilienfeld, 1996) in this study. Zero-order correlations were examined between PPI Factor 1 psychopathy (the personality and affective dimension) and PPI Factor 2 psychopathy (antisocial lifestyle) scores with the anxiety measures assessed in this study. Analyses were conducted that involved partialling out the effects of one factor from the other as recommended in the recent psychopathy literature (i.e., Patrick, 1994). Factor 1 psychopathy scores as measured by Lilienfeld's (1996) Psychopathic Personality inventory were significantly negatively correlated with anxiety as measured by the Welsh

Anxiety Scale (WAS), r (110) = -.33, p = .00, with trait anxiety as assessed by the STAI, r (88) = -.48, p = .00, and with state anxiety as assessed by the STAI, r (111) = -.29, p = .00. Factor 2 psychopathy scores as measured by Lilienfeld's (1996) PPI scale were significantly positively correlated with anxiety as measured by the Welsh Anxiety Scale (WAS), r (108) = .37, p = .00, with trait anxiety (STAI), r (85) = . 40, p = .00, and with state anxiety (STAI), r (109) = .27, p = .00, respectively.

Psychopathy & WAS Anxiety: Is there a moderating role of anxiety in BIS/BAS activity?

One area of interest in this study was to determine how anxiety impacts the relationship between psychopathy and risky decisions. To examine this set of questions, separate 2 x 2 Univariate ANOVA analysis with group status (psychopath, nonpsychopath) and median-split Welsh Anxiety level (low, high) as between-subject factors were conducted for the 3 dependent variables of interest—overall gambling task performance as a measure of risky decisions, self-reported BAS and self-reported BIS indices. To control for the effect of social desirability, Marlowe-Crowne scores were entered as a covariate in each of the three analyses. The first 2 x 2 ANOVA, assessing differential performance on the Iowa Gambling Task as a behavioral measure of BIS/BAS motivated activity, yielded a significant main effect for psychopathy group, a marginal main effect for WAS anxiety level and no significant Psychopathy X Anxiety interaction. Performance on the gambling task varied as a function of psychopathy group status, such that psychopathic analogues had significantly poorer (less advantageous) performance on this task as compared to nonpsychopath analogues, F (1, 106) = 8.40, p = .01 ($M_{\text{psychopath}} = 2.76$, $M_{\text{nonpsychopath}} = 20.83$). Performance on the gambling task also varied as a function of Welsh anxiety level, in that individuals with lower WAS scores performed better than individuals with

higher WAS scores, but this main effect marginally approached significance, F (1, 106) = 3.60, p = .06. Given that no significant Psychopathy X Anxiety interaction emerged, F (1, 106) = .67, p = .42, *ns,* this suggests that anxiety (as measured by Welsh scores) did not significantly moderate the relationship between psychopathy and risky behaviors on the gambling task.

The second 2 x 2 ANOVA, assessing differential endorsement of self-reported behavioral activation system (BAS) traits, yielded a significant main effect for psychopathy group, no main effect for anxiety, and no interaction. Endorsement of self-reported BAS activity varied as a function of psychopathy status, such that psychopath analogues endorsed significantly higher BAS scores as compared to nonpsychopath analogues, F (1, 106) = 12.30, p = .00 ($M_{\text{psychopath}}$ = 41.07, $M_{\text{nonpsychopath}}$ = 37.18). No significant main effect was obtained as a function of anxiety (WAS), F (1, 106) = .31, p = .58, *ns,* and anxiety scores did not significantly moderate the relationship between psychopathy and BAS activity, as the interaction between psychopathy and anxiety was nonsignificant, F (1, 106) = .00, p = .98, *ns.*

Finally, a third 2 x 2 ANOVA, assessing differential endorsement of self-reported behavioral inhibition system (BIS) traits, yielded a significant main effect of anxiety level, but no significant main effect for psychopathy status, and no psychopathy X anxiety interaction. Endorsement of self-reported BIS activity varied as a function of anxiety level, such that individuals with high (WAS) anxiety scores endorsed significantly higher BIS scores as compared to those in the low anxiety group, F (1, 106) = 22.01, p = .00 ($M_{\text{high Anxiety}}$ = 20.48, $M_{\text{low Anxiety}}$ = 17.70). No significant main effect was obtained as a function of psychopathy group, F (1, 106) = .65, p = .42, *ns,* and anxiety scores did not significantly moderate the relationship between psychopathy and BIS activity, as the interaction between psychopathy and anxiety was nonsignificant, F (1, 106) = .01, p = .91, *ns.*

Gray's model (BIS/BAS) & Hormone Relationships

Due to the potential confounding effects attributed to circadian rhythm variations in HPA activity across the afternoon hours, time of day for the baseline and post-task hormone level samplings (for cortisol and testosterone) were recorded and entered as a covariate in the GLM analyses. Across all analyses, Time 1 and Time 2 were unrelated to any of the tested dependent variables, including overall performance on the Iowa gambling task and endorsement of items on the self-report BIS and BAS (Carver & White, 1994) scales.

Hypothesis 7: Contrary to prediction, baseline and post-task cortisol were neither related to self-reported BIS activity (Carver & White's (1994) scale), r (113) = .03, *ns* and r (114) = -.08, *ns*, respectively, nor to BIS-motivated activity as reflected in advantageous IGT decisions, r (112) = .01, *ns*, and r (113) = .04, *ns.*

Hypothesis 8: Contrary to prediction, correlational analyses revealed that baseline cortisol was neither related to the state, r (113) = .035, *ns*. nor to the trait anxiety measures, r (89) = .06, *ns*. on the State Trait Anxiety Inventory. Similarly post-task cortisol levels were also unrelated to state, r (114) = -.10, *ns*. and trait anxiety levels, r (90) = -.05, *ns*. Correlational analyses also revealed no significant relationship between levels of baseline and post-task cortisol and self-reported anxiety as measured by the Welsh Anxiety Inventory, r = .08, *n.s* and r = .06, *ns.*, respectively.

Hypothesis 9: As predicted, baseline and post-task testosterone levels were negatively correlated with measures of anxiety. However all of these relationships were non-significant with one exception. Baseline testosterone level was significantly negatively correlated to trait anxiety, r (90) = -.21, p = .05.

Hypothesis 10: Contrary to prediction, Independent Samples T-test analyses revealed no significant differences in the means of the baseline and post-task log-transformed cortiosl values across psychopathic and nonpsychopathic analogues. Psychopathic participants did not exhibit lower levels of either baseline or peak arousal cortisol levels as compared to nonpsychopathic participants. Additionally, when a change score was calculated in level of post minus pre-task cortisol levels, a t-test also revealed no significant correlation between psychopathy and the change in cortisol level from baseline to peak arousal following the task.

Hypothesis 11: Contrary to prediction, baseline and post-task testosterone levels were neither positively related to self-reported BAS activity nor to BAS-motivated behavior based on performance (disadvantageous selections) on the gambling task. Contrary to prediction, Pearson correlation coefficients revealed that baseline and post-task testosterone were negatively correlated with self-reported BAS activity, $r\ (114) = -.23$, $p < .05$ and $r\ (114) = -.31$, $p < .01$, respectively.

Hypothesis 12:

Testosterone:

To examine any differential change in testosterone level across groups following the gambling task, a Repeated Measures 2 X 2 ANOVA with psychopathic group (psychopath, nonpsychopath) and anxiety group (low, high) as between-subject variables and baseline testosterone and peak post-task testosterone levels as the repeated measure (within-subject variables) was examined. This analysis failed to yield any significant within-subjects effects for a change in testosterone from baseline to post-task; however, a significant between-subjects effect was obtained for the Welsh anxiety variable, $F\ (1, 109) = 4.03$, $p = .047$, suggesting that level of anxiety was inversely related to testosterone level, such that participants with "low"

anxiety had significantly higher testosterone levels as compared to participants with "high" anxiety levels. No support was obtained for the prediction that psychopathic individuals would exhibit higher testosterone levels as compared to nonpsychopathic individuals.

Cortisol:

Likewise, to examine any differential change in cortisol level across groups following the gambling task, a Repeated Measures 2 X 2 ANOVA with psychopathic group (psychopath, nonpsychopath) and anxiety group (low, high) as between-subject variables and baseline cortisol and peak post-task cortisol levels as the repeated measure (within-subject variables) was examined. This analysis yielded a significant within-subjects main effect for a change in cortisol level across participants from baseline to post-task levels, $F (1, 108) = 20.01$, $p = .00$, such that cortisol level significantly decreased following the gambling task. Although no statistically significant within-subjects effect was obtained as a function of either psychopathy group, anxiety level, or the interaction of both of these between-subject factors, an effect that approached significance was observed for Cortisol Level X Anxiety Group, $F (1, 108) = 3.46$, $p = .066$. This marginal effect suggests that, whereas all participants regardless of anxiety level showed a significant decrease in cortisol level following peak arousal levels on the task, a trend emerged in that those in the "high" anxiety group seemed to have a larger decrease in cortisol levels than those in the "low" anxiety group.

Gender, Testosterone & Aggression

Hypothesis 13: As predicted, females exhibited lower levels of both baseline testosterone (log-transformed values), $t (111) = 9.03$, $p = .00$, ($M_{females} = 2.02$, $M_{males} = 2.42$) and post-task

testosterone, t (111) = 8.93, p = .00, ($M_{females}$ = 2.04, M_{males} = 2.45) as compared to male participants, but did not significantly differ from men in self-reported aggressive symptoms, t (106) = 1.73, p =.09, *ns*.

Psychopathy-Gender Interactions (BAS & BIS behaviors)

A multivariate 2 x 2 ANOVA examining the between-group effects of psychopathy group (psychopath, nonpsychopath) and gender (male, female) on the 3 BIS/BAS dependent variables (IGT performance on good – bad decks, self-report BAS, and self-report BIS indices) was conducted to assess hypotheses regarding the unique effects of gender on BIS/BAS activity. Although no specific a priori hypotheses were enumerated for the relationships between psychopathy and gender on the self-report BIS/BAS activity measures, if the BIS and BAS scales assess the same constructs assessed by the gambling task, then the same relationships obtained for the IGT would be expected based on the *response modulation model*. Findings demonstrated mixed support for these hypotheses. As predicted, no gender differences were obtained for BIS and BAS-motivated behavioral responses on the Iowa Gambling task. Univariate findings from a 2 x 2 (psychopathy group, gender) ANOVA with performance measured on good minus bad card selections across all decks failed to yield a main effect for gender, F (1, 106) = .49, p = .49, *ns,* but did yield the previously reported main effect for psychopathy group. Also as predicted, there were no significant psychopathy group X gender interactions for predicting advantageous and disadvantageous BIS/BAS motivated decisions on either the gambling task or the self-reported BAS and BIS dependent variables as will be described below.

