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Nicole Azores-Gococo April 18, 2011

Psychopathic Traits and Anxiety as Predictors of Aggression and Fear Processing in Young Males

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An abstract of
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Research in adult populations has indicated the presence of psychopathy subtypes largely differentiated by the presence of trait anxiety, which often correlates divergently with the affective/interpersonal component of psychopathy (CU) and the behavioral component (I/CP). Studies in children have demonstrated similar trends and indicate that emotional processing differences may underlie psychopathy and different types of associated aggression, specifically proactive and reactive aggression. The current study tested the hypotheses that anxiety measures would be differentially correlated with CU and I/CP. It was also hypothesized that CU and anxiety would interact to predict proactive aggression and a deficit in fear processing, while I/CP and anxiety would interact to predict reactive aggression and higher sensitivity in fear processing. The study used a community sample (n=88) of boys aged 7-11 and selected for their high levels of externalizing behaviors. Correlational analyses provided partial support for the hypothesis that anxiety was positively correlated with I/CP, but no significant correlations were found between CU and anxiety measures. Significant main effects of psychopathy subscales and anxiety were found in the prediction of reactive aggression, overall aggression, fear processing, and rule-breaking. However, regression analyses testing for hypothesized interaction effects revealed largely nonsignificant results. Findings are discussed in the context of developmental views on psychopathy and implications for the development of subtypes.

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Abstract

Research in adult populations has indicated the presence of psychopathy subtypes largely differentiated by the presence of trait anxiety, which often correlates divergently with the affective/interpersonal component of psychopathy (CU) and the behavioral component (I/CP). Studies in children have demonstrated similar trends and indicate that emotional processing differences may underlie psychopathy and different types of associated aggression, specifically proactive and reactive aggression. The current study tested the hypotheses that anxiety measures would be differentially correlated with CU and I/CP. It was also hypothesized that CU and anxiety would interact to predict proactive aggression and a deficit in fear processing, while I/CP and anxiety would interact to predict reactive aggression and higher sensitivity in fear processing. The study used a community sample (n=88) of boys aged 7-11 and selected for their high levels of externalizing behaviors. Correlational analyses provided partial support for the hypothesis that anxiety was positively correlated with I/CP, but no significant correlations were found between CU and anxiety measures. Significant main effects of psychopathy subscales and anxiety were found in the prediction of reactive aggression, overall aggression, fear processing, and rule-breaking. However, regression analyses testing for hypothesized interaction effects revealed largely nonsignificant results. Findings are discussed in the context of developmental views on psychopathy and implications for the development of subtypes.

Keywords: psychopathy, youth psychopathy, anxiety, proactive aggression, reactive aggression, preconscious fear processing

Psychopathic Traits and Anxiety
As Predictors of Aggression and Fear Processing in Young Males

Psychopathy has been thoroughly researched as a construct useful in understanding pathways toward antisocial behavior. Currently defined as a persistent personality constellation characterized by lack of guilt and empathy, callous interpersonal style, narcissism, and impulsive behavioral style (Kimonis, Frick, Fazekas & Loney, 2006), psychopathy has also been studied among children with antisocial tendencies. Findings have begun to identify more homogeneous groups of antisocial youth with affective, behavioral, and etiological distinctions. Such findings can inform interventions, addressing problem behaviors that pose a heavy burden on society and families (Brinkley, Newman, Widiger, & Lynam, 2004). Although most studies have used clinical and delinquent youth populations, community studies in adults have found that indicators of psychopathic personality style also appear in the general population. Examining the underlying processes of psychopathic traits bears relevance for understanding problem behaviors in several populations.

Psychopathic Subtypes: Studies in Adult Populations

Heterogeneity in the construct of psychopathy has posed a quandary for researchers since the syndrome's seminal formulation by Hervey Cleckley (1941). Cleckley and several subsequent researchers viewed the syndrome as a unitary construct with a distinct etiology and particularly high risk for aggressive, antisocial, and criminal behavior. Scores on diagnostic scales for psychopathy, most notably on the commonly used Psychopathy Checklist-Revised (PCL-R; Hare, 1991) do indeed predict criminal and antisocial activity more strongly than does a diagnosis of Antisocial Personality Disorder (ASPD). Furthermore, key behavioral correlates of psychopathy remain constant regardless of race or gender (Hart, 1998; Smith & Newman, 1990). Of note, however, a variety of laboratory procedures examining the physiological correlates of psychopathic traits (e.g. reduced skin conductance and startle response to aversive stimuli) (Arnett, 1997; Hare, 1978; Lykken, 1957; Ogloff, 1990; Patrick, Bradley, & Lang, 1993; Patrick, Cuthbert, & Lang, 1994) have elicited controversy concerning the veracity of low

anxiety as one of the core deficits in psychopathy. If all psychopaths have low anxiety, as Cleckley theorized, and this trait contributed to their inability to learn from punishment, one would expect all high-scorers on the PCL-R to have physiological manifestations of abnormally low anxiety and negligible response to intervention or punishment, indicating a constitutional deficit that produced such behavior. However, this is not the pattern of findings that has been noted in the empirical literature.

An early, influential explanation for variance in anxiety levels, affective deficits, and outcomes for those with psychopathic behavior was proposed by Karpman (1941) at the same time Cleckley published *The Mask of Sanity*. Karpman proposed that an underlying character neurosis could produce antisocial behavior similar to that seen in psychopathy. This type of individual, called a secondary or "neurotic" psychopath, was etiologically distinct from the primary psychopath defined by Cleckley. Whereas the callous and inadequately motivated antisocial behavior of the primary psychopath could not be traced to environmental causes, the secondary psychopath was likely to have experienced parental rejection and harsh punishment as a child. Karpman theorized that the secondary psychopath's antisocial behavior was more impulsive and reactive when violent or aggressive. These individuals were not deficient in affect and conscience, as primary psychopaths were, but acted similarly due to an affective disturbance and high levels of anxiety. At the time rejected by Cleckley, who believed that the syndrome was characterized by low anxiety underlying its behavioral components, Karpman's conceptualization has provided the basis for several recent studies addressing heterogeneity in psychopathy. Although research on psychopathy subtypes lacks etiological data to support the secondary psychopath conceptualization, findings on associated personality traits, clinical features, affective and cognitive deficits, and several other factors suggest distinct etiologies that underlie the psychopathy subtypes. A review of these research approaches in adult populations lays the groundwork for the possibility of studying such characteristics and distinctions in children.

While there is a precedent for differentiating primary and secondary psychopaths based on levels of trait anxiety (Skeem, Kerr, Johansson, Andershed, & Louden, 2007), more recent studies have used

broader approaches and thereby produced stronger, richer findings regarding the complex nature of subtypes. One approach is based on the two dimensions underlying psychopathy. The affective dimension (Factor 1 in the PCL-R) assesses a callous and remorseless use of others and deficient affective experience, while the behavioral dimension (Factor 2 in the PCL-R) assesses impulsivity, irresponsibility, and a deviant, antisocial lifestyle (Brinkley et al., 2004; Harpur & Hare, 1988; Skeem et al., 2003). Lykken (1995) and Zuckerman (1995) proposed that different patterns of Factor 1 and Factor 2 scores in psychopathic individuals were related to the primary-secondary distinction (Blackburn, 2008). The predominance of Factor 1 is considered the hallmark of Cleckleyan primary psychopathy that distinguishes it from other chronic antisocial behavior, while Factor 2 would logically be high in Karpman's secondary psychopath. A major impact of trait approaches to the Factor 1/Factor 2 (primary/secondary) distinction is their findings on trait anxiety, which should be markedly higher in secondary psychopaths than in primary psychopaths, according to Karpman's formulation (Brinkley et al., 2004). Hare's (1991) finding that trait anxiety is negatively associated with Factor 1 and positively associated with Factor 2 has been extended by studies of personality, affect, and behavior.

Lynam and colleagues (1998, 2003, 2005) advocate the assessment of psychopathy factors using the Five Factor Model (FFM), a useful model due to extensive studies in various populations that support its truly integrative, general assessment of personality (Brinkley et al., 2004; Costa & McCrae, 1988). Lynam and Widiger (1998) proposed that PCL-R domains assessing Factor 1 were associated with high antagonism and low neuroticism, while Factor 2 domains were more strongly characterized by low conscientiousness and high neuroticism, with some indications of high antagonism. These propositions are consistent with Hare's findings regarding trait anxiety, which is encompassed by the negative affectivity aspect of neuroticism. Findings supporting this conceptualization were found in a non-referred undergraduate sample (Miller & Lynam, 2003) and a sample of adolescent boys assessed in a longitudinal design (Lynam et al., 2005). If Factor 2 is considered an indicator of secondary psychopathy, Lynam et al.'s (2005) associations of Factor 2 with low conscientiousness and high neuroticism are consistent with

Karpman's conceptualization of the secondary psychopath, whose deviant and irresponsible lifestyle is theoretically based on character neuroses and negative affectivity.

A study by Blackburn, Logan, Donnelly, and Renwick (2008) used the Antisocial Personality Questionnaire (APQ) factors of impulsivity and withdrawal, parallel constructs to Factor 1 and Factor 2, to distinguish between primary and secondary psychopaths in a male forensic sample diagnosed as psychopathic by the PCL-R. Profiles derived from the APQ show that primary and secondary psychopaths are both high on the impulsive factor of aggression, but primary psychopaths are less withdrawn, displaying extraversion, self-confidence, and low to average anxiety. Secondary psychopaths, on the other hand, are introverted, socially anxious, and moody. This distinction follows Karpman's conceptualization quite closely and was mirrored by Blackburn et al.'s (2008) findings with other measures. Secondary psychopaths displayed more trait neuroticism, introversion, experience of abuse, and comorbid psychopathology. These findings support the behavioral similarity and distinct affective and etiological correlates of primary and secondary psychopathy.

