

AUTONOMIC NERVOUS SYSTEM FUNCTIONING AND CALLOUS-UNEMOTIONAL  
TRAITS IN CHILDHOOD-ONSET CONDUCT DISORDER

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DOCTOR OF PHILOSOPHY

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by  
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## ABSTRACT

Autonomic Nervous System Functioning and Callous-Unemotional Traits in Childhood-Onset

Conduct Disorder

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Although the current literature demonstrates relations between autonomic nervous system (ANS) functioning and conduct disorder (CD), there are inconsistencies across studies in the magnitude and direction of these associations, some of which may stem from heterogeneity within the CD diagnostic category. Considering callous-unemotional (CU) traits in research examining ANS functioning and CD relations could help to clarify these inconsistencies, given that CU traits identify a subgroup of youth with CD who exhibit a more severe and persistent course, as well as more negative correlates and sequelae than youth with CD without CU traits. However, there is a dearth of literature considering ANS processes among youth with CD with and without CU traits. Examining these relations, particularly during middle childhood when these processes may be amenable to intervention, has important implications for etiological, prevention, and intervention models. The present study examined relations among CD, CU, and ANS functioning among a sample of ethnic minority, urban children ( $N= 99$ ,  $M= 9.87 \pm 1.19$  years old; 48.5% male; 94.9% African-American, 3% Latino/a). Specifically, I examined whether CU traits moderated the relations between CD and (a) parasympathetic nervous system (PNS) functioning and (b) sympathetic nervous system (SNS) functioning. In addition, I

examined whether parenting behaviors (i.e., harsh parental discipline and parental warmth/involvement) influenced the relations between (a) CD and ANS functioning, and (b) CU and ANS functioning. Findings demonstrated that PNS functioning differed among children with high and low levels of CD symptoms depending on levels of CU traits. Within the current sample, among children with higher levels of CD symptoms, those with (a) higher CU symptom severity exhibited lower baseline respiratory sinus arrhythmia (RSA) and lower RSA reactivity (PNS withdrawal), compared to those with (b) lower CU symptom severity who demonstrated higher baseline RSA and higher RSA reactivity (PNS activation). Among children with lower CD symptom severity, those with (a) higher CU symptom severity exhibited higher baseline RSA and higher RSA reactivity, compared to those with (b) lower CU symptom severity who evidenced lower baseline RSA and lower RSA reactivity. Neither harsh parental discipline nor parental warmth/involvement moderated the relations between (a) CD and ANS functioning and (b) CU and ANS functioning. However, there were marginally significant associations between baseline RSA and (a) harsh parental discipline and (b) parental warmth/involvement, as well as between RSA reactivity and parental warmth/involvement in analyses examining CD, parenting, and ANS functioning. Furthermore, parental warmth/involvement tended to be associated with RSA reactivity in the analyses examining CU, parenting, and ANS functioning. Results have implications for facilitating the identification of children at risk for developing more pernicious subtypes of behavior problems, and contribute important information for the development of more individualized and potentially effective interventions for youth behavior problems, particularly among high-risk youth.

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## CHAPTER 1

### INTRODUCTION

Conduct disorder (CD) is defined by the *Diagnostic and Statistical Manual of Mental Disorders* as a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated (4th ed., text rev. [DSM-IV-TR], American Psychiatric Association, 2000). This pattern of behavior can manifest as aggressive behavior to other people or animals, property loss or damage, deceitfulness or theft, and serious violations of rules. The aggressive and antisocial behavior that characterizes CD is the most common impetus for referrals of children and adolescents to mental health treatment (Frick & Ellis, 1999; Lyman & Campbell, 1996). Furthermore, CD is associated with numerous negative correlates and sequelae, including individual, monetary, and societal costs (Frick, 1998; Frick & Ellis, 1999; Frick & Morris, 2004, Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005). Thus, much research has been devoted to the delineation of factors associated with the development of CD that could inform etiological models and in turn intervention and prevention efforts.

This line of research has illuminated a multitude of factors that confer risk for the development of aggressive and antisocial behavior among children, including dysfunctional family contexts, socioeconomic disadvantage, poor peer relations, neuropsychological deficits, deficiencies in autonomic nervous system functioning, and difficult temperamental traits (Dodge & Pettit, 2003; Frick, 2012; Frick & Marsee, 2006; Frick & White, 2008; Loeber & Farrington, 2000; Narhi, Lehto-Salo, Ahonen, & Marttunen, 2010; Odgers, Caspi, Russell, Sampson, Arseneault, & Moffitt, 2012; Raine, 2002). Research also has demonstrated that children with CD constitute a heterogeneous group (Cicchetti, & Rogosch, 1996; Frick, 2006; Frick & Ellis, 1999; Moffitt, 1993; Moffitt et al., 2008) with subgroups characterized by different CD symptoms

manifested, risk factors, presumed causal mechanisms, and developmental pathways (e.g., Frick & Ellis, 1999; Frick & Morris, 2004; Hawes, Brennan, & Dadds, 2009). This heterogeneity complicates our understanding of the manifestation and development of CD and, consequently, efforts to intervene and prevent associated negative sequelae. Thus, attempts have been made to disaggregate CD into meaningful subgroups to reduce this heterogeneity and advance our knowledge of this disorder.

Currently, the *DSM-IV-TR* specifies subtypes of CD based on age of onset and distinguishes between childhood-onset CD (onset of at least one symptom of CD prior to age 10) and adolescent-onset (absence of any symptom of CD before age 10). This distinction was based on a rich line of research documenting two distinct developmental trajectories of aggressive and antisocial behavior. This research demonstrated the existence of a group of children who evidence externalizing behaviors characteristic of oppositional defiant disorder (ODD; a pattern of negativistic, hostile, and defiant behaviors toward adults) early in life, and whose aggressive and antisocial behavior becomes progressively more severe over the course of middle childhood (ages six to twelve) and adolescence (ages thirteen to eighteen; Frick & Ellis, 1999; Lahey & Loeber, 1994; Moffitt, 1993). This developmental trajectory provided the basis for the childhood-onset subtype of CD.

In addition, this research identified a group of children for whom the onset of CD symptoms coincides with early adolescence, thus providing the basis for the adolescence-onset subtype of CD. Further research on these subtypes indicated that these groups differ along several dimensions. For example, compared with the childhood-onset type, those within the adolescent-onset subtype are less likely to be arrested and are less likely to be diagnosed with antisocial personality disorder as adults (Frick & Loney, 1999; Moffitt, 1993). Furthermore,

research has demonstrated increased cognitive and neuropsychological deficits such as lower verbal intelligence and poorer performance on tasks that tap executive functioning abilities (e.g., attention, planning, working memory) among children in the childhood-onset subtype compared to adolescence-onset subtype of CD (Moffitt, 1993). The homes of children with childhood-onset CD tend to be fraught with generalized dysfunction, parental psychopathology, and conflict (Frick, 1994; Frick & Ellis, 1999; Patterson, Reid, & Dishion, 1992). In addition, youth with childhood-onset CD manifest antisocial behavior that increases in severity over time and is more persistent, continuing through adolescence and into adulthood (Frick & Loney, 1999; Lahey & Loeber, 1994; Moffitt, 2003; Moffitt, Caspi, Harrington, & Milne, 2002).

Although these subtypes within the CD diagnostic category are supported by empirical research and are useful for creating more homogeneous CD subgroups, there still exists significant heterogeneity within the diagnostic category of CD. For example, in terms of developmental course, the CD diagnostic category does not reliably predict adult outcomes that may represent more pernicious versions of CD, such as identifying antisocial adults who are distinguished by affective/interpersonal deficits, or psychopathic traits (e.g., lack of guilt, empathy, and emotion; callous disregard for others and use of others for their own gain; Cleckley, 1976; Frick, O'Brien, Wootton, & McBurnett, 1994; Hare, 1970, 1980, 1991; Lynam et al., 2007). As Glenn, Raine, Venables, and Mednick (2007) note, relatively little is known about the antecedents of adult psychopathy. However, given the developmental sequelae, severity, and persistence of antisocial behavior among children with childhood-onset CD, it is likely that the emotional and behavioral precursors to adult psychopathy would be found within this subgroup (Frick & Ellis, 1999; Lynam, 1996).

## Callous-unemotional Traits

One method for identifying such a subgroup of youth with CD, advanced by Frick and colleagues (e.g., Frick, 1998; Frick, Barry, & Bodin, 2000a; Frick & White, 2008) and currently included as a proposed revision for the *DSM-5* (Frick & Moffitt, 2010), classifies CD subgroups in terms of the presence or absence of callous-unemotional (CU) traits (e.g., lack of guilt, lack of empathy, callous use of others for one's own gain). Among adults, the construct of psychopathy is comprised of an affective-interpersonal dimension characterized by egocentricity, a lack of empathy and guilt, superficial charm, shallow emotions, and an inability to form lasting relationships, as well as an impulsive/antisocial dimension characterized by behaviors such as aggression and criminality (Cleckley, 1976; Hare, 1970, 1980, 1993; Hare & Neumann, 2006; Lynam et al., 2007; Pardini & Loeber, 2007; Patrick, 2008). CU traits in childhood are thus analogous to the characteristics comprising the affective and interpersonal component of the construct of psychopathy in adulthood, and it is CU traits in particular that distinguish adults with psychopathy from the larger, more heterogeneous group of antisocial adults (Blair, 2005; Blair, Peschardt, Budhani, Mitchell, & Pine, 2006b; Cooke & Michie, 1997, 2001; Dadds, Fraser, Frost, & Hawes, 2005; Frick, 2006; Frick & White, 2008; Hare, 1993).

Children with CD who have higher levels of CU traits evidence deficits related to guilt and empathy consistent with the characteristics of adult psychopaths (Dadds et al., 2005; Frick et al., 1994; Frick, Bodin, & Barry, 2000b). Consistent with the adult literature on psychopathy, evidence suggests that CU traits among children are associated with more severe and persistent aggression and antisocial behavior (Frick, 2006; Frick & White, 2008; McMahon, Witkiewitz, Kotler, & Conduct Problems Prevention Research Group, 2010; Pardini & Fite, 2010; Rowe et al., 2009). For example, in several samples of adjudicated adolescents, those with CU traits were

more likely to reoffend, particularly with respect to violent crimes, and were quicker to reoffend following release compared to adolescents without CU traits (Brandt, Kennedy, & Patrick, 1997; Forth, Hart, & Hare, 1990; Toupin, Mercier, & Dery, 1995). Furthermore, longitudinal studies of children have demonstrated that CU traits prospectively predict increases in and/or higher rates of conduct problems and delinquency (Dadds et al., 2005; Frick et al., 2005). Although typically conceptualized as rare in childhood, recent research has demonstrated the presence of CU traits in clinic-referred and community-based samples of children (Christian, Frick, Hill, & Tyler, 1997; Dadds et al., 2005; Frick et al., 2000a; Frick, Cornell, Barry, Bodin, & Dane, 2003a; Frick et al., 1994; Kotler & McMahon, 2005). For example, findings from a multi-site cross-sectional study of 1136 community-based and 566 clinic-based youth show that 10%-32% of youth with CD and 2%-7% of those without CD met criteria for CU in the community-based sample, and 21%-50% of youth with CD and 14%-32% without CD met criteria for CU in the clinic-based sample (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). Taken together, this research demonstrates the utility of CU traits for distinguishing a subgroup of children with CD who exhibit an especially severe and persistent form of antisocial behavior that could reflect the antecedents to adult psychopathy.

Given that CU traits may be important developmental precursors and are useful for identifying a group of antisocial adults who manifest chronic patterns of severe and violent criminal behavior (Harpur, Hare, & Hakstian, 1989; Hemphill, Hare, & Wong, 1998), these traits could be similarly important for distinguishing an at-risk subgroup of children with CD (Frick & Marsee, 2006). In keeping with this possibility, in a proposal to the *DSM-5* Childhood Disorders and ADHD and Disruptive Behavior Disorders Workgroups, Frick and Moffitt (2010) provided evidence to support the addition of the specifier “with significant callous-unemotional traits”

within the CD diagnostic category for *DSM-5*. Although the CU specifier can be assigned to any youth meeting diagnostic criteria for CD, it is likely that youth with childhood-onset CD who also meet the criteria for the CU specifier are at greatest risk for exhibiting psychopathy in adulthood. In support of this distinction involving CU traits, children with CD and high levels of CU traits (CD+CU) differ from children with CD symptoms with low levels of or without CU traits (CD-only) on a variety of correlates, including intellectual functioning, social cognitive processing, aggression, interactions with peers, and contextual risk factors (e.g., parenting) (for reviews, see Frick, 2012; Frick & Moffitt, 2010; Frick & White, 2008).

Although there is extensive evidence of a link between intellectual deficits and CD among youth (e.g., Moffitt, 1993), children with CD+CU are less likely to exhibit intellectual deficits, particularly in verbal intelligence (Loney, Frick, Ellis, & McCoy, 1998; Salekin, Neumann, Leistico, & Zalot, 2004). Research also has documented differences in social information processing among antisocial children with and without CU traits. For example, the literature has consistently shown an association between aggression and antisocial behavior with hostile attribution biases (Dodge, 2003, 2006; Orobio de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002); however, recent research has demonstrated that children with conduct problems and CU traits actually make fewer hostile attributions in social situations than youth with conduct problems only (Frick et al., 2003b). Furthermore, the presence of CU traits is associated with positive outcome expectancies for aggressive solutions to resolving conflict (Marsee & Frick, 2007; Pardini, Lochman, & Frick, 2003).

In terms of aggression, research has shown that children with CD+CU manifest elevated levels of both reactive and proactive aggression (Crapanzano, Frick, & Terranova, 2010; Fanti, Frick, & Georgiou, 2009); however, there is also evidence for a unique association between

proactive forms of aggression and CU traits (Fite, Stoppelbein, & Greening, 2009; Frick et al., 2003a; Kerig & Stellwagen, 2010; Marsee & Frick, 2007). In addition, children with CD+CU engage in more bullying behaviors and are more likely to associate with deviant peers compared to children with CD-only (Fanti et al., 2009; Kimonis, Frick, & Barry, 2004). Lastly, although problematic parenting is associated with conduct problems more generally, problematic parenting behaviors are more strongly associated with conduct problems among children without CU traits than children with CD+CU (Frick, 2012; Oxford, Cavell, & Hughes, 2003; Wootton, Frick, Shelton, & Silverthorn, 1997), suggesting that children with CU traits are less responsive to contextual influences than children without CU traits.

