ABSTRACT

Title of Document: BIOBEHAVIORAL MECHANISMS UNDERLYING
                      EMOTIONALITY IN ANTISOCIAL PERSONALITY
                      DISORDER AND THE ROLE OF PSYCHOPATHIC
                      TRAITS

                      Marsha Nneka Sargeant, Master of Science 2008

Directed By: Stacey B. Daughters, Ph.D., Assistant Professor (Research),
                      Center for Addictions Personality and Emotion Research

The distinguishing features of Antisocial Personality Disorder (ASPD) include a general disregard for the rights of others in the form of irresponsible, impulsive, deceitful, remorseless, and aggressive behavior. The DSM-IV (APA, 1994) adds that individuals with ASPD exhibit a low tolerance for frustration and boredom. In support of this perspective, a recent study investigating the relationship between ASPD diagnosis and distress tolerance indicated that ASPD diagnosis was indeed related to low distress tolerance (DT; Daughters, Sargeant, Gratz, Bornovalova, & Lejuez, in press). This finding is interesting in light of the relation of ASPD to psychopathy, in which callous and unemotional (CU) traits manifest in emotional hyporeactivity. We examined whether psychopathic traits and accompanying hypoarousal are related to higher DT and low biological stress response in the form of cortisol reactivity to a laboratory stressor, which would indicate that low DT may be specific to a subset of individuals who have both ASPD as well as low levels of psychopathic traits. Therefore, given the hyper-reactivity in ASPD, it was hypothesized that ASPD would be associated with low DT and high

cortisol response to a laboratory stressor. Furthermore, given the hypo-reactivity associated with psychopathy, we hypothesized that psychopathic traits would be associated with high DT and blunted cortisol stress response. Results indicated that when considered together, ASPD and psychopathic traits predicted DT in expected directions. Specifically, ASPD was associated with lower DT and psychopathic traits with higher DT. Cortisol reactivity was not significantly related to ASPD or psychopathic traits, but exploratory analyses indicated that discrepant patterns of stress reactivity emerged for individuals with ASPD and high levels of psychopathic traits. These findings suggest unique contributions of ASPD and psychopathic traits to emotionality across behavioral and biological domains. Studies of this kind may assist in the development of more reliable and valid theoretical models of ASPD, with implications for treatment and intervention.

BIOBEHAVIORAL MECHANISMS UNDERLYING EMOTIONALITY IN
ANTISOCIAL PERSONALITY DISORDER AND THE ROLE OF PSYCHOPATHIC
TRAITS

By

Marsha Nneka Sargeant

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Advisory Committee:
Assistant Professor (Research) Stacey B. Daughters, Chair
Professor Carl W. Lejuez
Professor Jack Blanchard

Dedication

This is dedicated to my doozie of a mother, my sister (Schmelly), and dad, who have seen me through it all and have never faltered in their love and support, even on the darkest of days. Thank you also to Kobe, who has been there throughout these difficult days of graduate school and has given me support and encouragement. Finally, I'd like to thank Wanda "Eagle-eye" Stevenson, who gave me the initial boosts and necessary logistical and daily support to get this whole education idea going again.

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Chapter 1: Introduction

Overview

The distinguishing features of Antisocial Personality Disorder (ASPD) include a general disregard for the rights of others in the form of irresponsible, deceitful, impulsive, remorseless, and aggressive behavior. Moreover, ASPD is characterized by engagement in a series of aggressive, impulsive and chaotic behaviors as well as an inability to inhibit emotional responses and problem solve (APA, 2004). The lifetime prevalence of ASPD has been reported recently to be 3.6% (Compton, Conway, Stinson, Colliver, & Grant, 2005; Hall, 2003), with higher rates in men than in women (5.5% and 1.9%, respectively). This prevalence is associated with such negative outcomes as criminality (Hodgins, Mednick, Brennan, Schulsinger, & Engberg, 1996), domestic partner violence (Fals-Stewart, Leonard, & Birchler, 2005), and suicidality (Verona, Patrick, & Joiner, 2001). In individuals with ASPD, antisocial behavior manifests in childhood as conduct disorder and continues throughout adulthood (Gelhorn, Sakai, Price, & Crowley, 2007).

ASPD is often comorbid with numerous other disorders[1], the most common being mood and substance use disorders (Holdcraft, Iacono, & McGue, 1998). Individuals with ASPD who exhibit a comorbid disorder are susceptible to poorer outcomes and/or greater severity of impairment compared to individuals without ASPD (Goodwin & Hamilton, 2003; Holdcraft, Iacono, & McGue, 1998). Furthermore, ASPD is the most common psychiatric disorder among individuals with substance use disorders, with comorbidity rates of 30.3% with alcohol use disorders and 10.3% with drug use disorders (Compton,

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[1] For a more detailed discussion of ASPD Comorbidity, see Appendix A

et al., 2005). These rates are considerably higher than the 3.6% prevalence rate of ASPD

in the general population. Among individuals with substance use disorders, comorbid

ASPD is associated with greater psychological distress (Darke, 2004), greater severity of

substance use (Daughters, Sargeant, Bornovalova, Gratz, & Lejuez, in press) poorer

treatment outcomes (Compton, Cottler, Jacobs, Ben-Abdallah, & Spitznagel, 2003),

higher levels of needle and polydrug use, a higher prevalence of HIV infection, and

suicide rates (Brooner, Bigelow, Strain, & Schmidt, 1990) than individuals without

ASPD.

       Although much is known about the behavioral correlates and negative outcomes

associated with ASPD, little is known about the mechanisms that may be responsible for

the characteristics of this disorder. Research on the development of ASPD has identified

childhood aggression as one of the strongest risk factors for antisocial behavior in

adolescence and young adulthood (Broidy, Nagin, Tremblay, Bates, Brame, Dodge, et al.,

2003; Loeber & Hay, 1997); however, it remains unclear what mechanisms may be

responsible for engagement in aggression.

       In trying to understand mechanisms underlying ASPD, some researchers

developing comprehensive models of antisocial development have outlined dynamic

systems that involve parent-child and peer socialization processes (Frick & Loney, 2002)

as well as numerous other environmental risk factors such as deviant peers (Kasen,

Cohen, & Brook, 1998) and childhood victimization (Widom, 1997)[2,3]. There have also

been preliminary efforts to bridge behavioral and emotional factors involved in the

etiology of ASPD (Fowles, 2000; Herpertz et al., 2005; Smith, 2006; Vanyukov, Moss,

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[2] For a more detailed discussion of the developmental course of ASPD, see Appendix B
[3] For a more detailed discussion of the risk factors associated with ASPD, see Appendix C

Plail, Blackson, & et al., 1993); however, these studies have primarily examined emotion

regulation in children and adolescents with Conduct Disorder (CD) or with subclinical

levels of antisociality. Research in this area has found that conduct problems, particularly

aggressive behavior, may be attributable to the combination of self-regulation problems

and the propensity to respond poorly to distressing emotions including frustration and

anger (Deckard, Petrill, & Thompson, 2007). While much is known about self-regulation

deficits in individuals with externalizing problems, little is known about their poor

response to distressing emotions[4]. To this end, Daughters, et al. (in press) applied the

construct of distress tolerance (DT) and found that individuals with ASPD had lower

levels of DT than those without ASPD. Therefore, in trying to understand mechanisms

underlying ASPD, a useful starting point for examining behavioral and emotional

mechanisms may be the further investigation of distress intolerance in this disorder.

Antisocial Personality Disorder and Distress Tolerance

Low DT is characterized by an inability to persist in goal oriented behavior during

an aversive situation and is reflective of how one copes with the negative affect resulting

from environmental and interpersonal challenges (Brown, Lejuez, Kahler, Strong, &

Zvolensky, 2005). In order to measure DT, aversive challenge tasks are utilized that

capture both the valence and arousal aspects of emotion. The aversiveness of each task is

evidenced by changes in self-report negative affect during these tasks (Brown, Lejuez,

Kahler, & Strong, 2002; Brown, Lejuez, Kahler, Strong, & Zvolensky, 2005; Daughters,

Lejuez, Bornovalova et al., 2005; Daughters, Lejuez, Kahler, Strong, & Brown, 2005;

Daughters, Lejuez, Strong et al., 2005). Additionally, the ability of DT paradigms to

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[4] For a discussion of ASPD, Borderline Personality Disorder, impulsivity, and distress tolerance, see
Appendix D.

capture the arousal aspect of emotion is evidenced by elevation in heart rate and skin conductance during the challenge tasks (Brown, Lejuez, Kahler, & Strong, 2002); see also, Gratz, Rosenthal, Tull, Lejuez, & Gunderson, unpublished data).

One of the few studies examining behavioral mechanisms underlying ASPD in adults revealed an association between ASPD diagnosis and the inability to tolerate psychological distress (Daughters, Sargeant, Bornovalova, Gratz, & Lejuez, in press). Specifically, in a study examining the relation between DT and ASPD in residential treatment seeking substance abusers, patients with ASPD exhibited significantly lower levels of persistence on a psychological stress task (e.g., low distress tolerance) compared to non ASPD patients. This finding is consistent with the DSM-IV (APA, 1994) discussion of individuals with ASPD as having a low tolerance for frustration and boredom. Distress tolerance has also been hypothesized to underlie maladaptive behavior in individuals with Borderline Personality Disorder (BPD)[5]. One limitation of this work, however, is that the role of psychopathy, a set of traits that often co-occurs with ASPD, was not considered. Outlined next are several reasons that a clearer understanding of the link between DT and ASPD would require a better understanding of the contribution of psychopathy.

Antisocial Personality Disorder and Psychopathy

Based on the original account of this disorder (Cleckley, 1941), psychopathy is characterized by the type of social deviance present in ASPD (e.g., irresponsibility, social maladjustment, impulsive/aggressive behavior), as well as interpersonal and emotional deficits in the form of CU traits. Although this distinction has been made, the _DSM-IV_

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[5] For a more detailed discussion of ASPD and BPD, and distress tolerance see Appendix D.

equates the pattern of behavior characteristic of ASPD with psychopathy (APA, 1994), leading to some level of nosological confusion over its distinction from ASPD.

Critics of the *DSM-IV* point out that the ASPD criteria are overly focused on specific behavior, namely criminality and not enough on personality traits that may underlie such behavior (Hare, Hart, & Harpur, 1991). Moreover, although ASPD and psychopathy are not identical, psychopathy has been found to be comprised of two separate correlated factors (Hare, 1983), one reflecting criminality (i.e., antisocial behavior) and the other reflecting manipulative behavior and lack of empathy. Additionally, in a large epidemiologic study of psychiatric disorders, Robins and Regier (1991) found that few individuals with ASPD had difficulties with the law. Moreover, Hare (1983) found that half of all prisoners did not meet criteria for ASPD. Thus, criminality is not central to ASPD. These findings suggest that perhaps the personality traits that are associated with ASPD should be more closely examined.

In the *DSM-IV* field trial, there was mixed support for the proposal to include traditional traits of psychopathy in the criteria (Widiger et al., 1996). The decision not to include such criteria as lack of guilt and remorse was made for two reasons. First, the inclusion and exclusion of these criteria resulted in equally reliable assessments of ASPD. Second, there was a concern that inclusion of the criteria would lead to discriminant validity issues, particularly with respect to the distinction of ASPD and Narcissistic Personality Disorder. Therefore, there is no clear evidence arguing for the inclusion or exclusion of CU traits in ASPD. This ambiguity was further advanced by these two semi-independent bodies of literature that have developed with little attention to the overlap between the two categories. In the few studies that have attempted to

address ASPD and psychopathic traits, there have been conflicting findings regarding the overlap of the two disorders. While some purport that the conceptualization of ASPD is qualitatively different than psychopathy (Blackburn, 1988), others argue that psychopathic traits should not be ignored in the diagnosis of ASPD (Rogers, Duncan, Lynett, & Sewell, 1994). In fact, according to the Consequence Hypothesis, some have speculated that psychopathic traits may play a causal role in relation to antisocial behavior (Cooke, Michie, Hart, & Clark, 2004; McDermott et al., 2000). To this end, biological, psychological, and social risk factors conferred by psychopathy are thought to result in engagement in antisocial behavior.

The DSM diagnosis of ASPD has been consistently shown to be highly related to Hare's Psychopathy Checklist – Revised (1991) psychopathy ratings (Hare, Hart, & Harpur, 1991). Furthermore, in a psychometric review of the development of the PCL-R (Hare, Harpur, Hakstian, Forth, & et al., 1990), two correlated factors emerged. Factor 1 is characterized by interpersonal and affective deficits such as lack of remorse, superficial charm, and manipulative behavior. Factor 2, often referred to as the antisocial deviance component, comprises behavioral characteristics such as criminality, social deviance, impulsivity, and disinhibition. Factor 2, in turn, has been referred to in the literature as parallel to the DSM diagnosis of ASPD. Based on these findings, critics of the current *DSM* diagnostic criteria propose that ASPD may be a heterogeneous disorder, with individuals varying on psychopathic traits and patterns of comorbidity (Sher & Trull, 1994).

Indeed, the *DSM-IV* refers to psychopathy as merely a synonym for ASPD. Nevertheless, although the affective and interpersonal aspects of ASPD are described in

the DSM as central to the clinical presentation of the disorder (Harpur, Hart, & Hare, 1994; Rogers, Duncan, Lynett, & Sewell, 1994) these features are not incorporated into the diagnosis. Therefore, there has been little research on affective/emotional components of ASPD. Furthermore, due to the paucity of research on emotionality and ASPD, it is difficult to discuss ASPD in isolation, as the preponderance of the literature focuses on the antisocial behavior associated with psychopathy, not on the *DSM-IV* diagnosis of ASPD. Therefore, in considering the relationship between distress tolerance and ASPD, it is important to take into account the role of psychopathic traits. It may be the case that individuals with the presence/absence of psychopathic traits may be related to a different presentation of ASPD, with the absence of emotionality possibly related to a counterintuitive higher ability to tolerate distress.

Biobehavioral Mechanisms Underlying Emotionality in Psychopaths

Emotion is defined as affectively aroused behavior that is a response disposition or that underlies states of readiness for adaptive behavior (Lang, 1995). Affective arousal in organisms serves to prepare for self-preservative action (Lang, Bradley, & Cuthbert, 1990). This viewpoint of emotion delineates two brain motive systems: (1) an appetitive system responsible for approach and consummatory behaviors, and (2) an aversive system responsible for defensive reactions. Since these systems are involved in basic conditioning processes and allow emotional responses to be influenced by learning history, they play a key role in emotionality and affect the valence and arousal aspects of emotion. According to the low fear hypothesis, it is believed that psychopaths have a deficit in emotional reactivity to aversive events.

The preponderance of research on emotional reactivity in psychopaths focuses on the aversive system of emotion reaction and has assessed defensive emotional dispositions through the use of startle probe reflex in reaction to stimuli of various valence (i.e., pleasant, neutral, unpleasant) and aversive noise blast paradigms. Specifically, the core psychopathy traits of emotional detachment are associated with reduced or absence of a startle reflex potentiation and during exposure to threatening stimuli (Patrick, 1994; Patrick, Bradley, & Lang, 1993). Of note here is that this phenomenon was related to the affective features of psychopathy, but not to the antisocial/deviant aspect of the disorder. In fact, individuals with moderate levels of psychopathy, but who met criteria for ASPD showed a response pattern similar to that of normal controls. Additionally, it is notable that psychopaths and non-psychopaths did not differ in self-reports of the pleasantness or their arousal level when presented with the stimuli. Similar evidence of the importance of the detached/unemotional features of psychopathy was found in a study of startle reflex potentiation in prisoners classified as psychopathic, antisocial, detached or non-psychopathic (Patrick, 1994). The psychopathic and detached groups exhibited a diminished mean blink magnitude difference in anticipation of a noxious noise while the antisocial and non-psychopathic groups had similar and larger mean blink magnitude differences.