The primary and secondary psychopathy distinction may be clinically useful in that psychopathy factors are differentially related to internalizing and externalizing psychopathology. A recent study by Blonigen et al. (2010) assessed the clinical utility of self-reported psychopathy in a large sample of offenders. Externalizing symptomology, a latent variable comprising aggression and substance abuse, has been associated with both Factor 1 and Factor 2 on the PCL-R but with stronger positive associations with Factor 2, perhaps due to associated personality traits of disinhibition. Internalizing symptomology, on the other hand, was expected to be more prevalent in secondary psychopaths due to their increased vulnerability to anxiety, depression, and other signs of withdrawal and negative emotionality. Researchers administered the Psychopathic Personality Inventory (PPI), a self-report measure with two higher order factors corresponding to the affective-interpersonal criteria of Factor 1 (PPI-1) and the social deviance in Factor 2 (PPI-2). As hypothesized, internalizing behavior was negatively associated with the affective-

interpersonal factor and positively associated with social deviance as assessed by both the PPI and the PCL-R. Social deviance on both scales was positively associated with externalizing behavior.

Findings on correlations with personality and clinical symptoms highlight the role of anxiety, associated with neuroticism and internalizing symptoms, as a point of divergence in psychopathy factors. Findings from laboratory tasks and physiological measures also support this underlying distinction; Lykken (1957) found skin conductance lower than normal ranges in reaction to anxiety-evoking situations only in primary psychopaths, and Newman and Schmitt (1997,1998) found differences associated with trait anxiety in reward and punishment tasks among criminal psychopaths. Studies using behavioral self-report and laboratory tasks also help shed light on the impulsivity aspect of this distinction. Ray et al. (2009), for example, found in a drug treatment sample that PPI-2 was more strongly related to impulsivity-related traits than was PPI-1, including urgency, lack of premeditation and perseverance, and sensation-seeking. PPI-2 was also significantly associated with negative emotionality, while PPI-1 had a small, nonsignificant negative association. The authors suggest speculatively that the urgency component of impulsivity in secondary psychopaths may be related to violent reactions to negative emotions.

Anestis, Anestis, and Joiner (2009) expanded upon the role of negative urgency, the drive to quickly reduce negative affect, in primary and secondary psychopathy in an undergraduate sample. They found that negative urgency was positively related to secondary psychopathy scores on the Levenson Psychopathy Scales (1995), a finding that suggests a relationship between the negative affectivity and anxiety of secondary psychopaths and their impulsive behavior. This relationship does not appear to hold true for primary psychopaths. Experimental studies by Wilkowski and Robinson (2008) and Ali, Smarim, and Chamorro-Premuzic (2009) suggest that low cognitive control, with normal to high affective reactivity, may underlie the secondary psychopath's impulsive tendencies and inability to modulate maladaptive behavior.

In summary, the accepted two-factor structure of psychopathy suggests the influence of two distinct processes underlying the affective and behavior components of the psychopathic syndrome.

Divergent correlations of these two factors correspond to the primary and secondary subtypes respectively outlined by Cleckley (1941) and Karpman (1941). Personality and clinical approaches to this divergence highlight the association of secondary psychopathy with negative affectivity, increased comorbid psychopathology, particularly of the internalizing type, and environmental influences. Experimental and self-report findings have differentiated between affective and cognitive deficits in primary and secondary psychopathy and support Karpman's (1941) conceptualization in that secondary psychopaths' capacity for negative affect is intact, indicating that they have a disturbed conscience rather than an absent one. More recently a third factor has been proposed, comprising an arrogant and deceitful interpersonal style separate from the deficient affective experience (Cooke & Michie, 2001); correlational studies of traits and anxiety measures with this narcissistic factor have yet to be conducted.

The neurally based behavioral systems proposed by Gray (1976), called the Behavioral Inhibition System (BIS) and Behavioral Activation System (BAS) are also theorized to be differentially related to primary and secondary psychopathy. These systems may influence antisocial behavior through low inhibition, or failure to adjust behavior due to adverse consequences, and/or high activation, which drives behavior through increased sensitivity to potential rewards. The BAS facilitates responses to reward and nonpunishment cues and initiates behaviors through appetitive motivation (Newman et al., 2005). The BIS, on the other hand, is sensitive to punishment cues and facilitates passive avoidant behaviors by causing anxiety. The BIS/BAS system is assessed through self-report measures of associated behaviors and/or through physiological measures. Findings in adult populations suggest the presence of diminished BIS activity in primary psychopathy and of strong BAS in secondary psychopathy, supporting BIS and associated anxiety and negative affectivity as distinguishing factors of the secondary psychopath as conceptualized by Karpman (1941) (Hundt, Kimbrel, Mitchel, & Nelson-Gray, 2009; Newman, MacCoon, Vaughn, & Sadeh, 2005; Ross, Molto, Poy, Segarra, Pastor, & Montanes, 2007; Wallace, Malterer, & Newman, 2009). Furthermore, Newman and colleagues (2005) found that BAS activity characteristic of secondary psychopaths was associated with trait anxiety, and Wallace and colleagues

(2009) found that BAS functioning was positively associated with PCL-R Factor 2 while BIS functioning was negatively associated with PCL-R Factor 1.

Finally, some studies have combined these assessments of primary/secondary distinctive traits through the advanced technique of model-based cluster analyses to attain a more comprehensive understanding of psychopathy subtypes and their external correlates. These studies derive clustering variables from Karpman's (1941) conceptualization and have found support for the existence of his proposed subtypes. Clusters with characteristics of secondary psychopathy from both clinical and subclinical populations have displayed, in comparison to primary psychopaths, higher trait anxiety (Skeem et al., 2007), higher internalizing and externalizing psychopathology, impulsivity, and violent recidivism (Poythress et al., 2010), higher BAS functioning, and more frequent hostile, reactive aggression than proactive, instrumental aggression (Falkenbach, Poythress, & Creevy, 2008).

The literature described provides strong support for the existence of two subtypes of psychopathy with distinct behavioral, affective, and cognitive correlates. Given the etiological basis of Karpman's conceptualization and the paucity of etiological studies of psychopathy, studies of psychopathic characteristics in children are important for understanding the development of primary and secondary psychopathic traits.

The Assessment of Psychopathy Factors in Children and Adolescents

Findings from several studies have supported the importance of assessing psychopathic traits in youth. These traits appear to indicate more severe conduct problems and specific affective and cognitive characteristics of etiological significance (Salekin & Frick, 2005). Although research as early as that of Cleckley (1941) recognized the likelihood that psychopathic traits began early in life, the study of psychopathic traits in youth has been sporadic due to construct confusion and fear of mislabeling children with the pejorative term of "psychopath" (e.g. Quay, 1987). A systematic approach to assessment in children arose in the 1990's and has encouraged an increasing number of studies since then. The importance of such research in a forensic setting was evidenced recently by findings from Salekin and

colleagues (2008) who noted that among a sample of children and adolescents at a court assessment unit, psychopathy scores predicted both general and violent recidivism. In addition, some support has been found for psychopathy scores' improved predictive validity over disruptive behavioral disorder diagnoses (conduct disorder and oppositional defiant disorder) in regard to violent and nonviolent offenses in adolescence (Salekin, Neumann, Leistico, DiCicco & Duros, 2004).

Frick and colleagues (1994) developed a factor structure in child psychopathy parallel to that of adults, in which an impulsivity/conduct problems (I/CP) factor comprised impulsiveness, behavioral deviance, and inflated self-importance, and a callous-unemotional factor (CU) comprised interpersonal callousness and emotional insensitivity. In later work, Frick, Boden and Barry (2000) found support for dividing the I/CP factor into an "impulsive" and a "narcissistic" component. The CU factor of child psychopathy has been particularly influential in delineating a more severe subgroup within youth displaying conduct problems. These youth are thought to have deficits in behavioral inhibition that contribute to their undersocialization (Frick, 1998). If the CU factor is seen as the youth parallel to Factor 1, the interpersonal-affective factor of adult psychopathy, distinct correlates of child psychopathic factors may bear important implications for the study of etiological subtypes.

Like Factor 1 and Factor 2 in adults, the CU and I/CP factors demonstrate divergent correlations in a number of areas in children and. Lynam, whose correlational research on psychopathic traits and the FFM in adults has been cited as support for secondary psychopathy (e.g. Brinkley et al., 2004), extended his work to adolescents and found similar results (Lynam et al., 2005). By administering the Childhood Psychopathy Scale to a large sample of high-risk 13- and 16-year old boys, Lynam et al. replicated the finding that neuroticism was negatively associated with the CU dimension (Factor 1) and positively associated with the I/CP dimension (Factor 2). They theorize that low neuroticism and low agreeableness could underlie the emotional detachment of the CU dimension, while high neuroticism may underlie impulsivity and hostility.

Studies also demonstrate differences in social and cognitive processes underlying the two factors

of child psychopathy. In a sample of adjudicated youth, Pardini, Lochman and Frick (2003) found that the two factors differed in their correlations to fearfulness and emotional distress. CU traits were associated with increased positive emotion and focus on potential reward resulting from deviant behavior. The authors explain that an emotional processing deficit may buffer against personal distress resulting from deviant behavior and its negative consequences. The I/CP dimension, on the other hand, was positively associated with measures of fearfulness and personal distress. Similar findings were reported by Blair (1999) and Sharp, Van Goozen, and Goodyer (2006). In addition, reduced reactivity to distressing emotional stimuli was found to be associated with proactive aggression (Kimonis, Frick, Fazekas & Loney, 2006). As in the adult literature on psychopathy, Gray's BIS/BAS model has been applied as a possible basis for such distinctions. Assuming that both high impulsivity and low anxiety make young adults and children prone to deviant behavior, researchers examined associations of BIS and BAS measures to primary and secondary psychopathy in a sample of young adults (Ross, Molto, Poy, Segarra, Pastor & Montanes, 2007). While impulsivity, through positive associations with BAS measures, was seen in both subtypes, weak BIS was associated only with primary psychopathy. This distinction indicates that low trait anxiety may delineate a crucial difference between primary and secondary psychopathy in younger populations.

A notable trend in the comparison of adult literature and child/adolescent literature on psychopathy lies in the consistency of cognitive associations with psychopathic traits. In a review of neurocognitive models of psychopathy, Blair (2010) notes that impairment in the processing of emotional stimuli in reversal learning paradigms is more pronounced in adults than in adolescents when highly salient contingency changes of reward and punishment frequency are implemented; more overlap between age groups is seen when contingencies are less salient. Blair proposes that genetic underpinnings of psychopathy affecting the orbitofrontal cortex may do so in a progressive fashion as the child ages.