Clearly there is evidence that children with CD+CU differ from children with CD-only; nevertheless, the differences found between these two groups of youth may be influenced by the group-based approaches used in much of the literature (i.e., assigning children with CD to categories of CD+CU and CD-only). Yet, these discrete categories are somewhat artificial as they result from cut-off points—points above which the symptoms are considered clinically significant, followed by assignment to categories based on these cut-off points. However, even at levels below those required for a clinical diagnosis, symptoms of psychopathology can seriously impact individuals' functioning (Angold, Costello, Farmer, Burns, & Erkanli, 1999; Drabick, 2009). Furthermore, there are normative increases and decreases in these symptoms across development (Drabick, 2009; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004; Rutter & Sroufe, 2000). Therefore, changes in symptom severity and potentially group status would be anticipated over time and consistent with developmental expectations. These changes may lead individuals to experience different outcomes even if they start out within a particular group, such as childhood-onset CD. In addition, it is critical to take the child's context into account in order

to understand how particular child-specific factors can result in multiple outcomes (Drabick, 2009; Steinberg & Avenevoli, 2000). That is, although children with particular psychosocial profiles (e.g., deficits in executive functioning and verbal intelligence, increased hostile attribution biases, elevated levels of reactive aggression) are at increased risk of following a more severe and persistent antisocial pathway into adulthood, contextual factors at certain points in development may influence change in or otherwise modify this trajectory.

Taken together, it is important to understand CD within a developmental framework. For example, a developmental psychopathology framework emphasizes the importance of considering both categorical and dimensional approaches to symptoms, that when considered in conjunction with developmentally relevant processes, could help address constraints imposed by group-based approaches and facilitate the identification of more homogeneous groups within CD. In addition, this framework permits consideration of multifinality, which suggests that one pathway can result in multiple outcomes depending on other relevant factors (Cicchetti & Rogosch, 1996). A developmental psychopathology framework also supports the consideration of child-specific dimensions or correlates within CD, such as temperamental features, which would be useful in decreasing heterogeneity.

Thus, a developmental psychopathology perspective provides a useful guide for furthering our understanding of subgroups within childhood-onset CD. This framework can then help to illuminate the processes that distinguish the childhood-onset CD+CU subgroup from their CD-only counterparts and therefore help to establish more tailored intervention guidelines. Several principles of the developmental psychopathology perspective are important for research seeking to delineate the specific processes that differentiate childhood-onset CD+CU from CD-only, including the exploration of risk factors from multiple domains using multiple levels of

analysis, interactions between the child and the context in which the child is embedded, multifinality and equifinality, and the consideration of risk factors within the context of typical development (Cicchetti & Rogosh, 1996; Drabick & Steinberg, 2011; Rutter & Sroufe, 2000).

Given the current recommendation for the *DSM-5* diagnostic criteria for CD, as well as the likelihood that CU traits can aid in (a) reducing heterogeneity among antisocial youth, (b) inform etiological theories of CD, and (c) increase understanding of the processes that confer risk and resilience for CD sequelae, it is crucial to elucidate factors that may differentiate children with childhood-onset CD with and without CU traits. Although a good deal of literature has considered CD with and without CU traits among older youth, there is a paucity of research specifically examining risk processes and/or mechanisms underlying childhood-onset CD with and without CU traits, a crucial limitation given the negative prognosis associated with both childhood-onset CD and with CU traits. Although research on childhood-onset CD+CU traits has suggested several factors that may distinguish CD+CU from CD-only, distinct etiological or risk processes for these two subtypes have not been firmly established. However, as this area of research has grown, more is being learned about the biological underpinnings of CD+CU. One important candidate biological process that could aid in identifying more homogenous subtypes of children with CD and that may be amenable to intervention during the period associated with childhood-onset CD is autonomic nervous system (ANS) functioning. Children with CD+CU traits in childhood might manifest different patterns of ANS functioning compared to those without CU traits, which is one potential explanation for varying developmental trajectories within the childhood-onset CD subtype. Given (a) the dearth of research considering these issues and (b) evidence to indicate that even at sub-diagnostic levels, CD symptoms can have a deleterious effect on functioning, the following background and present study consider CD

symptoms and CU dimensionally. As such, the following sections refer to CD symptoms that arise in childhood more broadly (e.g., aggression, delinquency, criminality, disruptive behavior problems) rather than the more narrowly defined and circumscribed CD diagnosis.

### Temperamental Underpinnings of CU Traits

It is likely that temperamental differences, some of which may be associated with specific ANS processes, contribute to the development of CU traits and therefore underlie differences in psychosocial correlates among children with CD+CU and CD-only (Frick, 2006; Frick & Viding, 2009). One potential candidate temperamental style is characterized by low fearful inhibitions, an insensitivity to punishment, and less reactivity to negative stimuli (Dadds et al., 2005; Frick, 2006; Frick & Morris, 2004). Not surprisingly, children with CD symptoms and CU manifest several characteristics that reflect low fearful inhibitions. For example, children with early-onset CD+CU demonstrate an inclination toward thrill-seeking behavior (Frick et al., 2003b; Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999). In addition, Frick and colleagues (1999, 2003b) reported associations between CU traits and fearlessness (indexed by preference for sensation-seeking activities) among a clinic-referred sample of children aged 6 to 13 (Frick et al., 1999) and a community-based sample of children with an average age of 12 years (Frick et al., 2003b).

Evidence for a lack of fearful inhibitions among children with CD+CU also can be drawn from research indicating that youth with CD symptoms and high CU traits exhibit low levels of anxiety (Frick, 2006, 2012; Frick et al. 1994, 1999; Frick & Morris, 2004; Frick & White, 2008; Pardini & Fite, 2010). For example, in a sample of first through seventh grade boys followed over a two year period, Pardini and Fite found that CU traits prospectively predicted lower levels of anxiety, whereas attention-deficit/hyperactivity disorder and oppositional defiant disorder

(ODD) symptoms were related to higher levels of anxiety over time. Furthermore, Frick et al. (1994) found a negative association between CU traits and anxiety symptoms, but a positive association between ODD and CD symptoms with anxiety among a sample of 6- to 13-year-old children. Thus, children with CD+CU may be less likely to exhibit anxiety than CD-only children, consistent with dual pathway models of conduct problems and anxiety (e.g., Drabick, Ollendick, & Bubier, 2010; Garai, Forehand, Colletti, & Rakow, 2009; Ollendick, Seligman, & Butcher, 1999). One potential implication of these findings is that CD+CU children may be less distressed by the effects of their behaviors both on themselves and others because lower levels of anxiety at baseline may hinder their experiencing typical increases in anxiety associated with wrong-doing. Furthermore, these results suggest that low levels of anxiety may be associated with the development of CU traits (Drabick et al., 2010; Frick, 2006; Frick et al., 1999; Frick & White, 2008).

Children with CD+CU also exhibit insensitivity to punishment cues. For example, children with CD+CU emphasize rewards while minimizing punishment, which has been referred to as a reward-dominant cognitive style (Barry et al., 2000; Fisher & Blair, 1998; Frick, 2006; Frick & Morris, 2004; Frick & White, 2008; O'Brien & Frick, 1996). For example, O'Brien and Frick examined the response style of a sample of children ages 6-13 on a task assessing children's decision to pursue rewards despite increasing levels of punishment. Non-anxious children with elevated CU traits (both with and without conduct problems) played significantly more trials than children without CU traits, suggesting continued desire to obtain rewards regardless of the increasing rate of punishment (i.e., loss of points). Consistent with these results, Fisher and Blair (1998) reported that youth with higher psychopathy scores played more trials on a reward-dominant task than youth with lower psychopathy scores. Taken

together, these findings indicate that children with CU traits demonstrate a reward-dominant style. This style is consistent with the notion that children with CU traits exhibit insensitivity to punishment cues and potentially can differentiate children with CD+CU from children with CD-only who, though also reward-focused, are more likely than CD+CU children to modify their pursuit of rewards when faced with increasing punishment.

### Emotion Processes and CU Traits

Temperamental differences among youth with CU traits are also evident in terms of emotion-related processes (Frick, 2006, 2012), such as emotional reactivity. Emotional reactivity is typically thought of as an involuntary process, and refers to the "...speed and intensity of the initial activation of an emotion" (Cole, Martin, & Dennis, 2004, pp. 321-322; Rothbart & Bates, 2006). In contrast to emotional reactivity, *emotion regulation* occurs after an emotion has been activated and is described as "...changes associated with activated emotions" (Cole et al., 2004, p. 320). Changes may occur in the actual emotion or in related psychological processes, and these changes often result from particular behavioral strategies used by the individual (Cole et al., 2004; Thompson, 1994). Researchers have employed a variety of strategies for assessing emotion reactivity and regulation, including self-report, observational methods, physiological measures, and behavioral tasks.

#### *Emotional Reactivity*

In terms of emotional reactivity, whereas CD-only youth demonstrate increased reactivity in response to distress in others, children with CD+CU are less reactive to negative emotional stimuli, including emotionally distressing and threatening stimuli (Blair, 1999; Frick et al., 2003b; Kimonis, Frick, Fazekas, & Loney, 2006). For example, in a sample of children with an average age of 12 years, Frick et al. (2003b) found that children with high levels of CU

demonstrated reduced emotional reactivity, as indexed by slower response times or reduced attentional orienting, to negative emotional versus neutral words. Consistent with these findings, Kimonis et al. investigated emotional reactivity to visual stimuli using a dot-probe task among a sample of children ages 6-13 and found that children with psychopathic (including CU) traits and high levels of aggression demonstrated reduced reactivity to pictures with distressing emotional, compared to neutral, content. In contrast, children high on aggression but low on psychopathic traits demonstrated increased reactivity to distress.

#### *Emotion Recognition Deficits and CU*

Not only are CD+CU children less reactive to negative emotional stimuli than CD-only children, but CD+CU children are more likely to mistakenly label fearful expressions as another emotion, and take longer to recognize sadness in others compared to CD-only youth (Blair, Budhani, Colledge, & Scott, 2005; Blair, Colledge, Murray, & Mitchell, 2001; Dadds et al., 2006; Munoz, 2009; Stevens, Charman, & Blair, 2001). Stevens et al. found that children who scored high on psychopathic tendencies were less accurate in identifying facial expressions of fear and sad vocal affect compared to children low on psychopathic tendencies; however, these groups of children were comparable in recognizing facial expressions and vocal intonations of happiness and anger. Dadds and colleagues also found a negative association between CU traits and the ability to recognize fearful facial expressions among children, as well as a tendency to label neutral faces as angry. In an effort to extend findings to body postures, Munoz (2009) examined accuracy in identifying emotion from facial expressions and from body postures in a sample of boys aged 8-16, and found that boys with elevated CU traits were less accurate in labeling fear in both facial expressions and body postures. These studies provide robust evidence that children with CU traits exhibit a specific deficit in recognizing fear in others that

encompasses facial expression, vocal intonation, and body language, whereas CD-only youth do not exhibit these deficits (Pardini et al., 2003).

Researchers have hypothesized that difficulties labeling or recognizing fear among children high on CU traits, coupled with reduced reactivity to negative emotional stimuli, allows children with CU traits to more easily engage in antisocial behavior because they are not inhibited by the negative arousal associated with distress in others (Blair et al., 2005; Munoz, 2009). Moreover, these temperamental deficits in emotional reactivity and the insensitivity to punishment likely interfere with moral socialization, including the development of conscience that, within normative development, serves to inhibit children from engaging in antisocial behaviors (Blair, 2009; Pardini et al., 2003). Thus, these temperamental features likely underpin deficits in conscience and empathy development, each of which likely contributes to the development of CD+CU (Blair, 1995, 2005; Frick, 2006; Kochanska, 1993, 1997).

#### *Neural Functioning and CU*

Evidence from cognitive neuroscience is consistent with this possibility. Specifically, two neural regions associated with moral reasoning and empathy, the amygdala and the ventromedial prefrontal cortex (vmPFC; a region that is thought to be included within the orbitofrontal cortex (OFC) and to include parts of the medial OFC) (Greene, Sommerville, Nystrom, Darley, & Cohen, 2001; Koenigs & Tranel, 2007; Luo et al., 2006; Moll et al., 2002; Raine & Yang, 2006; Singer, 2006; Young & Koenigs, 2007), evidence dysfunction among adult psychopaths (Blair, 2004, 2007; Kiehl et al., 2001; Raine & Yang, 2006; Yang & Raine, 2009). The amygdala is involved in the recognition of emotion in others' facial expressions (Blair, 2004, 2007; Crowe & Blair, 2008; Hariri, Tessitore, Mattay, Fera, & Weinberger, 2002; Herba & Phillips, 2004; Jones, Laurens, Herba, Barker, & Viding, 2009; Lobaugh, Gibson, & Taylor, 2006), and plays a critical

role in aversive conditioning and associative (or instrumental) learning (Benes, 2006; Blair, 2004, 2007; Davidson, 2002; Davis, 2000; Killcross, Robbins, & Everitt, 1997; LeDoux, 1998; Patterson & Newman, 1993). The vmPFC appears to regulate the interaction of cognition and affect and is involved in reward-based decision-making (Bechara, Damasio, Damasio, & Lee, 1999; Blair, 2004; Blair et al., 2006a; Tranel, Bechara, & Damasio, 2000). More specifically, the vmPFC plays an important role in assessing the emotional value associated with a stimulus; this information is then communicated to the dorsolateral prefrontal cortex (DLPFC), a structure critically involved in self-regulation and selection of behavioral responses that continues to develop throughout childhood and into adulthood (Blair, 2004; Luciana, 2006).

The deficits in recognition of sad and fearful expressions among youth with CU traits reviewed earlier is likely a reflection of underactive amygdala functioning. In fact, functional brain imaging studies examining psychopathy in adulthood and CD+CU in adolescence and middle childhood have consistently reported amygdala hyporeactivity among individuals with elevated levels of psychopathy or CU traits (Birbaumer et al., 2005; Gordon, Baird, & End, 2004; Jones et al., 2009; Kiehl et al., 2001; Marsh et al., 2008; Veit et al., 2002). These findings are in contrast to findings from individuals who are primarily reactively, or impulsively, aggressive who demonstrate amygdala overreactivity to negatively valenced emotional (e.g., threatening) stimuli (Blair, 2010; Carre, Fisher, Manuck, & Hariri, 2012; Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Donegan et al., 2003; Lee, Chan, & Raine, 2008; New et al., 2009). In addition, as the amygdala is critically involved in aversive conditioning, it is not surprising that individuals with psychopathy exhibit deficiencies in aversive conditioning (Blair, 2005; Flor et al., 2002).