It is evident that the extant literature on emotional responding in psychopaths provides rich information for further study through its examination of emotional mechanisms at the biological level; however, little is known about such mechanisms in ASPD. Furthermore, there are few studies on biological markers of distress in ASPD. With respect to distress tolerance, a relevant biological indicator of distress is cortisol

reactivity. One study, examining the association between psychopathic traits and cortisol response to stress in college students found that high levels of psychopathic traits was associated with blunted cortisol response in males, but not in females (O'Leary, Loney, & Eckel, 2007). Another study found that cocaine addicts with ASPD exhibited decreased cortisol responsiveness to a stressor (Buydens-Branchey & Branchey, 2004). Although these findings are consistent with the idea of hypoemotionality in psychopathy, this study didn't consider possible differential effects of antisocial deviance and psychopathic traits. Therefore, one avenue of research that may prove fruitful is investigation into the role of HPA axis vulnerability in understanding emotional responding in individuals with ASPD. A related body of work focuses on children and adolescents with Conduct Disorder (CD), which is a required diagnosis for meeting ASPD criteria in adulthood. A brief review of this literature is provided in the next section.

Antisocial Behavior and Biological Markers of Distress

Exposure to stressful situations elicits an adaptive response in the nervous system. This response serves to maintain homeostasis and to return the body to equilibrium. A principal component of this stress-regulation system is the hypothalamic-pituitary-adrenal (HPA) axis, which controls the secretion of hormones for the pituitary and adrenal cortex, and can be indexed by changes in serum or salivary cortisol levels (de Kloet & Reul, 1987). Along these lines, HPA axis functioning has been found to explain individual differences in antisociality in children and adolescents by accounting for differences in emotional reactivity as measured by baseline cortisol levels as well as change in cortisol levels in response to stressful conditions (Hall, 2003; Loney, Butler, Lima, Counts, &

Eckel, 2006; McBurnett et al., 2005; van Goozen, Snoek, Matthys, van Rossum, & van Engeland, 2004; Vanyukov, Moss, Plail, Blackson, & et al., 1993).

Consistent with the fearlessness theory, reduced arousal has been found in children with externalizing disorders (Shirtcliff, Granger, Booth, & Johnson, 2005; Snoek, Van Goozen, Matthys, Buitelaar, & Van Engeland, 2004; Vanyukov, Moss, Plail, Blackson, & et al., 1993), suggesting that fearless children are more likely to engage in antisocial behavior because they are not afraid of the negative consequences. An alternative to this theory is that provided by the stimulation-seeking theory (Zuckerman, 1979), in which individuals with low arousal are more motivated to seek out stimulation in order to raise their arousal levels to an optimal level. Despite these findings, some research examining the relationship between cortisol levels and behavior problems have generated mixed findings (Granger, Weisz, & Kauneckis, 1994; Shirtcliff, Granger, Booth, & Johnson, 2005).

It is possible that callous and unemotional (CU) traits may account for these inconsistent findings. In a sample of adolescents, males with elevated CU traits exhibited lower cortisol levels than comparison groups (Loney, Butler, Lima, Counts, & Eckel, 2006). Females did not differ along this dimension. In another study examining cortisol reactivity in adolescent males in response to a challenge task (McBurnett et al., 2005), differences in salivary cortisol along a continuum of Conduct Disorder (CD) symptoms were only found when removing those in the top quartile of the number of CD symptoms endorsed. In this case, higher levels of salivary cortisol were related to higher levels of CD symptoms. It is possible that by removing those with the most extreme CD symptoms, that those with CU traits were also removed, thus affecting the results.

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As research examining HPA axis response has been limited to child and
adolescent samples with externalizing disorders, more work is needed to provide insight
into HPA axis functioning in adults with externalizing disorders like ASPD. Also, the
psychopathic traits of callousness and lack of emotion may be useful in examining
emotionality in individuals with ASPD.

Antisocial Personality Disorder and Callous and Unemotional Traits

Given the overlap of ASPD with psychopathy, the examination of psychopathic
traits may provide a starting point for further examining distress tolerance in individuals
with ASPD. It has been estimated that 25% of individuals with ASPD may meet criteria
for psychopathy (Blair, Mitchell, & Blair, 2005). One might expect that due to the
callous/unemotional (CU) feature of psychopathy, individuals with elevated psychopathic
traits might not experience distress in the same manner as other APSD individuals. They
may, due to emotional hypo-arousal, exhibit high distress tolerance. Furthermore, in line
with the theoretical framework provided by Gray's Behavioral Inhibition/Activation
Systems (BIS and BAS)[6], psychopaths exhibit non-avoidance and physiological
hypoarousal during aversive situations. This is consistent with a weak BIS system
(Fowles, 1980).

This information may be especially relevant for understanding the DT and ASPD
findings outlined above. Distress tolerance paradigms are able to create an aversive
situation in which one can choose to remain in order to achieve a goal, or to escape and
terminate the stressor immediately. Consistent with previous findings, one would thus
expect that "pure" ASPD traits would be associated with non-persistence and the inability

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[6] For a detailed discussion of the BIS and BAS systems, refer to Appendix E

to render the needed biological response to distress. However, when considering the role of psychopathic traits and specifically the lack of emotionality, one might instead predict a counterintuitive higher persistence on psychological challenge tasks, perhaps in the form of under-reactivity of the HPA axis. As such, it would be important to consider the overlap of ASPD with psychopathic traits in better understanding the role of ASPD and DT.

Current Study

Previous research indicates that ASPD is related to low distress tolerance (Daughters, et al., in press); however, theory and research indicate that certain types of individuals with ASPD, mainly those with elevated psychopathic traits, might evidence very different profiles of emotional vulnerability than other individuals with ASPD. Psychopathic traits may prove useful in better understanding emotion in individuals with ASPD, specifically the relationship between distress tolerance and ASPD. Thus the proposed study aims to replicate the distress tolerance findings and to extend those findings by examining the unique roles of ASPD and psychopathic traits in predicting emotionality across behavioral and biological domains.

The purpose of the current study is threefold. First, we attempted to replicate the previous results by Daughters, et al. (in press) by examining the relationship between DT and ASPD. We hypothesize that individuals with ASPD will evidence significantly lower levels of distress tolerance compared to individuals without ASPD as indexed by performance on two psychological challenge tasks. Second, we sought to extend these findings by examining the role of psychopathic traits in the relationship between distress

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tolerance and ASPD. We hypothesized that psychopathic traits and ASPD diagnosis would contribute to this relationship in opposite directions, such that psychopathic traits would predict higher levels of distress tolerance, independent of ASPD status, and psychopathic traits would predict lower levels of distress tolerance, independent of psychopathic traits. Specifically, we predict that psychopathic traits will function as a suppressor variable, with the magnitude of the relationship between ASPD and DT increasing, when these psychopathic traits (related to DT in the opposite direction as ASPD) are taken into account. Finally, given prior research that found a relationship between callous and unemotional traits and HPA axis functioning in children, we explored the unique roles of ASPD and psychopathic traits in predicting HPA axis functioning. Therefore, we predicted that ASPD diagnosis and psychopathic traits would divergently predict biological stress response as measured by total cortisol reactivity following stressor tasks (controlling for baseline cortisol levels).

One strength of the current study is that emotionality will be examined across both behavioral and biological domains, thereby reducing the limitations inherent in studies that rely solely on self-report. This is particularly helpful in light of the evidence in emotion studies of psychopathy, where self-report and physiological indices were contradictory (Patrick, 1994; Patrick, Bradley, & Lang, 1993; Patrick, Cuthbert, & Lang, 1994). Another strength of this study is that examining biological response to stress in ASPD and psychopathy is a novel approach to understanding emotionality in these disorders. Finally, this study seeks to replicate and extend previous findings relating ASPD to low DT.

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Chapter 2: Method

Participants

Participants were 91 consecutively admitted treatment-seeking men and women between the ages of 19 and 50 in the Harbor Light Salvation Army Residential Substance Abuse Treatment Center. Eighty-eight percent of participants were African American. Harbor Light requires that all residents undergo detoxification before treatment entry, thereby eliminating the possible residual effects of detoxification in our sample. Individuals who met criteria for Axis I disorders (except psychotic disorders) were not be excluded from the study in order to ensure generalizability of these findings to other individuals with ASPD.

Overall Design and Procedure

This study was conducted as part of a larger study conducted in the center examining distress tolerance and substance abuse treatment outcomes. Potential participants were approached once per week and asked if they were interested in participating in a study examining emotion and treatment outcome. The purpose and procedure were explained in detail to the participant. In particular, prospective participants were informed that the study would take approximately two hours during which they would be asked to complete four study components including: a) paper and pencil questionnaires, b) a clinical interview, c) two computer tasks, and d) provision of five saliva samples. Issues of confidentiality were explained in detail. Informed consent was then obtained from those who wished to participate. To accommodate those with

reading comprehension difficulties, efforts were made to ensure that all aspects of the experimental session were explained verbally.

Brief semi-structured interviews to determine DSM-IV psychiatric diagnoses were administered by trained graduate research assistants. Questionnaire data for the first 23 participants were initially collected using an audio-enhanced computer-assisted self-interviews software system (Audio CASI) on laptop computers in classrooms at the Harbor Light Facility. Due to difficulties with the system, we decided to collect data using paper and pencil questionnaires after 10% of the data was collected.

Experimenters led participants in a Progressive Muscle Relaxation exercise prior to providing the first saliva sample and beginning the DT tasks. The tasks were completed on laptop computers. Saliva samples were collected using the passive drool method, whereby saliva is passed through a straw and into a tube.

The order of the behavioral tasks was determined randomly for each participant to limit the influence of order effects. All participants were paid in the form of a $25 grocery store gift card for their participation. To ensure motivation during the computer tasks, participants were told that the amount of their payment was contingent upon their performance on the task. After the task, they were informed of how much money they earned and signed a receipt in that amount.

Inclusion / Exclusion Criteria and Design Considerations

There were several sample-related issues to consider for study inclusion/exclusion. First, the utilization of substance users in residential treatment has both strengths and limitations. The use of this sample is not representative of all individuals with ASPD and/or psychopathic traits, which could potentially limit

generalization of findings from this study. Also, the effects of detoxification could potentially affect self-reported, behavioral, and biological outcomes. Participants underwent detoxification before entry into the rehabilitation program; furthermore, all participants were free of drugs and alcohol during participation, as abstinence is mandatory and is frequently assessed throughout treatment. Finally, although all participants were substance users undergoing treatment, there were differences in substance use frequency and severity. Therefore, these factors were controlled for in the analyses as appropriate.

We also considered role of age in the examined relationships. The prevalence of personality disorders is higher in younger adults than older adults across both clinical and community samples (Ames & Molinari, 1994; Casey & Schrodt, 1989; Fogel & Westlake, 1990). Longitudinal studies have found similar patterns (Lenzenweger, Johnson & Willett, 2004), corroborating the hypothesis that personality disorders decline with age. There is also mounting evidence for the notion of heterotypic continuity. This refers to the idea that underlying personality characteristics remain stable with age but that the presentation of the characteristics change (e.g., Caspi, & Bem, 1990). For instance, one study examining personality disorder criteria across younger and older age groups found that, given equivalent personality disorder pathology, there was an age difference in the criteria endorsed (Balsis, Gleason, Woods, & Oltmanns, 2007). Also, in a study examining age-related personality differences in individuals with personality disorder, there were no age differences in the number of patients diagnosed with PD; however the severity of symptoms was found to decline with age (Molinari, Kunik, Snow-Turek, Deleon, & Williams, 1999). Evidence more pertinent to DT includes a

study which found that older adults with personality disorders reported lower levels of dysfunctional coping strategies than younger adults (Segal, Hook, & Coolidge, 2001). Moreover, irritability and aggressiveness have been found to decline with age in individuals with ASPD (Balsis, et al., 2007). Thus, we will constrain our sample to individuals aged 50 and younger.

The next consideration was with regard to diagnoses and psychotropic medications. Due to the high comorbidity of substance use with other Axis I disorders (Kessler, Chiu, Demler, & Walters, 2005; Kessler, Crum, Warner, Nelson, & et al., 1997; Merikangas et al., 1998), we did not exclude individuals diagnosed with co-occurring Axis-I disorders, with the exception of psychotic disorders. Psychotic symptoms have been found to affect the accuracy of self-report, insight and memory (Heinrichs & Zakzanis, 1998), so individuals with psychotic symptoms were excluded based on *DSM-IV* criteria. However, individuals on psychotropic medications were included even if these medications were prescribed to treat psychotic symptoms. Inclusion of individuals with Axis I disorders and on psychotropic medications reflects the general population of substance users, which increases generalizability to other populations of treatment-seeking substance users, and thereby increasing external validity. Additionally, Axis I disorders are controlled for in the analyses as appropriate.

Another consideration was measurement of psychopathy. Two measures, the Psychopathy Checklist – Revised (PCL; Hare, 1991) and the Psychopathic Personality Inventory (PPI; Lilienfield & Andrews, 1996), have been used extensively in the literature (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003). The PCL-R is geared toward individuals with criminal histories, thus limiting internal validity of the measure

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in assessing "successful psychopaths." Finally, while the PPI is a continuous measure, the PCL-R results in a dichotomous assessment of "psychopath" or "non-psychopath", thus failing to capture individual differences in disorder severity and other features of psychopathy that are not taken into account when an individual's score falls below the PCL-R threshold. Due to the above-mentioned deficiencies in the PCL-R and also due to collateral file data required to use the PCL-R, we decided to use the PPI, which has established adequate validity (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003).

Finally, it was necessary to address factors that would drastically affect HPA axis function. First, to address the possibility that participants were experiencing varying levels of stress, we chose to conduct a Progressive Muscle Relaxation (PMR) exercise prior to providing the first saliva sample. PMR has been shown to decrease levels of stress through proper relaxation and breathing techniques (Carlson, Bacaseta, & Simanton, 1988). Second, we chose the method of passive drool as opposed to cotton Salivette devices because the passive drool method has been shown to provide more accurate measures of salivary cortisol (Granger, Harmon, Hibel, & Rumyantseva, 2006). Also, we considered how to address the effect of smoking on cortisol. Smoking has been shown to have immediate effects on cortisol levels (Steptoe & Ussher, 2006). Previous studies at this facility revealed a high level of smoking among residents. We decided to incorporate a smoking questionnaire which addresses the quantity of cigarettes smoked on the day of the assessment as well as when participants had their last cigarette. We controlled for smoking quantity and the time since last cigarette in the analyses as appropriate. Additional potential confounds in measuring HPA axis function were minimized by the way that it was operationalized in the present study. That is, HPA axis

function was characterized as the *level of reactivity* to the distress tolerance tasks.

Therefore, variance in the baseline levels in and of themselves will be less of a confound.

Also, corticosteroids are known to affect cortisol levels (de Kloet & Reul, 1987), so

individuals taking these will be excluded from the study. Finally, in order to avoid the

potential confounding effects of daily fluctuations in cortisol levels, we collected data in

the evening, when evidence suggests that daily fluctuations have asymptoted.

The measures are categorized into four domains: (1) screening and diagnostic

assessment (which includes demographics and medications), (2) substance use, (3)

Emotionality/HPA Axis functioning including behavioral and biological measures, and

(4) potential covariates.