Regardless, such differences in the associations of psychopathic factors suggest that more homogeneous groups with distinct etiological bases may exist within the population of psychopathic

children and adolescents who display conduct problems. White and Frick (2010) highlight the significance of dividing the population according to the presence or absence of CU traits, because such traits are often associated with severe antisocial behavior. A review of several studies with independent samples found evidence of a predictive relationship between CU traits and aggressive, antisocial, and delinquent behavior, as well as an association with poorer treatment outcomes in community, clinic-referred, and forensic samples (Frick & Dickens, 2006). In studies of the association between dysfunctional parenting and conduct problems, findings also support a distinct etiology of CU traits. Wootton, Frick, Shelton and Silverthorn (1997) found in both nonreferred and clinic-referred youth that dysfunctional parenting practices, including low involvement, failure to use positive reinforcement, poor monitoring and supervision, inconsistent discipline, and corporal punishment, were strongly associated with conduct problems only in children with low CU traits. Edens, Skopp, and Cahill (2008) reported similar findings in a sample of juvenile offenders, specifically examining harsh and inconsistent discipline.

These research findings in youth correspond with the differences in psychopathic subtypes proposed by Karpman (1941). Given Karpman's emphasis on heightened anxiety in secondary, but not in primary psychopaths, it is important to examine the role of anxiety in children who display CU traits versus those who do not. Kochanska (1993) proposed that a subtype of children with antisocial behaviors lacks deviation anxiety, or negative arousal following wrongdoing and punishment, and that this low level of arousal impedes conscience development. This proposal may explain associations of psychopathy subtypes with measures of trait anxiety in the adult literature. If trait anxiety plays a role in different subtypes of psychopathic and antisocial individuals, it is also important to examine correlates of clinical symptoms of anxiety in youth. Studies of anxiety, both in trait form and clinical symptomology, and its correlations with antisocial behavior are described next.

Anxiety, Conduct Problems and Pathways to Antisocial Behavior

Psychopathy in youth is highly comorbid with the disruptive behavior disorders (DBDs), conduct disorder (CD) and oppositional defiant disorder (ODD). These in turn co-occur with other types of externalizing and internalizing psychopathology, including anxiety disorders (Lynam, 1997; Vincent &Hart, 2002; Seagrave & Grisso, 2002). Examining the discriminant validity of psychopathy amidst other forms of child psychopathology addresses Karpman's (1944) concerns regarding other forms of psychopathology resulting in psychopathy-like symptoms (Salekin, Neumann, Leistico, DiCicco & Duros, 2010). Using several measures of youth psychopathy in a sample of child and adolescent offenders (n=130), Salekin et al. (2010) found that psychopathy was indeed highly correlated with the DBDs, but that youth demonstrating symptoms of psychopathy also experienced symptomatology not included in Cleckley's original formulation, including depression, adjustment problems, and several forms of anxiety.

The finding that anxiety co-occurs with conduct problems is not an unexpected one. Several studies with clinical, forensic, and community samples have found higher rates of anxiety disorders in children and adolescents with antisocial tendencies than in samples without such behaviors (e.g. Robins & Price, 1991; Zoccolillo, 1992). Sareen, Stein, Cox & Hassard (2004) examined antisocial diagnoses and anxiety disorders in two large community surveys of adults (n=5877, 8116) and found that 36% of respondents with an antisocial diagnosis in one sample, as well as 47% of respondents in the other sample, also had a lifetime anxiety disorder. Respondents with this comorbidity were more likely to encounter problems such as alcohol or substance use disorders, depression, poor quality of life, and general impairment compared to respondents with one or neither diagnosis. Although all anxiety disorders were significantly associated with antisocial diagnoses, social phobia and posttraumatic stress disorder had the strongest associations. The authors provide multiple explanations for their findings. For example, socially anxious people may deceive others to avoid direct confrontation, or a common factor of high neuroticism might contribute to both anxiety and antisocial behavior, particularly the facet of impulsivity.

The latter explanation for the association between anxiety and antisocial behavior appears more fitting for children with co-occurring conditions. In a review of conduct disorder and comorbid conditions, Loeber and Keenan (1994) cite mixed findings on the impact of DSM-III-R anxiety disorders on CD. Walker et al. (1991) found that in a clinical sample of boys with CD, a comorbid anxiety disorder, particularly overanxious disorder, was associated with markedly fewer impairments, particularly in social functioning. Walker applies Gray's BIS/BAS model to the reduced deviance of these boys, explaining that anxiety is a direct manifestation of the BIS and contributes to inhibition of antisocial behavior. Campbell and Ewing (1990), however, found no significant difference in groups with and without anxiety disorders.

Anxiety disorders that commonly have their onset in childhood are separation anxiety and overanxious disorder (reclassified as generalized anxiety disorder in the DSM-IV), which may appear as early as preschool and decrease in prevalence with age (Cohen et al., 1993). Between 2% and 21% of children referred for anxiety also display externalizing behaviors (Russo & Beidel, 1994), with rates of comorbidity being highest in middle childhood and decreasing in adolescence. Evidence for very early comorbidity of anxiety and conduct problems was reported by Gregory, Eley and Plomin (2003), who conducted a large study with twins aged 2-4 (n=6783) and found that anxiety and conduct problems as reported by parents were correlated at .33 for boys and .30 for girls. Furthermore, genetic correlations for both anxiety and conduct problems were fairly low, although they were higher for boys than for girls. Higher correlations were found for shared environmental factors, which include parent-child relationships.

One underlying factor in the co-occurrence of anxiety and conduct problems is emotional reactivity. Frick and Morris (2004) reviewed temperamental vulnerabilities for the development of conduct problems, noting that both high and low levels of emotional reactivity may contribute to conduct problems. Temperamental vulnerabilities to conduct problems are typically associated with earlier onset and stable patterns of aggression in children (Brennan, Hall, Bor, Najman & Williams, 2003; Moffitt &

Caspi, 2001). Hence, differences in temperament, especially with regards to emotional reactivity for negative emotions, may delineate subgroups of aggressive children. For example, reactive aggression, a form of response to real or perceived provocation, is more strongly associated with angry reactivity than is proactive aggression, which is displayed to achieve a goal (Hubbard et al., 2002; Shields & Cicchetti, 1998). Intense emotional reactions of anger and frustration associated with conduct problems can inhibit socialization and contribute to long-term patterns of conduct problems and antisocial behavior; for example, children with high negative affect may have difficulty internalizing parental norms because of a high emotional reaction to discipline (Kochanska, 1993, 1995, 1997).

Proactive aggression, according to Frick and Morris (2004) is associated with low levels of emotional reactivity. Whereas reactive aggression is often retaliatory or impulsive, proactive aggression is unprovoked and aimed toward personal gain or coercion of others. Proactive aggression and covert conduct problems are associated with low levels of emotional arousal and may be a form of thrill-seeking behavior. Frick and Morris posit that such characteristics are consistent with the CU dimension of psychopathy, which is also associated with stable patterns of conduct problems because of inhibited conscience development. Although negative arousal at dysfunctional levels can lead to conduct problems, is also necessary for the internalization of norms and values (Kochanska, 1991). The low emotional reactivity of children with high CU traits can prevent “deviation anxiety,” or guilt and anxiety associated with wrongdoing. Like adult offenders, juvenile offenders with high CU traits are likely to have more severe and repeated patterns of violence, possibly due to a deficit in their capacity for negative emotional reactivity and anxiety.

Raine et al. (2006) also conducted a study that supports differential associations of proactive and reactive aggression in which a sample of 335 youth was assessed at age 7 and 16. At age 7, participants who were proactively aggressive at follow-up were characterized by initiation of fights, poor school motivation, several indicators of troubled family life, delinquency, and hyperactivity; at age 16 proactive aggression was associated with psychopathic personality, blunted affect, and serious violent offending.

Reactive aggression, on the other hand, was associated at age 16 with impulsivity, hostility-aggression, social anxiety, lack of close friends, sensation seeking, and early schizotypal indicators such as unusual perceptual experiences and paranoid ideation. These divergent associations coincide with distinctions of conduct problem youth with and without anxiety in terms of conduct and temperament.

Blair (2001) offers a cognitive neuroscience perspective for the basis of reactive and proactive aggression involved in psychopathy. Orbitofrontal cortex control over reactive aggression, according to the social response reversal hypothesis, operates through response to social cues, particularly expressions of anger. Processing of these expressions in others involves representations of previous social scenarios and assessment of, for example, hierarchical position in a modulation of reactive aggression. Judgments about an aggressive reaction are thus based on expectations of the other's social behavior and can be dissociated from a purely reward-punishment basis. Reward dominant individuals with less responsiveness to negative emotional valence in facial expressions, then, might be less likely to undergo this modulatory process. Although psychopathic individuals usually respond normally to angry facial expressions, their judgments regarding appropriate social behavior may be impaired. Blair proposes variations in individuals' violence inhibition mechanism as underlying instrumental aggression in psychopathic individuals. This mechanism responds to sad and fearful faces, and moral socialization occurs when these distress cues are paired with a representation of the act that caused them, such as an act of instrumental aggression. Instrumental aggression fails to be inhibited in psychopathic individuals because this moral conditioning does not occur, and autonomic response to sad and fearful faces is muted. Taken together, Blair's proposals suggest that an intact response to threat cues and inadequate modulation of social responses in psychopathic individuals may contribute to reactive aggression, while muted response to sad and fearful faces contributes to a failure of empathic conditioning that may lead to underlie instrumental or proactive aggression. Differential impairments in these mechanisms may lead to different frequencies of reactive and proactive aggression associated with primary and secondary psychopathic traits.

Although fearfulness and anxiety are more often associated with internalizing behaviors than with externalizing behaviors, such traits may also indicate underlying high levels of negative emotionality. Therefore examining their neural mechanisms and their association with externalizing behaviors is useful. In a review of brain mechanisms, emotion, and motivation associated with anxiety, Lang, Bradley, and Cuthbert (1998) compare characteristics of anxiety disorder patients to those of psychopaths and suggest important differences in their motivational systems. They review the paradigm of emotional picture processing and psychophysiology of anxiety disorder. The defensive startle reflex in response to emotionally valenced pictures is viewed as an indication of appetitive and aversive motivational systems, such that a heightened startle reflex occurs when the aversive system (BIS) is activated, as in fear and anxiety states, and an attenuated startle reflex occurs if the appetitive system (BAS) is activated. Several anxiety disorders, including panic disorder and PTSD, are associated with this startle sensitivity, which is also associated with negative affect.