Individuals with psychopathy also exhibit impairments in response reversal and gambling tasks (Blair et al., 2001; Budhani & Blair, 2005; Cools, Clark, Owen, & Robbins, 2002), each of which involves the vmPFC (Blair, 2004, 2005, 2007; Kimonis et al., 2007; Marini & Stickle, 2010; Marsh et al., 2008). Response reversal tasks require individuals to adapt their responses subsequent to a change in the valence of the stimulus (e.g., a previously rewarded response shifts so that the rewarded stimulus is now punished), whereas gambling tasks consist of choosing cards from advantageous decks (i.e., smaller gains and relatively smaller losses, resulting in net gains over time) and disadvantageous decks (i.e., larger gains and relatively larger losses, resulting in net losses over time). As the vmPFC appears to provide information about expectations of reward and changes in contingencies to other regions for use in the selection of a response, dysfunction in this region will negatively impact an individual's ability to regulate their emotions and behaviors (Blair, 2004, 2005). However, adults with psychopathy exhibit greater impairment on these tasks than do children with psychopathic tendencies (Blair et al., 2001; Blair, 2004, 2005). Blair has suggested that such evidence may reflect the developmental course of the disorder (Blair, 2004). Given the interconnections between the amygdala and the vmPFC, and amygdala hyporesponsivity evident in children with CU traits, it is possible that over time, the diminished afferent input from the amygdala to the vmPFC leads to decreased responsiveness of the vmPFC (Blair, 2004, 2007; Bublitz & Drabick, 2008). Thus, amygdala hyporesponsivity may be critically involved in the developmental progression of CD+CU in childhood to adult psychopathy not only because of its potential influence on other brain regions, but also because of the amygdala's role in moral socialization.

### *Moral Socialization and CU*

Moral socialization refers to the process wherein caregivers try to instill an understanding of moral conduct in their children by rewarding prosocial behavior and punishing behavior that is considered morally wrong (Blair, 2004; Brody & Shaffer, 1982; Kochanska & Aksan, 2006). Some accounts are based on the idea that moral socialization occurs through the use of punishment. That is, because punishment elicits a fear response, individuals learn to associate this fear response with the act that resulted in the punishment, which in turn serves to inhibit that act in the future (Patterson & Newman, 1993). However, alternative accounts suggest that moral socialization is actually achieved through the activation and promotion of empathy, and that empathy assists in moral development, whereas fear impedes moral socialization (Eisenberg, 2002; Hoffman, 1994). In their review of the influence of parents and peers on moral socialization, Brody and Shaffer found that, regardless of age, power-assertive discipline was negatively associated with moral socialization (i.e., lower scores on indices of children's moral attitudes and behaviors).

Returning to potential biological underpinnings, one account of moral socialization suggests that among typically developing individuals, seeing another individual's distress elicits increases in autonomic activity, which can be interpreted as physical cues of discomfort and therefore an aversive reaction (Blair, 1995; Eisenberg et al., 1990, 1996; Fabes, Eisenberg, & Eisenbud, 1993; Hastings, Zahn-Waxler, & McShane, 2006; Hastings, Zahn-Waxler, Robinson, Usher, & Bridges, 2000; Holmgren, Eisenberg, & Fabes, 1998). Similarly, Blair (1995, 2004, 2005; Blair, Jones, Clark, & Smith, 1997) suggests that moral socialization occurs through the process of aversive conditioning. Repeated pairings of moral transgressions with another individual's distress elicits an aversive response in the individual, which in turn results in a

learned association between the moral transgression and the aversive reaction and that the behavior is wrong or bad. Subsequently, the moral transgressions themselves elicit this aversive response (potentially characterized by increased autonomic activity) that should serve to inhibit behaviors that are morally wrong. Therefore, moral socialization is reliant on aversive conditioning and instrumental learning, each of which involves the amygdala (Blair, 2004, 2007; Fowles & Kochanska, 2000). Given these associations, deficits in amygdala functioning exhibited by adults with psychopathy and youth with CU traits may interfere with their ability to form aversive stimulus—reinforcement associations and consequently hinder socialization processes (Blair, 2004, 2005, 2007; Blair et al., 2005). Blair’s account underscores the important role of ANS functioning in moral socialization and the development of empathy as moral socialization depends, in part, on the ability to experience a visceral response to another’s distress, which is likely underpinned by ANS and limbic system functioning. Consistent with this possibility, the amygdala innervates the ANS via the hypothalamus and brainstem circuits and receives information about peripheral bodily states (Harper, Bandler, Spriggs, & Alger, 2000; Harper, Bandler, Spriggs, Lee, & Alger, 1998; LeDoux, 2000; Shirtcliff et al., 2009; Williams et al., 2005; Yang et al., 2009). In sum, hyporesponsivity to aversive stimuli (e.g., another person’s distress) may impede the socialization of moral emotions, subsequently leading to a lack of empathy manifested as CU traits. The combination of this hyporesponsivity to others’ distress and CU traits thus may facilitate the use of antisocial and aggressive behavior, thereby resulting in CD+CU (Blair, 1995, 2004, 2007; Blair et al., 2005).

Taken together, research to date suggests that childhood-onset CD+CU is associated with lack of fearful inhibitions and low emotional reactivity, whereas childhood-onset CD-only is associated with high emotional reactivity and low effortful control, defined as “...the ability to

inhibit a dominant response to perform a subdominant response” (Rothbart & Bates, 2006, p. 129). Given distinct temperamental features of children with childhood-onset CD+CU and the importance of identifying children at risk for serious and persistent antisocial behavior, it is crucial to refine and identify distinct characteristics of children with childhood-onset CD+CU using multiple levels of analysis, which may in turn elucidate the distinct etiology of CD+CU. Because temperament has a biological basis (Kagan, 1998; Rothbart & Ahadi, 1994; Rothbart, Ahadi, & Evans, 2000; Rothbart, Ahadi, & Hershey, 1994; Rothbart & Bates, 1998), it is likely that children with childhood-onset CD+CU could be distinguished from those without CU traits based on biological measures that index temperamental dimensions, such as ANS functioning (Kagan, 1998; Rothbart & Bates, 1998). Indeed, autonomic measures may be especially informative in this regard because they are thought to be less prone to bias and measurement error than self-report indices; also, they provide more sensitive indices of psychological states and the neural processes underlying the states and resultant behaviors (Lorber, 2004). Despite these issues and the growing literature seeking to validate and understand subgroups of children with CD, there is a dearth of literature examining differences in ANS functioning among children with CD with and without CU traits, particularly among childhood-onset CD.

#### Overview of the ANS

The ANS is the portion of the nervous system that controls basic visceral functions of the body such as cardiovascular activity, metabolism, digestion, and thermoregulation. The ANS consists of two branches, the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). This division is based on the location in the brain and spinal cord in which the autonomic nerves originate. Generally, the SNS regulates involuntary reactions to stress (e.g., increased heart and breathing rates) and prepares the body to respond to stressors,

whereas the PNS promotes growth and restoration of homeostasis. The regulation of the ANS has been conceptualized as a balance system because most organs that it innervates receive both sympathetic and parasympathetic input. Furthermore, the effects of SNS and PNS activation on an organ are generally antagonistic. This antagonistic relationship can be seen in patterns of cardiac activity that are primarily determined by the interaction of acceleratory SNS activation and deceleratory PNS activation. Thus, organs can receive simultaneous impulses that both increase and decrease their activity, which means that depending on the relative input of the PNS and SNS at any given time, the net effect of ANS activation may be either inhibitory or excitatory (Beauchaine, 2001; Brownley et al., 2000; Porges, 2003).

#### *PNS and SNS Indices*

Because heart rate (HR) has been found to be a robust biological correlate of antisocial behavior, many studies examining the link between biological processes and antisocial behaviors have utilized measures of HR (e.g., Baker et al., 2009; Ortiz & Raine, 2004; Posthumus, Bocker, Raaijmakers, Van Engeland, & Matthys, 2009). Researchers often measure levels of resting HR and heart rate reactivity (HRR; i.e., changes in heart rate in response to a stressor). However, HR is subject to both parasympathetic and sympathetic influences. As the separate influence of each of these branches cannot be parsed out within the HR construct, HR is not a particularly useful ANS index for distinguishing between childhood-onset CD+CU and CD-only. SNS and PNS functioning may be differentially related to CD+CU and CD-only; thus, the separate and concurrent consideration of both the PNS and SNS is important for advancing theoretical models about childhood-onset CD with and without CU.

Parasympathetic cardiac influences are indexed by respiratory sinus arrhythmia (RSA), which is the ebb and flow of heart rate across the respiratory cycle, or the variability of heart rate

for each respiratory cycle (Beauchaine, 2001; Porges, 1995). RSA is commonly regarded as an estimate of vagal tone (PNS influence on cardiac activity) because it serves as a proxy for processes that could only be measured via invasive procedures (Beauchaine, 2001). In terms of the relation between RSA and vagal tone, RSA results from *decreases* in vagal efference during inhalation, which increases heart rate, and *increases* in vagal efference during exhalation, which decreases heart rate (Beauchaine, 2001; Bubier, Drabick, & Breiner, 2009). However, “RSA” and “vagal tone” are not equivalent; RSA is an imperfect index of vagal tone because changes in respiration rate and tidal volume influence RSA independent of vagal tone (Beauchaine, 2001).

Electrodermal activity (EDA) is under exclusive control of the SNS. EDA describes the changes in the skin’s ability to conduct electricity, which is linked to changes in the hydration of the sweat glands. EDA often is operationalized as skin conductance (SC), which is an index of very small changes in the electrical activity of the skin, given that SC reflects the activity of the eccrine sweat glands (Erath, El-Sheikh, & Cummings, 2009) and an increase in sweating leads to an increase in SC activity (Scarpa & Raine, 1997). SC can be assessed by two components: a baseline component (skin conductance level), and a reactivity component that measures changes in SC from baseline in response to a stimulus (Erath et al., 2009). Research indicates that SC responses reflect an index of the amount of attention allotted to processing a stimulus (Dawson, Schell, & Fillion, 1990; Scarpa & Raine, 1997). Because the SNS is sensitive to stress and emotional arousal, SC activity can be used to assess stress reactivity to aversive or arousing events (Scarpa & Raine, 1997).

Last, SNS influences on cardiac activity also can be indexed by cardiac pre-ejection period (PEP). PEP refers to the time between the onset of the left ventricular depolarization and ejection of blood into the aorta (Beauchaine, 2001; Brenner, Beauchaine, & Sylvers, 2005). This

time interval is determined by beta-adrenergic influences that are in turn under the control of the SNS. Shorter time intervals reflect more sympathetic activity. Several theories linking ANS functioning and behavior have been derived from research using these various indices. It is to these theories that I now turn.

### Theories Linking the ANS and Behavior

#### *Polyvagal Theory*

According to Porges' Polyvagal Theory (1995), the evolution of the human ANS provides the neurophysiological substrates for and determines the range of emotional experiences and affective processes that are critical components of social behavior (Porges, 2003). Polyvagal Theory considers two distinct branches of the vagus, or tenth cranial nerve, which provide two sources of inhibitory input to the heart via the PNS. Each branch is posited to support a different adaptive behavioral strategy. These two branches include a phylogenetically older branch, which originates in the dorsal motor nucleus, and a newer branch that originates in the nucleus ambiguus. The dorsal motor nucleus controls what has been called the "vegetative vagus," which maintains unconscious reflexive cardiac activity, such as the slowing down of heart rate that occurs with the orienting reflex (Porges, 1995; Porges et al., 1996). This branch is thought to be rooted in the primary survival strategy of primitive vertebrates (reptiles and amphibians), which freeze when threatened. This illustrates the role of the vagus in suppressing metabolic demands under conditions of danger (Porges, 1995, 2001, 2003). The nucleus ambiguus branch, often referred to as the "smart vagus," is unique to mammals and mediates cardiac activity when environmental demands call for a more complex form of coping (Porges, 1995, 2001, 2003). Subsequent to orienting, mammals must choose to either engage with the threat or defer to fight-flight responding. Engaging with the threat necessitates sustained

attention, which is associated with inhibition of heart rate controlled by the vagus (Beauchaine, 2001; Suess et al., 1994; Weber, van der Molen, & Molendaar, 1994). The fight-flight response, however, is accompanied by rage and panic, and entails vagal withdrawal and heart rate accelerations controlled by the SNS (George et al., 1989; Porges, 1995, 1996). As the SNS is no longer opposed by the inhibitory vagal (PNS) influences, vagal withdrawal allows for large increases in cardiac output by the SNS that provide the individual with the resources needed to deal with dangerous circumstances. Thus, there is a functional purpose underlying the association between strong emotional experience and vagal withdrawal (Beauchaine, 2001; Beauchaine et al., 2007; Porges, 2001, 2003).

The smart vagus blocks SNS input to the heart, which accelerates heart rate, when social engagement is the most appropriate response, and allows the SNS influence on the heart to proceed unopposed when fighting or fleeing is the most appropriate response (Beauchaine et al., 2007; Porges, 1995, 2001). The mammalian ANS is presumed to function according to a phylogenetic hierarchical organization. Response strategies to threat are initially dictated by the newest neural structures, followed by the older structures if an initial response strategy fails. In other words, if vagally mediated social engagement behaviors preclude the individual's effective coping with a stimulus, response strategies shift to fight-flight behaviors controlled by the older SNS. If this response also fails, then immobilization behaviors occur. These are controlled by the vegetative vagus, the oldest response system. Polyvagal Theory posits that emotion regulation and social affiliation are emergent properties served by the smart vagus; thus, engagement of the newer vagal system suppresses the intense emotional reactions that characterize fight-flight responding (Beauchaine, 2001; Beauchaine et al., 2007; Porges, 1995, 2001, 2003).