Domain		Measure	Purpose
Screening and Diagnostic Assessment		Demographic Information	Information on age, race, education, marital status, and income
		Psychopathic Personality Inventory	Assessment of psychopathic traits
		Structrued Clinical Interview for DSM-IV (SCID-IV	Semi-structured interview designed to diagnose psychiatric disorders: Major Depression, Bipolar, Panic Episode, Alcohol and Substance Dependence, Anxiety, and Psychotic Disorders modules will be administered
		Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV)	Semi-structure interview to assess Antisocial and Borderline Personality Disorder
Emotionality and HPA Axis Function	Behavioral	Paced Auditory Serial Addition Task	Laboratory Challenge Tasks – Distress Tolerance
		Mirror Tracing – Computerized Version	
	Biological	Salivary Cortisol	An index of HPA activity in response to challenge tasks
Potential Covariates		Smoking Questionnaire	Last use of nicotine and quantity

<u>Screening and Diagnostic Assessments</u>

Demographic Information. Information was obtained regarding age, race, education, marital status, and income.

Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996). The PPI is a 187-item, self report measure designed to assess the primary personality traits of psychopathy as described by Cleckley (1941, 1988). This self-report measure has demonstrated high internal consistency (Cronbach's α = .89-.93).

The PPI yields a total score, which is interpreted as a global index of psychopathy, and scores on eight subscales, which reflect traits of impulsive nonconformity, blame externalization, Machiavellian egocentricity, carefree nonplanfulness, stress immunity, social potency, fearlessness, and coldheartedness. The PPI also contains validity scales intended to detect response styles of impression management, malingering, and random responding (i.e., Unlikely Virtues and Deviant Responding subscales). The PPI and PCL-R correlate at r = .54, suggesting a moderately high correlation between self-report and interview-based measures of psychopathy (Poythress, Edens, & Lilienfeld, 1998). For this study, we used the PPI total score to reflect the level of psychopathic traits.

Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1997). The SCID-IV was administered to assess for current Axis-I psychopathology. Specifically, the following disorders were assessed: Major Depressive Disorder, Bipolar I and II Disorders, Generalized Anxiety Disorder, Panic Disorder, Specific Phobia, Post-Traumatic Stress Disorder, Social Phobia, and Alcohol, Cocaine, Cannabis, Opioid, and Hallucinogen Dependence. Additionally, we screened for

psychotic disorders. Interviews were conducted by trained research assistants. Individuals who met criteria for psychosis were excluded as this may have affected their responses on the self report measures and performance on the challenge procedures.

Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV; Zanarini, Frankenburg, Sickel, & Yong, 1996). The DIPD-IV (Zanarini, Frankenburg, Sickel, & Yong, 1996) assesses 12 personality disorders including the 10 included in the DSM-IV as well as depressive and passive personality disorders. For this study, we used the DIPD-IV to assess for antisocial and borderline personality disorders. This interview has been found to compare favorably to the Structured Clinical Interview for DSM-III, with interrater coefficients ranging from .52 to 1.0 and test-retest reliability coefficients ranging from .46 to .85 (Zanarini, Frankenburg, Chauncey, & Gunderson, 1987).

Emotionality and HPA Axis Functioning

Behavioral Measures

Paced Auditory Serial Addition Task (PASAT-C). A modified computerized version of the PASAT (PASAT-C; Lejuez, Kahler, & Brown, 2003) was used as a psychological stressor in order to assess distress tolerance and HPA Axis response to distress.

In this task, numbers are sequentially flashed on a computer screen, and participants are asked to add the presented number to the previously presented number before the subsequent number appears on the screen. As the task was designed to limit the role of mathematical skill in persistence, the presented numbers only range from 0 to 20, with no sum greater than 20. Participants provided answers by using the mouse to click on their selection using pad displayed on the screen. Participants were told that their

score increases by one point with each correct answer and that incorrect answers or omissions will not affect their total score. The task consists of three levels with varying latencies between number presentations. Specifically, the first level of the PASAT provides a 3-s latency between number presentations (i.e., low difficulty), a 2-s latency during the second level (i.e., medium difficulty), and a 1-s latency during the final level (i.e., high difficulty). The first level lasts for 3 min and the second level lasts for 5 min. Following a 2-min brief rest period, the final level continues for up to 5 min, with the subject having a termination option. Specifically, participants were informed that once the final level began they could terminate exposure to the task at any time by pressing any button on the keyboard; however, they were told that the amount of money they would make at the end of the session depended upon their performance on the task.

Distress tolerance was indexed as latency to task termination. The experimental administration of a dysphoria scale (see below) occurs at the end of the second level of the PASAT to determine if the task increased psychological stress. This second administration occured at the end of second level of the PASAT as opposed to the end of the task to prevent confounds associated with termination latency.

Computerized Mirror-Tracing Persistence Task (MTPT-C; Strong, Lejuez, Daughters, Marinello, Kahler, & Brown, 2003). As a computerized version of the Mirror Tracing Persistence Task (MTPT; Quinn, Brandon, & Copeland, 1996), we used the MTPT-C. In this task, participants are required to trace a red dot along the lines of a star using the computer's mouse. To make the task similar to the original mirror tracing task, the mouse is programmed to move the red dot in the reverse direction. For example, if the participant moves the mouse to the left, the red dot moves to the right. To increase the

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difficulty level and frustration, if the participant moves the red dot outside of the lines of the star or if the participant stalls for more than two seconds, a loud buzzing noise sounds and the red dot returns to the starting position. Participants were told that they could end the task at any time by pressing any key on the computer keyboard, but that their performance on the task affected how much money they would earn. After receiving these instructions, the participants began the task and worked independently until the five-minute maximum time, at which point the task was terminated. The participants were not told the maximum duration that the task would last prior to beginning the task. Psychological distress tolerance was measured as latency in seconds to task termination. As an additional index of distress tolerance, the number of tasks that participants quit (i.e., quit neither, one, or both tasks) was also used.

Dysphoria. In line with previous studies using the distress tolerance tasks (Brown et al., 2002; Daughters, Lejuez, Bornovalova, et al., 2005b), we measured dysphoria using a four-item scale consisting of self-reported anxiety, difficulty concentrating, irritability, and frustration, with each item independently rated on a ten- point Likert scale on the PASAT and a 100-point scale on the mirror tracing task, with a total score derived by summing the score on each item. Reliability of this dysphoria scale has been demonstrated as acceptable ($\alpha = .77$). A baseline administration of the scale occurred at the start of the session and an experimental administration was administered after the second level of the PASAT. Because the MTPT-C only includes a single level, dysphoria was not assessed due to confounds of termination latency.

Biological Measures

Salivary Cortisol. Salivary cortisol was collected from each participant at five

time points. The first occurred immediately prior to the behavioral. The additional

collections occurred immediately after, 10, 20, and 30 minutes after the second task.

Together, these points provide information on: a) baseline cortisol/HPA functioning, b)

peak HPA reactivity, and c) latency (and ability) to return to baseline cortisol levels,

respectively.

Saliva was collected using the passive drool method[7]. In order to stimulate saliva

flow, participants were instructed to think of a favorite food. Participants allowed saliva

to collect under the tongue, and passed the saliva through a straw into a tube. The tubes

were sent for radioimmunoassay analyses.

We used Tai's (1994) Area Under the Curve method of calculating cortisol

reactivity to the distress tolerance tasks. Specifically, it is calculated by dividing the

abscissas into small rectangles whose areas are calculated from their geometric formulas.

The sum of these areas represents the total cortisol reactivity. This method of calculating

various metabolic and other biological indices of reactivity has numerous advantages

over traditional methods of calculating reactivity including better precision (Tai, 1994).

Potential Covariates

Smoking Measure. To control for the immediate effect of smoking on cortisol

levels (Canals, Colomina, Domingo, & Dominech, 1997; Steptoe & Ussher, 2006),

participants were asked when they smoked their last cigarette as well as the number

smoked on the last occasion.

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[7] For further discussion of the passive drool method, see Design Considerations

Chapter 3: Results

Descriptive Data. The sample consisted of 76 males and 15 females. The mean

age was 39 years (*SD* = 8.31). Sixty-one percent of the sample earned less than $20,000

annually. The majority of the sample identified as Black or African-American (88%). The

remaining racial/ethnic composition of the sample was as follows: 7.7% Caucasian, 1.1%

Hispanic/Latino, and 3.3% reported "other." Also, 65.9% of the sample earned a high

school degree or higher. A total of 27 individuals (29.7%) met criteria for ASPD. With

regard to mood and anxiety disorders, 4.7% of the sample met criteria for Bipolar

Disorder I, 1.2% for Bipolar II disorder, 22.6% for Major Depressive Disorder, 4.7% for

Panic Disorder, 1.2% for Social Phobia, 5.9% for Specific Phobia, 2.4% for Obsessive

Compulsive Disorder, 18.8% for Post-Traumatic Stress Disorder, 9.4% for Generalized

Anxiety Disorder, and 26.2% for Borderline Personality Disorder. With respect to

Substance Use Disorders, the most prevalent was Cocaine Use (51.8%), followed by

Alcohol Use (23.8%), Opioid Use (20.2%), then Hallucinogen Use (12.9%), then

Cannabis Use (9.4%). These results are presented in Table 1.

Demographic characteristics and mood disorders, anxiety disorders, and BPD

diagnoses for ASPD and non-ASPD groups. Comparisons of demographic characteristics

and psychiatric diagnoses were made between individuals who did and did not meet

criteria for ASPD. The results are presented in Table 1. Those with who met criteria for

ASPD did not significantly differ on any demographic variables. With respect to

psychiatric diagnoses, the two groups did not differ with respect to Major Depressive

Disorder, Bipolar I Disorder, Bipolar II Disorder, Social Phobia, Specific Phobia,

Obsessive Compulsive Disorder, Post Traumatic Stress Disorder, Generalized Anxiety

Disorder, or Borderline Personality Disorder. However, significantly more individuals with ASPD met criteria for Panic Disorder (13.3%) than those without an ASPD diagnosis (1.4%).

Substance Use Comparisons for ASPD and non-ASPD groups. Comparisons of DSM-IV current substance use dependence diagnoses were made between individuals who did and did not meet criteria for ASPD. The two groups did not differ with respect to diagnoses of Opioid, or Cannabis Use Dependence. However, significantly more individuals with ASPD met criteria for Alcohol (43.3%) and Cocaine Use Dependence (66.7%) than those without ASPD (13.9% and 45.2%, respectively). These results are presented in Table 1.

Relationship of psychopathic traits to demographic characteristics and mood disorders, anxiety disorders, and BPD diagnoses. A Pearson correlation was calculated to examine the relation of age to total PPI score and the following PPI subscales: Machiavellian Egocentricity, Social Potency, Fearlessness, Coldheartedness, Impulsive Non-Conformity, Alienation, Carefree Nonplanfulness, Stress Immunity, Deviant Responding, and the MPQ lie scale. Point biserial correlations were calculated to examine the relation of the remaining demographic variables and the DSM-IV diagnoses to the PPI subscales. Age was significantly negatively correlated with the PPI total score and Social Potency. Higher income was associated with higher Social Potency, Cold Heartedness, Stress Immunity, and lower Alienation scores. Higher level of educational attainment was associated with higher scores on the MPQ lie scale. Black/African-American ethnicity was associated with lower Fearlessness and higher Impulsive Nonconformity. Major Depressive Disorder diagnoses were associated with higher PPI

total scores, Machiavellian Egocentricity, Alienation, and Carefree Nonplanfulness. PTSD diagnoses were associated with higher Machiavellian Egocentricity and lower MPQ lie scores. Finally, BPD diagnoses were associated with higher Alienation and lower Stress Immunity. These results and the means and standard deviations for the PPI subscales are presented in Table 2.

Relationships between substance use diagnoses and psychopathic traits. Point biserial correlations were calculated to examine the relationship between psychopathic traits and current substance use dependence for each drug class. Alcohol dependence was related to higher PPI total scores higher Carefree Nonplanfulness scores, and lower scores on the MPQ lie scale. Cocaine dependence was related to lower Social Potency and Stress Immunity. There were no other significant associations. The results are presented in Table 3.

Distress tolerance tasks. Overall, participants persisted for an average of 201.79s (SD = 122.75) on the PASAT-C and 45% quit the task. With regard to MTPT-C, individuals persisted for an average of 189.88 (SD = 109.65 s) and 67% quit the task. Paired t-tests indicated a significant increase in dysphoria at the experimental administration of the dysphoria scale for the PASAT [$t(85)$ = -5.87, $p < .001$] and MT [t(87) = -5.94, p < .001], suggesting that the tasks were psychologically stressful. The two measures of psychological distress tolerance, PASAT-C and MTPT-C were significantly correlated, (r = .27, p <.01). Given issues related to skew, the DT score for each task was dichotomized as quit or no-quit. To simplify analyses as we have done in previous published work, we also utilized a combined variable (Daughters et al., 2005; in press). Using the dichotomous scores, we computed a composite distress tolerance variable,

indicating whether the participant quit neither, one or both tasks (i.e., 0, 1, 2). 25.3% of

participants quit neither task, 36.3% quit one task, and 38.5% quit both tasks. Skill level

was not related to quitting either task.

In order to examine the relationship between ASPD diagnosis and quitting the DT

tasks, a chi-square analysis was conducted. The relationship was not significant. These

results are presented in Table 4.

Cortisol reactivity (AUC). Nineteen participants declined provision of saliva

samples. Furthermore, there were two statistical outliers with respect to AUC (i.e.,

greater than 2.5 *SD*s above the mean), therefore, their scores were removed from the

analyses that involve AUC. Thus, all subsequent AUC analyses were conducted on 71

participants with a mean AUC of 0.46 μg/dL (*SD*=.30).

Relation of ASPD, distress tolerance and cortisol reactivity to demographic

characteristics, current mood disorders, anxiety disorders, and BPD. Point biserial

correlations were calculated to examine the relation of ASPD diagnoses, DT (using

dichotomous quit/no quit variables and the DT score [number of tasks quit]), and cortisol

reactivity (AUC) to demographic characteristics, current mood and anxiety disorders, and

BPD. ASPD was associated with Panic Disorder. Also, quitting the MTPT-C was

significantly associated with higher age. There were no other significant relationships, all

p's >.05. These results are presented in Table 5.

Relation of ASPD diagnosis, DT, and cortisol reactivity, to substance use dependence.

Point biserial correlations were computed to examine the relation of ASPD diagnosis, DT

(dichotomous variable and DT score), and AUC to substance use dependence. ASPD was

associated with both alcohol and cocaine dependence. Quitting more tasks was associated with

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opioid dependence. There were no other significant relationships. These results are presented in Table 6.

       Correlation between HPA Axis functioning and possible covariates for cortisol levels. There were no significant correlations between AUC and time since the participant's last cigarette or the number of cigarettes smoked, therefore these variables was not used as covariates in subsequent analyses involving AUC. These results are presented in Table 7.

Predictors of Distress Tolerance

       Unique contributions of ASPD and psychopathic traits to DT. To test the unique contributions of ASPD diagnosis and CU traits in predicting distress tolerance, we conducted multiple regression analyses in order to examine the effects of ASPD and PPI scores on the DT composite score. Opioid use was entered as a covariate in the first step. We entered ASPD diagnosis in the second step and PPI into the third step. The overall model was significant, $F(3,84)= 4.26$, $p<.01$, accounting for 13.8% of the variance in the distress tolerance score. The first step indicated that ASPD alone was not significant when predicting DT scores; however, the third step indicated that the unique contributions of ASPD and PPI score were significant and indicated that ASPD diagnosis was associated with quitting a greater number of tasks, while a high PPI score was associated with quitting a fewer number of tasks. The same general pattern of results emerges when examining the DT tasks separately, but with less robust associations. The results of this analysis are presented in Table 8.