However, the multivariate 2 x 2 ANOVA examining the between-group effects of psychopathy group (psychopath, nonpsychopath) and gender (male, female) on self-reported BAS and BIS indices (dependent variables) yielded significant main effects for psychopathy, $F(2, 106) = 7.74$, $p = .00$, and for gender, $F(2, 106) = 13.13$, $p = .00$, with no significant interaction. To further examine the between-group effects, the Univariate findings from the 2 x 2 ANOVA yielded a significant main effect for psychopathy group in predicting BAS activity, $F(1, 107) = 15.33$, $p = .00$, but no main effect for psychopathy in predicting self-report BIS activity. Additionally, Univariate findings yielded significant main effects for gender in predicting BAS activity, $F(1, 107) = 12.72$, $p = .00$, and in predicting BIS activity, $F(1, 107) = 12.65$, $p = .00$, respectively. No significant psychopathy group X gender interactions were obtained. However, contrary to prediction, gender yielded significant main effects on the self-reported measures of BAS, $F(1, 106) = 12.51$, $p = .00$, ($M_{\text{males}} = 37.30$, $M_{\text{females}} = 40.68$) and BIS activity, $F(1, 106) = 12.64$, $p = .00$, ($M_{\text{males}} = 17.91$, $M_{\text{females}} = 20.00$) respectively. Since this study predicted no unique differential gender differences in BIS/BAS activity, these findings were unexpected in that women endorsed more BAS and BIS activity items than men.

Testosterone Analyses by Gender:

Given the expected significant gender differences found in testosterone levels among men and women, a series of analyses were conducted to assess the effects of testosterone level, separately for men and women, on behavioral and self-report measures of BIS/BAS activity. In order to examine these relationships, median-split groups were formed using the baseline and post-task testosterone levels obtained for men separately, and then for women separately, to classify participants into high and low testosterone and cortisol groups. This procedure of

dichotomizing groups based on cortisol (and testosterone) concentrations using median-split scores has been used in previous studies (e.g., McBurnett et al., 2000). Multivariate 2 X 2 ANOVA analyses were then conducted examining the between-group effects of baseline testosterone levels (Low, High) and post-task testosterone levels (Low, High) on the 3 dependent variables reflecting BIS/BAS activity (overall IGT performance, and self-report BIS and BAS total scores). When this 2 x 2 ANOVA was assessed for men, no significant main effects or interactions were obtained for any of the BIS/BAS activity dependent variables. Likewise, when the same multivariate 2 X 2 ANOVA analysis was conducted using the median-split grouping variables for baseline (Low, High) and post-task testosterone levels (Low, High) for women, no significant main effects or interactions were obtained in predicting BIS/BAS behavioral and self-report indices as a function of testosterone level.

Moderator Relationships: Hormones, Risky Behaviors & Psychopathic Status

Hypotheses 14 & 15: Finally, Hierarchical Linear Regression analysis was used to assess the hypothesis that cortisol would moderate the relationship between testosterone and psychopathic behaviors. In particular, it was hypothesized that the relationship between testosterone and risky (psychopathic) behavior, as measured by card selections from disadvantageous decks, was expected to weaken when cortisol levels were high and strengthen when cortisol levels were low. The predicted moderator role of cortisol failed to reach statistical significance across these analyses. The first regression equation aimed to assess the moderating role of baseline cortisol level. Using hierarchical regression with risky behaviors as measured by selections from the disadvantageous decks (A and B) as the dependent (criterion) variable, the independent (predictor) variables were entered on separate blocks in the following order. Baseline

testosterone (log-transformed) level was entered on Block 1, then baseline cortisol (log-transformed) level on Block 2, and finally the interaction term (product) of the baseline testosterone and baseline cortisol indices on Block 3. This latter index, representing the testosterone X cortisol interaction was used to evaluate the moderation hypothesis. Baseline testosterone and baseline cortisol levels did not significantly predict differential performance on the gambling task, R^2 change = .005, p = .46, *ns*, and R^2 change = .001, p = .80, *ns,* respectively. Also, contrary to prediction, the interaction of baseline testosterone and cortisol levels significantly failed to predict differential performance on the gambling task, R^2 change = .02, F_{change} (1, 108) = 2.33, p = .13, *ns*. The regression equation including all of the aforementioned predictors was non-significant (baseline testosterone, baseline cortisol, and the interaction of testosterone x cortisol) accounting for only 3% of the variance, with cumulative R^2 = .03, p = .13, *ns*. When the same predictor variables were entered in a hierarchical linear regression examining overall performance on the gambling task (good minus bad card selections) as the criterion variable, comparable results were obtained.

The next hierarchical linear regression equation aimed to assess the moderating role of post-task cortisol level in the relationship between testosterone and psychopathic risky behaviors. As in the previous analysis, risky behaviors as measured by selections from the disadvantageous decks (A and B) served as the dependent (criterion) variable and the independent (predictor) variables were again entered on separate blocks in the following order. Post-task testosterone (log-transformed) level was entered on Block 1, then post-task cortisol (log-transformed) level on Block 2, and finally the interaction term (product) of the post-task testosterone and cortisol indices on Block 3. As previously mentioned, this latter index, representing the testosterone X cortisol interaction was used to evaluate the moderation hypothesis. Post-task testosterone and

post-task cortisol levels did not significantly predict differential performance on the gambling task, R^2 change = .006, p = .42, *ns*, and R^2 change = .003, p = .55, *ns*, respectively. Also, contrary to prediction, the interaction of post-task testosterone and cortisol levels did not significantly predict performance on the gambling task, R^2 change = .005, p = .47, *ns*.

Further Exploratory Analyses: Gender, Risky Behaviors & Hormone Relationships

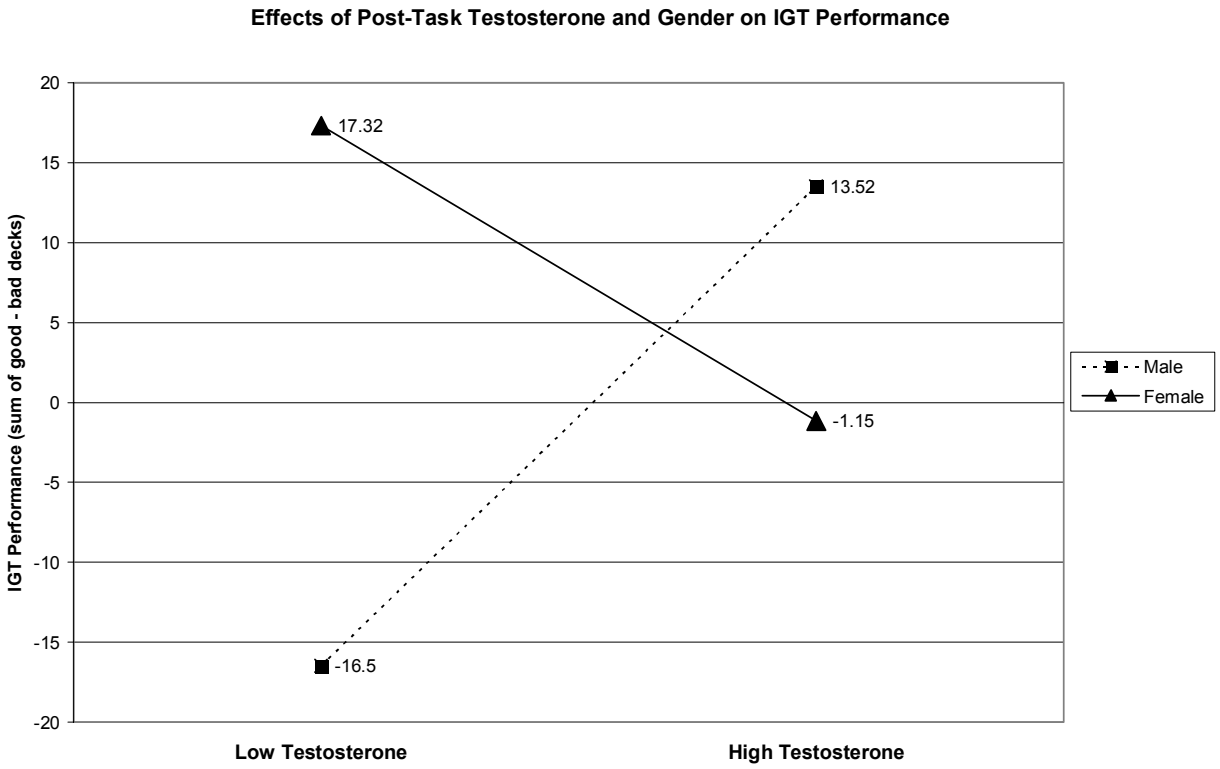
Post-hoc exploratory analyses yielded interesting and unexpected moderator relationships among hormones, gender, and gambling task performance. In the first analysis, a 2 (psychopath, nonpsychopath) x 2 (male, female), x 2 (low, high baseline testosterone) x 2 (low, high post-task testosterone) Univariate ANOVA was conducted examining the main effects and interactions among psychopathy, gender, and pre and post-task testosterone levels on overall performance on the gambling task (good minus bad selections on the IGT) as the dependent variable. Social desirability was entered as a covariate in this analysis. No significant main effects were obtained for group, gender, or median-split baseline and post-task testosterone groups in the univariate 2 x 2 x 2 x 2 analysis. However, a significant interaction emerged between post-task testosterone and gender in predicting overall performance on the gambling task, F (1, 111) = 12.33, p = .001. Given that gender is a static variable and baseline testosterone levels did not significantly interact with gender to predict task performance, it is likely that testosterone levels moderated performance and that the differential arousal level leading to these differences is attributed to reactions to the gambling task itself. Cell means depicting significant differences in gambling task performance in this interaction are presented in Table 1. Follow-up t-test analyses conducted to further decipher the results from this statistical interaction revealed that men in the high testosterone group exhibited a more advantageous pattern of card selections on the

gambling task as compared to men in the low testosterone group, t (50) = -2.44, p = .02. The opposite finding was obtained for female participants across post-task testosterone, such that females with low testosterone levels performed significantly better than those with high testosterone levels, t (58) = 2.09, p = .04. Although the sample size was unequal across these four cells, with a smaller number of men in the low testosterone cell and women in the high testosterone cell respectively, these data provide opportunities to examine gender differences in biologically "deviant" groups on a measure of risky, psychopathic behaviors. The graph in Figure 5 depicts this interaction.

Table 1. Cell means depicting the interaction between gender and testosterone in predicting gambling task performance.