Polyvagal Theory consequently can provide a model for relations between ANS functioning and CD symptoms, as well as how PNS activity may reflect emotion regulation abilities and the temperamental construct of effortful control. According to this theory, deficiencies of the smart vagus should put individuals at increased risk for emotion dysregulation and maladaptive social behavior. Furthermore, because ineffective functioning of the smart vagus would increase the tendency for individuals to rely on fight-flight behavioral responses that induce intense approach or avoidance emotional reactions, reduced cardiac vagal tone should be observed in disorders that are characterized by approach emotions, like anger (Beauchaine, 2001; Beauchaine et al., 2007). The literature is generally supportive of this proposition; for example, findings have shown that PNS functioning, as indexed by RSA, is reflective of individual differences in emotional reactivity and the ability to regulate arousal, emotion, or affect (Beauchaine, 2001; Calkins, 1997; Hastings et al., 2008; Kennedy, Rubin, Hastings, & Maisel, 2004; Porges, 1995; Santucci et al., 2008). High levels of basal RSA have been associated with social competence, empathy, and emotion regulation (Beauchaine, 2001; Eisenberg et al., 1995; Fabes, Eisenberg, & Eisenbud, 1993; Fox & Field, 1989). In contrast, low vagal tone is related to dysregulated affect and emotional lability, or a tendency toward extreme emotional responses, as well as externalizing behavior problems, including aggression, CD, and ODD symptoms, among at-risk and clinical samples ranging from early childhood through adolescence (Beauchaine, 2001; Beauchaine et al., 2007; Calkins & Dedmon, 2004; Donzella, Gunnar, Kreuger, & Alwin, 2000; El-Sheikh et al., 2001; Field et al., 1996; Gottman & Katz, 2002; Kennedy et al., 2004; Mezzacappa, Tremblay, Kindlon, & Saul, 1997; Pine et al., 1998; Porges, 2007). Moreover, vagal deficiencies (i.e., decreased baseline RSA and lower levels of RSA modulation) are related to externalizing behaviors among children and adolescents in

ethnically diverse samples (Beauchaine, Katkin, Strassberg, & Snarr, 2001; Crowell et al., 2006; Mezzacappa et al., 1997; Pine et al., 1998), as well as to internalizing symptoms (e.g., anxiety, depression) among youth (Lyonfields, Borkovec, & Thayer, 1995; Yeragani et al., 1993). This lack of specificity between vagal tone and psychological symptoms suggests that vagal deficiencies confer non-specific risk for a variety of difficulties (i.e., multifinality). Alternatively stated, although Polyvagal Theory provides a basis for understanding emotion dysregulation as the product of vagal deficiencies that lead to fight-flight responses that contribute to psychopathology, it does not explain why emotion dysregulation is manifested as externalizing behaviors among some individuals and internalizing behaviors among others. As discussed by Beauchaine (2001), to explain the predominant behavioral response set observed among various forms of psychopathology, sympathetic functioning must be considered in conjunction with parasympathetic functioning (Beauchaine, 2001; Beauchaine et al., 2007). In an effort to address this issue, Beauchaine (2001) posited a model that integrates Polyvagal Theory and Gray's Theory of Motivation (Gray, 1982, 1987; Gray & McNaughton, 2000) and outlines the interaction between the PNS and the SNS in the prediction of behavioral outcomes among youth.

#### *Beauchaine's Integrated Model of ANS Functioning and Behavior*

Beauchaine's (2001) integrated model serves to explain the conditions under which externalizing versus internalizing behaviors are likely to occur. This integrated model of ANS-behavior relations posits that the motivation underlying behavior is a reflection of the balance between the behavioral activation system (BAS, the reward system), and the behavioral inhibition system (BIS, the punishment system) and is under control of the SNS, whereas regulation-related abilities are controlled by the PNS. When behavioral responses are necessary, the BAS serves to maximize reward and minimize punishment, whereas the BIS produces

anxiety and fear, which serve to inhibit appetitive behaviors when aversive consequences (punishment) are anticipated. According to Beauchaine, an under-responsive reward system (BAS) combined with deficiencies in vagal modulation of emotion will result in the aggressive and sensation-seeking behaviors that characterize disruptive behavior disorders (e.g., ODD and CD). Alternatively, an over-responsive reward system (BAS), in combination with deficient vagal modulation of emotion, results in behaviors that are characteristic of internalizing disorders. Thus, Beauchaine suggests that aggressive behavior disorders, such as CD, should be characterized by attenuated SNS activity in conjunction with attenuated vagal tone and/or excessive vagal reactivity (Beauchaine, 2001). Beauchaine's model consequently has implications for elucidating the relation between ANS functioning and childhood-onset CD.

Based on Polyvagal Theory, different patterns of ANS functioning should be associated with childhood-onset CD with and without CU traits. CD-only should be a reflection of (a) low baseline RSA, which reflects negative emotionality or negative emotional traits; and (b) excessive RSA reactivity (vagal withdrawal), which is thought to characterize dysregulated emotional states. In contrast, childhood-onset CD+CU, which is presumed to be related to deficiencies in moral development rather than difficulties with regulating emotions (Frick, 2006; Frick & White, 2008), may be more likely to be associated with (a) low baseline RSA, consistent with negative emotional traits like anger; and (b) either normative levels of RSA reactivity or low RSA reactivity (PNS activation). However, according to Beauchaine's (2001) integrated vagal and motivational model, behavioral outcomes cannot be determined by one system alone. Therefore, SNS functioning must be considered in conjunction with the vagal system.

There are generally two models in the literature regarding how the SNS contributes to antisocial and aggressive behavior. One model suggests that aggression is a result of reward

dominance, which is associated with an overactive BAS, and posits that reactions to threatening stimuli are dominated by fight-flight responses accompanied by rage and panic. As noted above, fight-flight responding is characterized by the withdrawal of vagal (or PNS) influences and the dominance of SNS influences on cardiac activity. Based on this explanation, then, the SNS-dominated fight-flight responses to stressors would be expected to fuel aggressive responses to provocation (Beauchaine, 2001; Murray-Close & Crick, 2007; Porges, 1995, 2003; Scarpa & Raine, 1997). Moreover, this explanation implies that aggressive responses to provocation should be accompanied by heightened sympathetic activity and, consequently, reactive aggression. Thus, this explanation is likely more characteristic of childhood-onset CD-only than CD+CU.

A second model proposes that trait aggression reflects sensation seeking, which is a result of autonomic underarousal and reward insensitivity, consistent with an underactive BAS (Beauchaine et al., 2007). Two explanations have been posited to explain the relation between autonomic underarousal and aggressive behavior: sensation-seeking theory and fearlessness theory (Ortiz & Raine, 2004; Raine, 2002). From the sensation-seeking theoretical perspective, underarousal is experienced as an unpleasant state and, consequently, individuals engage in aggressive behavior to increase their physiological arousal to a more pleasant level (Kibler, Prosser, & Ma, 2004; Ortiz & Raine, 2004; Raine, 2002; Schneider, Nicolotti, & Delamater, 2002). The fearlessness explanation posits that autonomic underarousal reflects a lack of fear, which allows individuals to engage in aggression without regard for potential consequences (Ortiz & Raine, 2004; Raine, 2002). These theories are more effective in accounting for proactive (as opposed to reactive) aggression, suggesting that they may be more applicable to the development of CD+CU than CD-only.

## ANS Functioning and CD

Consistent with sensation-seeking and fearlessness theories, autonomic underarousal is a well-replicated correlate of aggressive behavior (e.g., Raine, 2002). For example, two associations between ANS functioning and childhood-onset CD have been well-documented: the association between lower resting HR and CD symptoms among youth (Ortiz & Raine, 2004; Raine, Venables, & Mednick, 1997) and between reduced EDA (or SC) at rest and CD symptoms (Delamater & Lahey, 1983; Lorber, 2004; Ortiz & Raine, 2004; Patrick, 2008). For example, studies have reported lower baseline HR and SC among children with CD and/or ODD (van Goozen et al., 1998; van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000), and the results of a meta-analysis conducted by Lorber (2004) indicate consistent associations between conduct problems and aggression and (a) low resting HR and (b) attenuated resting EDA among children. Furthermore, Van Bokhoven, Matthys, van Goozen, and van Engeland (2005) found that among a sample of children with disruptive behavior disorders, low resting SC was the best predictor of serious antisocial behavior in adolescence.

Research also demonstrates associations between attenuated autonomic reactivity and CD symptoms in childhood (McBurnett et al., 1993; van Goozen, Fairchild, Snoek, & Harold, 2007). Boyce and colleagues (2001) reported attenuated SNS reactivity (indexed by lower PEP reactivity) and externalizing behavior in a sample of 6- to 7-year-old children. Attenuated SNS reactivity also was found among a clinical sample of youth ages 8 to 12 with ODD and/or CD compared to controls with no psychiatric diagnoses (Mead et al., 2004, as cited in Beauchaine et al., 2007). In their meta-analysis, Kibler et al. (2004) found a negative association between HR reactivity (attenuated autonomic activity) and externalizing and aggressive behaviors among children. Taken together, these studies suggest that antisocial behavior and aggression are

associated with attenuated autonomic reactivity among youth. However, there is also empirical evidence demonstrating an association between CD symptoms and heightened autonomic reactivity, particularly in the context of provocation. For example, Williams, Lochman, Phillips, and Barry (2003) reported a relation between aggression and greater HR reactivity among boys aged 9-13 who were told that a peer with whom they were going to work was in a bad mood and wanted to pick a fight with them. Van Goozen et al. (1998) also found greater HR reactivity in response to peer provocation among a group of children 8-11 years old with ODD compared to a control group with no psychiatric diagnosis.

In addition to these inconsistencies in findings related to autonomic reactivity, the extant literature is limited by the fact that the majority of studies examining relations between ANS reactivity and CD symptoms focus on HR, HR reactivity, and SC activity. As discussed previously, HR and HR reactivity reflect activity of both the PNS and the SNS, whereas SC specifically reflects SNS activity. Thus, the independent contribution of the PNS is unclear. However, it is important to consider direct measures of PNS activity (e.g., RSA) because (a) the SNS and PNS operate together, not in isolation; (b) PNS activity may be an important correlate of antisocial behavior; and (c) patterns of joint action of the PNS and SNS may provide greater specificity in terms of associations between ANS activity and subtypes of children with CD symptoms.

Nevertheless, the literature linking PNS activity with CD symptoms is also characterized by inconsistencies. For example, although low baseline levels of RSA consistently are related to emotion dysregulation and CD symptoms (Beauchaine, 2001; Beauchaine et al., 2007; El-Sheikh, 2005; Fox & Field, 1989; Hinnant & El-Sheikh, 2009; Kennedy, Rubin, Hastings, & Maisel, 2004; Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007; Vasilev,

Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009), findings from some community-based samples do not report relations between low baseline RSA and externalizing behavior problems (Calkins et al., 2007; El-Sheikh et al., 2001; El-Sheikh & Whitson, 2006). Furthermore, the relation between RSA reactivity and CD symptoms is even less clear. High RSA reactivity, or decreases in RSA from baseline (i.e., greater vagal withdrawal), is associated with better emotion regulation and decreased externalizing and internalizing problems from young childhood through adolescence (Calkins, 1997; El-Sheikh et al., 2001; El-Sheikh & Whitson, 2006; Porges, 2007). Nevertheless, high RSA reactivity also is reportedly associated with internalizing symptoms (Boyce et al., 2001) and clinically significant behavior problems (Crowell et al., 2005; Beauchaine et al., 2001). Thus, moderate vagal withdrawal is adaptive because it allows the organism to attend to stressors and enact social engagement strategies to cope effectively with the stressor (Beauchaine, 2001; Porges, 1995, 2003, 2007). However, if the stressor is prolonged or if there are vagal system deficiencies (e.g., excessive vagal withdrawal), the inhibitory influence of the PNS may be largely removed, inhibiting the ability of the PNS to effectively manage the stressor. In this case, the SNS may be activated, leaving the individual subject to fight-flight responding and therefore unable to regulate associated emotional states of rage or panic. Thus, excessive vagal withdrawal or high RSA reactivity can reflect anger, which is related to externalizing behavior problems, or panic, which is more characteristic of internalizing symptoms, rendering RSA reactivity a non-specific index of emotion dysregulation in childhood (Beauchaine, 2001).

In contrast to predictions from Beauchaine's (2001) model, among community-based samples of young children, some studies have found an association between lower RSA reactivity (or RSA augmentation and PNS activation) and antisocial and aggressive behavior. For

example, in a sample of 5-6 year old children, Obradovic, Bush, Stamperdahl, Adler, and Boyce (2010) found an association between lower RSA reactivity (i.e., PNS activation) and externalizing symptoms (i.e., ODD symptoms, conduct problems, and overt hostility). In a sample of 5 year olds who were divided into an externalizing group, a mixed internalizing and externalizing group, and a low problem group, Calkins, Graziano, and Keane (2007) found that children in the externalizing group evidenced the least vagal withdrawal (i.e., the smallest decreases in RSA in response to challenge tasks), whereas the mixed problem group demonstrated the greatest decrease in RSA. Similarly, Boyce et al. (2001) found that 6-7 year old children with externalizing behavior problems showed low RSA reactivity, indicating activation of the PNS, whereas children with high levels of internalizing symptoms evidenced greater RSA withdrawal compared to low symptom children. Taken together, these findings suggest that co-occurring internalizing and externalizing behavior problems may contribute to inconsistencies in findings related to RSA reactivity. This possibility is consistent with ample evidence demonstrating significant co-occurrence of ADHD, ODD, and CD with anxiety disorders and depression, particularly among youth (Angold et al., 1999; Bubier & Drabick, 2009; Drabick, Gadow, & Sprafkin, 2006; Fleitlich-Bilyk & Goodman, 2004; Ford, Goodman, & Meltzer, 2003; Marmorstein, 2007). Thus, it is possible that specific relations between RSA and externalizing behaviors are obscured by failing to control for concurrent internalizing symptoms.

In addition, some research has found no differences in RSA reactivity among children with and without CD. For example, Mead et al. (2004, as cited in Beauchaine et al., 2007) examined RSA baseline and reactivity in 8-12 year olds with aggressive ODD and/or CD compared to a group of controls with no psychiatric diagnosis. The children engaged in a monetary incentive task after which they watched a film designed to evoke feelings of sadness

and empathy. Consistent with previous research, these researchers found attenuated baseline RSA in the aggressive ODD/CD group. However, the authors found no differences between the ODD/CD and control groups on RSA reactivity; both groups displayed similar vagal withdrawal, indexed by decreasing RSA, during the film.

These inconsistencies in the findings regarding RSA reactivity and CD symptoms may be a function of the age and type of samples examined; Crowell et al. (2005) examined a clinical sample of adolescents, whereas the normative samples examined by Boyce et al. (2001) and Calkins et al. (2007) were under the age of seven. Studies indicate moderate stability in baseline and reactivity measures of PNS and SNS by middle to late childhood; moreover, reactivity likely does not become stable until this developmental period (Bornstein & Suess, 2000; Calkins & Keane, 2004; Doussard-Roosevelt, Montgomery, & Porges, 2003; El-Sheikh, 2005, 2007; El-Sheikh et al., 2009; Hinnant & El-Sheikh, 2009; Hinnant, Elmore-Staton, & El-Sheikh, 2011). Furthermore, individual differences in SNS functioning may become apparent before those related to PNS functioning, and basal PNS stabilizes prior to PNS reactivity (Beauchaine et al., 2007; Bornstein & Suess, 2000; Bubier & Drabick, 2008; Calkins & Keane, 2004; El-Sheikh, 2005). Thus, the greater consistency among SNS, compared to PNS, findings may reflect the earlier stabilization of the SNS and variability in the findings related to PNS indices may reflect the developmental course of PNS functioning. Inconsistencies across findings related to ANS activity and CD symptoms also may be due to a lack of research examining the independent contributions of the SNS and PNS. Concurrent assessment of separate indices of the SNS and PNS, such as PEP and RSA respectively, would allow for a more comprehensive account of the differences among children with CD with and without CU, and could serve to clarify and potentially reduce the inconsistencies in the extant literature.