       We explored psychopathic traits as a possible suppressor variable in the relationship between ASPD and DT. According to the three variate case of suppression delineated by Conger (1974), there are three conditions that must be met in order to

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determine a case of suppression. First, the predictor and the suppressor should be significantly correlated. Second, the standardized beta coefficient of the predictor should be greater than the correlation between the predictor and the criterion variable. Third, $1-((r_{predictor, criterion})/ r_{suppressor, criterion})) > 1- (r_{predictor, suppressor})^2$. The relationships among ASPD, psychopathic traits, and DT meet these criteria. ASPD and PPI are significantly correlated ($r = .24$, $p <.05$). The standardized beta coefficient of ASPD ($\beta^* = .23$) is greater than the correlation between ASPD and the DT score ($r = .17$). The final condition was also met; therefore, psychopathic traits qualify as a suppressor variable in the relationship between ASPD and psychopathic traits. The implications of this finding are addressed in the discussion section.

Post-hoc analyses of the unique contributions of the PPI subscales in predicting DT. In order to further examine specific dimensions of psychopathic traits that are driving the relationship with DT, bivariate correlations were conducted between DT scores and each of the PPI subscales. These are presented in Table 9. Higher Fearlessness and scores were associated with higher DT scores. There were no other significant correlations. Also, in order to examine the independent contribution of each subscale in predicting DT, a multiple regression analysis was conducted. ASPD was entered into the first step and each of the eight PPI subscales were entered into the second step. The Fearlessness subscale was the only subscale to significantly predict DT scores ($\beta = -.03$, $SE = .01$, $p < .05$). These results are presented in Table 10.

Unique contributions of ASPD and psychopathic traits to cortisol reactivity. To test the unique contributions of ASPD diagnosis and CU traits in predicting cortisol reactivity, we conducted a regression analysis with ASPD diagnosis and PPI score

predicting AUC. The overall model was not significant, $F(2,68) = 1.80$, $p = .17$; however, the unique contribution of ASPD to the model approached significance ($p = .08$), indicating a trend for AUC being negatively related to AUC when controlling for psychopathic traits. The results of this analysis are presented in Table 11.

    Post-hoc analyses of cortisol response to DT tasks. We conducted repeated measures analyses of variance (ANOVAs) to explore differences in patterns of cortisol levels across the five time points (within group variables: pre-, post-, 10 min post-, 20 min post-, and 30 min post-task) between ASPD (n = 23) and non-ASPD (n = 48) individuals (controlling for psychopathic traits). No interaction was present. There was a significant main effect of time (F (1,68) =3.35, p<.05), such that cortisol decreased across each time point, and the between-group effect approached significance (F (1,68) = 3.21, p = .08); no interaction was present. The trend suggested that individuals with ASPD demonstrated lower cortisol levels regardless of time point. Average cortisol levels across time points for ASPD and non-ASPD individuals are presented in Figure 1.

Chapter 4: Discussion

Previous research indicated that individuals with ASPD may exhibit lower distress tolerance than those without ASPD (Daughters, Sargeant, Gratz, Bornovalova, & Lejuez, in press*).* This finding indicates that individuals with ASPD may have an inability to tolerate emotional distress while pursuing goal-directed behavior. This is consistent with the DSM-IV conceptualization of individuals with ASPD as having difficulty tolerating frustration. However, one limitation of this previous study is that psychopathic traits, which often co-occur with ASPD, were not considered, although such traits have consistently been associated with a lack of emotionality. This hypo-emotionality has been demonstrated in studies that found reduced or an absence of a startle reflex potentiation during exposure to threatening stimuli (Patrick, 1994; Patrick, Bradley, & Lang, 1993). Additionally, Patrick (1994) found that psychopaths had reduced blink magnitude in anticipation of a noxious noise, but antisocial individuals with moderate levels of psychopathy demonstrated blink magnitudes similar to non-psychopathic controls. These findings indicate that it is possible that individuals with ASPD and psychopathy may have different emotional profiles than those with ASPD without psychopathy. Therefore, the current study sought to consider the role of psychopathic traits in the relationship between ASPD and DT.

Substance use treatment-seeking individuals were administered two aversive laboratory tasks in order to assess their ability to tolerate distress. In an attempt to replicate the previous finding, it was hypothesized that ASPD would be associated with lower levels of distress tolerance. Further, as an extension of the previous finding, we hypothesized that this effect would be strengthened by considering the effects of

psychopathic traits on this relationship, whereby psychopathic traits would be associated with higher DT. When using the DT composite, significant findings emerged, as ASPD and psychopathic traits were related to DT in opposite and hypothesized directions, accounting for 11% of the variance in DT. Thus, ASPD was related to lower DT and psychopathic traits to higher DT. Further analyses revealed that the fearlessness dimension of psychopathy drove the relationship between psychopathic traits and DT. This is consistent with the idea that lack of fear is a core facet of psychopathy contributing to hypo-emotionality.

Psychopathic traits acted as a suppressor variable in the relationship between ASPD and DT. Therefore, when not included in the prediction of DT, the variance in ASPD accounted for by psychopathic traits suppresses the criterion-relevant part of variance accounted for by ASPD. To this end, the effect of ASPD on DT is partially blurred by criterion-irrelevant variance accounted for by psychopathic traits; however, psychopathic traits are not irrelevant. Their contribution represents a relationship of theoretical significance. Specifically, when psychopathic personality traits in some individuals with ASPD (which were hypothesized to be positively related to DT) are considered, more variance in DT is accounted for. This leaves variance unique to ASPD and not shared with psychopathic traits in predicting DT.

These findings suggest that individuals with ASPD and lower levels of psychopathic traits may regulate distress differently than those with higher levels of psychopathic traits. Indeed, extant literature on emotional responding in psychopaths has examined mechanisms at the biological/physiological level and has found consistent evidence of hypo-emotionality. Nevertheless, little is known about potential

biological/physiological processes that may be associated with coping with negative affect in individuals with ASPD. To examine this possibility, we examined the HPA axis functioning (as indexed by cortisol reactivity) as a biological indicator of distress during the stressor tasks.

It was hypothesized that ASPD would be associated with a high stress response and that psychopathic traits would be associated with a low stress response. However, the findings in this regard were complex. Indeed, there was an unexpected decrease in cortisol levels during the DT tasks. Specifically, exploratory analyses indicated that there was great variability in cortisol response in that the majority of the sample's salivary cortisol actually decreased from pre-task to post-task (70%), while others increased. This could be attributable to evidence that cocaine addicts with ASPD exhibited decreased cortisol response to stress (Buydens & Branchey, 2004).

Alternatively, this could be attributable to the physiological effects of chronic drug use and alcohol use, as prolonged alcohol use has been shown to be associated with blunted pituitary adrenal response to stressors (Rivier, Imaki, & Vale, 1990). Furthermore, chronic cocaine use has been associated with dysregulated HPA axis functioning even weeks after detoxification (Zorilla, Valdez, & Weiss, 2001). Persistent cocaine use results in depleted corticotropin releasing factor (CRF) and thus cortisol responses to stress become blunted (Zorilla, Valdez, & Weiss, 2000). Therefore, the capacity to handle stress becomes compromised. Although contrary to the hypotheses, the findings regarding decreased salivary cortisol across time points are not surprising.

Prior work on HPA axis functioning indicates that less cortisol response may indicate dysregulated biological emotional response and thus may affect one's response

to stress (Derijk, van Leeuwen, Klok, & Zitman, 2008). Nevertheless, the question still remains as to *how much* of a response is adaptive. Of course, a more parsimonious explanation for these findings is that salivary cortisol is not sensitive to the effects of a laboratory stressor task and that it would take a more intense stressor to elicit a cortisol response and the decrease in cortisol levels could merely reflect the natural decrease in cortisol levels over the course of the day. Moreover, given the high rates of the occurrence of traumatic events in this sample may have rendered the lab stressor less potent. Nevertheless, self-reports of increased negative affect in response to the task indicate that this is probably not the case.

The negative association between cortisol level and ASPD approached significance and suggested a trend toward individuals with ASPD having lower cortisol levels than those without ASPD. This was contrary to the hypothesized idea that ASPD would be associated with hyper-reactivity in the form of increased reactivity. This finding may be consistent with the stimulation-seeking theory (Zuckerman, 1979), wherein it is thought that individuals with externalizing disorders are physiologically under-aroused and that their externalizing behavior may serve as a means to seek optimal arousal. Furthermore, reduced arousal has been found in children with externalizing disorders (Shirtcliff, Granger, Booth, & Johnson, 2005; Snoek, Van Goozen, Matthys, Buitelaar, & Van Engeland, 2004; Vanyukov, Moss, Plail, Blackson, & et al., 1993). Despite these findings, some research examining the relationship between cortisol levels and behavior problems in children have generated mixed findings (Granger, Weisz, & Kauneckis, 1994; Shirtcliff, Granger, Booth, & Johnson, 2005).

Overall, there is great inconsistency in the HPA axis literature as to how best to

conceptualize cortisol stress response. Some would purport that it is normative for cortisol levels to increase in response to a stressor; however, it is unclear *how much* of an increase is normative. Also, cortisol reactivity has been conceptualized in another study as *change* cortisol levels (Sinha, Garcia, Paliwal, Kreek, & Rounsaville, 2006), without regard to whether that change was negative or positive. It is also difficult to control for cortisol levels that were not able to fluctuate due to chronically already being high at the beginning of the experiment.

Limitations

There were several limitations which should be noted. First, there may have been a selection bias in this study, as it is possible that individuals who chose not to participate or who started but did not complete the battery were characteristically different than those who completed the study. Given that the main construct of interest is DT, it is possible that the range of DT scores was restricted to those with relatively higher distress tolerance. The full battery was extensive, taking approximately three hours to complete. Another limitation was the homogeneity with respect to ethnicity in this sample. Although inner-city African-Americans are an underrepresented population, more studies will be needed in more heterogeneous samples in order to establish external validity. Also, the use of a substance use treatment-seeking sample further limits the generalizability of these findings.

Second, there is a validity issue with respect to the DT tasks. First, it is not clear the extent to which the two DT tasks tap the same construct, as quitting one task only correlated modestly with quitting the other at $r = .31$; however, both tasks' relations to the variables of interest approached significance in the same way. Also, self-report of

increases in negative affect from pre- to post- task suggest that both tasks do indeed induce distress. More research is needed to further explicate this construct and its behavioral measurement.

Third, there were limitations associated with the measurement of salivary cortisol, which is known to be affected by cigarette smoking. Specifically, in habitual smokers, smoking can attenuate cortisol reactivity to stressors (Rohleder & Kirschbaum, 2006). Only 18% of participants who provided saliva samples were non-smokers and 86% of individuals had smoked cigarettes and hour or less prior to participation. While neither the number of cigarettes smoked nor time since last cigarette were related to AUC, it is possible that smoking nevertheless introduced additional variance and pre- to post-task variability was affected. Additionally, the pattern of decreased cortisol levels across time points could have been attributable to small withdrawal effects during the course of the experiment could have also decreased cortisol levels (Steptoe & Ussher, 2006). Future studies, should consider other indices of HPA axis functioning that are not as vulnerable to the effects of smoking, such as corticotropin releasing hormone (Rohleder & Kirschbaum, 2005).

Fourth, the current study did not distinguish between psychopathic traits associated with callous and unemotional traits and those associated with social deviance. Recent studies have identified the PPI subscales associated with the traditional conceptualizations of PCL Factor 1 (interpersonal and emotional deficits) and Factor 2 (social deviance) (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Patrick, Edens, Poythress, Lilienfeld, & Benning, 2006). Therefore, this may have introduced redundancy in the measures of ASPD, which focuses on social deviance, and the PPI

which is thought to measure both social deviance and interpersonal/emotional deficits.

Finally, although attempts were made to accommodate individuals with reading difficulties, the low education level of this sample, coupled with possible effects of long-term substance use, may have led to lack of insight into their own behavior. This problem was compounded by the cultural irrelevance of some of the self-report items. Anecdotally, some participants stated that some of the items on the PPI were not applicable to inner city, low SES African-Americans or to individuals with severe substance use histories. Additionally, some of the questions were more applicable to older cohorts than to younger cohorts. All of these issues pose a significant threat to internal validity. Despite these potential barriers, the PPI demonstrated high internal consistency.

Conclusions and Future Directions

Evidence from the current study may highlight a distinct subset of individuals with ASPD (those with high levels of psychopathic traits) who may exhibit a different behavioral and emotional profile than individuals with ASPD who do not have psychopathic traits. Specifically, when considering ASPD and psychopathic traits together, there was evidence that ASPD was associated with low DT, while psychopathic traits were associated with high DT. Findings with cortisol, however, were less clear and require further development to understand their implications for ASPD and psychopathic traits and how they tie back to the behavioral measures. Future studies of individuals with ASPD should consider the impact psychopathic traits on emotion-related outcomes.

This study may be relevant when examining etiological factors associated with

ASPD. This study also leads to several more basic and clinically relevant lines. Although clinical lore maintains that ASPD and psychopathic traits are untreatable, Salekin's review of 42 psychopathy treatment studies (2002) suggested that there is little support for this idea. Instead, evidence suggested that current treatments are not well informed by basic research. Moreover, treatments have targeted other disorders related to ASPD and psychopathic traits and not known underlying mechanisms responsible for the clinical manifestation of the disorder. For example, many of the studies on ASPD are conducted in substance dependent, treatment-seeking samples. Therefore, the treatments target only substance use, not the reduction of other ASPD-related behavior. Given that low DT has been found to be related to substance use treatment dropout (Daughters, Lejuez, Bornovalova, Kahler, Strong, & Brown, 2005), perhaps incorporating treatment components designed to address tolerance of negative affect could be a viable avenue to pursue in substance abusing individuals with ASPD and low levels of psychopathic traits.

Tables

Table 1

Demographics, Axis I disorders, and BPD for overall sample, ASPD, and non-ASPD participants.

	Overall Sample	ASPD (*n* = 27)	No ASPD (*n* = 64)	Statistic
Demographics				
Mean Age (SD)	39	37.41	39.63	$t(89) = 1.17, p = .25$
	(8.31)	(8.94)	(8.02)	
Income (% > 20K)	58.2%	55.6%	59.4%	$\chi^2(1) = .11, p = .74$
Ethnicity (% African American)	87.9%	81.5%	90.6%	$\chi^2(1) = 1.49, p = .22$
Education (% High School Graduate)	65.9%	51.9%	71.9%	$\chi^2(1) = 3.39, p = .07$
Axis-I Disorders				
Major Depressive Disorder	22.6%	25.9%	21.1%	$\chi^2(1) = .25, p = .62$
Bipolar I Disorder	4.7%	7.4%	3.4%	$\chi^2(1) = .64, p = .42$
Bipolar II Disorder	1.2%	0%	1.7%	$\chi^2(1) = .47, p = .49$
Social Phobia	1.2%	7.4%	12.4%	$\chi^2(1) = .93, p = .63$
Specific Phobia	5.9%	7.4%	5.2%	$\chi^2(1) = .17, p = .68$
Obsessive Compulsive Disorder	2.4%	3.7%	1.7%	$\chi^2(1) = .31, p = .56$
Post-Traumatic Stress Disorder	18.8%	29.6%	13.8%	$\chi^2(1) = 3.02, p = .08$
Generalized Anxiety Disorder	9.4%	14.8%	6.9%	$\chi^2(1) = 1.36, p = .24$
Panic Disorder*	4.7%	14.8%	0%	$\chi^2(1) = 9.02, p < .01$
Borderline Personality Disorder	26.2%	33.3%	22.8%	$\chi^2(1) = 4.05, p = .13$
Substance Dependence				
Alcohol*	23.8%	44.4%	14.0%	$\chi^2(1) = 9.34, p < .01$
Cocaine*	51.8%	70.4%	43.14%	$\chi^2(1) = 5.49, p < .05$
Opioid	20.2%	26.9%	17.2%	$\chi^2(1) = 1.04, p = .31$
Cannabis	9.4%	11.1%	8.6%	$\chi^2(1) = .13, p = .71$
Hallucinogen	12.9%	7.4%	15.5%	$\chi^2(1) = 1.08, p = .30$

*p<.05, **p<.01

Table 2

Means and standard deviations for PPI scales and correlations between PPI scales and demographics, mood and anxiety disorders, and BPD.