		Gender	
		Men	Women
Testosterone	Low	-16.50	17.32
	High	13.52	-1.15

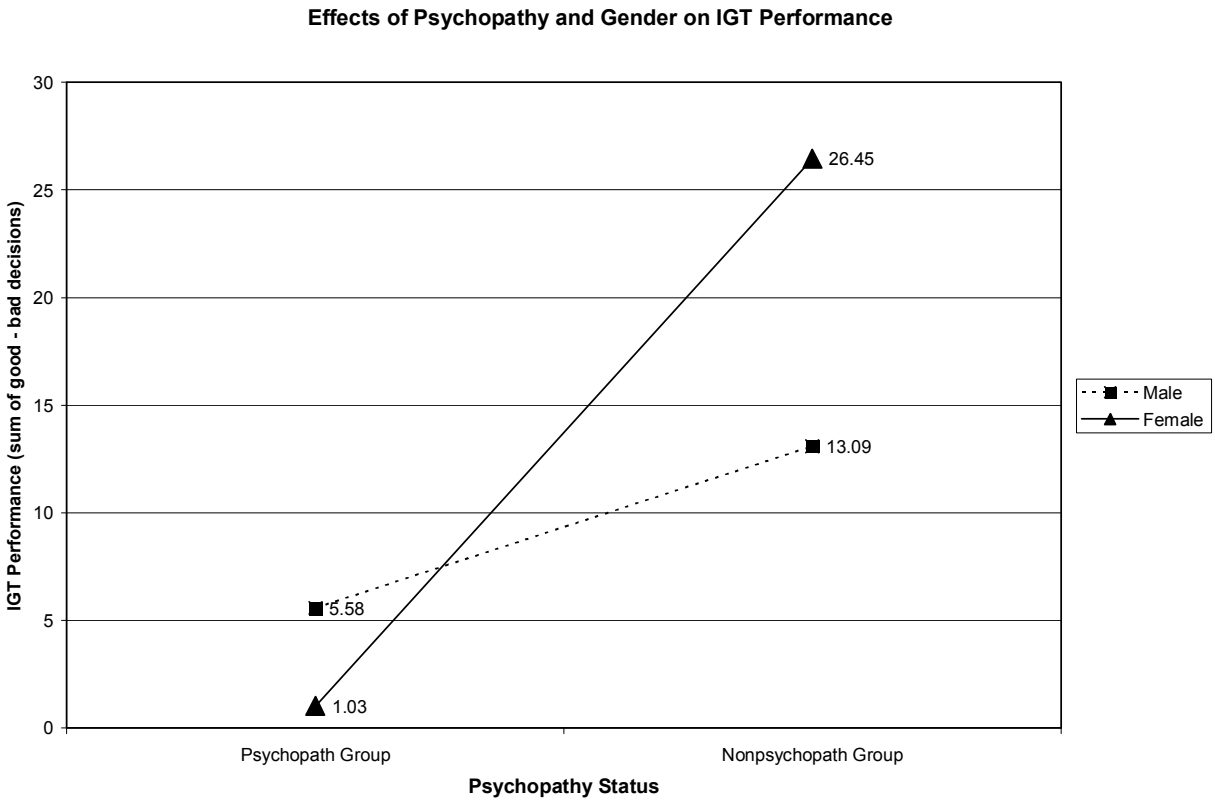
Figure 5. The Effects of Post-Task Testosterone and Gender on IGT Performance



An analogous analysis was conducted with median-split cortisol groups. A 2 (psychopathy group) x 2 (gender) x 2 (low, high baseline cortisol groups) x 2 (low, high post-task cortisol groups) Univariate ANOVA analysis was conducted assessing the main effects and interactions among psychopathy, gender and median-split cortisol levels on overall gambling task performance. A significant main effect was obtained for psychopathy group as previously described, such that nonpsychopathic participants made more advantageous decisions, whereas psychopathic participants made more risky, disadvantageous decisions on the gambling task, $F(1, 110) = 4.79$, $p = .03$. No main effects were obtained for gender. Also no main effects were obtained for either the median-split baseline (high, low) cortisol or post-task cortisol (high, low) groups. However, two significant interactions emerged in these analyses. A significant

psychopathic group X gender interaction was obtained in predicting overall performance on the gambling task, F (1, 110) = 4.36, p = .04, such that, as a group, nonpsychopathic female analogues made the best decisions on the task, as illustrated in the graph below (Figure 6). Follow-up t-test analyses revealed that among male participants, psychopathic status did not significantly impact performance, t (50) = -.80, p = .43, *ns* ($M_{psychopath}$ = 5.58, $M_{nonpsychopath}$ = 13.09). However, among females, nonpsychopathic participants performed significantly better on the gambling task than psychopathic participants, t (58) = -3.75, p = .00 ($M_{psychopath}$ = 1.03, $M_{nonpsychopath}$ = 26.45).

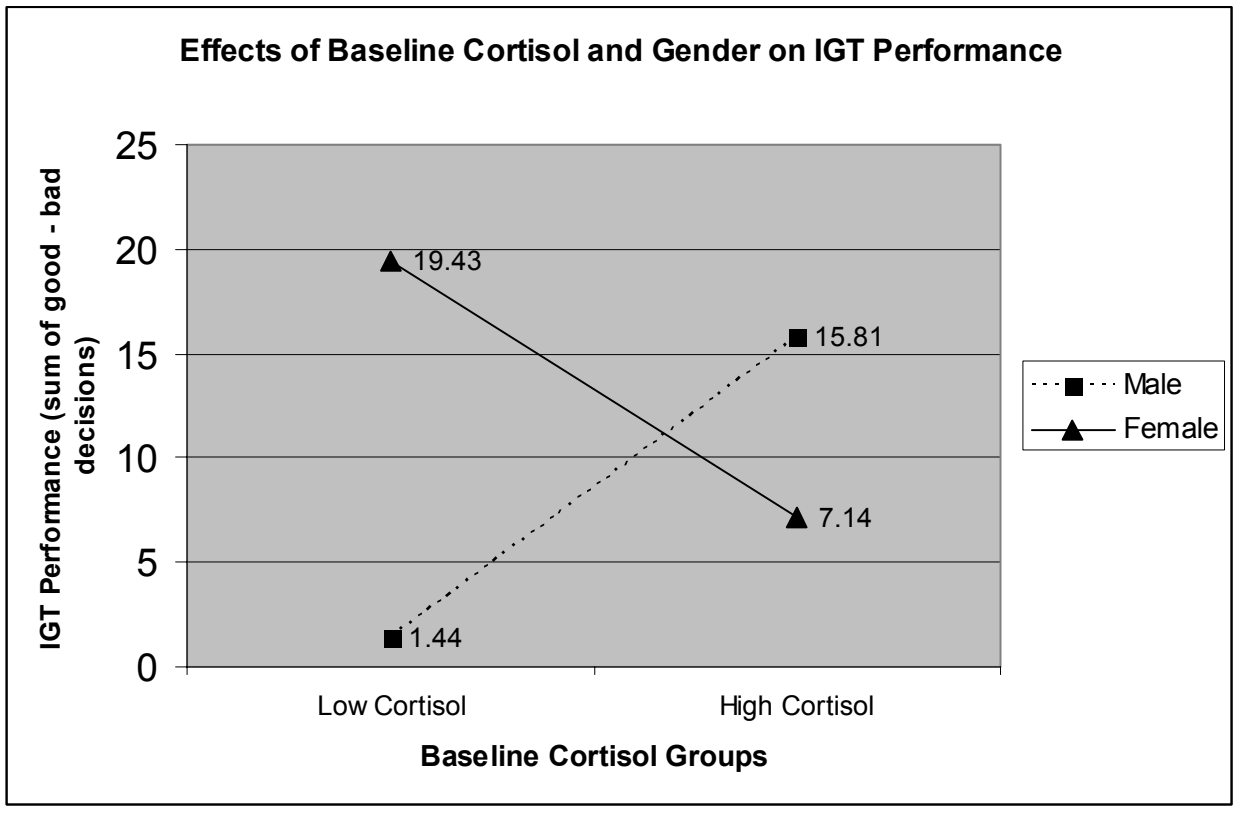
Figure 6. The Effects of Psychopathy and Gender on IGT Performance



Additionally, a significant interaction was obtained between gender and baseline cortisol levels in predicting overall performance on the gambling task, F (1, 110) = 3.87, p = .05, such that whereas men with low cortisol levels performed poorly on the task, the opposite was

observed for women. Women with low cortisol levels seemed to have optimal performance (see Figure 7 below). Follow-up t-test analyses revealed that the gender differences were only significant at low cortisol levels, such that women with low cortisol levels performed significantly better than men with low cortisol levels, t (53) = 2.07 (M_{males} = 1.44, $M_{females}$ = 19.43). The performance differences among men and women were not statistically different in the high cortisol group, t (54) = 1.10, p =.28, *ns* (M_{males} = 15.81 , $M_{females}$ = 7.14). Likewise, within gender, the performance of males did not significantly differ from one another at low versus high cortisol levels, t (50) = -1.57, p = .12, *ns,* and the same null finding for cortisol level was obtained within gender for females, t (57) = 1.64, p = .11, *ns.*

Figure 7. The Effects of Baseline Cortisol and Gender on IGT Performance



Discussion

This study aimed to extend the scope of findings from previous studies regarding etiological factors presumed to distinguish psychopathic from nonpsychopathic populations, by focusing on the combination of biopsychosocial constructs potentially underpinning the previously elucidated constructs of the behavioral inhibition and activation systems. The relationships between baseline and post-arousal hormone levels were examined among male and female college students screened and classified into psychopathic and nonpsychopathic analogue groups. Post-arousal samples were obtained following administration of a computerized behavioral task that has been reliably used initially to differentiate vesomedial brain lesioned patients and more recently psychopathic from nonpsychopathic populations. The Iowa gambling task (IGT) has been used to elicit BIS/BAS activity in response to mixed punishment and reward contingencies, demonstrating that psychopathic individuals respond to the task by utilizing high-risk, disadvantageous decisions, supporting the theory that psychopathic individuals exhibit low punishment and high reward sensitivity.

Consistent with previous findings on the IGT, a gambling task in which participants are asked to try to win as much money as they could, psychopathic analogues demonstrated both a hypersensitivity for reward, and a disregard for or disinhibition of responses that elicited punishment as exhibited by this group's higher monetary losses. Psychopathic analogues made less advantageous decisions than nonpsychopathic analogues, supporting previous evidence suggesting their attenuated punishment sensitivity, also described as passive avoidance deficits on a task involving punishment and reward cues. That is, as predicted by the *response modulation model* (Newman, 1987), the psychopathic analogues made more risky decisions guided by BAS or reward-motivated behavior and had difficulty inhibiting their responses

despite the presence of punishment cues. These findings also converge with Newman's operationalization of response perseveration errors made by this population, in that once they have developed a response set aimed at reward, psychopathic individuals seem to have an inability to appropriately pause to process and integrate peripheral cues related to punishment that may impede the successful attainment of their identified goal. Relatedly, the finding that psychopathic individuals display excessive BAS functioning as shown through their disadvantageous decisions on the gambling task is consistent with recent work suggesting the possibility of exaggerated BAS functioning in psychopathic individuals, especially while in pursuit of reward (e.g., Woodworth & Porter, 2002; Arnett et al., 1997). According to the response modulation deficit model, psychopaths are relatively deficient in processing and utilizing contextual cues that provide peripheral information to their dominant response set (i.e., aimed at maximizing reward) in order to regulate or alter their ongoing behaviors. Utilizing the Iowa gambling task as the main outcome measure of BIS/BAS motivated behavior, the present study's findings are consistent with previous behavioral findings attesting to deficient response modulation among psychopaths (e.g., Newman, 1998; Bernstein, Newman, Wallace, & Luh, 2000).

The computerized gambling task (IGT) was sufficiently sensitive to yield differential performance outcomes between participants classified as psychopathic and nonpsychopathic based upon two self-report measures of psychopathy used to screen participants in this study. Given the affective/anxiety deficits presumed to underlie Cleckley's conceptualization of primary psychopathy, a substantial research literature (e.g., Newman, Widom, & Nathan, 1985; Schmitt & Newman, 1999) has attested to the utility of using a well-established measure of anxiety/negative affectivity (WAS; Welsh, 1956) to parse the etiological heterogeneity between

high and low-anxious psychopathic individuals, especially since differential performance deficits have been obtained across these groups (Newman et al., 2005). Some studies have found that significant differences emerged between psychopathic individuals and controls only when low-anxious groups were compared to high-anxious groups, with a pattern in the literature of finding poorer passive-avoidance learning only in low-anxiety psychopathic individuals (e.g., Arnett et al., 1997; Newman et al., 1985). Since the WAS has been the most widely used instrument to assess the construct of anxiety among psychopathic populations (Hale et al., 2004; Newman & Kosson, 1986), the current investigation also used median-split scores on the WAS to differentiate psychopathic and nonpsychopathic participants into high and low-anxiety groups.