Inconsistencies also may be a consequence of failing to distinguish children with CD symptoms who exhibit CU traits from those that do not. Thus, examining ANS patterns related specifically to CU traits and/or to childhood-onset CD+CU could help to clarify the developmental mechanisms underlying the affective/interpersonal deficits that characterize children whose CD symptoms are particularly severe and who are at increased risk for developing adult psychopathy.

#### ANS Functioning and CU Traits

Although there is a large body of literature examining the psychophysiological characteristics of psychopathy among adults, there is a much smaller literature examining such correlates among children with CU traits, though findings among youth are consistent with findings among adults with psychopathy. For example, Fung et al. (2005) demonstrated reduced anticipatory SC responding to white noise bursts in a sample of 16 year olds with elevated psychopathy scores. Kimonis et al. (2008b) examined the relation between CU traits and EDA reactivity to a computerized provocation task among a sample of juvenile offenders ages 12-20. They found that higher scores (elevated levels of CU traits) were correlated with lower EDA reactivity to both high and low provocation. Although Fung et al. and Kimonis et al. utilized different types of stressors (white noise bursts vs. computerized provocation), both studies found an association between reduced SNS reactivity to a stressor and psychopathic traits among youth, which is consistent with associations found among adult psychopaths (Arnett & Newman, 1997; Fowles, 2000; Glenn et al., 2007; Lorber, 2004). Similar to Fung et al., Wang, Baker, Gao, Raine and Lozano (2012) assessed SC response and HR reactivity to white-noise bursts among a sample of 9-10 year old children and found an association between reduced anticipatory SC responding and higher psychopathic traits. Wang et al. also found that children with higher

psychopathic traits demonstrated increased HR acceleration in anticipation of the white-noise bursts, which is consistent with findings among adult psychopaths. Interestingly, reduced SC response was uniquely related to the interpersonal facet of psychopathy characterized by deceit and manipulation of others, whereas the greater HR acceleration was specifically related to callousness and impulsive behavioral traits. However, the authors did not distinguish between children who were (a) high on psychopathic traits and CD symptoms and (b) high on CD symptoms but low on psychopathic traits, a distinction that could have further clarified the findings. These findings do suggest that greater HR acceleration may reflect a diminished capacity to regulate both affect and behavior, but further reinforce the need to (a) examine the separate influence of the PNS and the SNS on cardiac activity, and (b) to distinguish between children with CD+CU (or psychopathic traits) and those with CD-only.

Although several studies have examined ANS functioning among youth with psychopathic features, studies comparing patterns of ANS functioning among children with CD+CU and CD-only are sorely lacking. In an examination of subgroups of hyperactive children, Delamater and Lahey (1983) found that children with high conduct problems and low anxiety (a common correlate of CU traits; Frick et al., 1994, 1999; Frick & Morris, 2004; Frick & White, 2008; Lynam et al., 2005; Pardini et al., 2007; Pardini & Fite, 2010) displayed the lowest ANS activity in terms of HR and SC baseline and reactivity. Similarly, among children in middle to late childhood with emotional and behavioral difficulties with and without CU traits, Blair (1999) compared SC responding to pictures depicting distress cues, threatening images, and neutral objects. Blair (1999) reported that children with psychopathic traits evidenced less SC reactivity to distress cues compared to both children with emotional and behavioral problems without psychopathic traits and children with neither emotional and behavioral problems or

psychopathic traits. In contrast, children with emotional and behavioral difficulties and low psychopathic traits did not demonstrate SCL hyporesponsivity to distress cues. These findings indicate that children with CD+CU evidence sympathetic underarousal, similar to adults with psychopathic traits. Anastassiou-Hadjicharalambous and Warden (2008) examined baseline HR and HR in reaction to an emotionally evocative film among children (ages 7-11) who were divided into a CD group, a CD with CU traits group, and a control group. Children with CD and CU traits demonstrated lower baseline HR and lower HR in reaction to the emotionally evocative film than both the CD-only group and controls. Taken together, these findings indicate (a) a general association between childhood-onset CD with CU traits and autonomic underarousal, and (b) that autonomic differences between childhood-onset CD+CU and CD-only are apparent by middle to late childhood. Furthermore, the autonomic underarousal demonstrated by children with CD+CU was in the context of cues of another's distress, consistent with findings from the adult psychopathy literature (Blair et al., 1997).

In sum, although the current literature examining relations among ANS functioning and childhood-onset CD is informative, it also is beleaguered by inconsistencies. In addition, with only two studies to date, research specifically examining differences in ANS functioning between children with CD+CU and those with CD-only is clearly only in its infancy. The difficulties within this literature make drawing clear conclusions regarding the relations among ANS functioning and CD with and without CU traits challenging, if not impossible. However, based on the theories and the evidence presented, several predictions can be made regarding the relations among ANS functioning and CD with and without CU traits.

Children with early-onset CD-only are characterized by high emotional reactivity, deficits in emotion regulation, and disinhibition (Frick, 2012; Frick & Viding, 2009). In contrast,

children with early-onset CD+CU are characterized by a lack of fearful inhibitions, deficits in emotional reactivity, and insensitivity to punishment cues (Frick, 2012; Frick & White, 2008; Frick & Viding, 2009). Among children with CD+CU, antisocial behavior more broadly, and instrumental aggression in particular, likely stems from deficits in moral or conscience development, as opposed to deficits in emotion regulation that are more likely to characterize children with CD-only. It is therefore likely that children who develop CD+CU would manifest low baseline RSA but normative levels of RSA reactivity, reflecting negative emotional traits but age-appropriate emotion regulation abilities. A child with this PNS profile would be expected to exhibit angry and hostile affect, but would be able to regulate these emotions in response to stressors. Children with CD-only are posited to exhibit low baseline RSA and excessive RSA reactivity, reflecting negative emotional traits and emotion dysregulation. A child with this PNS profile is expected to be characterized as angry and hostile, but unlike youth with CD+CU, unable to regulate these emotions so that when confronted with a stressor, the child would be expected to exhibit an angry, impulsive reaction.

In terms of SNS activity, children with CD+CU are expected to be characterized by an underactive BAS, which would be manifested as longer PEP at baseline and less PEP reactivity. This SNS profile is thought to reflect a reduced sensitivity to reward, and thus a motivation to obtain larger rewards without being deterred by the threat of punishment that would contribute to the sensation-seeking behaviors and fearlessness characteristic of childhood-onset CD+CU. Combined with the profile of PNS activity described above, this profile of SNS activity could serve to explain the behavioral (use of instrumental aggression) and emotional (callousness/lack of empathy) presentation of childhood-onset CD+CU. Alternatively stated, because of their profile of SNS activity, such individuals are motivated to seek out rewards and are insensitive to

punishment, which puts these children at increased risk of experiencing deficits in the development of empathy. Combined with a PNS profile that (a) predisposes the child to demonstrate negative affect, but (b) also equips them with the ability to regulate their anger in the context of stressors, these correlates of SNS activity may facilitate the use of instrumental aggression and violence among these youth. Specifically, these youth are capable of being more planful and less impulsive with their use of aggression, and when seeking a goal or reward, are not deterred by potential aversive consequences associated with aggression.

In contrast, childhood-onset CD-only is likely characterized by overactive BAS activity, which would be reflected in shorter baseline PEP and heightened PEP reactivity. Thus, this profile of SNS activity is posited to reflect a heightened sensitivity to rewards, and the impulsivity and disinhibition associated with CD. Combined with the PNS profile described above (i.e., low baseline RSA and excessive RSA reactivity), this SNS profile is reflected in a behavioral profile among CD-only children in which children are highly impulsive and lack the emotion regulatory abilities to control their negative reactivity. Thus, such children would be highly emotionally reactive, especially to negative stimuli; when combined with disinhibition, this predisposition is likely to lead to reactive aggression. However, relative to CD+CU youth, CD-only youth are more likely to exhibit anxiety, or inhibition, to distress cues that is associated with empathy, and therefore they do not demonstrate high levels of CU traits.

#### *Parenting Behaviors and CD with and without CU*

Based on the principle of multifinality, not all children with these particular patterns of ANS functioning would be expected to demonstrate childhood-onset CD with and without CU traits. Thus, contextual factors such as parenting behaviors may interact with children's ANS functioning to exacerbate or buffer the child's risk for CD with and without CU. There is ample

evidence that negative parental behaviors, which include coercive acts, negative emotional expressions, and verbal and physical aggression that parents direct at their children, are associated with CD symptoms among youth (Compton, Snyder, Schrepferman, Bank, & Shortt, 2003; Cote, Vaillancourt, Barker, Nagin, & Tremblay, 2007; Erath, El-Sheikh, & Cummings, 2009; Leve, Kim & Pears, 2005; Patterson, 2002; Pettit & Arsiwalla, 2008; Silk, Sessa, Morris, Steinberg, & Avenevoli, 2004; Stormshack, Bierman, McMahon, Lengua, & Conduct Problems Prevention Group, 2000). Thus, for children with the ANS profiles thought to be associated both with CD+CU and CD-only, harsh parental behaviors would exacerbate children's CD symptoms. Among children with attenuated PNS functioning at baseline, excessive PNS withdrawal, and heightened PEP activity, it is likely that negative parental behaviors will exacerbate children's CD symptoms. According to coercion theory (Patterson, 1982, 2002), children learn to interact aggressively with others through coercive, or hostile and emotionally negative, parent-child interactions. Children with an ANS profile predicted to be associated with CD-only (i.e., excessive PNS withdrawal in response to stressors) exhibit emotion dysregulation, increasing the likelihood that these children will elicit harsh parenting and continue to engage in coercive interchanges with parents. Thus, the parent-child relationship may spiral into a pattern of escalating coercive exchanges (Keenan & Shaw, 1995; Patterson, 1982, 2002; Scaramella & Leve, 2004). When emotional and physiological dysregulation is reinforced by coercive interchanges with parents, these children fail to learn effective emotion regulatory strategies and are instead socialized to be emotionally (and perhaps autonomically) labile (Patterson, 1982; Scaramella & Leve, 2004). In turn, these coercive interchanges with parents and the resultant dysregulation are associated with various maladaptive outcomes, including externalizing behavior problems, problematic peer relations, and academic difficulties (Morris, Silk, Steinberg,

Myers, & Robinson, 2007; Patterson & Capaldi, 1990; Scaramella & Leve, 2004). Indeed, children who are emotionally dysregulated may have difficulties with attending to schoolwork, abiding by classroom rules, and negotiating peer processes effectively (Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003; Laird, Jordan, Dodge, Pettit, & Bates, 2001; Scaramella & Leve, 2004; Schwartz & Proctor, 2000), instead behaving in ways that serve to escalate the emotional arousal rather than diffusing and resolving conflict (Kopp, 1989). Children who are exposed to harsh discipline often learn that aggression is an acceptable way of obtaining desired outcomes, controlling others, and solving problems (Bandura, 1973; Gershoff, 2002; Pardini et al., 2007); thus, both children with CD+CU and CD-only learn to utilize aggression to solve their problems through their exposure to harsh parenting behaviors.

Although harsh parental behaviors would likely exacerbate CD symptoms among children at risk for CD+CU and CD-only based on their patterns of ANS functioning, low parental warmth may confer risk specifically for children who are physiologically at risk for CD+CU. Parenting behaviors characterized by warmth are important for moral socialization, and in particular, the development of empathy and the internalization of prosocial norms (Fowles & Kochanska, 2000; Kochanska, 1997). Higher levels of parental warmth and responsiveness in infancy prospectively predict increased empathy (Kiang, Moreno, & Robinson, 2004) and guilt in response to wrong-doing (Kochanska, Forman, Aksan, & Dunbar, 2005). In fact, research has shown that positive parenting practices characterized by warmth and involvement are associated with decreases in CU traits over time (Frick, Kimonis, Dandreaux, & Farell, 2003c). It is especially likely that among children with attenuated baseline parasympathetic functioning, normative parasympathetic reactivity, and attenuated SNS functioning at baseline and reactivity, lower parental warmth would be uniquely associated with increased risk for CD+CU. Based on

their hypothesized ANS functioning, these children would not be expected to exhibit negative arousal or anxiety in response to parental punishment or another individual's distress, each of which is theorized to be necessary for proper moral socialization. Combined with a lack of parental warmth, these youth are therefore at increased risk for attenuated development of empathy and development of CU traits (Blair, 1995; Blair et al., 1997, 2001; Kochanska, 1993; Newman, 1987). In fact, findings also have demonstrated a unique relation between low levels of anxiety and the development of CU traits among children with parents who are low on warmth and involvement (Pardini et al., 2007).

In contrast, children with the ANS profile predicted to be associated with CD-only exhibit more normative anxiety responses to punishment and other individuals' distress; as such, they are expected to be more receptive and responsive to parental moral socialization efforts and consequently less likely to develop impairing levels of CU traits. Therefore, children who experience excessive PNS withdrawal and heightened PEP activity are likely to be impulsive and have difficulty with emotion regulation, which makes these children prone to anger. These problems may result in an increased likelihood of engaging in impulsive antisocial behavior and reacting aggressively in the context of increased emotional arousal (Frick, 2006; Loney et al., 2003). However, their physiological make-up affords these children the capacity to experience anxiety and, consequently, remorse, guilt, or other emotions in response to their antisocial behavior and its attendant interpersonal consequences, which would be expected to inhibit future antisocial behavior. Nevertheless, their profile suggests that they are unable to control their CD symptoms because of their poor regulation abilities and impulsivity (Frick, 2006, 2012; Pardini et al., 2003). It is these difficulties with emotion regulation and impulsivity that differentiate CD-only and CD+CU children. Thus, among children whose ANS functioning is characterized by

excessive PNS withdrawal and heightened PEP, negative parental behaviors will exacerbate children's risk for CD but will not confer risk for the CU traits. For children with attenuated baseline parasympathetic functioning, normative parasympathetic reactivity, and attenuated SNS functioning at baseline and reactivity, harsh parental behaviors and low parental warmth will confer risk for CD+CU. Thus, negative parental behaviors characterized by low warmth, low involvement, and harsh discipline may differentially interact with particular patterns of ANS functioning, exacerbating children's risk of developing CD-only or CD+CU.