Furthermore, Thomas et al. (2001) examined amygdala response to neutral and fearful faces in anxious and depressed children using functional and structural Magnetic Resonance Imaging (MRI). They found that children with anxiety disorders exhibited a heightened amygdala response to fearful faces in comparison to neutral faces, while depressed children had a blunted response to fearful faces. This finding suggests that heightened sensitivity to fearful expressions is a characteristic of anxiety and not a broader internalizing dimension.

O'Brien and Frick (1996) assessed reward dominance in association with anxiety, conduct problems, and psychopathy in clinically-referred children and a control group (n=132) using a task of competing rewards and punishment. Reward dominance is associated with increased BAS activity, while anxiety manifested in clinical levels is indicative of BIS activity. As expected, conduct problem diagnoses were significantly associated with anxiety disorders, and a group with comorbid diagnoses was compared to a conduct problem group without anxiety disorders. In addition, subjects with high CU traits were grouped into those with and without a co-occurring anxiety disorder consisting of both clinical and

normal sample subjects. Researchers found that non-anxious children with conduct problem diagnoses had significantly higher reward dominance than did anxious children, supporting the idea that low trait anxiety indicates a subgroup of children with conduct problems. Similarly, when psychopathic traits were analyzed, these alone were not related to reward dominance until anxiety was taken into account. Children with psychopathic traits and low anxiety exhibited the most reward dominance. O'Brien and Frick (1996) interpreted these findings as a suggestion that BIS activity in anxious children with conduct problem diagnoses and/ or psychopathic traits may counteract reward dominant tendencies. These findings run counter to those of Newman et al. (2005) in adults, in which BAS activity was positively associated with trait anxiety. O'Brien and Frick's (1996) finding indicates that in children, the interaction of anxiety and psychopathic factors may not necessarily result in very aggressive or antisocial tendencies as they might in secondary psychopathic adults, perhaps because BAS-related reward dominant tendencies are more malleable in younger individuals than in older, consistently antisocial individuals. Aggression that does occur in anxious children with psychopathic traits, according to Raine et al. (2006), is likely to be reactive and impulsive and nature.

Similarly, Vitale et al. (2005) examined behavioral inhibition in children with low anxiety and high psychopathic traits. They proposed poor response modulation as underlying psychopathic individuals' tendencies toward poor passive avoidance and other behavioral disinhibition. With a community sample of both males (n=164) and females (n=144) all aged 16, researchers administered a passive avoidance task and a picture word Stroop task to assess the activity of automatic response modulatory processes. Their results, which replicated those found in low-anxious psychopathic adult offenders, showed that low-anxious, high-psychopathic boys and girls displayed more passive avoidance errors and reduced interference from modulatory processes compared to low-anxious, low-psychopathic youth. This reduced response modulation could make them likely to engage in antisocial behavior because they are less likely to reflect on their behavior and to alter it in response to environmental

feedback, possibly interfering with socialization and increasing risk for adult psychopathy (Newman, 1998).

Finally, a study conducted by Frick, Lilienfeld, Ellis, Loney and Silverthorn (1999) analyzed associations between trait anxiety and psychopathy dimensions in children, also proposing subgroups of antisocial youth distinguished by differing anxiety levels. 143 clinically-referred children, aged 6-13 and predominantly male, completed the DISC diagnostic interview and measures for trait anxiety, childhood psychopathy, and fearlessness. While the I/CP dimension of psychopathy exhibited positive correlations with trait anxiety, CU traits had nonsignificant negative correlations with these measures. There was a moderate significant correlation between the two psychopathy dimensions. Therefore researchers assessed correlations of each dimension while controlling for the other. Notably, negative associations between CU traits and trait anxiety became significant when controlling for the I/CP dimension, and associations between I/CP and trait anxiety became more strongly positive when controlling for the CU dimension. These results indicate that the correlation between psychopathy dimensions, if not statistically controlled, may suppress divergent correlations between these domains and anxiety.

The Current Study

Given the strong support for the existence of subtypes in youth with conduct problems and antisocial behaviors, and the associations of psychopathic dimensions and anxiety measures in these youth, the current study aims to further investigate processes that underlie aggression and conduct problems in these youth. It tests whether subclinical manifestations of anxiety disorders in youth have the same associations with psychopathic traits and conduct problems that have been demonstrated with trait anxiety. Past studies have used experimental tasks such as picture processing and computerized card games as indicators of BIS and BAS activity, but no study has yet examined preconscious processing of fear in pictures of others. The preconscious level of processing may indicate another source of the distinction between an impulsive, anxious subtype of conduct problem youth and a high CU, less anxious subtype. Sensitivity to fearful faces as evidenced by a short reaction time in a preconscious processing

task may serve as an index of BIS sensitivity to punishment cues, which is experienced as anxiety at a conscious level. This proposition is in keeping with findings reviewed by Frick and Dickens (2006) in which antisocial youth without CU traits exhibit emotional dysregulation through self-reported anxiety, reactivity to the distress of others in social situations, and general reactivity to negative emotional stimuli (Loney et al., 2003; Kimonis et al., 2001). On the other hand, a deficit in sensitivity to fearful expressions, such as that seen in previous studies of CU traits, may indicate weakened BIS input, which would allow BAS sensitivity to reward cues to dominate behavior in the absence of deviation anxiety. We propose that mixed findings regarding BAS activity and externalizing behaviors as indicators of secondary psychopathy, in which similar outcomes for these dimensions are sometimes found for primary and secondary psychopaths, are due to the failure of previous studies to differentiate between types of aggressive outcomes. Negative emotionality and impulsivity typical of youth with secondary psychopathic traits have also been associated with reactive aggression, perhaps due to increased threat sensitivity that could be evidenced by sensitivity to fearful faces, as well as negative urgency (Anestis et al., 2009). Lower levels of emotional reactivity found in association with CU, on the other hand, are associated with proactive aggression (Kimonis et al., 2006). We predicted that proactive aggression would be highest in a distinct subset of high-CU children distinguished by low anxiety, indicating particularly low emotional reactivity. Additionally, both parent and child measures of several study variables are employed to reduce effects of reporter bias.

Specifically, the current study tests the following hypotheses:

1. The I/CP dimension of psychopathy will be positively associated with anxiety when controlling for the CU dimension.

2. The I/CP dimension and high levels of anxiety will interact to predict a) high preconscious fear processing and b) reactive aggression.

3. High preconscious fear processing will mediate the relationship between I/CP and anxiety with reactive aggression.

4. The CU dimension of psychopathy will be negatively associated with anxiety when controlling for the I/CP dimension.

5. The CU dimension and low levels of anxiety will interact to predict a) low preconscious fear processing and b) proactive aggression.

6. Low preconscious fear processing will mediate the relationship between CU and anxiety with proactive aggression.

Method

Participants

Data were gathered as part of a larger study (see Sylvers, in press) using a community sample of 88 boys aged 7 to 11 years (m=8.88, SD=0.98). The sample was 45.5% (n = 40) Caucasian, 44.3% (n = 39) African American, 6.8% (n = 6) Asian, and 3.4% (n = 3) Hispanic. Flyers were mailed to 15,000 families living in the Greater Atlanta metropolitan area and posted at university-affiliated medical clinics, Boys and Girls Clubs, and YMCA's. These recruitment flyers indicated that the study was looking to study families with 8 - 10 year old sons who were "handfuls" and got into trouble at home and school. If parents consented to the study, the study was explained to the boys and their assent was requested. Diagnostic interviews and screening measures indicated that 6.8% of the sample (n=6) was positive for conduct disorder; 10.2% (n=9) was positive for attention deficit hyperactivity disorder; 4.5% (n=4) was positive for oppositional defiant disorder, and 44.8% (n=39) met screening cutoffs for a possible anxiety disorder. For the DBDs, higher percentages of children in our sample fell in the intermediate diagnostic range as compared to the positive diagnostic range. Exclusionary criteria included severe asthma, heart conditions, autism spectrum disorders, bipolar disorder, and mental retardation. This research was approved by the Emory University institutional review board.

Measures

Questionnaires.

Aggression Scale (AS; Orpinas & Frankowski, 2001). The AS is an 11-item self-report questionnaire that measures a child's overt aggressive behavior, including verbal (e.g., I threatened to hurt or hit someone) and physical (e.g., I pushed or shoved other students) aggression. Respondents are asked to indicate how many times that they have engaged in the behaviors over the previous week. The 2-year stability of the AS is moderate to high, with values ranging from 0.50 to 0.63. Studies have found positive associations between AS scores and school violence and weapon carrying (Escobar-Chavez, Tortolero, Kelder, & Kapadia, 2002). Internal consistency for the AS was adequate in this sample (Cronbach's α = 0.74). For the purposes of the current study, proactive and reactive subscales were constructed following Washburn, McMahon, King, Reinecke, & Silver (2002) based on operational definitions of reactive and proactive aggression by Dodge (1991). The reactive scale included four items describing aggressive reactions to anger, while the proactive scale included seven items describing purposeful, instrumental aggression. Internal consistency for the reactive subscale was also acceptable (Reactive: Cronbach's α =.72). Internal consistency for the proactive scale, however, was low, and results should be interpreted with caution (Cronbach's α=.58). As the AS proactive scores were skewed in this sample (SPSS skewness statistic=2.04), these data were natural log transformed to reduce skew (transformed SPSS skewness statistic = .47).

Antisocial Process Screening Device – Child and Parent Form (APSD; Frick & Hare, 2001). The APSD is a 20-item, 3-point Likert-type scale that assesses psychopathic traits in children. The item content of the APSD is based largely on the Psychopathy Checklist – Revised (PCL-R; Hare, 1991), a widely used and well validated measure of psychopathy in adult prison populations. Items from the APSD make up three subscales: impulsivity/conduct problems (ICP; e.g., acts without thinking); narcissism (NAR; e.g., brags excessively), and callous/unemotional (CU; e.g., does not show emotions). The NAR subscale was not assessed in the current study because there was no compelling evidence for types of aggression associated with narcissism. Several studies have found that the APSD possesses adequate internal consistency, test-retest reliability, and convergent validity with the PCL-R (e.g., Christian Frick,

Hill, Tyler & Frazer, 1997; Fite, Greening, Stoppelbein, & Fabiano, 2009; Frick & Hare, 2001; Lee, Vincent, Hart, & Corrado, 2003).