Whereas dividing psychopathic individuals into high and low-anxiety groups has lead to differential passive-avoidance learning deficits among low-anxiety psychopathic individuals in previous studies (i.e., Arnett, 1997), contrary to prediction, Welsh anxiety scores did not moderate the relationship between psychopathy and risky behaviors in this study. Schmitt, Brinkley, and Newman (1999) was the first published study to examine psychopathy and IGT performance, hypothesizing differential performance patterns on the IGT in their sample of high and low anxious incarcerated psychopathic and nonpsychopathic individuals. However, they failed to obtain their predicted hypotheses, and found that the IGT was only sufficiently sensitive in differentiating between high and low-anxious offenders more generally. In the current study, performance on the gambling task also varied as a function of Welsh anxiety level, in that individuals with lower WAS scores performed better than individuals with higher WAS scores, but this main effect only marginally approached significance. Good performance on the gambling task entails a propensity for more conservative, less risky card selections. Although it was expected that individuals who endorse higher levels of anxiety would be more likely to

make less risky decisions, the opposite finding was obtained. Anxiety did not improve, but impaired performance, which suggests that anxiety or perhaps increased arousal level impairs performance on this task. Additionally, given that no significant Psychopathy X Anxiety interaction emerged, this null finding suggests that anxiety (as measured by Welsh scores) did not significantly moderate the relationship between psychopathy and risky behaviors on the gambling task in this sample. As expected, endorsement of self-reported BIS activity varied as a function of anxiety level, such that individuals with high (WAS) anxiety scores endorsed significantly higher BIS scores as compared to those in the low anxiety group.

In addition to an examination of the role of psychopathy and psychopathy as a function of anxiety levels in predicting differential BIS/BAS driven behaviors, this study also sought to examine the link between psychopathy and response modulation deficits as a function of gender. No gender differences were predicted, as differences in response modulation or BIS/BAS motivated behaviors on the gambling task were expected to be theoretically driven by idiographic differences characterized by psychopathy and unrelated to gender, per se. As predicted, based on the obtained main effects, gender differences were unrelated to differential BIS/BAS motivated activity on the gambling task. These findings converge with previous robust findings for attenuated punishment learning observed in a "high psychopathic" participant group. In a sample of 8 male and 8 female participants, the gambling performance of individuals classified in the "high psychopathic" group was similar to that of orbitofrontal patients, in comparison to the intact punishment learning exhibited by individuals in the "low psychopathic" group (van Honk et al., 2002), and unrelated to gender. The present study's results also converge with those obtained in a mixed gender sample examining punishment sensitivity in psychopathic analogues (van Honk et al., 2003). As previously enumerated, regardless of biological gender

differences, differential BIS/BAS activity was presumed to be robustly related to psychopathic traits.

Despite this study's converging finding that gender alone was unrelated to overall performance on the gambling task, some divergent findings were obtained when the effects of gender were examined on the self-reported BIS and BAS indices. Contrary to prediction, gender differences emerged on the self-reported measures of BAS and BIS activity, respectively. Women in this study endorsed more BAS and more BIS activity items, as compared to men. Such findings suggest that the self-report BIS/BAS measures likely measure a different construct than that measured by the gambling task, which was the behavioral measure of BIS/BAS motivated behavior used in this study. Data obtained in this study provides further support for the latter statement, in that correlational analyses failed to obtain significant correlations between the behavioral and self-report BIS/BAS indices. Specifically, neither the BIS nor the BAS self-reported items significantly correlated with the behavioral BIS and BAS indices (as measured by advantageous and disadvantageous selections on the IGT), respectively. These findings provide evidence for the lack of convergent validity between the behavioral and self-report trait measures of the BIS and BAS constructs, suggesting that these are two very different outcome measures. Also contrary to prediction, when correlational analyses were examined between self-reported BAS activity as measured by Carver and White's scales (1994) and testosterone, baseline and post-task testosterone levels were negatively correlated with both self-reported BAS and BIS activity, again attesting to some potential problems in the construct validity of these scales. This was a counter-intuitive finding because based on theoretical grounds, if the self-reported BIS and BAS scales demonstrated reasonable discriminant validity, then testosterone would be expected to be positively related to BAS and negatively related to BIS items, but this was not the case.

Besides indicating potential construct validity problems, the increased endorsement of BIS and BAS items by women may be an artifact of gender-role socialization theories, which are outside of the scope of this study. In brief, due to the differential societal roles that women espouse as compared to men, their responses may represent a balance of both reward-motivated behaviors and an increased proclivity to be responsive to fear cues, inhibitions, and less risky behaviors. These data might suggest that gender may serve as a protective mechanism as measured by an increased proclivity toward making decisions based on attending to both rewarding and punishing cues. Given Trasler's (1978) account of the integral role of avoidance learning in the socialization of human behavior, the increased BIS responses may reflect that women are more attentive to punishment cues to guide their decision-making processes as compared to men in general. However, in light of other findings in this study, this protective factor may be overshadowed by the effects of psychopathy in that both male and female psychopathic analogues showed an increased proclivity toward more risky decisions and an attenuated ability to make use of punishment cues to make more advantageous decisions.

In addition to the relationships explored between gender and psychopathy in predicting risky behaviors, a number of predictions were made for the role of hormones, namely cortisol and testosterone, in potentially underlying the differential behaviors of psychopathic and nonpsychopathic participants in an approach-avoidance learning task. Based on the tenets of Gray's (1987) model for the role of the BIS and BAS in motivating behavior, this study sought to build upon Gray's model by examining the potential neuroendocrinological correlates implicated in the body's arousal states that guide subsequent behaviors. Although the literature is sparse and results have not always converged, some previous research has yielded an inverse relationship between basal cortisol levels and risky decisions on the Iowa gambling task (e.g.,

Van Honk et al., 2003). Contrary to predictions, baseline and post-task cortisol level alone, which are physiological indicators of HPA axis activity activated by stress, fear, and anxiety cues, failed to differentiate psychopathic from nonpsychopathic behaviors in this study. Whereas psychopathic analogue participants were predicted to have lower levels of cortisol at baseline and during peak arousal as compared to nonpsychopathic participants, cortisol level failed to differentiate between the psychopathic and nonpsychopathic analogue groups. Additionally, there was no significant correlation between psychopathy and the change in cortisol level from baseline to peak arousal following the task. Also, whereas it had been predicted that cortisol would be related to both behavioral manifestations of BIS activity as assessed by choices made on the gambling task and by self-reported BIS activity on Carver and White's (1994) scale, cortisol was unrelated to both of these indices of BIS activity.

One potential explanation for the null findings between cortisol level and BIS/BAS motivated behaviors may be that the gambling task did not elicit sufficient stress to alter cortisol levels. In support of this explanation, although it was predicted that the post-task cortisol level, which was operationalized as a measure of peak HPA activity during the gambling task, would increase from baseline levels, the results of the repeated measures ANOVA indicated that cortisol levels actually decreased across time. Additionally, participants had no strong financial incentive to perform in a particular way on the task because all participants regardless of their performance on the task, had been told that they would receive $5 as compensation for their participation in this study. These null results may also be due to the fact that the gambling task confounds punishment and reward contingencies, and subsequently both BIS and BAS motivated activity. Gray's model postulates that the activation of one system should theoretically inhibit the other system, but both systems are simultaneously involved on the IGT, potentially activating

both cortisol and testosterone reactivity. Both baseline and post-task cortisol levels were significantly positively related to testosterone levels among participants in this study, attesting to the potential confounding effects of these hormones. Future research would benefit by developing a way to tap into these systems independently, as the simultaneous reward and punishment cues inherent in the task likely activate both cortisol and testosterone making it difficult to examine the independent effects of these hormones.

A closer examination of the correlations between pre and post-task hormone levels separately across gender revealed differential hormone-behavior patterns across men and women. Among men in the sample, baseline testosterone levels were significantly positively related to both baseline and post-task cortisol levels, perhaps suggesting that male hormones were activated prior to the gambling task and that these hormonal or arousal levels were maintained throughout the duration of the task. Previous studies have found that in sporting competitions, men typically experience an increase in testosterone in anticipation of competition (Booth, Shelly, Mazur, Tharp, & Kittok, 1989). This rise has been attributed to making male competitors more willing to take risks (Daltzman & Zuckerman, 1980). Although the present study did not involve a sporting competition, participants were asked to try to acquire as much money as possible on a gambling task, which involved risky behaviors. Given biological differences in the secretions of hormones between men and women, women may have a different pattern of relationships among testosterone, cortisol, and risk-taking or competitive behaviors, as is illustrated in the current study.

Among women in the sample, baseline hormone levels were not correlated, but post-task cortisol and testosterone levels were significantly positively related. Minimal research is available examining testosterone and cortisol levels among female participants. In one study,

which examined anticipatory and post-game testosterone and cortisol levels among a sample of female rugby players, which likely differ greatly from the current study's sample (Bateup, Booth, Shirtcliff, & Granger, 2002), the following results and explanations were described and compared in light of previous hormone findings among male competitors. These authors review potential reasons for the differences between hormone-behavior links in women and men noting that women produce 5-7 times less testosterone as compared to men. Whereas the primary source of testosterone for men is through the activation of the hypothalamic-pituitary-gonadal (HPG) axis, rises in testosterone levels in women occur as a result of HPA activation and adrenal gland secretions. Higher pre-game rises in both testosterone and cortisol in women as compared to men were attributed to the fact that the primary source of both hormones is the same in women (the adrenal glands). Although baseline cortisol and testosterone levels were not found to be correlated among women in the current study, this divergent finding from that obtained in Bateup et al. (2002) is likely due to the vastly different design and demand characteristics employed in both studies. In their study (Bateup et al., 2002) women's pre-game hormone levels were assessed as a measure of anticipatory arousal prior to a competitive, highly aggressive sport that rewards physical domination and aggressive behaviors. In contrast, the current study's initial hormone measure was a measure of baseline levels that likely entailed very minimal anticipatory arousal levels. Despite the divergent design characteristics, similar to the rugby study's findings, post-task cortisol and hormone levels among women in this study were also positively correlated as was found for women during the rugby game. Hence, although hormone levels were not significantly related to differential performance on the gambling task, it is possible that the task activated an adrenal gland response from women as an explanation for the correlated post-task cortisol and testosterone levels.