### Gaps in the Literature

There are several gaps in the literature regarding the relations between ANS functioning and CD with and without CU traits. First, there is a dearth of research examining relations among childhood-onset CD and ANS functioning that utilize direct assessments of both sympathetic and parasympathetic influences on cardiac activity concurrently. Given that these branches do not influence behavior in the same way (Beauchaine, 2009), it would enhance our understanding of children's behavior, including CD with and without CU, to delineate the specific contributions of each branch and the specific sympathetic and parasympathetic patterns that ultimately result in these particular types of behavior. Furthermore, given that emotion regulation abilities, which are reflected in PNS activity, may be key factors in distinguishing children with CD+CU from those with CD-only, it is important to assess both SNS and PNS functioning in examinations of childhood-onset CD.

It is also important to note that of the studies that have assessed separate measures of PNS and SNS functioning, the majority have utilized RSA and EDA. However, it has been argued that it is theoretically and methodologically better to consider measures of PEP and RSA as opposed to measures of EDA and RSA. This argument is based on an empirically supported

model of the joint action of the SNS and PNS called the doctrine of autonomic space (Berntson et al., 1997; Berntson & Cacioppo, 2004), which conceptualizes reactivity in the two branches as flexible, as opposed to earlier models suggesting that increased activity in one branch was associated with decreased activity in the other branch (Beauchaine, 2009; Berntson, Cacioppo & Quigley, 1991; Berntson, Cacioppo, Quigley, & Fabro, 1994; Cannon, 1939). Thus, this model better characterizes joint action between the SNS and PNS, suggesting that it can be reciprocal or non-reciprocal. Adoption of this doctrine leads one to consider PEP instead of EDA/SC for several reasons. First, it is unclear whether the doctrine of autonomic space can be applied to skin conductance levels (SCLs), as this model was intended to explain autonomic control of concurrently innervated target organs such as the heart. SC is an electrodermal measure of SNS activity, not a cardiac one. Second, there is much evidence that EDA increases in response to a variety of both positive and negative stimuli, which renders it a non-specific marker of physiological arousal (Anders, Lotze, Erb, Grodd, & Birbaumer, 2004; Beauchaine, 2009; Beauchaine, Katkin, Strassberg, & Snarr, 2001; Lorber, 2004). Third, PEP reactivity has been found to be specific to reward conditions, which is particularly applicable to examining childhood-onset CD, which is characterized by strong approach or reward-seeking behaviors (Beauchaine, 2009; Beauchaine, Hong, & Marsh, 2008; Brenner et al., 2005). Thus, according to the doctrine of autonomic space model, efforts to understand the specific influences of the ANS on behavior should focus on the separate influences of the SNS and the PNS on one target organ, the heart. To do this effectively, one must use measures that reflect the influence of each on cardiac activity (i.e., PEP and RSA). Thus, the present study utilized measures of RSA and PEP in order to examine the relations between ANS functioning and CD with and without CU traits.

Furthermore, and more directly related to the current study, there have been no studies to date that examine the associations between CD, CU and ANS functioning using separate and concurrent assessment of PNS and SNS functioning among children in middle childhood. Middle childhood is a critical period for considering CD with and without CU for a variety of reasons. Middle childhood is a period of both increasing stability and change. First, temperamental constructs such as reactive processes and effortful control appear to stabilize by middle childhood (Eisenberg, Smith, Sadovsky, & Spinrad, 2004; Rothbart & Bates, 2006). Although levels of CU traits are not yet stable among children with antisocial behavior (Fontaine, McCrory, Boivin, Moffitt, & Viding, 2011), CU traits may evidence increasing stability during this developmental period, given evidence of relatively high stability from late childhood through adolescence (Frick et al., 2003c; Lynam, Charnigo, Moffitt, Loeber, & Stouthamer-Loeber, 2009; Munoz & Frick, 2007; Obradovic et al., 2007). Second, the ANS also undergoes significant changes from infancy through middle childhood. Although differences in SNS-related functioning between children with CD symptoms and those without appear to emerge earlier in development, differences in vagal tone and reactivity between children with behavior problems and those without appear to arise during middle childhood (Beauchaine et al., 2007; Posthumus, Bocker, Raaijmakers, van Engeland, & Matthys, 2009). By middle to late childhood, there is moderate stability of SNS and PNS baseline and reactivity levels, which become increasingly stable subsequent to this period (Beauchaine et al., 2007; Bornstein & Suess, 2000; Calkins & Keane, 2004; El-Sheikh, 2005, 2007; El-Sheikh et al., 2009). For example, by late childhood, children with CD+CU demonstrate SNS hypoarousal relative to youth with CD-only (Blair, 1999).

Third, given expected developmental changes in empathy levels, differences among youth with and without conduct problems in terms of empathy become more pronounced during this period. Although children with behavior problems do not differ from typically developing children in their levels of empathy in the preschool years, by the beginning of the middle childhood period (around 6-7 years old), this pattern changes such that children with behavior problems have significantly lower levels of concern for others (Hastings et al., 2000; Kochanska, 1991; Zahn-Waxler, Cole, Welsh, & Fox, 1995). These differences in empathy are likely underpinned by SNS hypoarousal in the context of others' distress, an established correlate of adult psychopathy that is evident by middle to late childhood (Anastassiou-Hadjicharalambous & Warden, 2008; Blair, 1999), suggesting that it is especially important to evaluate these two emergent phenomena during this developmental period.

Fourth, middle childhood corresponds to the period during which childhood-onset CD is diagnosed. As noted, children who manifest early (e.g., childhood-onset) behavioral problems tend to demonstrate antisocial behavior that increases in rate and severity through adolescence; moreover, these children demonstrate higher rates of CU traits and their antisocial behavior is more likely to continue through adulthood compared to those whose antisocial behavior begins later in life (Frick & Loney, 1999; Lahey & Loeber, 1994; Moffitt, 1993; Moffitt & Caspi, 2001). Thus, early identification of and intervention for behavior problems during middle childhood, when the mechanisms involved in conferring risk for more severe antisocial behavior may still be malleable, could aid in protecting at-risk children from more deleterious outcomes in adolescence and adulthood (Frick, 2006, 2012; Lahey & Loeber, 1994).

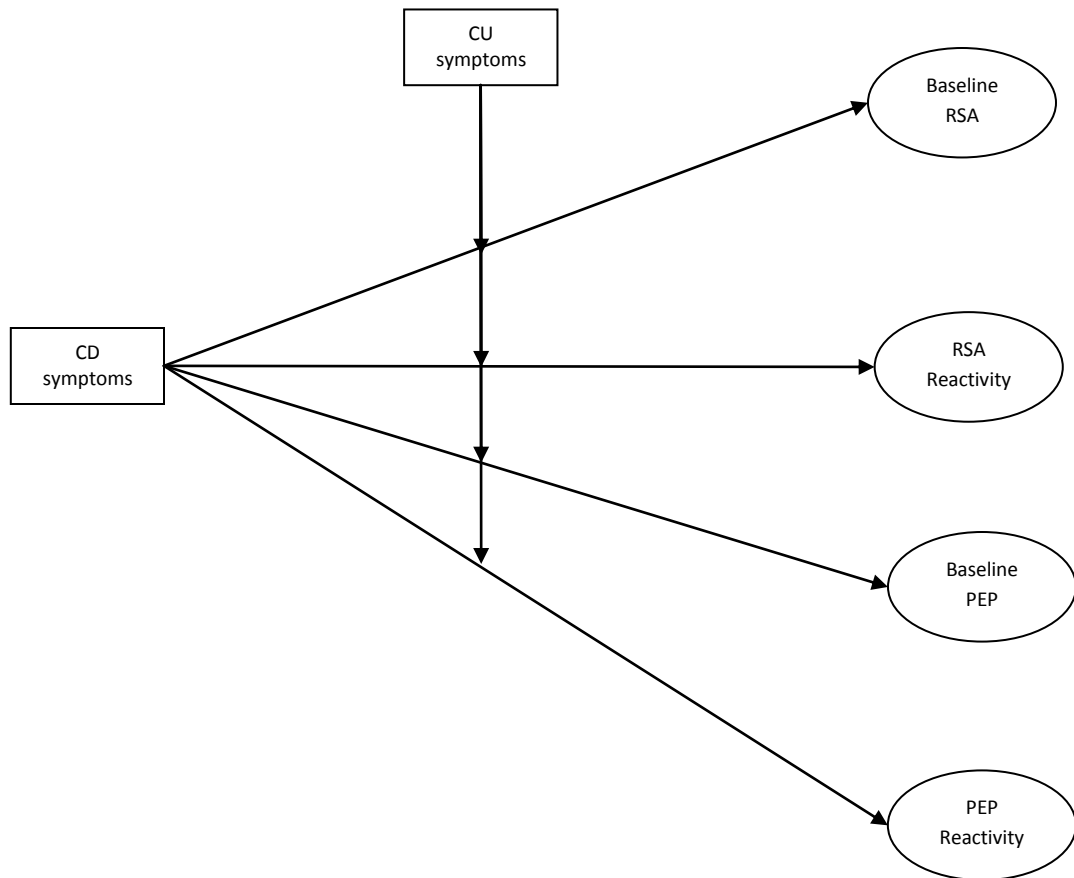
Another important gap in the literature is that despite being at increased risk for CD and the associated negative correlates, there is a dearth of research examining these processes among

samples of children living in low income, urban neighborhoods. Evaluating CD with and without CU traits among low income, urban children is critical for numerous reasons. First, there is ample evidence indicating a positive relation between CD symptoms and residing in disadvantaged neighborhoods, characterized by high crime and low SES (Aneshensel & Sucoff, 1996; Attar, Guerra, & Tolan, 1994; Farrington, 1998; Farrington, Ullrich, & Salekin, 2010; Kimonis, Frick, Munoz, & Aucoin, 2008a; Odgers et al., 2012; Schonberg & Shaw, 2007; Thornberry, Huizinga, & Loeber, 1995). Second, the impact of risk factors for CD is likely exacerbated in contextually disadvantaged environments, and ethnic minority individuals, who are overrepresented in low income, inner-city neighborhoods, are more likely to experience impairment associated with CD symptoms (Ezpeleta, Keeler, Erkanli, Costello, & Angold, 2001; Leventhal & Brooks-Gunn, 2000). For example, Lynam, Caspi, Moffitt, Wikstrom, and Loeber (2000) found that the effect of impulsivity on delinquency in adolescence was greater for lower SES neighborhoods. Consistent with these results, the association between both impulsivity and CU traits with delinquency is stronger among youth residing in neighborhoods characterized by lower levels of either informal social control and/or social cohesion (Meier, Slutske, Arndt, & Cadoret, 2008). Third, children residing in contextually disadvantaged neighborhoods are at elevated risk for exposure to physical and psychosocial stressors, maltreatment, and violence, each of which has been associated with maladjustment (Evans & English, 2002; Gorman-Smith & Tolan, 1998; McLoyd, 1998). Fourth, exposure to stressful environments in childhood heightens stress reactivity (Boyce & Ellis, 2005; Steinberg & Avenevoli, 2000), which also confers risk for negative psychological and behavioral outcomes (Attar et al., 1994; Boyce & Ellis, 2005; Ellis & Boyce, 2008; Evans & English, 2002). Given the adverse outcomes associated with contextual disadvantage, examination of the relations among CD, CU, and ANS

functioning among children residing in low income, urban environments is especially important and could have implications for models of risk and resilience, as well as prevention and intervention efforts among at-risk children.

### The Current Study

Given these gaps in the current literature, the present study examined associations among CD and CU symptoms with children's ANS functioning in a sample of low income, ethnic minority children in middle childhood (see Figure 1). In addition, I examined contextual factors that have the potential to influence these relations. The current study entailed an analysis of data collected at the baseline interview of Phase II of the Child Health and Behavior Study (CHBS), a longitudinal investigation of approximately 100 families residing in North Philadelphia. CHBS was designed to understand children's social, physical, and psychological adjustment. CHBS examines how factors specific to the child (e.g., temperament) and the child's many contexts (e.g., home, school, neighborhood) influence their adjustment, and which factors confer risk for psychological problems or buffer children from developing such problems. The constructs taken from CHBS that were utilized in the current study include parent-reported CD symptoms, parent-reported CU traits, ANS functioning (i.e., baseline RSA, RSA reactivity, baseline PEP and PEP reactivity), parent-reported parenting behavior (e.g., warmth/involvement and harsh discipline), and child demographics (e.g., age, sex, family income).



*Figure 1.* Model of the prediction of autonomic nervous system (ANS) functioning from childhood-onset conduct disorder (CD) symptoms, moderated by callous-unemotional (CU) symptoms.

In the current study, I examined whether the relations between children’s CD symptoms and (1) PNS functioning and (2) SNS functioning differ based on levels of CU traits. First, I examined whether CU traits moderated the relation between CD symptoms and children's PNS

functioning at both baseline and reactivity. Second, I investigated whether CU traits moderated the relation between CD symptoms and children's SNS functioning at both baseline and reactivity. In addition, given that contextual factors may confer risk or resilience, I examined the influence of parenting behaviors on the relations between ANS functioning with (a) CD symptoms and (b) CU traits.

### *Hypotheses*

***Aim 1:*** To examine differences in the relation between CD symptoms and PNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.

***Hypothesis 1a:*** It was predicted that among children with higher CD symptom severity, those with both high and low CU symptom severity would show attenuated baseline RSA.

***Hypothesis 1b:*** It was predicted that among children with higher CD symptom severity, those with low CU symptom severity would show low RSA reactivity (excessive RSA/PNS withdrawal) compared to those with high CU symptom severity.

***Aim 2:*** To examine differences in the relation between CD and SNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.

***Hypothesis 2a:*** It was predicted that among children with higher CD symptom severity, those with high CU symptom severity would show attenuated baseline PEP (longer baseline PEP), whereas those with low CU symptom severity would show heightened baseline PEP (shorter baseline PEP).

***Hypothesis 2b:*** It was predicted that among children with higher CD symptom severity, those with high CU symptom severity would show attenuated PEP reactivity (less PEP shortening), whereas those with low CU symptom severity would show heightened PEP reactivity (more PEP shortening).

***Aim 3:*** To examine whether (a) harsh parenting behaviors and (b) parental warmth/involvement moderate the relations between CD symptoms and ANS functioning and between CU traits and ANS functioning.

***Hypothesis 3a:*** It was expected that harsh parenting behaviors would moderate relations between ANS functioning and CD symptoms, but not moderate relations between ANS functioning and CU traits.

***Hypothesis 3b:*** It was expected that parental warmth/involvement would moderate relations between ANS functioning and CU traits, but not moderate relations between ANS functioning and CD symptoms.