In this study, both parent and child versions of the APSD were administered. However, child-reported scores for CU and I/CP dimensions had low internal consistency. Mother reported CU and I/CP exhibited acceptable internal consistency (CU: Cronbach's α=.64; I/CP: Cronbach's α=.63). All APSD total and factor scores exhibited skewness within acceptable limits in this sample (SPSS skewness within +/- 1.0).

Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL is widely used in clinical and research settings. It includes 118 items on a 3-point Likert-type scale that assess overall psychological functioning in children. The current study used the problem items, which ask parents to rate children's behavioral and emotional problems by frequency. Several studies have found that the CBCL possesses adequate reliability and moderate to high correlations with other commonly used measures of childhood behavioral disorders (e.g., Achenbach, 1991). In this study, the Anxious/Depressed (Anx/Dep) syndrome scale raw score and Anxiety Disorders scale raw score were used as indices of anxiety. The Aggressive Behavior syndrome scale raw score was used as an index of general aggression. The Rule-Breaking raw score was also used in exploratory analyses of conduct problems. These scores served as exploratory outcome variables and a parent counterpoint to child reports of aggression. They could not be used in tests of the main hypotheses, as no distinction between reactive and proactive aggression is given on the CBCL.

Computerized Diagnostic Interview Schedule for Children (C-DISC; NIMH, 1996). The C-DISC is a widely used structured clinical interview that assesses DSM-IV Axis I diagnostic criteria in children. Psychometric studies of the C-DISC suggest adequate reliability and construct validity (Schwab-Stone et al., 1996). The ADHD, ODD, and CD modules were administered in this study.

Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher, Cully, Balach, Kaufman & McKenzie-Neer, 1997). The SCARED is a self-report instrument for children and their

parents that screens for DSM anxiety disorders. The SCARED includes five factors: somatic/panic, generalized anxiety, separation anxiety, social phobia, and school phobia. It has been found to be correlated with internalizing behaviors, state and trait anxiety in clinical populations, and to significantly distinguish between children with and without anxiety disorders (Monga et al., 2000). The SCARED total score and Generalized Anxiety Disorder (GAD) score were used as indices of child anxiety [in keeping with Frick et al. (1999), who operationalized trait anxiety as the total score for anxiety disorder symptoms as well as those specific to GAD. The total score exhibited adequate reliability in this sample (Cronbach's α=.74). The GAD score was also sufficiently reliable (Cronbach's α=.63).

Experimental task.

Modified Continuous Flash Suppression (mCFS) – The mCFS is a pre-attentive processing paradigm modified from Yang, Zald, and Blake's (2007) for use in children. It was used in this study to quantify fear processing. It measures participants' pre-attentive processing of neutral, happy, fearful, and disgusted facial expressions taken from the standard set of Ekman expressions (Ekman & Friesen, 1976). The Ekman images were cropped to remove all features outside of the face. The current paradigm was modified from the original paradigm by using NuVision 60GX (McNaughton Inc., Beaverton, OR) stereoscopic goggles rather than mirror stereoscopes.

The current paradigm also included a fixation cross in the middle of the stimulus area, which was designed to help participants focus on the appropriate area of the screen prior to beginning each trial. Stimuli were presented in the center of the video monitor (800 X 600 resolution) and were viewed against a uniform grey background. In the initial 1000 ms, one eye was presented with a full contrast dynamic mondrian image and the other eye viewed a face image, with increasing contrast at 2% every 20 ms. Once the face image reached full contrast (at 1s), the contrast of the mondrian image decreased at 2% every 100 ms for 5100 ms. The face image was presented in one of four quadrants in the stimulus square, and participants were asked to push a button (using a 4-button pad) corresponding to the quadrant that the face was presented in as soon as they recognized any part of a face in that quadrant. Prior to beginning the

experiment, the buttons and tasks were explained to participants, and participants were quizzed regarding which buttons corresponded to which quadrants. After participants exhibited an understanding of the task, the task was initiated. Trials were terminated when the participant pushed a button, and reaction time (RT) in ms and accuracy (correct quadrant versus incorrect quadrant) were recorded. The task consisted of 100 trials, with 25 repeated presentations of each stimulus type (neutral, disgust, fearful, and happy expressions). The overall accuracy was high in this sample (M = 93.99% correct, SD = 12.5%). Reaction times for all emotional expressions exhibited skewness within acceptable limits (SPSS skewness statistic within +/- 1.0).

Procedure

Upon entering the laboratory, study staff obtained the mother's consent. They then described the study to the child, answered any questions, and requested verbal assent. The mother and child were then taken to separate rooms and given walkie-talkies, which they could use to communicate at any point during the study. Mothers were also able to see but not to hear the child during the study. Mothers completed a battery of questionnaire measures by hand, while study staff verbally administered questionnaire and semi-structured interview measures to children. Child participants were reminded of the nature of each task before beginning, and task-specific assent was requested. Following the C-DISC interview and questionnaire measures, children completed the mCFS task as part of a series of other experimental tasks which were not assessed in the current study.

Results

Preliminary Analyses

Descriptive statistics for all variables are given in Table 1. Due to its association with aggressive behavior scores, we controlled for African American ethnicity (dummy coded as 0 or 1) in all analyses examining aggressive outcomes. Before calculating hypothesized correlations and interaction effects, intercorrelations of anxiety and aggression measures, as well as correlations between anxiety and aggression measures, were calculated (see Table 2). Child reports of anxiety on the SCARED were

positively but nonsignificantly correlated with parent reports of anxiety on the CBCL. Similarly, child reports of aggression on the AS were nonsignificantly correlated with parent reports of aggression on the CBCL, with one correlation between child report of proactive aggression and parent report of total aggression being in the negative direction (r=-.08, *p*=.50). Child reports of aggression were negatively but nonsignificantly associated with rule-breaking, as reported by mothers on the CBCL. Parent reports of aggression and rule-breaking, however, were significantly associated in the positive direction (r=.67, *p*<.001).

Correlations were also calculated for each of the measures with fear processing differences, as assessed by reaction time to fearful faces subtracted from reaction time to neutral faces in the flash suppression task (see Table 2). I/CP was not significantly correlated with fear processing when controlling for CU. CU, however, was significantly correlated with fear processing when controlling for I/CP. Although no anxiety measures were significantly associated with fear processing, a significant negative correlation was noted between fear processing and proactive aggression.

Linear regression techniques controlling for ethnicity were used to examine main effects of anxiety and psychopathy measures in predicting both aggression and fear processing (See Table 3). Notably, both SCARED total and GAD scores significantly predicted reactive aggression. CU consistently predicted fear processing in the negative direction. Both psychopathy factors consistently predicted aggression and rule-breaking as reported on the CBCL. I/CP was a stronger predictor of aggression than was CU.

Hypothesis Tests

Hypothesis 1 stated that the I/CP factor of psychopathy would be positively associated with measures of child anxiety. To test this hypothesis, zero-order correlations were calculated between the I/CP and all anxiety measures (see Table 1). No significant correlations were found between the I/CP factor of psychopathy and self-report measures of anxiety completed by the child. Significant positive associations were found, however, between I/CP and two parent-reported anxiety-related factors on the

CBCL. These factors were Anxiety/Depression (Anx/Dep) and Anxiety Disorders. These associations decreased slightly in strength but remained significant when partial correlations were conducted to control for the CU factor of psychopathy. Hypothesis 1 was therefore partially supported.

Hypothesis 2a predicted that I/CP and anxiety would interact to predict reactive aggression. Regression analyses were conducted for each measure of anxiety with reactive aggression as the dependent variable. Interaction terms were calculated for I/CP with each of these factors. These analyses all controlled for the CU factor and ethnicity. They also controlled for proactive aggression, which was positively associated with reactive aggression. Main effect terms and statistical controls were entered into the first block of the analysis, and the interaction term was entered in the second block to assess its independent association with reactive aggression. No significant interaction effects were found using these analyses (see Table 4).

Additional, exploratory regression analyses were also conducted to assess whether anxiety and I/CP interacted to predict CBCL aggression and rule-breaking scores. Ethnicity and ASPD CU scores were controlled for in all analyses. Results are presented in Table 4. A significant interaction was found between I/CP and child self-reported GAD in predicting aggression. Post hoc regression analyses splitting the sample into those low and high for child GAD revealed a much stronger association between impulsivity and aggression for those children with low anxiety (b=.69), compared to those with high anxiety (b=.29).

Hypothesis 2b predicted that the interaction between I/CP and anxiety would predict fear processing sensitivity, as evidenced by a shorter reaction time to fearful faces compared to neutral faces during the flash suppression task. Regression analyses were conducted using the interaction terms for I/CP with each measure of anxiety (see Table 4). No significant interactions were found. Hypothesis 2b was therefore disconfirmed and the mediation hypothesis (Hypothesis 3) was negated.

Hypothesis 4 predicted that the CU factor of psychopathy would be negatively associated with measures of child anxiety. Table 2 displays partial correlations between CU and child anxiety measures

controlling for the I/CP factor. Associations were all nonsignificant, with some in the negative direction and others positive. Hypothesis 4 was therefore not supported.

Hypothesis 5a predicted that CU and anxiety would interact to predict proactive aggression. Because the data distribution for proactive aggression was negatively skewed, a log 10 transformation was performed. Interaction terms were calculated for CU with each measure of anxiety. Regression analyses controlled for I/CP and reactive aggression. No significant interaction effects or main effects were found and hypothesis 5a was therefore not supported (see Table 4). As with the I/CP factor, additional regression analyses were conducted using the CBCL factor of aggression as a dependent variable. No significant interactions were found (see Table 4).

Hypothesis 5b stated that the interaction between CU and anxiety would predict low preconscious fear processing sensitivity, as evidenced by longer reaction times to fearful faces compared to neutral faces. Regression analyses were conducted using the interaction terms for CU with each measure of anxiety. No significant interaction effects were found (see Table 4); Hypothesis 5b was therefore not supported, and the mediational hypothesis 6 was negated as well.

Because of the possibility of ADHD-related attentional factors contributing to experimental tasks, post-hoc analyses were conducted controlling for ADHD symptom scores, but no significant differences in results were detected when ADHD was controlled.

Discussion

This study examined psychopathic traits and anxiety in a community sample of boys and tested the associations between these factors, aggressive outcomes, and preconscious fear processing. While hypotheses were derived from a well-documented literature, drawing from both theory and empirical findings on secondary psychopathy and BIS/BAS characteristics observed in several populations, the specific hypotheses of the current study have not been previously tested. Hypotheses were largely unsupported.