Whereas previous research has noted a significant relationship between cortisol and anxiety levels (e.g., Dabbs et al., 1991), also contrary to prediction, cortisol levels were unrelated to self-report anxiety indices in this study. Both baseline and post-task cortisol levels were unrelated to state-trait anxiety measures and to Welsh anxiety scores in this study. One explanation for the null cortisol findings may be that the population of college students used in this study may not exhibit sufficient variability in the activation of their HPA axis to capture differences in cortisol level. That is, most studies reporting a relationship between cortisol and anxiety or other psychological disturbances, assess these relationships within clinically diagnosed disorders, such that increased HPA activity has been found in depression, obsessive-compulsive disorder, and childhood abuse (Tsigos & Chrousos, 2002). Although in one study in which students were also used, cortisol level was significantly inversely related to choice of risky decks (Van Honk et al., 2002), studies in which cortisol is correlated with anxiety among healthy student populations are more limited. Dabbs and Hooper (1990) found significant relationships between cortisol and anxiety as measured by the NEO-PI in a student population. However, the difference between their sample and the present study's sample is that they conducted their analyses with participants in the top and bottom 10% of the cortisol distribution and their sample only consisted of male undergraduates. These procedures were not employed in the current analyses because groups based on upper and lower deciles would be too small and concordantly would lack sufficient power to detect statistical differences. McBurnett et. al (2000) found higher cortisol levels among boys with comorbid high levels of anxiety and aggression, and lower cortisol levels in boys with high levels of aggression and lower anxiety levels, but sample characteristics may likely be attributed to these findings as their study was conducted with inpatient sex-offending boys. In the present study, the two groups delineated as psychopathic

and nonpsychopathic analogues may not differ enough from each other given the shared variance in their college student identity and the associated college lifestyle stressors, to capture a wide enough range on a neuroendocrine variable such as cortisol. That is, the college students who comprised the participant base in this study may share more in common given their shared lifestyle characteristics in regards to stress levels and subsequent HPA activity that may be accounting for the null cortisol findings.

Another potential explanation for some of the null hormone findings is related to the mechanisms governed by the endocrine system. Whereas the nervous system is primarily responsible for fast-acting, short-duration responses to changes in the body, the endocrine system controls the slow-acting responses of long duration (Taylor, 1999), for example leading to long-term effects in individuals with chronically high stress levels. This may explain why it may be difficult to reliably activate and detect cortisol changes, given the nature of the system. Given IRB standards and ethical codes of contemporary research, it may be difficult to engage participants in a task that leads to significant changes in endocrine functioning. However, an interesting trend did emerge for a statistically marginal finding for an interaction between cortisol level and anxiety levels in the repeated measures analysis. Individuals classified in the high anxiety group exhibited a larger decrease in cortisol levels than those in the low anxiety group. Such a finding may be attributed to a common finding in anxiety treatment research for anxiety levels to "peak and pass," such that those who endorsed higher anxiety levels presumably had higher baseline cortisol levels that decreased across the experimental session.

Although cortisol was generally unrelated to anxiety, given the presumed anxiety deficits presumed to underlie psychopathy or at least a subset of psychopathic offenders in the literature, the relationship between anxiety and psychopathy was further assessed. On first glance, the

psychopath group as a whole seemed to endorse significantly higher levels of self-reported anxiety as compared to the nonpsychopath group. While such a finding seems inconsistent with the theoretical construct of psychopathy and its characteristic emotional/affective deficits as described in classic earlier research findings (e.g., Lykken, 1957), it is nonetheless not too surprising. More recent empirical findings using psychometrically state-of-the art instruments have found the previously elucidated inverse relationship between psychopathy and anxiety to be equivocal at best (Hale et al., 2004). Several studies have failed to obtain the theoretically predicted relationships for an inverse relationship between PCL-R psychopathy and various measures of fear and anxiety (e.g., Patrick, 1994; Schmitt & Newman, 1999). Attempts to clarify the relationship between psychopathy and anxiety have been more informative when the partial correlations are examined to assess the unique relationship between anxiety and Factor 1 and Factor 2 psychopathy separately, in order to determine the correlation between anxiety and the personality/affective components versus the antisocial/lifestyle components of psychopathy. For example, Patrick (1994) reported that measures of fear and emotional distress were negatively related to PCL-R Factor 1 scores after controlling for the effects of PCL-R Factor 2, and positively related to PCL-R Factor 2 scores after controlling for Factor 1. As recommended by recent studies (Patrick, 1994; Hale et al., 2004), the unique relationship between anxiety measures and the factor scores underlying the psychopathy measures used in this study were further examined. These analyses served to explain the initially surprising finding of greater levels of anxiety endorsed by the psychopathic analogue group as compared to the controls. Convergent with previous research findings, Factor 1 psychopathy as assessed by Lilienfeld's (1996) Psychopathic Personality Inventory – Short version was significantly negatively correlated with anxiety as measured by the Welsh Anxiety Scale (WAS) and with trait and state

anxiety as assessed by the STAI. Additionally, as expected Factor 2 psychopathy as measured by the PPI was significantly positively correlated with anxiety as measured by the WAS, and with trait and state anxiety as assessed by the STAI.

The findings obtained for the inverse relationship between self-reported anxiety and the affective/personality (Factor 1) dimension of psychopathy as well as the direct relationship between anxiety and the antisocial/lifestyle (Factor 2) dimension of psychopathy as measured by the PPI, an instrument used to assess sub-clinical psychopathy in a non-offending population are important in a number of ways. First of all, these findings replicate previously obtained findings and lend support to our understanding of psychopathy and the types of deficits that may differentially distinguish individuals characterized by higher or lower Factor 1/Factor 2 characteristics. Differentiating psychopathic individuals by their factor scores has greatly informed recent promising treatment outcome research (APLS conference presentation, Skeem et al, March 2005), aimed at reducing recidivism rates among a population of psychopathic offenders, a population that has long been considered not amenable to treatment. Based on their preliminary treatment outcome findings, they conclude that differentiating individuals based on factor scores greatly increases the specificity needed to develop more appropriate, tailored approaches to use with psychopathic offenders. Previous studies have suggested that if variants of psychopathy could be identified reliably and supported empirically, the increase in the specificity of the construct of psychopathy would likely improve our ability to understand, manage, and treat individuals who have generally been regarded as both dangerous and untreatable (Skeem, Poythress, Edens, Lilienfeld, and Cale, 2003). Given the differential relationships obtained for anxiety levels among high Factor 1 versus high Factor 2 groups, for example, such information could be utilized in informing different treatment approaches. As

previously discussed, human socialization processes aimed at the development of prosocial behaviors are a result of learning acquired through fear conditioning and reward paradigms (e.g., Trasler, 1978; Mowrer, 1960). So, the results of this study may support the merit of utilizing treatment approaches aimed at helping individuals with high Factor 2 scores positively related to anxiety to recognize the relationship between anxiety levels and risky behaviors. For example, psychoeducational interventions can be focused on the usefulness of recognizing and utilizing individuals' innate physiological fear and anxiety responses to guide and motivate more effective future decisions and behaviors.

In addition to guiding the development of more specifically tailored treatment approaches based on the relationship between anxiety and the dimensions of psychopathy, this study's findings also lend credence to the importance of studying the construct of psychopathy in a non-offending population. Additionally, it supports the utility of such instruments as the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996), the Primary and Secondary Psychopathy Scales (Levenson et al., 1995), and the Self-Report Psychopathy Scale III (SRP-III; Hare, Paulhus, & Hemphill, 2002) in capturing an analogous dimensional set of personality and behavioral constructs as that operationalized by the gold-standard (PCL-R; 1991) to assess the construct of psychopathy in non-offending populations. This study's findings obtained with a non-adjudicated group of psychopathic and nonpsychopathic analogues as classified by these measures converged with findings obtained with psychopathic and nonpsychopathic offenders in jail/prison populations, attesting to the construct validity of these measures. These findings also converge with seminal conceptualizations of psychopathy (Cleckley, 1941), which did not consider criminal behavior to be an essential feature of psychopathy.

Cleckley viewed the interpersonal and affective dimension as "core" features of psychopathy, elucidating that these may or may not be associated with actual criminal, antisocial types of behaviors (Wilson, Frick, and Clements, 1999). Cleckley made the distinct point that psychopathy does not necessarily involve criminal activity as illustrated through his numerous case presentations of individuals cutting across social class, gender, ethnicity, and career path (Brinkley, Schmitt, Smith, & Newman, 2001). Despite Cleckley's enumeration of psychopathy as a dimensional personality construct that can be assessed in both offending and non-offending populations, the bulk of the literature in psychopathy has been conducted using the PCL-R in institutional, correctional settings. This inadvertently constrains our knowledge base on psychopathy by only studying the unsuccessful criminal psychopaths, who have been apprehended for their antisocial behaviors, ignoring the subset of psychopaths who do not commit or at least have not been apprehended for their actions (Edens, Poythress, & Watkins, 2001). Numerous studies have emphasized the importance of studying psychopathy across noninstitutionalized samples to gain a more comprehensive understanding of the construct of psychopathy as originally conceptualized (e.g., Edens et al., 2001; Brinkley et al., 2001; Benning et al., 2003; Ishikawa, Raine, Lencz, Bihrle, & LaCasse, 2001). Given the inconsistent findings in the recent literature, future studies should continue to examine the relationship between anxiety and psychopathy, as affective deficits have been described as one of the core features of primary psychopathy.

Also as an extension of Gray's model, this study sought to examine the potential role of testosterone and its relationship with BAS-motivated behaviors and the associated deficits in learning that have been reported among psychopathic individuals in previous literature. Although testosterone was predicted to be positively related to self-reported BAS activity and

BAS-motivated behavior based on disadvantageous selections on the gambling task, baseline and post-task testosterone levels were not significantly correlated with differential performance on the gambling task. Change in testosterone level from baseline to post-task was also unrelated to overall performance on the gambling task. No support was obtained for the prediction that psychopathic individuals would exhibit higher testosterone levels as compared to nonpsychopathic individuals. To further examine any potential relationship between testosterone and task performance, a Repeated Measures ANOVA with psychopathic group (psychopath, nonpsychopath) and anxiety group (low, high) as between-subject variables and baseline testosterone and post-task testosterone levels (peak arousal) as the repeated measure (within-subject variables) was examined. No significant within-subjects effects for a change in testosterone from baseline to post-task were obtained. However, results suggested that level of anxiety has an influence on testosterone level, such that participants with "low" anxiety had significantly higher testosterone levels as compared to participants with "high" anxiety levels. Based on the studies reviewed, to this author's knowledge, previous studies have not explored or reported this inverse relationship between low anxiety and high testosterone levels. However, such findings seem logical given the demonstrated link between testosterone and aggressive, psychopathic behaviors, and the mixed findings for an inverse relationship between psychopathy and anxiety. That is, previous meta-analytic findings have elucidated a small but significant relationship between testosterone and aggression, and to a lesser degree antisocial behaviors. Also, although research results are mixed, a core feature of theoretical conceptualizations of psychopathy is reduced anxiety. Hence, the present study's finding of an inverse relationship between testosterone and anxiety follows from the past literature base. Future research should

continue to explore the relationships between testosterone and anxiety, especially as related to risky, deviant types of behaviors.

Research assessing the relationship between testosterone and psychopathy, in particular, has been limited. As previously described, the studies have focused on a wide range of populations including offender populations, individuals with antisocial personality disorders, and juvenile delinquents, and less so among non-institutionalized, successful psychopathic individuals. As was speculated for the null cortisol findings, it may be more difficult to detect distinct differences in testosterone, a neuroendocrinological marker among a college population given the shared lifestyle variance among this group. As reviewed, meta-analytic results indicate a small effect size between testosterone and aggression, more generally in studies with men (Mazur & Booth, 1998).