	Mean (SD)	Gen	Age	Inc	Educ	Ethn	MDD	Bipo 1	Bipo 2	GAD	Soc Phob	OCD	PTSD	Spec Phob	Panic	BPD
Psychopathic Traits																
PPI-Total	443.90 (39.60)	.04	**-.25***	.13	-.01	-.13	**.39****	.21	-.03	.07	.01	-.05	.19	-.11	.03	.14
PPI Subscales																
PPI-ME	70.79 (13.06)	-.05	-.18	.02	-.01	.09	**.29****	.07	-.04	.04	.08	.11	**.26***	-.05	.05	.08
PPI-SP	66.30 (8.82)	.02	**-.38****	**.37****	.10	.06	-.04	.07	-.04	-.10	-.15	.00	-.13	-.05	-.08	-.01
PPI-F	49.46 (10.11)	.09	-.18	.09	.02	**-.31****	.21	.19	-.05	.02	.08	.11	.10	-.08	.10	.11
PPI-CH	44.95 (9.70)	.01	.08	**.24***	.12	.05	.10	.06	.08	-.04	-.20	-.09	-.08	.01	-.04	-.09
PPI-IN	38.16 (8.15)	.02	-.12	-.01	-.03	**.31****	.21	.21	-.08	.13	.10	.02	.17	-.13	-.09	.10
PPI-A	43.77 (9.28)	-.04	-.03	**-.23***	-.19	-.04	**.29****	-.11	-.08	.18	.10	.06	.16	.06	.10	**.33****
PPI-CN	39.52 (8.66)	.01	.03	-.17	-.08	-.06	**.30***	.18	.11	.14	.04	-.05	.13	-.11	.08	.08
PPI-SI	30.00 (5.39)	.15	-.17	**.28****	.16	.06	-.14	.08	.02	-.20	-.18	-.10	-.07	-.04	-.09	**-.24***
PPI-DR	24.77 (3.85)	-.10	.04	-.04	-.06	-.09	.16	.02	-.05	.09	.08	.18	.08	.01	.12	.16
PPI-MPQ	34.90 (5.30)	.04	**.21***	-.02	**-.21***	.05	-.17	-.12	.02	-.13	-.02	-.00	**-.26***	-.05	.07	.00

*p<.05, **p<.01

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Table 3

Relationships between substance use diagnoses and psychopathic traits.

	Alcohol	Cocaine	Cannabis	Opioid	PCP/Halluc
Psychopathic Traits					
PPI-Total	**.35****	-.06	-.10	.06	.02
PPI Subscales					
PPI-ME	.19	.02	-.08	.10	.03
PPI-SP	.10	**-.27***	-.01	-.10	.18
PPI-F	.20	-.07	-.14	-.04	-.07
PPI-CH	.12	-.02	.13	.13	-.07
PPI-IN	.21	-.02	-.06	-.03	-.03
PPI-A	.12	.08	-.15	-.04	.16
PPI-CN	**.28****	.20	.00	.14	-.15
PPI-SI	-.06	**-.25***	-.03	.01	-.01
PPI-DR	.12	.13	.08	.01	-.02
PPI-MPQ	**-.35****	-.09	-.06	.03	-.11

*p<.05, **p<.01

Table 4

Relationship between ASPD diagnosis and quitting DT tasks.

Number of Tasks Quit	Overall Sample	ASPD (*n* = 27)	No ASPD (*n* = 64)	Statistic
				$\chi^2(2) = .2.95, p = .23$
0	25.3%	18.5%	28.1%	
1	36.3%	29.6%	39.1%	
2	38.5%	51.9%	32.8%	

*p<.05, **p<.01

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Table 5

Relation of ASPD, distress tolerance and cortisol reactivity to demographic characteristics and mood disorders, anxiety disorder, and

BPD.

	Gen	Age	Income	Educ	Ethn	MDD	Bipo 1	Bipo 2	GAD	Soc Phob	OCD	PTSD	Spec Phob	Panic	BPD
ASPD	.03	-.12	.04	-.19	-.13	.05	.08	-.07	.13	-.08	.06	.19	.04	**.33****	.18
Distress Tolerance															
PASAT-C	-.07	.07	-.05	-.14	.20	.02	.02	-.10	.12	.17	.02	-.01	.08	.02	-.04
MTPT-C	.00	**.27****	-.07	-.11	.10	-.16	-.19	.08	.15	-.02	-.05	-.10	-.14	.04	-.19
DT Score	-.08	.19	-.06	-.15	.19	-.09	-.10	-.02	.16	.09	-.02	-.07	.03	.04	-.13
HPA Axis Functioning															
AUC	.05	.15	-.09	.08	.07	-.10	-.01	.00	-.07	-.06	-.02	-.11	-.01	-.04	-.03

*p<.05, **p<.01

Table 6

Relation of ASPD diagnosis, DT, AUC, and psychopathic traits to substance use dependence.

	Alcohol	Cocaine	Cannabis	Opioid	PCP/Halluc
ASPD	**.33****	**.25***	.04	.11	-.11
Distress Tolerance					
PASAT-C	-.10	.06	.03	.21	.08
MTPT-C	-.18	.05	-.02	.18	-.09
DT Score	-.18	.05	.05	**.23***	.03
HPA Axis Functioning					
AUC	-.12	-.21	-.07	-.11	-.02

*p<.05, **p<.01

Table 7

Correlation between HPA Axis functioning and possible covariates for cortisol levels.

	Time Since Last Cigarette (in minutes)	Number of cigarettes Smoked
AUC	-.16	-.08

*p<.05, **p<.01

Table 8

Multiple regression analysis predicting number of tasks quit.

Variable	B	SE	sr^2
Step 1*			
Opioid Dependence*	.23	.11	.05
Step 2			
Opioid Dependence*	.21	.11	.05
ASPD	.25	.18	.02
Step 2**			
Opioid Dependence*	.22	.10	.05
ASPD Diagnosis*	.39	.19	.05
CU Traits*	-.01	.00	.06

*p<.05, **p<.01

Table 9

Correlations between DT scores and PPI subscales.

	DT score
Psychopathic Traits	
PPI-Total	-.19
PPI Subscales	
PPI-ME	.01
PPI-SP	-.16
PPI-F	**-.31****
PPI-CH	-.02
PPI-IN	-.19
PPI-A	-.04
PPI-CN	**.10**
PPI-SI	-.13
PPI-DR	-.12
PPI-MPQ	**-**.08

*p<.05, **p<.01

Table 10

Regression analyses of DT onto the PPI subscales controlling for ASPD diagnosis.

Variable	B	SE	sr^2
Step 1*			
ASPD	.29	.18	.03
Step 2			
ASPD	.33	.19	.03
PPI-ME	.01	.01	.00
PPI-SP	.01	.01	.00
PPI-F*	-.03	.01	.06
PPI-CH	-.01	.01	.01
PPI-IN	-.01	.02	.00
PPI-A	-.00	.01	.00
PPI-CN	.01	.01	.00
PPI-SI	-.02	.02	.00

*p<.05, **p<.01

Table 11

Regression analysis predicting AUC.

Variable	B	SE	sr^2
ASPD Diagnosis	-.14	.08	.04
PPI total	.00	.00	.00

*p<.05, **p<.01

Figures

Figure 1

Mean cortisol levels of ASPD versus non-ASPD groups across time points.
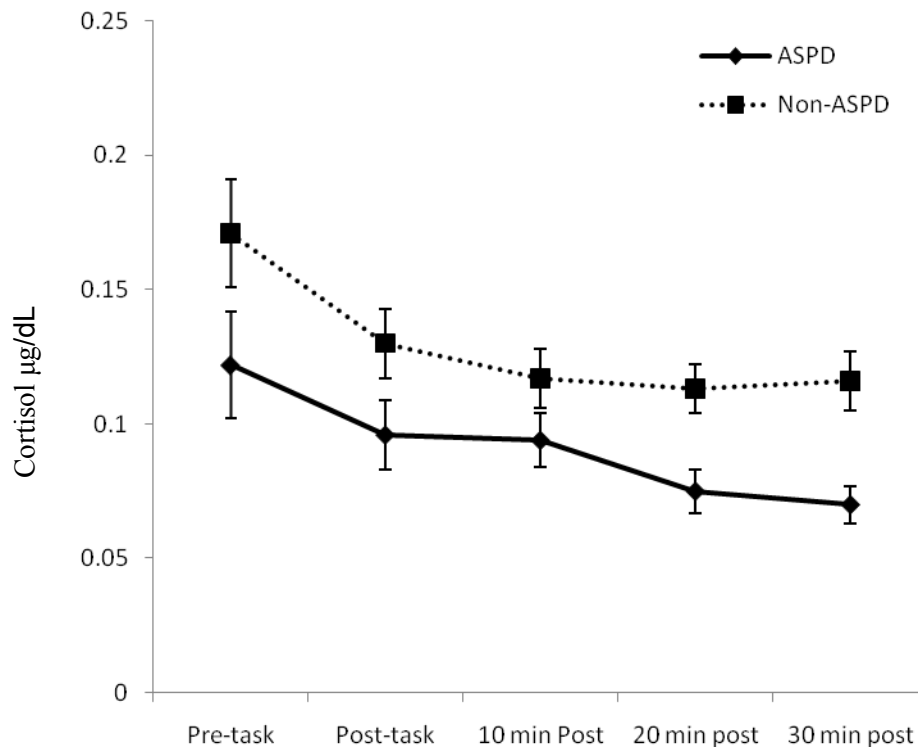
Appendix A

Comorbidity with Substance Use Disorders

Although the temporal precedence of the occurrence of ASPD and alcohol use disorders has not yet been established, research suggests that a common traits of disinhibitory psychopathology and deviance proneness are responsible for the relation between alcohol use disorders and ASPD (Sher & Trull, 1994). Others conjecture a common biological basis based on genetic-environmental factors (Van den Bree, Svikis, & Pickens, 1998).

Given the high comorbidity of ASPD with substance use disorders, and the negative individual and societal outcomes associated with the co-occurrence of these disorders, it is important to examine ASPD within the substance using population. Insight in this area may assist in understanding the mechanisms underlying ASPD that may be responsible for the poor substance use treatment outcomes.

Comorbidity – Other Disorders

Co-occurring ASPD and depression influence the course and severity of alcoholism and is associated with negative treatment outcome and higher likelihood of drug use. Moreover, comorbid ASPD with depression is associated with higher negative emotionality and lower constraint scores on the Multidimensional Personality Questionnaire (Holdcraft, Iacono, & McGue, 1998). Additionally, over 50% of individuals with ASPD present with lifetime comorbid anxiety disorder (Goodwin & Hamilton, 2003). As with depression, the comorbidity of these disorders is associated with a higher risk of substance use disorders.

ASPD is more prevalent among individuals with schizophrenia than in the general population (Robins & Regier, 1991). Furthermore, this comorbidity is associated with negative outcomes such as high risk for criminal activity and violence (Hodgins, Toupin, & Cote, 1996; Moran & Hodgins, 2004), higher severity of substance use disorders, more psychiatric impairment, and aggression (Mueser, Drake, Ackerson, Alterman, & et al., 1997).

Borderline Personality Disorder (BPD) and ASPD share features of impulsivity, aggressiveness (Zanarini & Gunderson, 1997), abuse, neglect, and alienation (Zanarini, Gunderson, Marino, Schwartz, & et al., 1989), suggesting a shared underlying etiology (Widom, 1997; Zanarini & Gunderson, 1997). These two disorders demonstrate comorbidity rates as high as 16% in females and 48% in males (Zanarini et al., 1998).

Pathological gamblers with ASPD experience greater severity of gambling, medical, psychiatric and social problems compared to pathological gamblers without ASPD (Pietrzak & Petry, 2005). One genetic study suggested a possible shared genetic vulnerability (Slutske et al., 2001), thus opposing the idea that antisocial behavior is purely a consequence of being a pathological gambler.

Appendix B

Developmental Course

A diagnosis of ASPD requires a diagnosis of Conduct Disorder (CD) before the age of 15. Childhood behavior problems have long been considered precursors to criminality in adulthood, including violent offending. Indeed, CD is one of the most prevalent disorders of childhood (Kessler, McGonagle, Zhao, Nelson, & et al., 1994; Krueger, Caspi, Moffitt, & Silva, 1998) with prevalence rates of 10-12% and is associated with such negative outcomes as substance abuse and adult psychiatric disorders, primarily ASPD (Krueger, Caspi, Moffitt, & Silva, 1998).

Childhood aggression has been found to be the most significant risk factor for antisocial behavior in adolescence and childhood (Loeber & Hay, 1997). Nevertheless, not all children who are aggressive exhibit antisocial behavior in childhood (Maughan & Ruter, 1998) and furthermore, not all children with Conduct Disorder go on to develop ASPD in adulthood. This finding, coupled with the existence of late-onset of antisocial behavior (Marmorstein & Iacono, 2005) suggest the presence of different trajectories to adult antisocial behavior. Indeed, research suggests that there is a high level of heterogeneity in the developmental course of ASPD (Schaeffer et al., 2006). Nevertheless, one requirement for a diagnosis of ASPD is a diagnosis of conduct disorder in childhood (APA, 1994).

Numerous developmental models have been proposed based on different key features of antisocial behavior (e.g., aggressive behavior, personality traits, and biological correlates). One key feature that has received considerable attention is physical

aggression, both due to its pervasiveness in individuals with ASPD and to its social consequences. It not yet is not clear whether physical aggression is a distinct risk factor for later violence or whether the constellation of behavior problems present in conduct disorder has more predictive utility. A longitudinal study of six sites in three countries indicated that childhood physical aggression in boys was a risk factor for later criminality; however, this relationship was not found in girls (Broidy, et al, 2003). Other developmental trajectories toward antisocial behavior have been proposed that involve different types of delinquency, criminal involvement, and time and length of onset.

Prospective and retrospective examinations of the developmental course of disruptive behavior (Loeber et al., 1993) resulted in the delineation of three developmental pathways: (1) overt, which involves high levels of aggression in childhood and leads to violence in adolescence and adulthood, (2) covert, which involves secretive antisocial behavior in childhood (e.g., stealing) and results in property crimes in adolescence and adulthood, and (3) authority conflict, which highlights the more oppositional behaviors and leads to later status offending.

Another model (Patterson, 1989) outlined two trajectories based on time of onset. The "early starters" were characterized by failure in school, coercive parenting, and antisocial behaviors beginning in childhood. The "later starters" began in adolescence and were characterized by poor parental monitoring, defiance of authorities, and the presence of deviant peers.