## CHAPTER 2 METHOD

### *Participants*

Participants were 99 children ( $M = 9.87 \pm 1.19$  years old; 48.5% male; 94.9% African-American, 3% Latino/a) and their primary caregivers (86.7% biological mothers) drawn from five elementary schools in North Philadelphia. The neighborhoods from which families were drawn can be characterized as an inner city area with high levels of crime, poverty, and homogeneity in terms of ethnic minority status. In terms of family configurations, 67.7% of children lived in single-parent households, 18.8% lived in intact (two biological parent) households, 1.0% lived in foster homes or with a guardian other than a biological parent, and 12.5% lived in other family configurations. In terms of annual household income, 67.4% earned less than \$20,000, 9.8% earned between \$20,000 and \$30,000, and 22.8% earned \$30,000 or more. Thirty-four percent of the children lived in families receiving public assistance. Forty-seven percent of the primary caregivers had completed high school, 24% less than high school, and 28% beyond high school.

### *Procedure*

The present study is part of a larger research project designed to follow contextually at-risk children and their caregivers over time. The study was approved by Temple University's Institutional Review Board. The project director obtained permission from the School District of Philadelphia and subsequently from the principals of five elementary schools to send information regarding the project to primary caregivers (hereafter "parents") of third- through fifth-grade children. Families were mailed a description of the study, which stated that we were interested in learning about children's social, physical, and emotional adjustment; a parental consent form;

and a self-addressed, stamped postcard. If interested in participating in the project, parents were asked to either return the self-addressed stamped postcard or call to make an appointment. The sample characteristics (i.e., ethnicity, sex, family SES) are representative of the schools from which the families were drawn; however, due to confidentiality requirements, no information was available to compare those who self-selected into the project and those that did not.

Children and their primary caregivers were invited to our research lab for two visits, each lasting approximately 2.5 hours. Prior to participation, parents and children provided consent and assent, respectively. Parents completed questionnaires related to their child's behaviors, their interactions with their child, their household income, and their experience with familial stressors. The child participated in a protocol designed to measure autonomic functioning, described below.

### *Measures*

*Demographics.* Parents provided background information about their household income, as well as their child's sex and age. Household income was measured on a scale from 1 (\$0-9,999) to 5 (*Over \$40,000*).

*Autonomic functioning.* Child autonomic functioning was measured using Bio-Impedance Technology's HIC-2000 (Bio-Impedance Technology, Inc., Chapel Hill, NC, n.d.), a noninvasive instrument for detecting and monitoring bioelectric impedance signals. An external electrocardiographic (ECG) cable was added to the HIC-2000 to increase the flexibility for electrode positioning and ease of detecting the ECG signal. The HIC-2000 recorded RSA and PEP with a constant 5 V potential across seven, pre-gelled electrodes that have circular contact areas with 1 cm diameters. Disposable spot electrodes were applied to the child's neck, back, stomach, and shoulder (Qu, Zhang, Webster, & Tompkins, 1986). Cardiac signals were

monitored by and interfaced to a PC-based computer. Both RSA and PEP were measured during tasks chosen to provide a range of stressors (i.e., social, cognitive, physical, and emotional; Alkon et al., 2003; Bubier & Drabick, 2008). The protocol has been shown to be a reliable and valid method for examining sympathetic and parasympathetic responses to challenge among children ranging in age from 3 to 11 (Alkon et al., 2003; Bubier & Drabick, 2008; Bubier et al., 2009). For each child, the order of the tasks was as follows: social (3 min), cognitive (3 min), physical (1 min), and emotional (3 min). The social challenge involved engaging the child in conversation about the child's school, family, and interests. During the cognitive challenge, the child was asked to repeat a list of two to six numbers presented orally by the experimenter. In the physical challenge, after the experimenter placed several drops of lemon juice on the child's tongue, the child was asked to taste and identify the liquid. For the emotional challenge, the child was asked to watch two brief video clips chosen to evoke emotional reactions (i.e., fear and sadness). The "fear" video depicts a young boy who is frightened during a thunderstorm and the "sadness" video depicts a child and her mother addressing the loss of the child's pet bird (Alkon et al., 2003; Eisenberg et al., 1988). To establish baseline measures (i.e., indices of RSA and PEP at rest), age-appropriate books were read to the child before and after the challenge tasks. Each baseline task was 3 min in length.

During baseline and each of the tasks, the child's behavior and physiological reactions (i.e., HR, RSA, and PEP) were monitored. This standardized protocol took approximately 20 min to administer. Sympathetic-linked cardiac activity was indexed by PEP, measured as the interval from the beginning of ventricular depolarization (ECG Q wave) to the onset of ventricular ejection (impedance cardiographic B wave; Sherwood et al., 1990). Waveforms were collected

via the spot electrode configuration described above (Qu et al., 1986). PEP data were ensemble-averaged in Cop-Win 6.0 H software in 30-s epochs.

Parasympathetic cardiac activity was assessed using spectral analysis via Nevrokard's Long-Term Heart Rate Variability (LT-HRV) software (Nevrokard, Ljubljana, Slovenia, n.d.), which separates heart rate variability time series into component frequencies using fast-Fourier transformations (Berntson et al., 1997). High frequency spectral power ( $>.15$  Hz) was extracted to measure RSA. This high frequency band is a better index of cardiac vagal control, as compared to low frequency ( $<.04$  Hz) or midfrequency (.04 to .15 Hz) variability (Houtveen & Molenaar, 2001; Mezzacappa, Kindlon, Earls, & Saul, 1994). Spectral densities were calculated in 30-s epochs. The log of RSA was used to index parasympathetic functioning, which is a transformation commonly used to normalize spectral analytic data (Crowell et al., 2006). Mean scores for PEP and RSA were calculated at baseline and the difference score (mean across each of the four challenge tasks minus mean at baseline) was used as a measure of autonomic reactivity (Alkon et al., 2003; Bubier & Drabick, 2008). Therefore, reactivity refers to the shortening of PEP and vagal withdrawal. Participants were included in the analyses if they had at least 50% scorable epochs within each task and during baseline. This decision was made to maximize the number of participants included while maintaining an adequate number of epochs (Bubier & Drabick, 2008; Bubier et al., 2009). As described below, missing data were imputed using full information maximum likelihood estimation for the primary analyses. For the present study, the following variables were utilized: baseline RSA, RSA reactivity, baseline PEP, and PEP reactivity.

*CD symptoms.* Parents rated child CD symptoms using the Child and Adolescent Symptom Inventory-4: Parent Checklist (CASI-4; Gadow & Sprafkin, 1994, 2002), which includes the behavioral symptoms of most childhood emotional and behavioral disorders described in the *Diagnostic and Statistical Manual of Mental Disorders-Fourth edition (DSM-IV;* APA, 1994). Individual items have a one-to-one correspondence with *DSM-IV* symptoms. Parents are asked to endorse on a scale from 0 (*never*) to 3 (*very often*) how well each statement describes their child's overall behavior. Examples of CD symptoms include, "Bullies, threatens or intimidates others," "Starts physical fights," and "Has deliberately destroyed others' property." In light of research demonstrating that symptoms of behavioral disorders such as CD can be impairing even at levels below those necessary to garner a clinical diagnosis (Angold et al., 1999; Drabick, 2009), CD symptoms were examined dimensionally in the present study. Thus, items will be summed to create a symptom severity score for CD (16 items,  $\alpha = .78$ ). Findings of numerous studies (Gadow & Sprafkin, 2010) indicate that CASI-4 subscales demonstrate adequate psychometric properties, including internal consistency, test-retest reliability, convergent and divergent validity with respective scales of other relevant measures, agreement with structured interview or clinician diagnoses, sensitive indicators of treatment effects, and clinical utility (e.g., Gadow & Sprafkin, 1995, 2002, 2005, 2008).

In comparison to scores derived from the community-based samples used to norm the CASI-4 (Gadow & Sprafkin, 1994, 2002), the current sample of low income, ethnic minority, urban children demonstrate higher levels of CD symptoms for both boys ( $M = 1.0$  vs.  $M = 3.13$ , respectively; one-sample  $t(45) = 3.85$ ,  $p < .05$ ) and girls ( $M = 0.7$  vs.  $M = 1.69$ , respectively; one-sample  $t(47) = 2.69$ ,  $p < .05$ ). As another strategy to compare the current sample to the normative sample, the CASI-4 was used to create symptom count (categorical) scores, for which scores are

recoded such that 0 corresponds to *never/sometimes*, and 1 corresponds to *often/very often*; thus, a symptom is considered clinically significant only if it is rated as occurring *often* or *very often*. When the total symptom count score meets or exceeds the number of symptoms outlined by the *DSM-IV* as consistent with a clinical diagnosis (i.e., 3 CD symptoms), the child receives a screening cutoff score of “yes,” meaning that they exhibit the requisite number of symptoms for the diagnosis of CD. The number of children in the current sample who received parent-rated screening cutoff scores for CD ( $\geq 3$  symptoms), compared to the total number of children in the sample ( $N = 99$ ) is  $n = 6$  (6.4%).

*CU traits.* Caregivers rated their child’s demonstration of remorse, sympathy, empathy, and emotion expression using the Inventory of Callous/Unemotional Traits (Frick, 2003), a 24-item instrument. Items were rated on a scale from 0 (*not at all true*) to 3 (*definitely true*). Previous research with community and detained, ethnically diverse samples of youth has shown that all items load onto a general CU factor, and the results of these studies provide support for utilizing the total ICU score in analyses (Essau, Sasagawa, & Frick, 2006; Fanti et al., 2009; Kimonis et al., 2008b; Roose, Bijttebier, Decoene, Claes, & Frick, 2009; White, Cruise, & Frick, 2009). The ICU demonstrates good convergent, construct, and criterion validity. For example, scores on the ICU demonstrate significant positive associations with established measures designed to tap similar personality traits including the Antisocial Process Screening Device (Frick & Hare, 2001) and the Childhood Psychopathy Scale (Lynam, 1997), and with measures designed to assess risk factors for criminal and antisocial behavior (Kimonis et al., 2008b; Roose et al., 2009; White et al., 2009). Furthermore, scores on the ICU are negatively related to measures of empathy and prosocial attitudes, as well as positively associated with antisocial behavior, bullying, sensation seeking, delinquency, and aggression (Essau et al., 2006; Fanti et

al., 2009; Kimonis et al., 2008b; Roose et al., 2009). Given that there are no currently accepted categorical approaches for defining CU traits and that CU traits are most frequently considered dimensionally in the existing literature, items were summed to create a symptom severity score for CU (24 items,  $\alpha = .79$ ).

*Parenting.* Caregivers reported on their parenting behaviors using the Structured Interview of Parent Management Skills and Practices (Capaldi & Patterson, 1989, 1994), a semi-structured interview that assesses parenting behaviors in a variety of domains, including monitoring, discipline, positive reinforcement, and involvement. This interview was administered by trained graduate-level research assistants. Parents were asked how frequently they engage in particular parenting behaviors and how they would handle a variety of hypothetical situations involving their child. Two scales, parental warmth/involvement and parental harsh discipline, were developed for the present study in consultation with Capaldi and colleagues. Scales were developed using data from CHBS in an iterative process with attention to item construct validity, factor loadings, scale internal consistency, and previous research using this interview.

For items that were included in the parental warmth/involvement subscale, parents were asked to rate how often they engaged in the particular behavior on a five-point Likert scale ranging from 1 (*never*) to 5 (*always*). Sample items include, “How often do you show your child you like it when s/he helps around the house without being told?” and “How often do you talk with your child about his/her plans for the coming day (like what’s happening with school or friends)?” Items were summed to create a parental warmth/involvement scale (5 items,  $\alpha = .72$ ). Higher scores indicate higher levels of parental warmth and involvement. For items that were included in the harsh discipline subscale, parents were asked to respond to questions about their

disciplinary behaviors (e.g., how they would handle various situations involving their child) in forced choice response formats. Sample hypothetical behaviors presented to parents included, “Physically fights (push, kick, shove) with brothers or sisters or other kids,” and “Ignores you when you ask him/her to do something.” Parents’ responses were then assigned to a category (e.g., harsh, physical discipline). Examples of responses that comprise the harsh, physical discipline category include, “Spank,” “Wash mouth,” and “Threaten physical or psychological harm.” Items were recoded so that responses assigned to the harsh, physical discipline category received a 1 and all other response categories received a 0. The recoded items were then summed to create a harsh parental discipline scale (30 items,  $\alpha = .79$ ). Higher scores indicate higher levels of harsh discipline.

#### *Statistical Analyses*

The primary questions and respective hypotheses were examined using multiple regression analyses that were conducted using the statistical software Mplus 5.1 (Muthén & Muthén, 1998-2007). Prior to analysis, all variables were examined for missing values and fit between their distributions and the assumptions of multiple regression analysis including normality, linearity, and homoscedasticity of residuals using SPSS/PASW statistical software. Given the presence of missing data, parameter estimates and model tests were conducted using Full Information Maximum Likelihood (FIML) methods as implemented in Mplus. Unlike listwise or casewise deletion that may bias the dataset, FIML is a useful approach to missing data given that it estimates values for missing data based on all of the available data (Graham, 2009; Little & Rubin, 2002).

To determine whether the variables’ distributions met the assumption of normality, I examined univariate indices of skewness and kurtosis to determine if the absolute value of any of

these indices is greater than 2.0. Multivariate normality was then assessed using a  $p < .001$  criterion for Mahalanobis distance. Bivariate correlations were also conducted to examine the relations among the study variables.

To minimize multicollinearity, the independent variables were standardized ( $M = 0$ ,  $SD = 1$ ) before inclusion in the regression equations, and these standardized variables were used to create a  $CD \times CU$  cross-product interaction term (Aiken & West, 1991). For significant interaction terms, post hoc probing of moderational effects was conducted using procedures outlined by Holmbeck (2002). Following these procedures, two new conditional moderator variables ( $\pm 1 SD$  from the mean) were created, in addition to two new interaction terms using the new conditional variables. Subsequently, two post-hoc regressions were conducted. Each of these regressions involved concurrent entry of the  $CD$  variable, the  $CU$  variable, and the  $CD \times CU$  cross-product interaction term. The results of these analyses allowed me to compute unstandardized betas (slopes) and a regression equation for children experiencing high (1  $SD$  above the mean) and low (1  $SD$  below the mean) levels of  $CU$  symptoms. Significant unstandardized betas associated with the  $CU$  variable would indicate that the slope of the line for high or low  $CU$  symptoms was significantly different from zero. Specific tests of the hypotheses are described below.