Based on several previous findings on divergent correlations of psychopathy factors with anxiety and fear processing in adults and children (e.g. Blackburn et al., 2008; Lynam & Widiger, 1998; Lynam et al., 2005; Pardini, Lochman, & Frick, 2003), it was hypothesized that the I/CP dimension of psychopathy would be positively associated with anxiety while the CU dimension would be negatively associated with anxiety. The first of these hypotheses was partially supported. Significant zero-order correlations were found between I/CP and parent reports of anxiety, remaining significant when controlling for CU, but this finding did not hold true for child reports. The second correlational hypothesis was not supported, as correlations of CU with anxiety were nonsignificant, with some positive and some negative in direction. These findings are partially consistent with those of Frick et al. (1999) who found similar correlations to those in this study, in which I/CP and other measures of conduct problems were positively associated with trait anxiety and CU was nonsignificantly and negatively correlated with anxiety. When controlling for I/CP, however, the anxiety and CU associations became significantly negative, a finding that was not replicated in the current study. The assessment of trait anxiety was similar to that of the current study, in which both parent and child reports of anxiety symptoms were assessed and the Anxiety/Depression subscale of the CBCL was used as an additional measure. Failure to replicate results after controlling for the presence of I/CP may be due to sample differences. The sample used in Frick et al.'s (1999) study consisted of children referred to an outpatient mental health clinic. Severity of CU and anxiety in their sample may have been higher in comparison to the current study's community sample, allowing the relationship between forms of psychopathology to be observed in a wider range of symptomology. In addition, Frick et al.'s sample was not specifically recruited for externalizing behaviors, making parent reporters more likely to report internalizing and anxiety in their children.

It was also hypothesized in the current study that interactions between the two psychopathy factors and anxiety would predict fear processing differences. Specifically, I/CP was hypothesized to interact with anxiety to predict more sensitive preconscious fear processing, as evidenced by shorter reaction times in response to fearful faces on the flash suppression task. CU, on the other hand, was

hypothesized to interact with anxiety to predict slower reaction times to fearful faces, indicating a preconscious fear processing deficit. Support for this deficit, while not assessed through the same flash suppression task, has been evidenced in previous literature and is theoretically based on Gray's (1976) BIS/BAS distinction. A relationship between CU, low anxiety, and decreased sensitivity to fearful stimuli would support a weak BIS, while the I/CP dimension's association with increased fear sensitivity and higher anxiety would demonstrate a normal to high-functioning BIS and stronger BAS characteristic of a secondary psychopath. These hypotheses were not supported. There were no significant interactions of I/CP with anxiety in predicting fear processing. The interaction between I/CP and Anx/Dep approached significance, but when probed in post-hoc analyses, no significant differences were found between high and low anxious groups. Additionally, no significant interactions were found between CU and anxiety measures in predicting fear processing. In preliminary analyses, differing associations of each psychopathy factor with fear processing were found, consistent with findings of Pardini et al. (2003) regarding decreased fear processing associated with CU. Specifically, CU was significantly, negatively correlated with fear processing reaction time differences, indicating reduced sensitivity to fear stimuli. This correlation, however, did not appear to be moderated by anxiety levels as hypothesized.

Hypothesized interactions of psychopathy dimensions and anxiety in predicting different types of aggression were also not supported. These hypotheses were based on a finding by Kimonis et al. (2006) in which reduced reactivity to distressing emotional stimuli, found to be associated with the CU dimension (e.g. Frick & Morris, 2004), was also associated with increased proactive aggression, while the angry reactivity associated with reactive aggression appears to be related to impulsivity (Raine et al., 2006). Interactions of CU and I/CP with anxiety, however, did not differentially predict proactive and reactive aggression. Due to these nonsignificant interactions, mediation hypotheses of fear processing as mediators of the psychopathy and anxiety interaction predicting types of aggression were negated.

Reactive aggression was found to be associated with child-reported anxiety, while proactive aggression was associated with a deficit in fear processing as evidenced by slower reaction times to

fearful faces. These trends were expected due to previous findings on correlates of these two types of behavior (e.g. Raine et al., 2006). They may also suggest that fear processing distinctions may be seen only in proactive aggression, as they are only seen in the CU dimension of psychopathy; I/CP and reactive aggression may not be associated with fear sensitivity as captured by the mCFS task. Such suggestions, however, require further studies.

Additional analyses using parent reports of general aggression and rule-breaking yielded one significant interaction, in which I/CP and GAD, as reported by the child, interacted to predict aggression, such that I/CP was more strongly related to aggression when anxiety was low. This finding did not directly relate to hypotheses, as parent reports of aggression had no distinction between reactive and proactive aggression; the aggression affected by the interaction of I/CP and GAD may have encompassed both categories. This finding is consistent with literature in children regarding the modulatory effect of anxiety on psychopathic traits in children. BAS tendencies such as reward dominance and poor response modulation are attenuated by BIS inhibitory mechanisms in children with anxiety disorders and conduct problems, as compared to nonanxious children with conduct problems (O'Brien & Frick, 1996; Vitale et al., 2005). It stands contrary to studies with adult offenders in which BAS tendencies are positively related to anxiety and Factor 2 of psychopathy (Newman et al., 2005), suggesting that anxiety may play a larger role in modulating aggression in younger age groups, compared to older and more severely aggressive populations. This finding did not hold true for parent-reported anxiety on the CBCL or with child's report of total anxiety on the SCARED, which may indicate that GAD is a specific component in this modulatory effect and that a broader internalizing dimension does not consistently modulate aggression. Indeed, findings on the comorbidity of anxiety disorders with externalizing behaviors in children have been mixed, with some studies reporting less impairment associated with comorbidity and others reporting no significant differences (Loeber & Keenan, 1994). Specific mechanisms for the modulatory effect of anxiety should be explored in future research.

Overall, anxiety and psychopathy did not appear to interact to predict aggression in this sample. Main effects of these variables in predicting aggression and rule-breaking, however, indicate that they are nevertheless important variables to assess in relation to externalizing behaviors. Psychopathy factors consistently predicted externalizing behaviors as reported by the parent, a finding that is consistent with the body of literature regarding the predictive value of psychopathic traits in predicting aggression, delinquency, and recidivism (e.g. Farrington, 2005; Salekin, 2004). The finding that I/CP is a stronger predictor of aggression than is CU, while both are equally strong predictors of rule-breaking, is an interesting one that may bear implications for children with externalizing problems. Although both factors may indicate proneness to more serious delinquency and violence as children age, treatment approaches to rule-breaking and aggression might focus on different underlying personality dimensions. The strength of I/CP as a predictor of aggression, as well as its interaction with GAD, indicates that much of the aggression seen in this sample might be a product of impulsivity rather than callousness toward others. These findings may also mean that callous children are not necessarily aggressive, or that their aggression is covert. Examination of specific instances of aggression and rule-breaking would help shed light on associated factors that can be targeted in treatment.

Methodological differences from previous studies of similar constructs may partially account for the lack of significant interactions. The primary outcome variables of proactive and reactive aggression were assessed based on child report and the proactive scale suffered from relatively low internal reliability. The proactive-reactive distinction was derived from only one paper (Washburn, McMahon, King, Reinecke & Silver, 2002), which classified items from the AS based on operational definitions of proactive and reactive aggression from Dodge (1991). Although parent reports of aggression and rule-breaking were included, these did not include a proactive-reactive distinction. The other primary outcome variable of fear processing was assessed by a single measure. The flash suppression task is a fairly novel experimental tool. Its use, therefore, has not been documented extensively in previous studies.

Reporter effects may pose a problem in interpreting the current study's findings. Discrepancies between parent and child report of both anxiety and aggression may indicate biases from both parties and pose difficulty in determining the true nature of these variables. Interestingly, the one significant interaction involved a child report of anxiety and parent reports of psychopathic traits and aggression, indicating that there are benefits to having multiple reporters in detecting true effects for internalizing and externalizing symptoms. Although self-report data were gathered for child psychopathy, these proved to be unreliable and could not be used. Child self-reports of psychopathy may be difficult to gather due to high face validity of the instrument used; social desirability may prevent children from answering honestly on such questions. Parent reports of child psychopathy were sufficiently reliable, but given the affective dimension of psychopathy and the self-selected nature of this sample, mothers may also not be the ideal source for assessment of child psychopathy. Similar issues may hold true for reports of anxiety. Parent oversight of child anxiety might have posed an issue in this sample because they were recruited specifically for externalizing behaviors (their primary complaint concerning their children), which may have accounted for some of the discrepancy between parent and child reports of anxiety.

Confounding variables may also have affected our findings. No measure of IQ was included in the study. Given recent cognitive theories for the etiology of psychopathic traits and aggressive behaviors (e.g. Sadeh & Verona, 2008), such a measure might play an important role in variables of interest. Peer interactions may also play a role in modulating aggressive behaviors and psychopathic traits. A final possibility for the study's failure to replicate trends in psychopathic traits, aggression, and anxiety may be our use of a community sample, rather than a clinical sample. Severity of problems, particularly aggressive behaviors, which were negatively skewed in child reports, may have produced restriction of range and prevented detection of such trends.

The role of fear processing in psychopathic traits in youth requires further examination. Blair (2010) provides an alternative explanation for reactive aggression in psychopathy that may explain the nonsignificance of fear processing as an intermediary variable in this study. Rather than highly

responsive threat circuitry, Blair proposes that a tendency toward frustration may underlie psychopathic individuals' proneness to reactive aggression. Psychopathic individuals may experience increased response to frustrating events and increased likelihood of encountering frustrating events because they are more likely to respect a rewarding effect that is not fulfilled. Alternatively, Sadeh and Verona (2008) applied a cognitive control and working memory approach to differences in primary and secondary psychopathy traits in nonincarcerated. They found that some characteristics of primary psychopathy, including low anxiety and social dominance, were associated with diminished attentional capacity, while some secondary psychopathy characteristics were associated with impaired working memory function. Although more support is needed for such findings, they indicate an attentional component in both types of psychopathy that was not captured by our preconscious fear processing task and measures assessing affective factors.