Other models (Moffitt, 1993) propose the existence of two distinct groups: (1) life persistent offenders and (2) adolescence-limited offenders. This model was elaborated by including subtypes of these two groups based on the presence or absence of Attention

Deficit Hyperactivity Disorder and the desistence of aggressive behavior either during elementary or in adolescence or late adulthood (Loeber & Stouthamer-Loeber, 1998).

In a prospective longitudinal study of urban, primarily African-American boys, Schaeffer (2003) integrated each of the prevailing developmental models and delineated four trajectories. The first two, which combined are parallel to the life course persistent offenders in other models, were characterized by chronically high levels of aggression in childhood, and the other with increasing moderate levels of aggression throughout childhood and into adolescence. These groups exhibited high rates of antisocial behavior in young adulthood. In contrast, another group was identified as having low levels of aggression throughout childhood with lower rates of antisocial behavior in young adulthood. Finally, the non-aggressive group exhibited little aggression and this pattern remained stable. This group had the lowest rate of young adulthood antisocial behavior. There was no evidence of a high aggression group whose antisocial behavior decreased over time.

Studies of antisocial behavior in children have also focused on correlates of the development psychopathic traits, namely callousness. The genesis of such traits have been associated with cruelty to animals during childhood (Dadds, 2006), which has been found to be a behavior present in the histories of a disproportionately high rate of violent offenders (Dadds, Turner, & McAloon, 2002).

Appendix C

Risk Factors for ASPD

Aside from Conduct Disorder and aggressive behavior in childhood, other risk factors for the development of ASPD include parental alcoholism (Kuperman, Schlosser, Lidral, & Reich, 1999), ADHD (Loeber, 1988), and deviant and poor peer relationships in early and middle childhood (Bagwell, 2004; Coie, 2004). Familial factors are perhaps the most influential environmental factors that contribute to the development of CD and later ASPD. Specifically inconsistent supervision accompanied by harsh punishment (Loeber, 1990) and violence and neglect within the family (Widom, 1997) have been found to be the most powerful predictors of CD and later ASPD.

Evidence from twin studies, twins reared apart, and adoption studies have revealed that there are genetic influences on antisocial behavior. Specifically, behavior genetic studies indicate that externalizing disorders in general are 80% heritable (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). Estimates of the heritability of ASPD have been 69% (Fu et al., 2002). Studies of the heritability of psychopathic traits in children indicate a heritability of 30% for antisocial behavior without callous/unemotional (CU) traits and 80% for antisocial behavior with CU traits. These estimates highlight the need to investigate biological vulnerability for the development of antisocial behavior and psychopathic traits.

Ten percent of families in any given community account for more than 50% of the crime in that community (Farrington, Barnes, & Lambert, 1996; Farrington, Jolliffe,

Loeber, Stouthamer-Loeber, & Kalb, 2001). Although this speaks to the influence of genes on antisocial behavior, it also highlights the importance of genetic-environment interactions in the development and persistence of ASPD. In fact, studies of biosocial interactions for the development of ASPD indicate that biological and social and biological risk factors converge to exponentially increase risk (Raine, 2002). Specifically, adoption studies have found that negative parenting behaviors coupled with risk for antisocial behavior by biological parents result in increased risk in comparison to either the biological or environmental risk alone.

Many studies have found that psychophysiological risk for antisocial behavior is stronger in those from social backgrounds that lack the typical psychosocial risk factors. For example, despite the well-replicated finding of autonomic hyporeactivity antisocial groups across numerous psychophysiological indices in children, adolescents (Raine, Venables, & Williams, 1990, , 1995), and adults (Patrick, Bradley, & Lang, 1993; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000; Raine, Venables, & Williams, 1990), resting heart rate has been found to be lower in individuals from higher social classes (Raine & Venables, 1984), those from privileged middle class backgrounds attending private schools (Maliphant, Hume, & Furnham, 1990), and those from intact homes (Wadsworth, 1976) than those in comparison groups who exhibit antisocial behavior. Similarly, reduced skin conductance activity during electrodermal classical conditioning was found in antisocial adolescents in high but not low social classes (Raine & Venables, 1981). Despite these finding, little work has been done examining biological correlates of antisocial behavior in adults.

Appendix D

ASPD, Impulsivity, and Distress Tolerance

Impulsivity has been defined in numerous ways in personality theory. One conceptualization that is particularly applicable to ASPD and distress tolerance is one that defines impulsivity as the preference for immediate over delayed gratification and the pursuance of easier means of obtaining self-gratification. Behavioral definitions conceptualize impulsive behavior as the choice of a small, immediate reward over a larger but more delayed reward (Ainslie, 1975). In this way, impulsivity in individuals with ASPD is often described in terms of reward. In other words, these individuals engage in impulsive behavior such as stealing, fighting, etc. for perceived gains (e.g., material and social status).

Although not clearly indicated in current theories of impulsivity in individuals with ASPD, a definition of impulsivity that includes variables controlling emotionally mediated engagement in impulsive behavior may inform the understanding of mechanisms underlying ASPD. Along these lines, it may be useful to consider avoidance as a negative reinforcer in its impact on behavior, particularly self-control. In thinking about the impact of emotion on impulsivity in individuals with ASPD, distress intolerance may describe a facet of impulsivity that is associated with emotion regulation as well as perceived gains (i.e., negative reinforcement coupled with positive reinforcement). Along these lines and given the high levels of negative emotionality in antisocial individuals (Hicks & Patrick, 2006), these individuals may have a low threshold for tolerating distress and may engage in behaviors that serve to reduce distress

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(e.g., ceasing effortful goal-directed behavior) rather than persisting in effortful goal directed behavior which culminates in a valuable gain. Therefore, individuals with ASPD my exhibit distress intolerance due to an emotionally-motivated type of impulsivity and examining emotionality in individuals with ASPD may provide insight into mechanisms underlying antisocial behavior.

ASPD, Borderline Personality Disorder, and Distress Tolerance

Distress tolerance has been hypothesized to underlie maladaptive behavior in individuals with Borderline Personality Disorder (Linehan, 1993). ASPD and BPD have been described as "mirror image disorders" (Paris, 1997). This notion is supported by their common symptoms, personality dimensions (Widiger & Costa, 1994), risk factors (childhood abuse, parenting styles, etc), and neurobiological substrates (Volm, Richardson, Stirling, Elliott, Dolan, Chaudhry, et al., 2004). As such, both ASPD and BPD are marked by impulsive traits and irritability (DSM-IV, 1994), and are thought to have common etiologies, differing mostly by gender-specific factors (Paris, 1997). Specifically, gender differences in socialization, childhood antecedents, hormones, personality traits, and autonomic indicators have all been implicated in differentiating pathways to ASPD and BPD. Also, in women, childhood antisocial symptoms have been found to predict the development of affect-related BPD criteria in adulthood (Goodman, Hull, Clarkin, Yeomans, 1999), further suggesting an etiological link between the two disorders. Furthermore, while both disorders are marked by impulsive traits and hostility, they differ in the direction of hostility, with BPD being associated with introverted hostility and ASPD associated with extroverted hostility (Hotzitaskos, Soldatos, Sakkas,& Stefanis, 1997).

Furthermore, recent empirical evidence suggests that acts of self-harm by individuals with BPD reflect attempts to regulate negative affect (Gratz, 2003). Similarly, given shared features of ASPD and BPD, distress intolerance may explain studies that suggest that violent and aggressive acts may serve to modulate emotion (Bushman, Baumeister, & Phillips, 2001). This finding is consistent with aggression as a key feature of ASPD, as well as the other core features described in the *DSM-IV* (1994) described above. Moreover, impulsive traits may serve to bolster the relationship between distress intolerance and aggressive acts. Along these lines, individuals with ASPD, when faced with negative emotionality, may engage in impulsive decision-making, such as ceasing goal-directed behavior or by engaging in impulsive acts such as violence and aggression.

Appendix E

Behavioral Inhibition System / Behavioral Activation System

Gray's (1978) two factor learning theory proposed two systems involved in learning and motivation, the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS). The BAS is responsible for approach behavior related to responses to cues or stimuli. The BIS, in turn inhibits the BAS in situations of response-contingent punishment. Since this seminal work, others have used this framework to explain the emotional deficit present in psychopathy. The BIS/BAS systems serve as a well-supported theoretical framework from which to explain the impulsive and fearless behavior present in psychopathy.

According to this perspective, psychopaths exhibit a weak BIS, the system responsible for response contingent behavior, namely avoidance of punishment. As such, the weakened BIS accounts for both the aggressive behavior and the unemotional characteristics (i.e., callous behavior and fearlessness) of individuals with psychopathy. The weakened BIS would result in a dominant BAS, which in turn would result in approach behavior in conflict situations (aggression).

From the theoretical perspective that the BIS modulates anxiety related responses, a weakened BIS would also result in lower anxiety and fearlessness in conflict and other anxiety- or fear-producing situations. Furthermore, this lowered anxiety has been specifically associated with callous and unemotional traits, rather than the deviant behavior components of psychopathy (Bare, Hopko, & Armento, 2004). Following this theoretical perspective, the weakened BIS associated with callous and unemotional traits

would also result in persistence in or non-avoidance of aversive situations. For example, a weakened BIS may result in persistence on psychological challenge tasks designed to produce frustration due to the lack of an emotional response. Furthermore, in line with previous work done examining psychophysiological responses to aversive situations, individuals with callous and unemotional traits would exhibit attenuated stress responses to aversive situations, such as that response created by psychological challenge tasks.

Appendix F

Psychopathic Personality Inventory

This test measures differences in personality characteristics among people that is, how people differ from each other in their personality styles. Beginning on the next page, read each item carefully, and decide to what extent it is false or true <u>as applied to you.</u> Then mark your answer in the space provided to the left of each item using the scale provided below.
1) False 2) Mostly False 3) Mostly True 4) True
Even if you feel that an item is neither false nor true as applied to you, or if you are unsure about what response to make, try to make some response in every case. If you cannot make up your mind about the item, select the choice that is <u>closest</u> to your opinion about whether it is false or true as applied to you. Try to be as honest as you can, and be sure to give your own opinion about whether each item is false or true as applied to you.

1) With one smile, I can often make someone I've just met interested in getting to know me better.
2) I like my life to be unpredictable, even a little surprising.
3) Members of the opposite sex find me "sexy" and appealing.
4) I am very careful and cautious when doing work involving detail.
5) Physically dangerous activities, such as sky-diving or climbing atop high places, frighten me more than they do most other people.
6) I tend to have a short temper when I am under stress.
7) Even when others are upset with me, I can usually win them over with my charm.
8) My table manners are not always perfect.

9) If I'm at a dull party or social gathering, I like to stir things up.
10) I weigh the pros and cons of major decisions carefully before making them.
11) Being rich is much less important to me than enjoying the work I do.
12) I've always considered myself to be something of a rebel.
13) I sometimes worry about whether I might have accidentally hurt someone's feelings.
14) I find it difficult to make small talk with people I do riot know well.
15) I think a fair amount about my long—term career goals.
16) I would not mind wearing my hair in a "mohawk."
17) I occasionally forget my name.
18) I rarely find myself being the center of attention in social s'ituations.
19) It might be fun to belong to a group of "bikers" (motorcyclists) who travel around the country and raise some hell.
20) I tell many "white lies."
21) I often hold on to old objects or letters just for their sentimental value.
22) I am a good conversationalist.
23) A lot of people in my life have tried to stab me in the back.
24) I am so moved by certain experiences (e.g., watching a beautiful sunset, listening to a favorite

piece of music) that I feel emotions that are beyond words.

25) I often find myself resenting people who give me orders.

26) I would find the job of movie stunt person exciting.

27) I have always been extremely courageous in facing difficult situations.

28) I hate having to tell people bad news.

29) I think that it should be against the law to seriously injure another person intentionally.

30) I would be more successful in life had I not received so many bad breaks.

31) It bothers me (or it would bother me) quite a bit to speak in front of a large group of strangers.

32) When I am faced with a decision involving moral matters, I often ask myself, Am I doing the right thing?-"

33) From time to time I really blow up" at other people.

34) Many people think of me as a daredevil.

35) It takes me a long time to get over embarrassing or humiliating experiences.

36) I usually feel that people give me the credit I deserve.

37) I've never really cared much about society's so- called va1ues of right and wrong."

38) If someone mistreats me, I'd rather try to forgive him or her than get even.

39) It would bother me to cheat on an examination or assignment even if no-one got hurt in the process.

40) I become deeply upset when I see photographs of starving people in Africa.

41) I rarely monopolize conversations.

42) Making a parachute jump would really frighten me. __

43) At times I have been envious of someone.

44) I become very angry if I do not receive special favors or privileges I feel I deserve.

45) I often find myself worrying when a friend is having serious personal problems.

46) I pride myself on being offbeat and unconventional.

47) Keeping in touch with old friends is very important to me.

48) I usually strive to be the best at whatever I do.

49) I almost always feel very sure of myself when I'm around other people.

50) I look down at the ground whenever I hear an airplane flying above my head.

51) I could make an effective "con artist" if the situation required it.

52) I wouldn't mind spending my life in a commune and writing poetry.

53) I have had "crushes" on people that were so intense that they were painful.

54) I like to stand out in a crowd.

55) I'm not intimidated by anyone.

56) Before I say something, I first like to think about it for a while.

57) I would enjoy hitch-hiking my way across the United States with no prearranged plans.

58) I am a guilt-prone person.

59) I bet that it would be fun to pilot a small airplane alone.

60) When I want to, I can usually put fears and worries out of my mind.

61) Never in my whole life have I wished for anything that I was not entitled to.

62) I generally prefer to act first and think later.

63) I am easily flustered in pressured situations.

64) I often make the same errors in judgment over and over again.

65) I always look out for my own interests before worrying about those of the other guy.

66) I smile at a funny joke at least once in a while.

67) People have often criticized me as being unruly.

68) I almost always promptly return items that I have borrowed from others.
69) I sometimes have difficulty standing up for my rights in social situations.
70) If I want to, I can influence other people without
their realizing they are being manipulated.
71) My opinions are always completely reasonable.
72) I become embarrassed more easily than most people.
73) When I'm in a frightening situation, I can "turn off" my fear almost at will.
74) It bothers me greatly when I see someone crying.
75) Frankly, I believe that I am more important than most people.
76) I frequently have disturbing thoughts that become so intense and overpowering that I think I
can hear claps of thunder or crashes of cymbals inside my head.
77) If I do something that causes me trouble, I'm sure to
avoid doing it again.
78) I often place my friends' needs above my own.

79) I like having my vacations carefully planned out.
80) People whom I have trusted have often ended up "double-crossing" me.
81) I often become deeply attached to people I like.

82) I've been the victim of a lot of bad luck in my life.

83) I have at times eaten too much.
84) I sometimes question authority figures "just for the hell of it.".
85) When my life becomes boring, I like to take some chances to make things interesting.
86) I tend to be "thin-skinned" and overly sensitive to criticism.

87) I've quickly learned from my major mistakes in life.
88) When someone is hurt by something I say or do, I usually consider that to be their problem.
89) I like to dress differently from other people.
90) If I really wanted to, I could convince most people .of just about anything.
91) I get restless and dissatisfied if my life becomes too routine.
92) I generally feel that life has treated me fairly.
93) Ending a friendship is (or would be) very painful for me.
94) When I am under stress, I often see large, red, rectangular shapes moving in front of my eyes.
95) I often do favors for people even when I know that I will probably never see them again.
96) I have sometimes "stood up" a date or a friend because something that sounded like more fun
came up.
97) I haven't thought much about what I want to do with my life.
98) Looking down from a high place gives me "the jitters."
99) I feel that few people in my life have taken advantage of me.
100) I can't imagine being sexually involved with more than one person at the same time.
101) I'm never concerned about whether I'm following the "rules" in social situations; I just
make my own rules.