Additionally, this study also failed to find support for the hypothesis that psychopathic individuals would exhibit higher testosterone levels as compared to nonpsychopathic individuals, even when controlling for gender. However, given the robust findings for gender differences in the literature, it was predicted that females would have lower levels of testosterone and of self-reported aggression as compared to men, but the relationships between psychopathy, hormones and behaviors should be salient despite these gender differences. Based on human biological or more specifically neuroendocrinological processes, testosterone relationships were expected to vary by gender. Testosterone levels were expected to be higher among male participants, regardless of psychopathy status. As predicted, females exhibited lower levels of both baseline testosterone and post-task testosterone as compared to male participants, although these levels were not predictive of subsequent task performance.

In addition to the lack of obtained main effects for any of the baseline and post-task hormone (testosterone and cortisol) levels in predicting advantageous or disadvantageous performance on the gambling task, cortisol levels also failed to significantly moderate the relationship between baseline testosterone level and risky behaviors within the sample. Given the role cortisol plays in activating the HPA in response to stress or fear-inducing cues, it had been predicted that perhaps individuals classified by high scores on measures of psychopathy engage in more risky behaviors because of a deficiency in their avoidance-learning mechanisms, potentially mitigated by decreased cortisol, or similarly decreased HPA activity in response to high-risk, stressful situations. Given previous findings for a link between testosterone and aggressive, risky types of behaviors, it was predicted that increased cortisol levels may moderate the relationship between testosterone and risky behaviors, such that high cortisol levels may weaken the relationship, whereas low cortisol levels may strengthen the relationship between testosterone and risky behaviors on the gambling task. Regression analyses failed to obtain a significant interaction between only cortisol and testosterone levels in predicting risky, disadvantageous decisions on the gambling task. Based on this author's review of the literature, few studies have examined the potential moderating role of cortisol between testosterone and risky or aggressive, competitive types of behaviors, and those studies have yielded mixed findings. In one study, Salvador, Suay, Martinez-Sanchis, Simon, and Brain (1999) examined the relationships between testosterone, cortisol, and behavior exhibited during judo contests. Among their sample of 28 male judo fighters, their findings paralleled those of the present study's findings, in that they also failed to obtain support for a moderator role of cortisol in inhibiting violent behaviors.

In contrast, Dabbs et al. (1991) assessed salivary testosterone and cortisol levels among late adolescent (ages 17-18) male offenders in a state prison. Their study is one of the very few

studies found that have examined the interaction between testosterone and cortisol, predicting a moderator role of cortisol between testosterone and violence level of crime, similar to the current study's predicted relationships. In their study, testosterone was positively correlated to violence of crime. No main effects emerged for cortisol, but cortisol was found to moderate the relationship between testosterone and violence of crime. In their sample of 113 male offenders, their effect size was significant but very small, in that the interaction between cortisol and testosterone in predicting violence accounted for only 4% of the variance. Their significant cortisol findings are likely in part due to the situational stress-laden environment of a prison, especially that the inmates in their study were assessed during the first few weeks of their incarceration when stress levels are probably at their peak. Given that hormone levels and namely cortisol is sensitive to stress, the stress-laden prison environment may be contributing to their obtained moderator relationships. In contrast, with the current study's combination of the behavioral task and the nature of a college sample, it is likely that stress levels were not sufficiently high enough to capture cortisol differences as had been predicted.

To further assess the potential moderating roles of hormones, a few post-hoc exploratory analyses yielded unexpected moderator relationships among hormones, gender, and gambling task performance. Median-split scores across the four hormone levels assessed in the lab were used to classify participants into high and low testosterone and cortisol groups. Exploratory analyses were conducted examining the main effects and interactions among psychopathy, gender, and testosterone and cortisol separately on overall performance on the gambling task. No significant main effects were obtained for level of testosterone or for gender on IGT performance. However, a significant interaction emerged between post-task testosterone level and gender in predicting overall performance on the gambling task. Men in the high testosterone

group exhibited a more advantageous pattern of card selections on the gambling task as compared to men in the low testosterone group. The opposite finding was obtained for female participants across post-task testosterone, such that females with low testosterone levels performed significantly better than those with high testosterone levels. Although the sample size was unequal across these four cells, with a smaller number of men in the low testosterone cell and women in the high testosterone cell respectively, these data, although preliminary, provide opportunities to examine gender differences in biologically "deviant" groups on a measure of risky, psychopathic behaviors.

As previously mentioned, men are expected to have significantly higher levels of testosterone than women, and accordingly women are expected to have 5-7 times less testosterone than men. Although the majority of the sample did exhibit this biologically expected pattern, a subset of men and of women exhibited atypical hormone patterns. These two biologically "deviant" groups, men in the "low testosterone" group and women in the "high testosterone" group based on median-split scores across the sample, drove this interaction effect for a significant relationship between risky behaviors and gender among these deviant groups. That is, whereas men and women in the typical hormone groups (men in the high testosterone group and women in the low testosterone group) exhibited good performance on the gambling task, women in the high testosterone and men in the low testosterone group exhibited poor performance, marked by overall monetary losses on the tasks. Such a finding is particularly intriguing in its potential implications of a biological marker for deviant or risky behaviors among both men and women with characteristically atypical testosterone levels. These findings also suggest that higher testosterone levels seem to serve as a protective factor, at least for men in making better, more conservative decisions on the gambling task. Interestingly, these findings were not obtained for

high and low baseline levels of testosterone, which suggests that the task might be activating the release of testosterone. For men who were classified as having the higher levels of testosterone in response to the task, this hormonal change seemed to benefit their performance. As previously elucidated with athletes in a judo competition, those athletes that made the most competitive attacks and offensive behaviors subsequently exhibited higher testosterone levels (Salvador et al., 1999). Perhaps, there is an optimum level of adrenal gland activity in releasing testosterone in men that improves decision-making skills and behaviors, whereas the opposite is true for women given their biological composition. These preliminary interaction data suggesting a potential hormonal marker for deviant behaviors merit further pursuit in future research endeavors.

Likewise, the main effects and interactions among psychopathy, gender and cortisol levels on overall gambling task performance were also assessed. A significant main effect was obtained for psychopathy group, such that nonpsychopathic participants made more advantageous decisions as previously elucidated, whereas psychopathic participants made more risky, disadvantageous decisions on the gambling task. No main effects were obtained for gender or for cortisol level (median-split baseline and post-task cortisol groups) in predicting overall performance. However, two significant interactions emerged in these analyses. First, an interaction was obtained between psychopathic group and gender in predicting overall performance on the gambling task, such that females in the nonpsychopathic group outperformed the females in the psychopathic group in overall gains on the gambling task. However, among male participants, psychopathic status did not significantly impact performance as it did for female participants. Additionally, a significant interaction emerged between gender and baseline cortisol levels in predicting overall performance on the gambling task. Performance on the

gambling task significantly varied across gender based on the level of baseline cortisol. Whereas low basal cortisol level was associated with improved female performance, it was associated with significantly inferior male performance. The performance of females in the higher basal cortisol group was not statistically significant from that of men in the high cortisol group. These findings have yielded interesting differential relationships between gender and hormones in their interactive or moderating role on a task that activates BIS and BAS motivated behaviors.

The hormone relationships obtained in this study seem to qualify previous findings (e.g. Van Honk et al., 2003) of a disadvantageous pattern of decisions on the gambling task among participants with the lowest cortisol levels, in that the present study yielded cortisol-gender interaction relationships on performance. Cortisol seemed to have differential associations in men versus women. Whereas women who made better decisions had lower baseline cortisol levels, men's performance was impaired under conditions of low cortisol levels. Based on previous literature, cortisol is viewed as a marker of HPA activity, generally activated in response to stressors and generally construed as a marker of one's fear, anxiety, or stress reactivity index. Whereas the results of this study suggest that women's performance is optimized when their resting state neuroendocrinological responses reflect a more relaxed state, men's performance on a task involving risky decisions seems to benefit from higher baseline levels of HPA activation. Since cortisol is often associated with higher anxiety, these findings may imply that men make better decisions with increased anxiety levels, whereas anxiety as measured by cortisol activation impairs female performance and decision-making abilities.

Across the various relationships obtained for hormones in moderating the relationships between gender and approach-avoidance behaviors, a unifying construct seems to unfold. The emergent gender differences may be indicative of overall arousal level differences associated

with differential task performance in males versus females. That is, males made better decisions marked by increased monetary gains at higher levels of arousal as reflected by higher testosterone and cortisol levels, whereas females made better decisions with lower levels of arousal overall as reflected in their improved performance at lower levels of cortisol and testosterone. Perhaps higher levels of arousal, at least within the domain of hormonal activity measured in this study, have a more adaptive effect on men's behaviors. In contrast, women's behavioral choices seem to benefit from lower levels of arousal as measured by lower adrenal gland and HPA hormonal activity levels. Whereas the findings for testosterone are more intuitive given the constitutional differences in the production of testosterone in men versus in women, the findings for cortisol are particularly compelling because normal, healthy men and women are not expected to differ significantly in their levels of cortisol. The current study's findings concur with previous hormone findings, in that men and women did not significantly differ in level of cortisol; however, level of cortisol, potentially a marker of arousal differences, moderated differential performance across gender. Future research should continue to explore these relationships, given the potential implications of these findings. Identifying hormonal markers that lead to differential behaviors across gender could influence behavioral, pharmacological, and developmental practices in altering men's and women's proclivity for risky and non-risky behaviors.

Given the support obtained for Gray's model of the BIS/BAS in differentiating psychopathic and nonpsychopathic behaviors within a noninstitutionalized, nonoffender population, the findings of this study begin to address some of the gaps in the literature regarding the dearth of research on non-offender based populations. As Cleckley (1976) initially conceptualized, psychopathic individuals, that is individuals who possess the core interpersonal, affective, and

impulsive behaviors of psychopathy, but who function adaptively and avoid legal encounters, do exist in our community. His case histories featured examples of successful noncriminal psychopathic individuals, including doctors, research scientists and other esteemed professionals (Benning et al., 2003). However, to date, minimal research has attempted to identify and study nonincarcerated individuals who exhibit core features of psychopathy. Hence, this study seems to be taking a step in the right direction.

In their paper, Brinkley, Newman, Widiger, and Lynam (2004) urge psychologists to focus research efforts on advancing knowledge regarding the specific manifestations and etiologies of psychopathy, in order to facilitate the development of more effective interventions. The current study has sought to do just that—to further extend previous research on etiological explanations of psychopathy by examining factors such as behavioral activation and inhibition, hormones, and anxiety across gender in further elaborating upon the presumed response modulation deficits that have been identified as serving an etiological role in distinguishing psychopathic from nonpsychopathic behaviors. The investigator of this study concurs with Brinkley et al. (2004) regarding the need for increased specificity in the heterogeneous construct of psychopathy via the identification of distinct etiological variants of psychopathy, which would likely be integral to the development of more effective treatment approaches.

Based on the overall findings from this study, a number of conclusions, limitations, and further questions to explore in future research can be enumerated. First, this study utilized an analogue psychopathic and non-psychopathic sample identified through two self-report screening questionnaires. A number of previous studies have outlined the potential dangers of using self-report measures to identify groups, but especially psychopathic groups, based on idiosyncratic personality characteristics such as their proclivity toward being deceptive, manipulative, etc.