Unexpectedly, the current study found no significant associations found between fear processing and measures of anxiety; most associations, in fact, were nonsignificant and negative. Anxiety as assessed in subclinical symptoms, then, appears not to be associated with fear processing and may not, in fact, underlie the remorselessness and affective deficit that underlies CU traits. These results are inconsistent with those of Thomas et al. (2001) who found increased amygdala response to fearful faces in children with anxiety disorders as assessed by the SCARED. This difference in findings, however, may be due to the fact that Thomas et al.'s study also assessed depression, which was not assessed in the current study except as combined with anxiety on one subscale of the CBCL. It is also possible that increased amygdala activation may not correspond with faster reaction time to unconscious processing of fearful stimuli.

Recent studies have examined factors that differentially affect conscious and preconscious fear processing. Perez-Edgar et al. (2007) found that attention alters the increased amygdala activation associated with behaviorally inhibited youth such that anxious youth did not show increased amgydala activation in a passive viewing condition. These researchers also point out that anxiety arising from

behavioral inhibition may be distinct from anxiety that emerges through other routes, a consideration that is useful for the current study. Because the current study was based on BIS/BAS-related hypotheses, assessment of anxiety directly related to behavioral inhibition would have been ideal. The measures used assessed more general types of anxiety that may not have reflected BIS functioning. Furthermore, the current study's measurement of fear processing did not use neuroimaging, instead requiring children to press a button once they recognized faces. Amygdala reactivity to fearful faces is linked to the uncertainty provided by that stimulus (Dugas et al., 2004), which may have resulted in a slower reaction time regardless of amygdala activation in the current sample.

One study also found that administering testosterone to healthy young adult women reduced the unconscious emotional responses to fearful faces as measured by a masked emotional Stroop task (van Honk, Peper & Schutter, 2005). Self-reported measures of anxiety, however, were unaffected by the administration of testosterone. Although these results are not directly applicable to our all-male, preadolescent sample, they do call attention to the differential effects of testosterone levels on the experience of fear and anxiety. Given the host of biological factors that could not be controlled in our study, including age and physiological maturation, it is possible that boys with high baseline testosterone levels had a suppressed reaction to fearful faces regardless of their anxiety levels.

The findings regarding testosterone point to an important distinction between fear and anxiety. Construct confusion throughout the literature, as well as overlapping items on measures of fear and anxiety, may account for null findings in the current study. Given the often nonsignificant association between fearlessness and trait anxiety (Frick et al., 1999), the relationship between anxiety and fear processing with regards to psychopathic traits may not be as straightforward as hypothesized. Sylvers, Lilienfeld, and LaPrairie (2011) conducted a meta-analysis to detect associations between measures of fear and anxiety and found significant content overlap between the two constructs. Their literature review showed that emotional arousal is involved in both anxiety and fear, but that distinct neural pathways underlie these two types of response. DSM-IV classifications of anxiety disorders contribute to construct

confusion in that they group fear-based disorders, such as phobias, with anxiety-based disorders that are more highly characterized by internalizing symptoms (Krueger, 1999).

A developmental perspective may also contribute to interpreting this study's findings. A study by Frick, Cornell, Bodin, Dane, Barry & Loney (2003) indicated that CU traits are distinct due to associated pathways to behavioral problems. Regardless of whether or not they had conduct problems, nonreferred children with CU traits exhibited a lack of behavioral inhibition, while a hostile attribution bias was associated with conduct problems only in the absence of CU traits. The longstanding effect of behavioral inhibition and hostile attribution bias on aggression is unlikely to be captured when data for the predictor and outcomes measures are gathered at the same point in time. Frick et al. also state that trait anxiety may co-occur with CU traits despite low fearfulness and low behavioral inhibition because of impairments due to conduct problems. This co-occurrence might make the distinctive correlates of anxiety with CU and I/CP difficult to decipher when predicting behaviors. Blair's (2010) proposal that of genetic effects progressively contributing to orbitofrontal cortex distinctions in psychopathy may provide a neurocognitive basis for this developmental perspective. Whether genetic or environmental in nature, with accumulated failure of social learning through antisocial and aggressive behavior, the development of psychopathic traits seems to entail a complex network of factors. Longitudinal studies in which the effect of conduct problems and impairments on anxiety is taken into account might address these issues.

Strengths and Limitations

This study's strengths included its collection of questionnaire data from parents and children, an ethnically diverse sample, and its inclusion of only pre-adolescent children. Its limitations, however, are important to note. The unreliability of child reports on the APSD was unfortunate and likely gave us an incomplete picture of affective dimensions of psychopathy that would be captured in self-report. This limitation suggests that other psychopathy measures might be better suited to collecting self-report data, such the Psychopathy Checklist Youth Version (PCL-YV, Forth, Kosson & Hare, 2003). Another

limitation may arise from the item overlap between conduct problem items on the I/CP score and measures of aggression and rule-breaking, which may account for some significant associations.

As mentioned previously, a stronger measure for reactive and proactive aggression administered to parents might have provided more reliable results than those obtained with the child-report AS. Although the CBCL provided reliable data for aggression and rule-breaking, we were unable to see distinctions in types of aggression from this measure.

Low correlations between parent and child reports on anxiety and aggression may indicate a limitation in our measurements. Although they assessed the same constructs, parent and child measures of anxiety included very different items, with CBCL items omitting somatic indices of anxiety and the SCARED including limited items for general internalizing. In future studies, a parent and youth version of the same measure might both be administered in addition to the CBCL so that more concordance might be obtained between reporters. Although assessment of anxiety disorder symptoms has been used as an indication of trait anxiety in previous studies (e.g. Frick et al., 1999), measurements based on trait approaches to anxiety such as the neuroticism on the FFM might capture different trends that do not rely on diagnostic criteria. Inclusion of a BIS/BAS functioning measure might also have allowed more direct testing of hypotheses; namely, that normal to high BIS functioning and high BAS functioning would be associated with I/CP, predicting more reactive aggression and higher sensitivity in fear processing, while low BIS functioning and normal to high BAS functioning would be associated with CU, predicting more proactive aggression and lower sensitivity in fear processing.

The self-selected nature of our sample might also have lent to an underlying bias in parent reports. Children were enrolled in our study due to parents' perception of conduct problems, whether these were mainly attentional, aggressive, or delinquent in nature. It is possible that parents did not accurately report anxiety in their children because they perceived externalizing behaviors as their children's primary problem. In addition, parent reporters were all mothers, and their reports of their sons

might have been limited to role-specific observations. Including a father, teacher, or other caregiver report might provide more impartial and comprehensive data for future studies.

In addition, only one measure of fear processing was included in this study. The flash suppression task is a new experimental tool and includes only Caucasian faces. Due to the ethnically diverse composition of our sample, a task including ethnically diverse faces would be more appropriate to as to reduce uncertainty in rating outgroup faces compared to ingroup faces (e.g. Beupre & Hess, 2006). This study also did not include measures of IQ or SES, both of which predict aggressive behavior (e.g. Lefkowitz, Eron, Walder, & Huesmann, 1977).

The number of analyses conducted increases the chance that a Type 1 error occurred. On the other hand, our findings might be affected by Type 2 error given the relatively small sample size used in this study. A review by McClelland and Judd (1993) provides several reasons why our study may have had inadequate statistical power for detecting moderator effects. Nonexperimental field studies often report difficulty in detecting statistically significant moderator effects due to the nature of the multiplied regression coefficient for hypothesized interactions. Errors in measuring two individual factors are exacerbated when they are multiplied, and theoretical constraints tend to restrict the magnitude of the coefficient. The effect of restriction of range and low variance in our variables may have also been exacerbated by the calculation of interaction terms. McClelland and Judd explain that extreme scores may be necessary for detecting moderator effects. The considerations they note are particularly applicable to the community sample in our study, whose diagnostic scores on DBDs fell much more frequently in the intermediate range than in the positive range.

Implications for Future Research

This study reaffirms the importance of assessing psychopathy in children as a co-occurring condition with other child behavior problems. The findings of this study bear implications for both the assessment of psychopathy specifically in children and for theoretical bases for the etiology of psychopathy. Some support was found for the associations of childhood psychopathy traits with anxiety

that underlie the primary-secondary psychopathy distinction as conceptualized by Karpman (1941). The lack of consistently negative associations between CU and anxiety measures may indicate that the primary psychopathy distinction arises later in life and may rely on failed social learning through more severe antisocial and aggressive behaviors. Alternatively, it may be based on neurocognitive processes becoming less malleable as individuals reach adulthood.

Based on associations of psychopathy with the FFM, Salekin et al. (2005) suggested that anxiety and neuroticism may only be associated with psychopathy (total score) at an early age, when overall comorbid psychopathology is more common. In a review of adolescent psychopathy measurement, Farrington (2005) explains that facets of the FFM elucidate mixed findings regarding this association. Neuroticism includes impulsiveness and angry hostility, which may underlie aggressive behavior associated with both psychopathy factors assessed in the current study. However, neuroticism also includes self-consciousness, with the glibness and shamelessness associated with CU at the negative end, and vulnerability, with the primary psychopathy hallmark of fearlessness at the negative end. The issue of psychopathy's correlations with neuroticism-related traits, including impulsivity, hostility, anxiousness, and depression, remains a contentious one due to theoretical inconsistencies and mixed results, and this study contributes to the body of literature that aims to clarify these distinctions. Examining the two psychopathy factors as distinct constructs remains a useful method for distinguishing personality traits and clinical manifestations of anxiety.

Although multiple studies have examined a fear-processing deficit in association with types of aggression and psychopathic traits, few studies have examined the interrelations between psychopathic traits, aggression, fear processing, and anxiety. The current study combined a personality approach and a clinical comorbidity approach to the role of anxiety in the prediction of aggression while interacting with psychopathic traits. Results revealed that the complex relationship between anxiety and psychopathic traits is not directly reflected in fear processing tendencies or in types of aggression. Fear processing was, however, related to variables of interest such as CU traits and proactive aggression in directions consistent

with trends in previous studies, indicating that our assessment of fear processing on the preconscious level is relevant to the study of psychopathy and aggression. Few studies have examined the relationship between psychopathic traits and anxiety disorders, with even fewer such studies in children and adolescents. Studies with both adult and youth populations have produced mixed findings, often assessing associations with psychopathy total score or only with the CU dimension. Children with psychopathic traits often demonstrate zero correlations with anxiety and depression while adult psychopaths exhibit negative correlations, suggesting that younger individuals with psychopathic traits have yet to develop a "mask" of sanity and are more likely to exhibit negative affect than are psychopathic adults (Sevecke & Kosson, 2010; Salekin, 2010). Despite the presence of a fear processing deficit in association with CU traits, then children with such traits may not act aggressively due to low anxiety.