102) I find it easy to go up to someone I've never met and introduce myself.
103) I often feel very nostalgic when I think back to peaceful moments in my childhood.
104) When I go to a restaurant, I carefully look over the menu before deciding what to order.

105) Some people seem to have gone out of their way to make life difficult for me.
106) I have always been completely fair to others.
107) I get a kick out of startling or scaring other people.
 108) I generally try to pay attention when someone important speaks to me directly.
109) I feel very bad about myself after telling a lie.

110) I enjoy watching violent scenes in movies.
111) I would not enjoy being a race-car driver.
112) I am very careful about my manners when other people are around.
113) I feel that very few people have ever understood me.

114) I'm hardly ever the "life of the party."
115) I have occasionally felt discouraged about something.
116) I agree with the motto, "If you are bored with life, risk it."

117) I am a squeamish person.
118) I enjoy (or I would enjoy) participating in sports involving a lot of physical contact (e.g., football, wrestling)
119) I do not enjoy loud, wild parties and get-togethers.

120) I often push myself to my limits in my work.
121) I am easily "rattled" at critical moments.
122) In school or at work, I sometimes try to "stretch". the rules a little bit just to see how much I can get away with.
123) On occasion, I've had to restrain myself from punching someone.
124) I wouldn't mind belonging to a group of people who "drift" from city to city, with no permanent home.
125) I have at times been angry with someone.
126) If I were growing up during the 1960's, I probably would have been a "hippie" (Or, I was a "hippie" during the 1960's).
127) When a friend says hello to me, I generally either wave or say something back.
128) While watching a sporting event on TV, I sometimes wince when I see an athlete get badly injured.
129) I'm good at flattering important people when it's useful to do so.
130) I sometimes become deeply angry when I hear about some of the injustices going on in the world.
131) I'm not very good at talking people into doing favors for me.
132) Seeing a poor or homeless person walking the streets at night would really break my heart.
133) When someone tells me what to do, I often feel like doing exactly the opposite just to spite them.
134) I always tell the entire truth.
135) I prefer rude, but exciting people to nice, but boring people.
136) I can remain calm in situations that would make many other people panic.
137) I usually enjoy seeing someone I don't like get into trouble.
138) When I'm in a group of people who do something wrong, somehow it seems that I'm usually the one who ends up getting blamed.
139) People are almost always impressed with me after they first meet me.
 140) I like to (or would like to) wear expensive, "showy" clothing.
141) In the past, people who were supposed to be my "friends" ended up getting me in trouble.
142) I might enjoy flying across the Atlantic in a hot-air balloon.

143) I don't take advantage of other people even when it's clearly to my benefit.
144) I'm the kind of person who gets "stressed out" pretty easily.
145) Sometimes I'm a bit lazy.
146) I sometimes like to "thumb my nose" at established traditions.
147) During the day, I generally see the world in color rather than in black-and-white.
148) When I am doing something important (e.g., taking a test, doing my taxes) I usually check it over at
least once or twice to make sure it is correct.
149) When I'm among a group of people, I rarely end up being the leader.
150) To be perfectly honest, I usually try not to help people unless I think there's some way that they can
help me later.
151) Many people probably think of my political beliefs as "radical."
152) I sometimes lie just to see if I can get someone to believe me.

153) I have to admit that I'm a bit of a materialist.
154) I think that it might almost be exciting to be a passenger on a plane that appeared certain to crash, yet somehow managed to land safely.
155) In social situations, I sometimes act the same way everyone else does because I don't want to appear too different.
156) Never in my whole life have I taken advantage of anyone.
157) I can hold up my end of a conversation even if the topic is something I know almost nothing about.

158) I often tell people only the part of the truth they want to hear.

References

Ames, A., Molinari, V. (1994). Prevalence of personality disorders in community-living elderly. *Journal of Geriatric Psychiatry and Neurology, 7,* 189-194.

APA. (1994). *Diagnostic and statistical manual of mental disorders (4th ed).* Washington, DC,US: American Psychiatric Publishing, Inc.

Bagwell, C. L. (Ed.). (2004). *Friendships, peer networks, and antisocial behavior.* Washington, DC,US: American Psychological Association.

Balsis, S., Gleason, M. E. J., Woods, C. M., & Oltmanns, T. F., (2007). An item response theory analysis of DSM-IV personality disorder criteria across younter and older age groups. *Psychology and Aging, 22,* 171-185.

Bare, R. L., Hopko, D. R., & Armento, M. E. A. (2004). The relation of psychopathic characteristics and anxiety in noncriminals: physiological and cognitive responses to guided imagery. *Journal of Psychopathology and Behavioral Assessment, 26*(4), 225-232.

Barriga, A. Q., Morrison, E. M., Liau, A. K., & Gibbs, J. C. (2001). Moral cognition: Explaining the gender difference in antisocial behavior. *Merrill-Palmer Quarterly, 47*(4), 532-562.

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

Blackburn, R. (1988). On moral judgements and personality disorders: The myth of psychopathic personality revisited. *British Journal of Psychiatry, 153*, 505-512.

Blair, J., Mitchell, D., & Blair, K. (2005). *The Psychopath: emotion and the brain*. Maldon, MA: Blackwell Publishing.

Broidy, L. M., Nagin, D. S., Tremblay, R. E. (2003). Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: a six site, cross-national study. *Developmental Psychology, 39,* 222-245.

Brooner, R. K., Bigelow, G. E., Strain, E., & Schmidt, C. W. (1990). Intravenous drug abusers with antisocial personality disorder: increased HIV risk behavior. *Drug and Alcohol Dependence, 26*(1), 39-44.

Brown, R. A., Lejuez, C. W., Kahler, C. W., & Strong, D. R. (2002). Distress tolerance and duration of past smoking cessation attempts. *Journal of Abnormal Psychology, 111*(1), 180-185.

Brown, R. A., Lejuez, C. W., Kahler, C. W., Strong, D. R., & Zvolensky, M. J. (2005). Distress tolerance and early smoking lapse. *Clinical Psychology Review, 25*(6), 713-733.

Bushman, B. J., Baumeister, R. F., & Phillips, C. M. (2001). Do people aggress to improve their mood? Catharsis beliefs, affect regulation opportunity, and aggressive responding. *Journal of Personality and Social Psychology, 81*(1), 17-32.

Buydens-Branchey, L., Branchey, M. (2004). Cocaine addicts with conduct disorder are typified by decreased cortisol responsivity and high plasma levels of DHEA-S. *Neuropsychobiology, 50,* 161-166.

Canals, J., Colomina, M. T., Domingo, J. L., & Dominech, E. (1997). Influence of

smoking and drinking habits on salivary cortisol levels. *Personality and

Individual Differences, 23*(4), 593-599.

Carlson, C. R., Bacaseta, P. E., & Simanton, D. A. (1988). A controlled evaluation of

devoltional meditation and progressive relaxation. *Journal of Psychology and

Theology, 16,* 362-368.

Casey, D. A., Schrodt, C. J. (1989). Axis II diagnoses in geriatric inpatients. *Journal of

Geriatric Psychiatry and Neurology, 2,* 87-88.

Caspi, A., Bem, D. J. (1990). Personality continuity and change across the life course. *In:

Handbook of personality: theory and research.* Pervin, L.A., New York, NY, US.

Guilford Press, 549-575.

Cleckley, H. (1941). *The mask of sanity: an attempt to reinterpret the so-called

psychopathic personality*. Oxford,England: Mosby.

Coie, J. D. (Ed.). (2004). *The impact of negative social experiences on the development of

antisocial behavior*. Washington, DC,US: American Psychological Association.

Compton, W. M., Conway, K. P., Stinson, F. S., Colliver, J. D., & Grant, B. F. (2005).

Prevalence, correlates, and comorbidity of DSM-IV antisocial personality

syndromes and alcohol and specific drug use disorders in the United Sates: results

from the Ntional Eidemiologic Srvey on Acohol and Rlated Cnditions. *Journal of

Clinical Psychiatry, 66*(6), 677-685.

Compton, W. M., Cottler, L. B., Jacobs, J. L., Ben-Abdallah, A., & Spitznagel, E. L.

(2003). The role of psychiatric disorders in predicting drug dependence treatment

outcomes. *American Journal of Psychiatry, 160*(5), 890-895.

Connor, D. F., Steingard, R. J., Anderson, J. J., & Melloni, R. H., Jr. (2003). Gender differences in reactive and proactive aggression. *Child Psychiatry & Human Development, 33*(4), 279-294.

Cooke, D. J., Michie, C., Hart, S. D., & Clark, D. A. (2004). Reconstructing psychopathy: Clarifying the significance of antisocial and socially deviant behavior in the diagnosis of psychopathic personality disorder. *Journal of Personality Disorders, 18*(4), 337-357.

Daughters, S. B., Lejuez, C. W., Bornovalova, M. A., Kahler, C. W., Strong, D. R., & Brown, R. A. (2005). Distress tolerance as a predictor of early treatment dropout in a residential substance abuse treatment facility. *Journal of Abnormal Psychology, 114*(4), 729-734.

Daughters, S. B., Lejuez, C. W., Kahler, C. W., Strong, D. R., & Brown, R. A. (2005). Psychological distress tolerance and duration of most recent abstinence attempt among residential treatment-seeking substance abusers. *Psychology of Addictive Behaviors, 19*(2), 208-211.

Daughters, S. B., Lejuez, C. W., Strong, D. R., Brown, R. A., Breen, R. B., & Lesieur, H. R. (2005). The relationship among negative affect, distress tolerance, and length of gambling abstinence attempt. *Journal of Gambling Studies, 21*(4), 363-378.

Daughters, S. B., Sargeant, M. N., Gratz, K. L., Bornovolova, M. A., & Lejuez, C. W. (in press). The relationship between distress tolerance and antisocial personality disorder among male inner-city treatment seeking substance users. *Behavior Research and Therapy.*

de Kloet, E. R., & Reul, J. M. (1987). Feedback action and tonic influence of

    corticosteroids on brain function: A concept arising from the heterogeneity of

    brain receptor systems. *Psychoneuroendocrinology, 12*(2), 83-105.

Deckard, D., Petrill, S. A., Thompson, L. A. (2007). Anger/frustration, task persistence,

    and conduct problems in childhood: a behavioral genetic analysis. *Journal of*

    Child Psychology and Psychiatry, 48, 80-87.

Derijk, R. H., van Leeuwen, N., Klok, M. D., & Zitman, F. G. (2008). Corticosteroid

    receptor gene variants: modulators of the stress response and implications for

    mental health. *European Journal of Pharmacology, 585,* 492-501.

Fals-Stewart, W., Leonard, K. E., & Birchler, G. R. (2005). The occurrence of male-to-

    female intimate partner violence on days of men's drinking: the moderating

    effects of antisocial personality disorder. *Journal of Consulting and Clinical*

    Psychology, 73(2), 239-248.

Farrington, D. P., Barnes, G. C., & Lambert, S. (1996). The concentration of offending in

    families. *Legal and Criminological Psychology, 1*(1), 47-63.

Farrington, D. P., Jolliffe, D., Loeber, R., Stouthamer-Loeber, M., & Kalb, L. M. (2001).

    The concentration of offenders in families, and family criminality in the

    prediction of boys' delinquency. *Journal of Adolescence, 24*(5), 579-596.

Flynn, P. M., Craddock, S. G., Luckey, J. W., Hubbard, R. L., & Dunteman, G. H.

    (1996). Comorbidity of antisocial personality and mood disorders among

    psychoactive substance-dependent treatment clients. *Journal of Personality*

    Disorders, 10(1), 56-67.

Fogel, B. S., & Westlake, R. (1990). Personality disorder diagnoses and age in inpatients with major depression. *Journal of Clinical Psychiatry, 51,* 232-235.

Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology, 17*(2), 87-104.

Fowles, D. C. (2000). Electrodermal hyporeactivity and antisocial behavior: Does anxiety mediate the relationship? *Journal of Affective Disorders, 61*(3), 177-189.

Frick, P. J., & Loney, B. R. (Eds.). (2002). *Understanding the association between parent and child antisocial behavior*. New York, NY,US: Kluwer Academic/Plenum Publishers.

Fu, Q., Heath, A. C., Bucholz, K. K., Nelson, E., Goldberg, J., Lyons, M. J., et al. (2002). Shared genetic risk of major depression, alcohol dependence, and Cannabis dependence: Contribution of antisocial personality disorder in men. *Archives of General Psychiatry, 59*(12), 1125-1132.

Gelhorn, H. L., Sakai, J. T., & Price, R. K. (2007). DSM-IV conduct disorder criteria as predictors of antisocial personality disorder. *Comprehensive Psychiatry, 48,* 529-538.

Goldstein, R. B., Powers, S. I., McCusker, J., Mundt, K. A., & et al. (1996). Gender differences in manifestations of antisocial personality disorder among residential drug abuse treatment clients. *Drug and Alcohol Dependence, 41*(1), 35-45.

Goodman, G., Hull, J. W., Clarkin, J. F., & Yeomans, F. E. (1999). Childhood antisocial behaviors as predictors of psychotic symptoms and DSM-III-R borderline criteria

among inpatients with borderline personality disorder. *Journal of Personality Disorders, 13,* 35-46.

Goodwin, R. D., & Hamilton, S. P. (2003). Lifetime comorbidity of antisocial personality disorder and anxiety disorders among adults in the community. *Psychiatry Research, 117*(2), 159-166.

Granger, D. A., Harmon, A. G., Hibel, L. C., & Rumyantseva, O. (2006). Measuring salivary cortisol in studies of child development: Watch out – what goes in may not come out of commonly used saliva collection devices: Penn State University, Department of Biobehavioral Health, Behavioral Endocrinology Laboratory.

Granger, D. A., Weisz, J. R., & Kauneckis, D. (1994). Neuroendocrine reactivity, internalizing behavior problems, and control-related cognitions in clinic-referred children and adolescents. *Journal of Abnormal Psychology, 103*(2), 267-276.

Gratz, K. L. (2003). Risk factors for and functions of deliberate self-harm: An empirical and conceptual review. *Clinical Psychology: Science and Practice, 10*(2), 192-205.

Hall, L. M. (2003). Hypothalamic pituitary adrenal axis functioning in disruptive behavior disorders: An examination of the relationship between cortisol levels and antisocial behavior. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 63*(7), 3509.

Hare, R. D. (1983). Diagnosis of antisocial personality disorder in two prison populations. *American Journal of Psychiatry, 140*(7), 887-890.

Hare, R. D., Harpur, T. J., Hakstian, A. R., Forth, A. E., & et al. (1990). The revised Psychopathy Checklist: Reliability and factor structure. *Psychological Assessment, 2*(3), 338-341.

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

Harpur, T. J., Hart, S. D., & Hare, R. D. (Eds.). (1994). *Personality of the psychopath*. Washington, DC,US: American Psychological Association.

Hatzitaskos, P. K., Soldatos, C. R., Sakkas, P. N., & Stefanis, C. N. (1997). Discriminating borderline from antisocial personality disorder in male patients based on psychopathology patterns and type of hostility. *The Journal of Nervous and Mental Disease, 185,* 442-446.

Heinrichs, R. W., & Zakzanis, K. K. (1998). Neurocognitive deficit in schizophrenia: A quantitative review of the evidence. *Neuropsychology, 12*(3), 426-445.