Despite these potential validity concerns in distinguishing two groups into psychopathic and nonpsychopathic groups based on self-report measures, the findings from this study and from previous research have corroborated the finding that analogue psychopath groups respond in analogous ways to psychopathic groups identified within correctional settings, such that these individuals demonstrate similar psychophysiological deficits as found in offender populations. Additionally, as this study and previous studies have illustrated (e.g., Van Honk et al., 2002, 2003, 2004), psychopathic analogues also make equally high risk decisions on behavioral tasks as made by adjudicated psychopathic offenders. With these considerations in mind, what do the current findings really mean? Because analogue psychopaths evidence similar patterns of psychophysiological responding to anticipatory types of stressors and active and passive avoidance tasks, and display similar behavioral choices on laboratory tasks assessing sensation-seeking and proclivity toward reward as opposed to punishment learning based decisions, does this mean that analogue psychopaths are likely or more likely to commit the types of heinous crimes found among psychopathic offenders? These are the types of questions that are receiving more attention in the recent literature, namely distinguishing between unsuccessful and successful psychopathic individuals, highlighting similarities and differences between these groups.

As Cleckley (1976) and others (Ishikawa et al., 2004) have noted, the study of both successful psychopathic individuals in the community as well as unsuccessful psychopathic individuals who are adjudicated, is integral to gaining a fuller understanding of the continuous construct of psychopathy. Because of the limited data available, it is unclear at this time whether individuals classified as analogue psychopaths within this college population are necessarily at greater risk for engaging in illegal crimes. To this author's knowledge, no longitudinal data

exists following the trajectory from an analogue psychopath to subsequent offending behaviors. It is possible that analogue psychopaths are more like individuals classified as "successful psychopaths" in previous studies (i.e., Ishikawa et al., 2001). In Ishikawa and colleagues' study, successful psychopaths in the community (as determined by the individual's lack of conviction for a crime) demonstrated greater autonomic reactivity and greater executive functioning than unsuccessful psychopaths and controls. It was concluded that perhaps successful psychopaths are better able to avoid conviction because of their superior capability for assessing cues in risky situations and making more appropriate decisions based on processing punishment-laden cues.

Given the behavioral findings obtained in the present study for analogue psychopaths to exhibit the response modulation deficits characteristic of psychopathic offenders, it would be fascinating to compare the performance of analogues and "true psychopaths" in future studies as Ishikawa et al.'s (2001) study did in examining neuropsychological functioning, namely executive function and memory, among these groups. However, the findings from the present study also highlight some potential limitations in the use of hormones as a biological marker for BAS and BIS-motivated behaviors among an analogue sample. Although the expected behavioral differences emerged, most of the predicted hormone relationships that were expected to differentiate psychopathic and nonpsychopathic analogues failed to emerge. Furthermore, although some interesting moderator relationships emerged between gender, hormones and risky decisions on the behavioral task, these differences generally emerged among a smaller subset of the sample that were examined as atypical or biological deviants, females with high testosterone and males with low testosterone levels. Given these circumscribed findings among a subset of the sample and given the expense associated with conducting hormone assays, it is recommended that future studies aimed at assessing hormone differences focus on a more pathological or

deviant group than that available through an analogue group. The biological or endocrinological markers used in this study (testosterone and cortisol) as markers of HPA activity are likely not sensitive enough to capture differences in analogue samples as this study sought to do. Such differences are more likely to emerge in groups characterized by much higher levels of aggressiveness or anxiety, or among particular athletes who are in highly competitive or aggressive types of sporting competitions, or potentially among an adjudicated sample of deviant offenders as Dabbs et al. (1991) found that cortisol served as a moderator in the relationship between testosterone and the violence level of offenders' crimes.

This study aimed to identify the role of hormonal substrates in relation to Gray's (1987) model of the behavioral inhibition (BIS) and activation (BAS) systems—constructs proposed to serve a role in the etiology and/or maintenance of psychopathy. In summary, although this study did not yield all of the predicted relationships, especially in regards to hormone-behavior relationships, a number of the obtained relationships not only corroborated but also extended the scope of earlier research findings. As a group, psychopathic analogues exhibited the *response modulation deficits* espoused by Newman et al. (1987), in that the performance of psychopathic analogues on the gambling task revealed a tendency to perseverate on reward, while failing to attend to punishment cues to effectively modulate behaviors. The findings from the current study attest to the robust pattern of response modulation deficits differentiating psychopaths from nonpsychopaths, given that these relationships were obtained within a non-offending college sample screened for psychopathic-like traits. Given the parallels in the behavior choices between patients with ventromedial frontal lobe deficiencies and psychopathic individuals on the Iowa Gambling task, future research would benefit from pursuing psychophysiological, skin-conductance response (SCR) measures with psychopathic and nonpsychopathic controls while

participants are administered the IGT. Previous research (Bechara, 2000) has documented that anticipatory skin conductance responses exhibited by normal controls before selecting cards from disadvantageous decks are virtually absent in patients with ventromedial lesions. The absence of anticipatory SCR's among VM patients (and potentially psychopathic individuals) may be an indication that their ability to experience expected somatic, emotional states that are presumed to guide behavior, is greatly compromised. It would be interesting to explore whether these psychophysiological deficiencies in response to the IGT are also evident among psychopathic individuals who make poor, risky, comparable decisions to those made by VM patients, and furthermore to assess if these differences are robust across non-offender populations.

This study also sought to examine the moderating role of anxiety on the performance of psychopathic and nonpsychopathic analogues. Anxiety did not significantly moderate performance across groups, but a marginal relationship was obtained for anxiety. Good performance on the IGT entails more conservative, less risky card selections. One would expect that individuals who endorse higher anxiety would make less risky decisions, but the opposite finding was obtained. This finding suggests that perhaps anxiety impaired performance on the task, leading high anxious participants to make more disadvantageous decisions. Furthermore, the findings obtained for an inverse relationship between primary psychopathy (Factor 1) and anxiety, and a direct relationship between secondary psychopathy (Factor 2) and anxiety, corroborates the importance of differentiating between these two dimensions of anxiety. This level of specificity could more aptly inform the development of specialized treatment interventions for these two different clusters of psychopathic individuals.

Finally, this study made a number of unique contributions in regards to the role of hormones in serving as potential neuroendocrinological markers for activating differential behaviors across gender. Exploratory analyses provided evidence for an unexpected link between hormone levels and gender in predicting subsequent reward-seeking, risky behaviors. Whereas testosterone was not specifically related to the risky decisions attributed to an imbalance in BIS-BAS mediated responses among psychopathic participants, and whereas cortisol was not directly related to BIS-motivated responses among nonpsychopathic participants per se, an examination of gender elucidated the conditions by which hormones are associated with a differential proclivity toward risky and non-risky behaviors. In accordance with the tenets of Damasio's (1996) *somatic marker hypothesis,* differential arousal states across gender, potentially mitigated by testosterone and cortisol levels, were associated with differential decision-making on the gambling task. Specifically, advantageous decisions were associated with higher testosterone and cortisol levels among males, and with lower cortisol and testosterone levels among females.

Given the potential contributions of this line of research, hormones may be useful to explore in differentiating psychopathic from nonpsychopathic participants' responses to differential BIS/BAS driven behaviors among a more clinically deviant population. Such a link could inform treatment approaches in working with psychopathic offenders—a group that has traditionally been resistant to treatment. Namely, this finding could facilitate research on altering hormone levels with psychopharmacological interventions. The differential hormone findings obtained in this study across gender could inform differential treatment implications for male versus female deviant offenders. Future research would also benefit by developing a way to tap into these BIS/BAS systems independently, as the simultaneous reward and punishment cues inherent in the Iowa Gambling task likely activate both cortisol and testosterone making it

difficult to examine the independent effects of these hormones. As demonstrated in this study, by examining moderator relationships between psychopathy, hormones, gender and measures of BIS/BAS motivated behaviors, a number of interesting findings that enrich the previous literature emerged regarding the conditions under which hormones influence behavior. Further research aimed at replication and extension of these findings would likely lead to an increasingly specific and comprehensive biopsychosocial model for understanding the mechanisms underlying psychopathic behaviors. Subsequently, an understanding of the etiological and/or maintenance factors underlying psychopathy would likely inform the development of more tailored treatment interventions to reduce recidivism in a traditionally treatment-resistant population.

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Appendix A
Saliva/Health Screening Phone Eligibility Questions

1. **Are you currently under a physician's care for any illness?**
    a. **Exclude if any of the following:** any form of cancer including testicular cancer, or any endocrinological disease
    b. Assess for Asthma and/or known allergies that require meds. If Asthma (see later note)
2. **How has your health been in the past few days? Have you had a fever of OVER 102 in the last few days?**
    a. **Exclude:** If currently or frequently runs fever of 102+. Ask them to reschedule if get a fever of 102+ within 48 hours of their scheduled appointment.
3. **Are you currently taking any over-the-counter or prescribed medications, or any vitamin supplements, or any other pills that may contain steroid hormones?**
    a. **Exclude: If intaking any known steroid hormones (Exs:** frequent use of inhaler, Flonase (for allergies), any hydrocortisone creams, DHEA supplements, andristerone)
    b. **If taking** prescribed medication, vitamin supplements or any other drug, **ask them for the exact name/dosage, reason for it?**
        i. **If unclear regarding eligibility, then follow the UNSURE rules** (i.e, say you will have to consult with supervisor regarding eligibility, find out best times for scheduling them, and tell them you will notify them either way via email/phone call.)

   c. **If use asthma inhaler,** must ask: How frequently do you need to use your inhaler?
      i. **If rare** (i.e., once a week, a couple of times a month, etc.), then **OKAY**
      ii. **However, if person needs to use inhaler on a daily basis,** probably Exclude.
      iii. **Basically, as long as person doesn't need inhaler for 1 day before the study, then that is fine and they can be included.**

4. **Are you a smoker?**
   a. IF Yes, assess how much? Basically if the person can avoid having a cigarette for 2 hours prior to their scheduled appt time, then we can include them.
   b. **Exclude if:** person smokes approx 1 pack or more per day, such that it is unlikely that they could go for 2 hours without a cigarette.
5. **If scheduling,** make sure to ask participants to schedule their appointment on a day in which their stress level is no greater than usual. Tell them that we want them to come in on what would be considered a "normal/typical day" and NOT on a day in which they have several exams, a presentation, following a family/relationship crisis, or any other unusual experiences. Should any of these come up on the day of the study, encourage them to let you know and to reschedule their appointment.

If Eligible & Willing to schedule, say: "Great, we're excited that you'll be joining our study. We just have a couple of more things that we'll ask of you for the day of your appointment, in order for us to have useful saliva samples. On the day of the study, please try to drink a cup of water within the hour before your appointment if possible. If possible, we're hoping that you avoid eating a full meal 2 hours before the study. If you have eaten anything or chewed

gum/candy, etc, please try to have some water to rinse out any residue before coming in. If you are a smoker, would it be possible to NOT smoke at least 2 hours before your appointment? If you are planning to have alcohol the night before, is it possible to limit your intake to the legal limit, namely not more than 1 drink per hour on the night before your appointment? If you exceed this amount (and/or are hung over the morning of the study), please let us know and we could try to reschedule. Finally, we ask that you try not to brush your teeth within the hour before coming in to the study. We will remind you of these things in an email on the night before your appointment. Thanks again and we look forward to seeing you on day/time."