Salekin and Frick (2005) advocate a developmental approach to child psychopathy that places it within the realm of other types of child psychopathology. This perspective is especially important given that psychopathic traits are less stable in youth than they are in adults. As part of this process, Salekin and Frick suggest that the presence of psychopathic traits should be documented according to both its normative and non-normative characteristics, as is the case for other types of child psychopathology. Because this study was conducted with a non-referred sample with a broad range of conduct problems and other behavioral problems, we were able to assess both normative and non-normative behaviors associated with the presence of psychopathic traits. Longitudinal research in the future may benefit from the use of community samples with a variety of externalizing problems so as to examine the developmental processes that underlie behaviors associated with psychopathic traits. Such studies might inform intervention approaches to child psychopathy, about which little is known.

Conclusion

Despite the lack of support for many of its hypotheses, this study provides valuable perspective on psychopathic traits and anxiety in the context of child psychopathology. It extended theories of

psychopathic subtypes, as well as research on comorbidity of anxiety and conduct problems in children, by applying them to a preconscious fear processing task and examining associations to different types of aggressive outcomes. Although relationships between these variables did not follow hypothesized patterns, important relationships did emerge. The findings of this study suggest that specific types of anxiety, such as that characterized in GAD, may play a role in aggression. In addition, reduced sensitivity to fear stimuli was associated with CU and proactive aggression, although these relationships require further examination. Further studies on co-occurrence of anxiety disorders and aggression in similar populations may lead to more sophisticated hypotheses and findings on the development and impact of psychopathic traits on child psychopathology and behavior.

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Table 1

Descriptive Statistics for All Variables Entered

	M (SD)	Min-Max
APSD I/CP	6.29 (2.01)	1-10
APSD CU	3.92 (2.17)	0-9
SCARED total	26.25 (15.27)	2-82
SCARED GAD	6.08 (3.78)	0-18
CBCL Anx/Dep (raw score)	4.71 (4.49)	0-15
CBCL Anxiety Disorder (raw score)	2.12 (2.16)	0-8
AS Reactive	5.07 (5.69)	0-24
AS Proactive	3.45 (4.83)	0-24
CBCL Aggression	12.31 (7.71)	0-33
CBCL Rule-breaking	5.06 (3.52)	0-14
mCFS Fear difference (neutral – fear reaction time) (MS)	-771.33 (536.59)	-2238-621

Note: APSD=Antisocial Process Screening Device, I/CP=Impulsivity/Conduct Problems score, CU= Callous-Unemotional score; SCARED=Screen for Child Anxiety Related Emotional Disorders, GAD=Generalized Anxiety Disorder score; CBCL= Child Behavior Checklist, Anx/Dep= Anxious/Depressed syndrome score; AS= Aggression Scale; mCFS=modified Continuous Flash Suppression

Table 2

Correlations between All Variables Entered

	2	3	4	5	6	7	8	9	10	11
1)APSD I/CP	.23(p=.008)*	-.00 (p=.992)	-.04 (p=.762)	.37 (p=.001)*	.30 (p=.008)*	.01 (p=.904)	-.10 (p=.396)	.62 (p<.001)*	.54(p<.001)*	.01 (p=.900)
Partial correlations controlling for APSD CU		.05 (p=.693)	-.03 (p=.777)	.35 (p=.002)*	.29 (p=.009)*	.03 (p=.823)	-.13 (p=.276)	.57 (p<.001)*	.47 (p<.001)*	.10 (p=.391)
2)APSD CU		-.15 (p=.195)	-.01 (p=.917)	.13 (p=.252)	.06 (p=.597)	-.04 (p=.753)	.07 (p=.519)	.40 (p<.001)*	.54 (p<.001)*	-.26 (p=.023)*
Partial correlations controlling for APSD I/CP		-.16 (p=.178)	-.002 (p=.989)	.02 (p=.838)	-.03 (p=.787)	-.04 (p=.715)	.11 (p=.346)	.28 (p=.013)*	.47(p<.001)*	-.27 (p=.016)*
3) SCARED Total			.80 (p<.001)*	.05 (p=.668)	-.09 (p=.454)	.29 (p=.010)*	.06 (p=.619)	-.11 (p=.342)	-.14 (p=.224)	.07 (p=.573)
4) SCARED GAD				.02 (p=.838)	.03 (p=.809)	.32 (p=.005)*	.18 (p=.113)	-.09 (p=.439)	.00 (p=.998)	-.08 (p=.501)
5) CBCL Anx/Dep					.88 (p<.001)*	-.14 (p=.231)	-.07 (p=.545)	.46 (p=.001)*	.21 (p=.065)	-.05 (p=.687)
6) CBCL Anxiety Disorder						-.18 (p=.118)	-.05 (p=.674)	.34 (p=.002)*	.11 (p=.333)	.01 (p=.918)
7) AS Reactive Aggression							.42 (p<.001)*	.05 (p=.697)	-.05 (p=.671)	-.21 (p=.070)
8)AS Proactive Aggression								-.06 (p=.583)	-.001 (p=.992)	-.24 (p=.035)*
9) CBCL Aggression									.66 (p<.001)*	-.06 (p=.581)
10) CBCL Rule-Breaking										-.07 (p=.538)
11) CFS Fear Difference (ms)										

*p<.05

Table 3

Main Effects for Aggression and Fear Processing Variables Regressed on Psychopathy and Anxiety Variables

Dependent variable	Independent variables	β	*p*
Reactive Aggression (controlling for proactive)	I/CP	.08	.424
	CU	-.04	.683
	SCAREDtotal	.26	.018*
	I/CP	.10	.332
	CU	-.08	.449
	SCAREDGAD	.23	.029*
	I/CP	.13	.270
	CU	-.08	.469
	Anx/Dep	-.16	.160
	I/CP	.13	.236
	CU	-.09	.409
	CBCL Anxiety	-.20	.063
Proactive Aggression (controlling for reactive)	CU	-.14	.200
	I/CP	.12	.288
	SCAREDtotal	-.02	.852
	CU	.12	.268
	I/CP	-.14	.211
	SCAREDGAD	.08	.442
	CU	.12	.273
	I/CP	-.15	.214
	Anx/Dep	.04	.707
	CU	.13	.257
	I/CP	-.16	.176
	CBCL Anxiety	.08	.472
mCFS Fear difference (ms)	CU	-.29	.014*
	I/CP	.09	.460
	SCAREDtotal	-.00	.976
	CU	-.29	.012*
	I/CP	.08	.466
	SCAREDGAD	-.10	.375
	CU	-.29	.015*
	I/CP	.14	.254
	Anx/Dep	-.06	.609
	CU	-.29	.015*
	I/CP	.12	.315
	CBCL Anxiety	-.01	.939

CBCL Aggression	CU	.22	.017*
	I/CP	.54	<.001*
	SCAREDtotal	-.08	.382
	CU	.24	.011*
	I/CP	.53	<.001*
	SCAREDGAD	-.08	.343
	CU	.23	.008*
	I/CP	.44	<.001*
	Anx/Dep	.26	.005*
	CU	.24	.007*
	I/CP	.48	<.001*
	CBCL Anxiety	.17	.059
CBCL Rule Breaking	CU	.41	<.001*
	I/CP	.42	<.001*
	SCAREDtotal	-.09	.335
	CU	.42	<.001*
	I/CP	.41	<.001*
	SCAREDGAD	.01	.928
	CU	.42	<.001*
	I/CP	.41	<.001*
	Anx/Dep	-.01	.883
	CU	.42	<.001*
	I/CP	.43	<.001*
	CBCL Anxiety	-.06	.484

*****p<.05

Table 4

Interaction Effects for Aggression and Fear Processing Variables Regressed on Psychopathy * Anxiety Variables

Interaction Terms	Dependent Variable	ΔR^2	ΔF	p
I/CP * SCAREDTotal	Reactive Aggression	.00	0.42	.52
	Total Aggression	.03	2.83	.50
	CBCL Rule Breaking	.00	0.00	.98
	CBCL Aggression	.01	1.60	.21
	mCFS Fear Difference (ms)	.01	0.87	.35
I/CP* SCAREDGAD	Reactive Aggression	.00	0.13	.72
	Total Aggression	.00	0.12	.73
	CBCL Rule Breaking	.01	1.09	.30
	CBCL Aggression	.04	5.13	.03*
	mCFS Fear Difference (ms)	.01	1.09	.30
I/CP*Anx/Dep	Reactive Aggression	.00	0.32	.57
	Total Aggression	.00	0.22	.64
	CBCL Rule Breaking	.02	2.38	.13
	CBCL Aggression	.00	0.02	.89
	mCFS Fear Difference (ms)	.05	3.81	.06
I/CP * CBCLAnxiety	Reactive Aggression	.01	0.73	.40
	Total Aggression	.00	0.10	.76
	CBCL Rule Breaking	.01	1.99	.16
	CBCL Aggression	.00	0.05	.83
	mCFS Fear Difference (ms)	.01	1.06	.31
CU * SCAREDTotal	Proactive Aggression	.03	2.74	.10
	Total Aggression	.00	0.06	.80
	CBCL Rule Breaking	.00	0.07	.79
	CBCL Aggression	.00	0.41	.52
	mCFS Fear Difference (ms)	.01	1.17	.28
CU * SCAREDGAD	Proactive Aggression	.00	0.01	.92
	Total Aggression	.00	0.31	.58
	CBCL Rule Breaking	.00	0.33	.57
	CBCL Aggression	.00	0.24	.63
	mCFS Fear Difference (ms)	.00	0.00	.98
CU *Anx/Dep	Proactive Aggression	.00	0.39	.54
	Total Aggression	.01	0.86	.36
	CBCL Rule Breaking	.01	1.27	.26
	CBCL Aggression	.02	3.68	.06
	mCFS Fear Difference (ms)	.01	0.70	.41
CU * CBCLAnxiety	Proactive Aggression	.00	0.02	.89
	Total Aggression	.00	0.18	.68
	CBCL Rule Breaking	.00	0.57	.46
	CBCL Aggression	.02	2.54	.12
	mCFS Fear Difference (ms)	.01	1.20	.28

*p<.05