Herpertz, S. C., Mueller, B., Qunaibi, M., Lichterfeld, C., Konrad, K., & Herpertz-Dahlmann, B. (2005). Response to emotional stimuli in boys with conduct disorder. *American Journal of Psychiatry, 162*(6), 1100-1107.

Hicks, B. M., Krueger, R. F., Iacono, W. G., McGue, M., & Patrick, C. J. (2004). Family transmission and heritability of externalizing disorders: A twin-family study. *Archives of General Psychiatry, 61*(9), 922-928.

Hodgins, S., Toupin, J., & Cote, G. (Eds.). (1996). *Schizophrenia and antisocial personality disorder: A criminal combination*. Springfield, IL, US: Charles C. Thomas Publisher, Ltd.

Hodgins, S., Mednick, S. A., & Brennan, P. A. (1996). Mental disorder and crime: evidence from a Danish birth cohort. *Archives of General Psychiatry, 53,* 489-496.

Holdcraft, L. C., Iacono, W. G., & McGue, M. K. (1998). Antisocial personality disorder and depression in relation to alcoholism: A community-based sample. *Journal of Studies on Alcohol, 59*(2), 222-226.

Kasen, S., Cohen, P., & Brook, J. S. (1998). Adolescent school experiences and dropout, adolescent pregnancy, and young adult deviant behavior. *Journal of Adolescent Research, 13*(1), 49-72.

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV dsorders in the National Comorbidity Survey replication. *Archives of General Psychiatry, 62*(6), 617-627.

Kessler, R. C., Crum, R. M., Warner, L. A., & Nelson, C. B.(1997). Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry, 54*(4), 313-321.

Kessler, R. C., McGonagle, K. A., Zhao, S., & Nelson, C. B. (1994). Lifetime and 12-month prevalence of DSM-III--R psychiatric disorders in the United States: Results from the National Comorbidity Study. *Archives of General Psychiatry, 51*(1), 8-19.

Krueger, R. F., Caspi, A., Moffitt, T. E., & Silva, P. A. (1998). The structure and stability of common mental disorders (DSM-III-R): A longitudinal-epidemiological study. *Journal of Abnormal Psychology, 107*(2), 216-227.

Kuperman, S., Schlosser, S. S., Lidral, J., & Reich, W. (1999). Relationship of child

    psychopathology to parental alcoholism and antisocial personality disorder.

    Journal of the American Academy of Child & Adolescent Psychiatry, 38(6), 686-

    692.

Lang, P. J. (1995). The emotion probe: Studies of motivation and attention. *American*

    Psychologist, 50(5), 372-385.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle

    reflex. *Psychological Review, 97*(3), 377-395.

Lejuez, C. W., Kahler, C. W., & Brown, R. A. (2003). A modified computer version of

    the Paced Auditory Serial Addition Task (PASAT) as a laboratory-based stressor.

    The Behavior Therapist, 26, 290-293.

Lenzenweger, M. F., Johnson, M. D., & Willett, J. B. (2004). Individual growth curve

    analysis illuminates stability and change in personality disorder features: the

    longitudinal study of personality disorders. *Archives of General Psychiatry, 61,*

    1015-1024.

Levenston, G. K., Patrick, C. J., Bradley, M. M., & Lang, P. J. (2000). The psychopath as

    observer: Emotion and attention in picture processing. *Journal of Abnormal*

    Psychology, 109(3), 373-385.

Lilienfeld, S. O., & Andrews, B. P. (1996). Development and preliminary validation of a

    self-report measure of psychopathic personality traits in non-criminal populations.

    Journal of Personality Assessment, 66, 488-524.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality*

    disorder. New York, NY,US: Guilford Press.

Loeber, R. (1990). Disruptive and antisocial behavior in childhood and adolescence: Development and risk factors. 233-257.

Loeber, R. (Ed.). (1988). *Behavioral precursors and accelerators of delinquency*. Leiden,Netherlands: E. J. Brill.

Loney, B. R., Butler, M. A., Lima, E. N., Counts, C. A., & Eckel, L. A. (2006). The relation between salivary cortisol, callous-unemotional traits, and conduct problems in an adolescent non-referred sample. *Journal of Child Psychology and Psychiatry, 47*(1), 30-36.

Maliphant, R., Hume, F., & Furnham, A. (1990). Autonomic nervous system (ANS) activity, personality characteristics and disruptive behaviour in girls. *Journal of Child Psychology and Psychiatry, 31*(4), 619-628.

Marmorstein, N., & Iacono, W. G. (2005). Longitudinal follow-up of adolescents with late-onset antisocial behavior: A pathological yet overlooked group. *Journal of the American Academy of Child & Adolescent Psychiatry, 44*(12), 1284-1291.

Martens, W. H. J. (2000). Antisocial and psychopathic personality disorders: Causes, course, and remission--a review article. *International Journal of Offender Therapy and Comparative Criminology, 44*(4), 406-430.

McBurnett, K., Raine, A., Stouthamer-Loeber, M., Loeber, R., Kumar, A. M., & Kumar, M. (2005). Mood and hormone responses to psychological challenge in adolescent males with conduct problems. *Biological Psychiatry, 57*(10), 1109-1116.

McDermott, P. A., Alterman, A. I., Cacciola, J. S., Rutherford, M. J., Newman, J. P., & Mulholland, E. M. (2000). Generality of Psychopathy Checklist--Revised factors

over prisoners and substance-dependent patients. *Journal of Consulting and Clinical Psychology, 68*(1), 181-186.

Merikangas, K. R., Mehta, R. L., Molnar, B. E., Walters, E. E., Swendsen, J. D., & Auilar-Gaziola, S. (1998). Comorbidity of substance use disorders with mood and anxiety disorders: Results of the international consortium in psychiatric epidemiology. *Addictive Behaviors, 23*(6), 893-908.

Molinari, V., Kunik, M. E., Snow-Turek, A. L., Deleon, H., & Williams, W. (1999). Age-related personality differences in inpatients with personality disorder: a cross-sectional study. *Journal of Clinical Geropsychology, 5,* 191-202.

Moran, P., & Hodgins, S. (2004). The correlates of comorbid antisocial personality disorder in schizophrenia. *Schizophrenia Bulletin, 30*(4), 791-802.

Mueser, K. T., Drake, R. E., Ackerson, T. H., & Alterman, A. I. (1997). Antisocial personality disorder, conduct disorder, and substance abuse in schizophrenia. *Journal of Abnormal Psychology, 106*(3), 473-477.

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

O'Leary, M. M., Loney, B. R., Eckel, L. A. (2007). Gender differences in the association between psychopathic personality traits and cortisol response to induced stress. *Psychoneuroendocrinology, 32,* 183-191.

Paris, J. (1997). Antisocial and borderline personality disorders: two separate diagnoses or two aspects of the same psychopathology? *Comprehensive Psychiatry, 38,* 237-242.

Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights.

Psychophysiology, 31(4), 319-330.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath:

Startle reflex modulation. *Journal of Abnormal Psychology, 102*(1), 82-92.

Patrick, C. J., Cuthbert, B. N., & Lang, P. J. (1994). Emotion in the criminal psychopath:

Fear image processing. *Journal of Abnormal Psychology, 103*(3), 523-534.

Pietrzak, R. H., & Petry, N. M. (2005). Antisocial personality disorder is associated with

increased severity of gambling, medical, drug and psychiatric problems among

treatment-seeking pathological gamblers. *Addiction, 100*(8), 1183-1193.

Poythress, N. G., Edens, J. F., & Lilienfeld, S. O. (1998). Criterion-related validity of

thePsychopathic Personality Inventory in a prison sample. *Psychological*

Assessment, 10, 426-430.

Quinn, E. P., Brandon, T. H., Copeland, A. L. (1996). Is task persistence related to

smoking and substance abuse? The application of learned industriousness theory

to addictive behaviors. *Experimental and Clinical Psychopharmacology, 4,* 186-

190.

Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and

adults: A review. *Journal of Abnormal Child Psychology, 30*(4), 311-326.

Raine, A., Lencz, T., Bihrle, S., LaCasse, L., & Colletti, P. (2000). Reduced prefrontal

gray matter volume and reduced autonomic activity in antisocial personality

disorder. *Archives of General Psychiatry, 57*(2), 119-127.

Raine, A., & Venables, P. H. (1981). Classical conditioning and socialization--a biosocial interaction. *Personality and Individual Differences, 2*(4), 273-283.

Raine, A., & Venables, P. H. (1984). Electrodermal nonresponding, antisocial behavior, and schizoid tendencies in adolescents. *Psychophysiology, 21*(4), 424-433.

Raine, A., Venables, P. H., & Williams, M. (1990). Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. *Archives of General Psychiatry, 47*(11), 1003-1007.

Raine, A., Venables, P. H., & Williams, M. (1995). High autonomic arousal and electrodermal orienting at age 15 years as protective factors against criminal behavior at age 29 years. *American Journal of Psychiatry, 152*(11), 1595-1600.

Rivier, C., & Imaki, T. (1990). Prologed exposure to alcohol: effect on CRF mRNA levels, and CRF- and stress-induced ACTH secretion in the rat. *Brain Research, 18,* 1-5.

Robins, L. N., & Regier, D. A. (1991). *Psychiatric disorders in America: The Epidemiologic Catchment Area study*. New York: Free Press.

Rogers, R., Duncan, J. C., Lynett, E., & Sewell, K. W. (1994). Prototypical analysis of antisocial personality disorder: DSM-IV and beyond. *Law and Human Behavior, 18*(4), 471-484.

Rohleder, N., & Kirschbaum, C. (2006). The hypothalamic-pituitary-adreanal (HPA) axis in habitual smokers. *International Journal of Psychophysiology, 59,* 236-243.

Salekin, R. T. (2002). Psychopathy and therapeutic pessimism. Clinical lore or clinical reality? *Clinical Psychology Review, 22,* 79-112.

Schaeffer, C. M., Petras, H., Ialongo, N., Masyn, K. E., Hubbard, S., Poduska, J., &
Kellam, S. (2006). A comparison of girls' and boys' aggressive-disruptive
behavior trajectories across elementary school: Prediction to young adult
antisocial outcomes. *Journal of Consulting and Clinical Psychology, 74*(3), 500-
510.

Segal, D. L., Hook, J. N.,& Coolidge, F. L. (2001). Personality dysfunction, coping
styles, and clinical symptoms in younger and older adults. *Journal of Clinical
Geropsychology, 7,* 201-212.

Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology:
Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology,
103*.

Shirtcliff, E. A., Granger, D. A., Booth, A., & Johnson, D. (2005). Low salivary cortisol
levels and externalizing behavior problems in youth. *Development and
Psychopathology, 17*(1), 167-184.

Sinha, R., Garcia, M., Paliwal, P., Kreek, M. J., & Rounsaville, B. J. (2006). Stress-
induced cocaine craving and hypothalamic-pituitary-adrenal responses are
predictive of cocaine relapse outcomes. *Archives of General Psychiatry, 63,* 324-
331.

Slutske, W. S., Eisen, S., Xian, H., True, W. R., Lyons, M. J., Goldberg, J., & Tsuang, M.
(2001). A twin study of the association between pathological gambling and
antisocial personality disorder. *Journal of Abnormal Psychology, 110*(2), 297-
308.

Smith, A. (2006). Cognitive empathy and emotional empathy in human behavior and
  evolution. *Psychological Record, 56*(1), 3-21.

Snoek, H., Van Goozen, S. H. M., Matthys, W., Buitelaar, J. K., & Van Engeland, H.
  (2004). Stress responsivity in children with externalizing behavior disorders.
  Development and Psychopathology, 16(2), 389-406.

Steptoe, A., & Ussher, M. (2006). Smoking, cortisol and nicotine. *International Journal
  of Psychophysiology, 59*(3), 228-235.

Van den Bree, M. B. M., Svikis, D. S., & Pickens, R. W. (1998). Genetic influences in
  antisocial personality and drug use disorders. *Drug and Alcohol Dependence,
  49*(3), 177-187.

van Goozen, S. H. M., Snoek, H., Matthys, W., van Rossum, I., & van Engeland, H.
  (2004). Evidence of fearlessness in behaviourally disordered children: A study on
  startle reflex modulation. *Journal of Child Psychology and Psychiatry, 45*(4),
  884-892.

van Lier, P. A. C., Vitaro, F., Wanner, B., Vuijk, P., & Crijnen, A. A. M. (2005). Gender
  differences in developmental links among antisocial behavior, friends' antisocial
  behavior, and peer rejection in childhood: Results from two cultures. *Child
  Development, 76*(4), 841-855.

Vanyukov, M. M., Moss, H. B., Plail, J. A., Blackson, T., & et al. (1993). Antisocial
  symptoms in preadolescent boys and in their parents: Associations with cortisol.
  Psychiatry Research, 46(1), 9-17.

Verona, E., Patrick, C. J., & Joiner, T. E. (2001). Psychopathy, antisocial personality, and
  suicide risk. *Journal of Abnormal Psychology, 110*(3), 462-470.

Vollm, B., Richardson, P., Stirling, J., Elliott R., Dolan, M., Chaudhry, I., Del Ben, C.,

 McKie, S., Anderson, I., & Deakin, B. (2004). Neurobiological substrates of

 antisocial and borderline personality disorder: preliminary results of a functional

 fMRI study. *Criminal Behavior and Mental Health, 14,* 39-54.

Wadsworth, M. E. J. (1976). Delinquency, pulse rate, and early emotional deprivation.

 British Journal of Criminology (16), 245-256.

Widiger, T. A., Cadoret, R., Hare, R., Robins, L., Rutherford, M., Zanarini, M.,

 Alterman, A., Apple, M., Corbitt, E., Forth, A., Hart, S., Kultermann, J., Woody,

 G., & Francis, A. (1996). DSM--IV antisocial personality disorder field trial.

 Journal of Abnormal Psychology, 105(1), 3-16.

Widom, C. S. (Ed.). (1997). *Child abuse, neglect, and witnessing violence*. Hoboken, NJ,

 US: John Wiley & Sons, Inc.

Zanarini, Frankenburg, F. R., Chauncey, D. L., & Gunderson, J. G. (1987). The

 Diagnostic Interview for Personality Disorders: Interrater and testetest reliability.

 Comprehensive Psychiatry, 28(6), 467-480.

Zanarini, Frankenburg, F. R., Dubo, E. D., Sickel, A. E., Trikha, A., Levin, A., &

 Reynolds, V. (1998). Axis II comorbidity of borderline personality disorder.

 Comprehensive Psychiatry, 39(5), 296-302.

Zanarini, Frankenburg, F. R., Sickel, A. E., & Yong, L. (1996). *The Diagnostic Interview*

 for DSM-IV Personality Disorders. Belmont, MA: McLean Hospital and Harvard

 Medical School.

Zanarini, & Gunderson, J. G. (Eds.). (1997). *Differential diagnosis of antisocial and*

 borderline personality disorders. Hoboken, NJ, US: John Wiley & Sons, Inc.

Zanarini, Gunderson, J. G., Marino, M. F., Schwartz, E. O., & et al. (1989). Childhood experiences of borderline patients. *Comprehensive Psychiatry, 30*(1), 18-25.

Zorrilla, E. P., Valdez, G. R., & Weiss, F. (2001). Changes in levels of regional CRF-like-immunoreactivity and plasma corticosterone during protracted drug withdrawal in dependent rats. *Psychopharmacology, 158,* 374-381.

Zuckerman, M. (1979). *Sensation Seeking: Beyond the Optimal Level of Arousal*. Hillsdale, NJ: Erlbaum.