Appendix B
Final Pre-Screening Questions on the Day of the Study

When participants come in on the day of the study, research assistants will ask these pre-screening questions:

1. Have you smoked within the past hour?
2. Did you drink alcoholic beverages last night to the point of becoming drunk and/or being hung-over this morning?
3. Have you had a fever of 102 + in the past 48 hours?
4. Are you currently under a physician's care for any type of endocrine disorder or cancer?
5. Have you been taking any over-the-counter or prescribed medications, or any other pills that may contain steroid hormones within the past 48 hours?
6. Have you used an asthma inhaler in the past 24 hours?
7. Has your mood today been any different than usual? Explain. (if much worse, reschedule)
8. Has today been any more stressful/hectic/emotionally distressing than is typical for you?
9. Have you had a full meal within the past hour before you came in today?

Appendix C

Daily Inventory of Stressful Events (DISE)

1. Did you have an *argument or disagreement* with anyone since this time yesterday?
   A – No **B – Yes**

2. If NO to above, mark A for "0." If YES to above, how stressful was this for you?

   B – Not at all **C – Not very** **D – Somewhat** **E –Very**

3. Since this time yesterday, did anything happen that you *could have argued* about but you decided to let pass in order to avoid a disagreement?
   A – No **B – Yes**

4. If NO to above, mark A for "0." If YES to above, how stressful was this for you?

   B – Not at all **C – Not very** **D – Somewhat** **E –Very**

5. Since this time yesterday, did anything happen at *work or school* (other than what you have already mentioned) that most people would consider stressful?
   A – No **B – Yes**

6. If NO to above, mark A for "0." If YES to above, how stressful was this for you?

   B – Not at all **C – Not very** **D – Somewhat** **E –Very**

7. Since this time yesterday, did anything happen at *home* (other than what you have already mentioned) that most people would consider stressful?
   A – No **B – Yes**

8. If NO to above, mark A for "0." If YES to above, how stressful was this for you?

   B – Not at all **C – Not very** **D – Somewhat** **E –Very**

9. Many people experience *discrimination* on the basis of such things as race, sex, or age. Did anything like this happen to you since this time yesterday?
   A – No **B – Yes**

10. If NO to above, mark A for "0." If YES to above, how stressful was this for you?

    B – Not at all **C – Not very** **D – Somewhat** **E –Very**

11. Since this time yesterday, did anything happen to a *close friend or relative* (other than what you have already mentioned) that turned out to be stressful for you?

A – No **B – Yes**

12. If NO to above, mark A for "0." If YES to above, how stressful was this for you?
B – Not at all **C – Not very** **D – Somewhat** **E –Very**

13. Did *anything else* happen to you since yesterday that most people would consider stressful?
A – No **B – Yes**

14. If NO to above, mark A for "0." If YES to above, how stressful was this for you?
B – Not at all **C – Not very** **D – Somewhat** **E –Very**

Appendix D

Table 2. Means and Standard Deviations for Independent and Dependent Variables by Psychopathy Group and Gender

	Psychopath Group						Nonpsychopath Group					
Variable/Scale Name	Men			Women			Men			Women		
	N	M	SD	N	M	SD	N	M	SD	N	M	SD
PPI-Total Score	27	137.74	13.36	29	132.24	13.63	22	122.64	12.45	27	120.37	18.66
PPI-Factor 1_Z (Personality)	29	.49	1.79	30	-.21	2.25	23	-.03	2.17	28	-.33	2.46
PPI-Factor 2_Z (Lifestyle)	28	1.34	2.16	30	1.36	2.13	22	-1.81	2.62	28	-1.56	2.57
Aggression Q	27	81.56	14.80	31	73.06	18.68	23	14.87	2.94	30	17.03	1.69
Welsh Anxiety	29	49.72	6.80	31	54.00	14.86	23	48.83	8.56	30	47.80	6.75
State Anxiety	29	33.28	6.56	31	36.74	10.14	23	32.04	7.67	30	32.53	2.73
Trait Anxiety	21	35.95	7.05	27	39.41	8.69	21	33.67	6.67	20	35.75	7.61
BIS	29	18.07	3.26	31	19.87	2.72	23	17.83	3.64	30	20.00	2.73
BAS	29	40.34	5.03	31	41.87	5.39	23	34.30	4.43	30	39.27	4.73
Baseline Cortisol	29	.34	.27	30	.31	.26	23	.25	.13	30	.28	.15
Post-task Cortisol	29	.25	.14	31	.25	.19	23	.23	.09	30	.24	.12
Baseline Testosterone	29	315.52	202.92	31	131.97	104.45	23	289.25	141.24	30	117.23	65.26
Post-task Testosterone	29	284.13	139.29	31	138.68	77.68	23	389.18	341.15	30	118.40	63.21
IGT (good-bad)	29	5.59	31.52	31	1.03	29.51	23	13.08	36.16	29	26.45	22.14
Levenson Psychopathy	29	36.86	5.64	29	34.00	9.94	23	16.74	6.90	30	14.10	4.89
Self-Report Psychopathy	28	84.25	10.27	31	72.87	12.01	22	52.00	16.74	29	51.79	12.03
MC Social Desirability	29	47.38	4.47	30	49.70	4.83	23	53.00	5.19	30	53.00	4.78

Table 3. Correlation Matrix

Variable	1 Psychopathy	2 PPI	3 PPI-1	4 PPI_2	5 Agg Q	6 Gender	7 IGT_good	8 IGT_bad	9 STAI	10 BIS	11 BAS	12 Pre-Testo	13 Post_Testo	14 Pre-Cort	15 Post-C
1. Psychopathy	____														
2. PPI	-.425**	____													
3. PPI_1	-.075	.656**	____												
4. PPI_2	-.543**	.789**	.092	____											
5. Agg Q	-.492**	.507**	.199*	.556**	____										
6. Gender	.049	-.137	-.123	-.001	-.166	____									
7. IGT_good	.282**	-.179	-.054	-.223*	-.295**	.070	____								
8. IGT_bad	-.282**	.179	.052	.226*	-.294**	-.072	-.999**	____							
9. STAI	-.232**	.184**	-.460**	.349**	.140	.172	-.191	.192	____						
10. BIS	.001	-.312**	-.568**	.061	-.007	.310**	-.049	.046	.472**	____					
11. BAS	-.353**	.374**	.330**	.302**	.401**	.262**	-.081	.082	-.122	.051	____				
12. Pre-Testo	-.037	.020	.074	-.116	.155	-.651**	-.070	.070	-.182	-.348**	-.231*	____			
13. Post_Testo	.019	-.020	.070	-.124	.047	-.647**	-.078	.077	-.152	-.382**	-.305**	.897**	____		
14. Pre-Cort	-.062	-.014	-.124	.075	.070	.013	.009	-.007	.070	.027	-.014	.242**	.127	____	
15. Post-Cort	.000	-.022	-.020	-.056	.005	-.022	.035	-.036	-.060	-.079	-.012	.260**	.245**	.780**	____

Note: Psychopathy = Grouping variable based on high/low psychopathy quartile scores on the Self-Report Psychopathy Scale (SRP)and on Levenson's Primary and Secondary Psychopathy Scales, coded such that 1 = "psychopath" and 2 = "nonpsychopath. PPI sum = Psychopathic Personality Inventory; PPI_1 = Factor 1

PPI; PPI_2 = Factor 2 PPI; Agg Q = The Aggression Questionnaire; Gender = male/female; IGT_good = sum of Iowa Gambling task good/advantageous decisions (decks C+D); IGT_bad = sum of Iowa Gambling task bad/disadvantageous decisions (decks A+B); STAI = sum of Stait-Trait Anxiety Inventory; BIS = sum of Carver and White's self-report Behavioral Inhibition Scale; BAS = sum of Carver and White's self-report Behavioral Activation Scale; Pre-Testo = Baseline Testosterone level; Post-Testo = Post-task Testosterone level; Pre-Cort = Baseline Cortisol level; Post-Cort = Post-task Cortisol level.
(*) Correlation is significant at the .05 level (2-tailed).
(**) Correlation is significant at the .01 level (2-tailed).

Table 4. Internal Consistency Coefficients of Measures Used in This Study

Measures	**Cronbach's Alpha**
STAI ("State")	.88
STAI ("Trait")	.87
STAI Full Scale	.90
PPI Full Scale	.85
PPI Factor 1	.83
PPI Factor 2	.87
Self-Report Psychopathy Scale (SRP)	.87
Buss Aggression Q	.91

VITA

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Education

December 2005—Doctor of Philosophy (Clinical Psychology), Pennsylvania State University,
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Clinical Experience

APA-Approved Internship, West Los Angeles VA Healthcare Center — Aug 2004 – August 2005
Therapist, Counseling and Psychological Services (CAPS), Penn State University — Aug 2002 – May 2004
Therapist, The Psychological Clinic, Penn State University — Jan 1999 – May 2004
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Neuropsychological Assessment, The Psychological Clinic, Penn State University — Sept 2001 – June 2002

Research Experience

Clinical Psychology/Psychopathy—Pennsylvania State University — Aug 2001 – Present
Research with Dr. Peter Arnett in etiological models of psychopathy. Dissertation defended August 2005.
Graduate Research Assistant , Family Life Project—Pennsylvania State University
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Graduate Research Assistant—Dept. of Psychology, Pennsylvania State University — Jan 1998 – July 2004
Clinical/Forensic Psychologyy—Pennsylvania State University — Aug 1998 – July 2001
Research with Dr. Gordon Nagayama Hall in etiological models of sexual aggression. Masters defended July 2001.
Clinical Psychology/Child Abuse—University of Southern California — Summer 1999
Research assistant for Dr. Thomas Lyon, J.D., Ph.D.
Forensic Psychology—University of California, Los Angeles — Sept 1997 – June 1998
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Clinical Psychology/Family Therapy Process—The University of Utah — Summer 1997
Research with Dr. James Alexander in the area of Family Process research for adolescent delinquency.
Neuroscience/Cortical Analysis—University of California, Los Angeles — Spring 1997
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Teaching Experience:

Instructor/Lecturer of Psychology of Adjustment Course—Pennsylvania State University — Jan – Aug 2002
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Research and Graduate Studies Office Travel Grant Award — (2002 & 2003)
Fellowship for Graduate Studies, the Pennsylvania State University — (1998-1999)
Student Award for Research Training , UCLA — (Winter & Spring 1997)
Dean's Honors List, UCLA — (Fall & Winter 1996-1997; Spring 1995)
Annual College Scholarship Service Award recipient for academic excellence, UCLA — (1994-1997)
Recipient of The Guy T. Ellis Scholarship — (a full-tuition award, 1994 & 1995)

Publications

Erian, M., Lin, C., Patel, N., Neal, A. & Geiselman, R. E (1998). Juror verdicts as a function of victim and defendant attractiveness in sexual assault cases. *American Journal of Forensic Psychology. Vol 16(3),* 25-40. Menard, K.S., Hall, G.C.N., Phung, A., Ghebrial, M.E., & Chow, L. (2003). Gender differences in sexual coercion and harassment among college students: Developmental, individual, and situational determinants. *Journal of Interpersonal Violence, 18.*
Ghebrial, M.E. & Hall, G.C.N. (2004). Gender-role socialization, depression, and psychopathy in a university sample. Manuscript being prepared for resubmission.
Ghebrial, M.A. & Arnett, P.A. (2004). Examination of BIS and BAS functioning in psychopathic offenders. Manuscript submitted for publication.