***Aim 1:** To examine differences in the relation between  $CD$  symptoms and  $PNS$  functioning based on whether children exhibit high or low levels of  $CU$  traits relative to other youth in the present sample.*

***Hypothesis 1a:** It was predicted that among children with higher  $CD$  symptom severity, those with both high and low  $CU$  symptom severity would show attenuated baseline  $RSA$ .*

To test this hypothesis, multiple regression analysis was used to examine the relations among CD symptoms, CU traits, and children's PNS functioning. For all regressions, child sex, age, and household income were entered into the first step to control for these variables. To examine whether the relation between CD symptoms and baseline RSA differed for children with high and low levels of CU traits, CD symptom severity, CU trait severity, and the CD×CU interaction term were entered into the second step of the multiple regression equation for which baseline RSA was the dependent variable.

***Hypothesis 1b:** It was predicted that among children with higher CD symptom severity, those with low CU symptom severity would show low RSA reactivity (excessive RSA/PNS withdrawal) compared to those with high CU symptom severity*

Similar to tests for Hypothesis 1a, to examine the relations among CD symptoms, CU traits, and RSA reactivity, CD symptom severity, CU trait severity, and the CD×CU interaction term were entered into the second step of a multiple regression equation for which RSA reactivity was the outcome.

***Aim 2:** To examine differences in the relation between CD and SNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.*

***Hypothesis 2a:** It was predicted that among children with higher CD symptom severity, those with high CU symptom severity would show attenuated baseline PEP (longer baseline PEP), whereas those with low CU symptom severity would show heightened baseline PEP (shorter baseline PEP).*

Multiple regression analysis was used to examine the relations among CD symptoms, CU traits, and children's SNS functioning. For all regressions, child sex, age, and household

income were entered into the first step in order to control for these variables. To examine whether the association between CD symptoms and baseline PEP differed for children with high and low levels of CU traits, CD symptom severity, CU trait severity, and the CD×CU interaction term were entered into the second step of a multiple regression equation for which baseline PEP was the outcome.

***Hypothesis 2b:*** *It was predicted that among children with higher CD symptom severity, those with high CU symptom severity would show attenuated PEP reactivity (less PEP shortening), whereas those with low CU symptom severity would show heightened PEP reactivity (more PEP shortening).*

Similar to tests of Hypothesis 2a, to examine the relations among CD symptoms, CU traits, and PEP reactivity, CD symptom severity, CU trait severity, and the CD×CU interaction term were entered into a multiple regression equation for which PEP reactivity was the outcome variable.

***Aim 3:*** *To examine whether (a) harsh parenting behaviors and (b) parental warmth/involvement moderate the relations between CD symptoms and ANS functioning and between CU traits and ANS functioning.*

***Hypothesis 3a:*** *It was predicted that harsh parenting behaviors would moderate relations between ANS functioning and CD symptoms, but not moderate relations between ANS functioning and CU traits.*

***Hypothesis 3b:*** *It was predicted that parental warmth/involvement would moderate relations between ANS functioning and CU traits, but not moderate relations between ANS functioning and CD symptoms.*

I next conducted a set of analyses examining whether parenting factors (i.e., harsh discipline or warmth) moderate the relation between either (a) CD symptoms and/or (b) CU traits and child ANS functioning. Given the sample size, I was under-powered to test a 3-way interaction among CD, CU, and the parenting variables, and wanted to minimize the total number of regression analyses conducted given issues related to Type I error. Thus, these moderational analyses were conducted only for ANS variables for which CD or CU main effects, or the CD×CU interaction term were significant based on the results of the analyses for the first two aims. For each parenting variable, cross-product interaction terms with either CD symptoms or CU traits were created. To examine whether the associations among CD or CU symptom severity and the ANS outcome differ based on the parenting variable, multiple regression analyses were conducted for which the ANS variable was the outcome. Child sex, age, and household income were entered into the first step in order to control for these variables, and CD or CU symptom severity, the parenting variable, and the CD or CU × parenting interaction term were entered into the second step. Post hoc probing was accomplished using the techniques described above (Aiken & West, 1991; Holmbeck, 2002).

#### *Power Analysis*

A power analysis was conducted in order to determine the sample size necessary to test the hypotheses of the current study. With six predictors, as were tested in all hypotheses, sufficient power ( $.80$ ,  $\alpha = .05$ ) to detect medium-to-large effect sizes ( $f^2 = .2$ ) is attained with a sample size of 75 (Faul, Erdfelder, Lang, & Buchner, 2007). Therefore, the present sample provided adequate power for conducting the analyses involving CD, CU, parenting, and ANS functioning.

## CHAPTER 3

### RESULTS

#### *Descriptive Statistics*

Means, standard deviations, and number of participants for all study variables are presented in Table 1. Based on examination of skewness and kurtosis, one extreme outlier was identified and removed from all further analyses. Multivariate normality was also examined and no outliers were identified. Given these results, all further analyses were conducted without transforming or otherwise modifying the variables (excluding the log transformation of RSA variables discussed previously). Bivariate correlations for study variables are presented in Table 2. CD symptom severity was positively associated with CU symptom severity. CD symptom severity was also positively associated with baseline PEP.

Table 1

#### *Means, Standard Deviations, and Number of Participants for Study Variables*

Variable	<i>M</i>	<i>SD</i>	<i>n</i>
Age	9.87	1.19	99
CU traits	23.60	10.48	85
CD symptoms	2.39	3.26	94
PEP Baseline	100.65	11.48	70
RSA Baseline	4.04	.45	62

Table 1 Cont.

*Means, Standard Deviations, and Number of Participants for Study Variables*

Variable	<i>M</i>	<i>SD</i>	<i>n</i>
PEP Reactivity	.54	3.64	70
RSA Reactivity	.09	.20	61
Parental Harsh Discipline	3.77	3.55	99
Parental Warmth/Involvement	21.92	3.09	95

*Note.* CU = callous-unemotional, CD = conduct disorder, PEP = pre-ejection period, RSA = respiratory sinus arrhythmia.

Table 2

*Bivariate Correlations among Study Variables*

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Age	-										
2. Sex	.00	-									
3. Household Income	-.01	.03	-								
4. CU Traits	-.01	-.21	-.23*	-							
5. CD Symptoms	-.06	-.22*	-	.54	-						
6. RSA Baseline	-.01	.05	.22	-.03	-.04	-					
7. RSA Reactivity	-.13	.14	-.13	-.04	-.06	-.23	-				
8. PEP Baseline	.13	-.17	-.24*	.18	.26*	-.13	-.07	-			
9. PEP Reactivity	.05	-.00	.09	.04	.01	.07	.12	-	-		
10. Parental Harsh Discipline	-.05	.13	.05	.13	.07	.22	-.03	.24	-.09	-	
11. Parental Warmth/Involvement	.09	.01	-.02	-.23*	-.05	-.18	-.13	-.06	-.14	-.34**	-

*Note.* CU = callous-unemotional, CD = conduct disorder, RSA= respiratory sinus arrhythmia,

PEP = pre-ejection period.

\*\* $p < .01$ , \*  $p < .05$ .

***Aim 1:** To examine differences in the relation between CD symptoms and PNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.*

***Hypothesis 1a:** It was predicted that among children with higher CD symptom severity, those with both high and low CU symptom severity would show attenuated baseline RSA.*

Multiple regression analyses examining the relations among CD symptoms, CU traits, and baseline RSA were conducted. Results for these regression analyses are presented in Table 3. These analyses revealed that after controlling for child sex, age, and household income, the CD×CU interaction term was significantly associated with baseline RSA ( $\beta = -0.44, p < .05$ ). To explore this relation, post hoc probing procedures were conducted (Aiken & West, 1991; Holmbeck, 2002). From these analyses, I derived unstandardized betas (slopes) and a regression equation for children presenting high (1 *SD* above the mean) and low (1 *SD* below the mean) CU trait scores. For low CU trait scores the slope was significantly different from zero ( $B = 1.41, t(56) = 2.64, p < .05$ ), indicating that among children with lower CU trait severity, higher CD symptoms were associated with higher baseline RSA, and lower CD symptoms were associated with lower baseline RSA (Figure 2). For high CU trait scores, the slope also was significant ( $B = -1.27, t(56) = -2.79, p < .05$ ), indicating that among children with higher CU trait severity, higher CD symptoms were associated with lower baseline RSA, and lower CD symptoms were associated with higher baseline RSA (Figure 2).

Table 3

*Multiple Regression Analysis Summary for Variables Predicting Baseline RSA*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.05	.05	.05
Age	-.01	.05	-.02			
Sex	.05	.12	.06			
Household Income	.07	.04	.22			
Step 2				.21*	.16	.27
CD symptoms	.12	.08	.27			
CU traits	.00	.07	.00			
CD symptoms $\times$ CU traits	-.13	.05	-.44**			

*Note.* CD = conduct disorder, CU = callous-unemotional.

\*  $p < .05$ , \*\*  $p < .01$ .

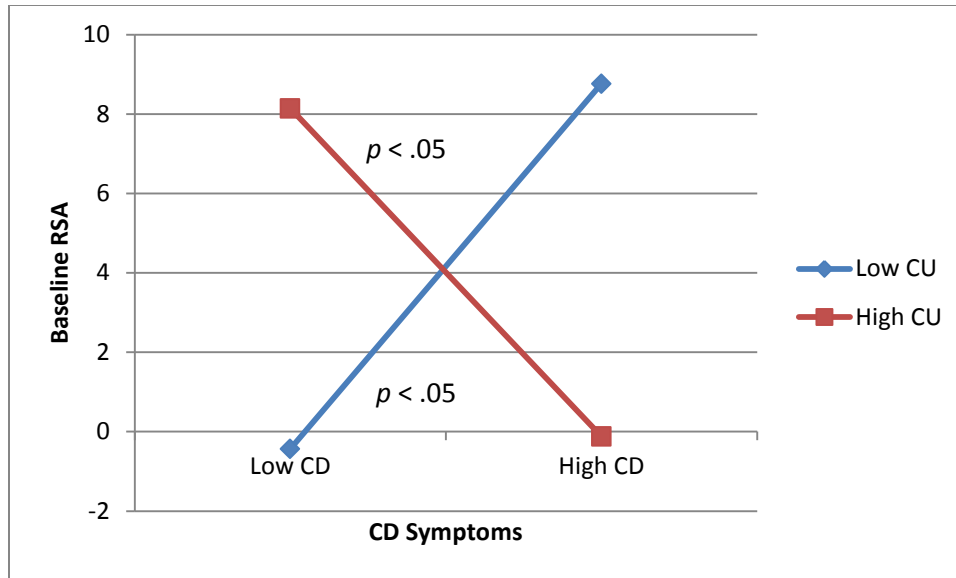


Figure 2. Relation between CD symptoms and baseline RSA among children with high (1 SD above mean) vs. low (1 SD below mean) levels of CU traits.

**Hypothesis 1b:** It was predicted that among children with higher CD symptom severity, those with low CU symptom severity would show low RSA reactivity (excessive RSA/PNS withdrawal) compared to those with high CU symptom severity.

Multiple regression analyses examining whether the relation between CD symptoms and RSA reactivity differed for children with high and low levels of CU traits were conducted (Table 4). After controlling for child sex, age, and household income, the CD×CU interaction term was significantly associated with RSA reactivity ( $\beta = -0.50, p < .05$ ). Post-hoc probing demonstrated that the slopes for both high ( $B = -0.56, t(55) = -2.97, p < .05$ ) and low ( $B = 0.66, t(55) = 3.00, p < .05$ ) CU traits were significantly different from zero, indicating that the relation between CD symptom severity and RSA reactivity differs depending on CU trait severity. Examination of Figure 3 suggests that among children with higher CU trait severity, higher CD symptoms were associated with lower RSA reactivity, and lower CD symptoms were associated with higher RSA

reactivity. Contrary to prediction, among children with lower CU trait severity, higher CD symptoms were associated with higher RSA reactivity, and lower CD symptoms were associated with lower RSA reactivity.

Table 4

*Multiple Regression Analysis Summary for Variables Predicting RSA Reactivity*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.06	.06	.06
Age	-.03	.02	-.15			
Sex	.07	.05	.16			
Household Income	-.02	.02	-.14			
Step 2				.28**	.22	.39
CD symptoms	.06	.03	.31			
CU traits	-.01	.03	-.03			
CD symptoms $\times$ CU traits	-.06	.02	-.50**			

*Note.* CD = conduct disorder, CU = callous-unemotional.

\*\*  $p < .01$ .

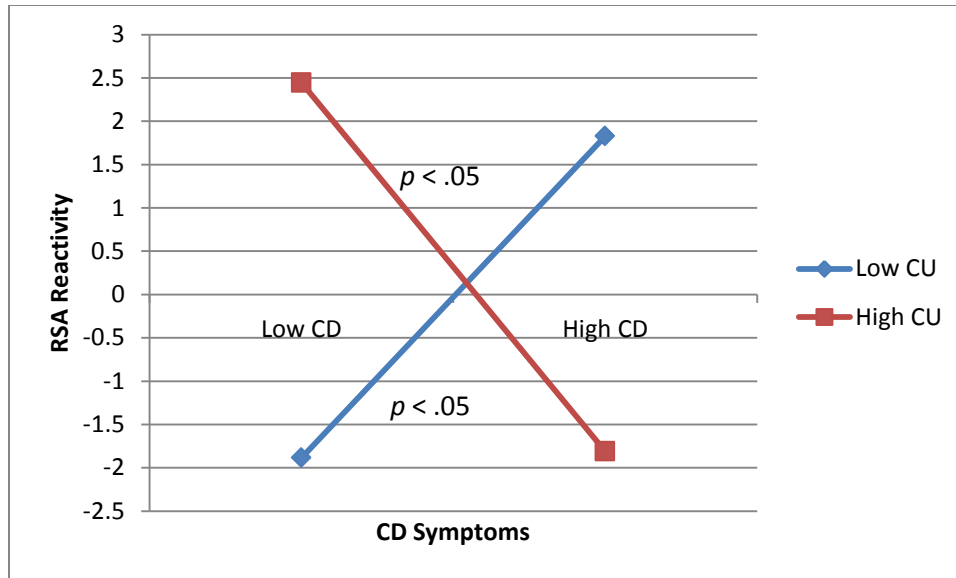


Figure 3. Relation between CD symptoms and RSA reactivity among children with high (1 *SD* above mean) vs. low (1 *SD* below mean) levels of CU traits.

*Aim 2: To examine differences in the relation between CD and SNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.*

*Hypothesis 2a: It was predicted that among children with higher CD symptom severity, those with high CU symptom severity would show attenuated baseline PEP (longer baseline PEP), whereas those with low CU symptom severity would show heightened baseline PEP (shorter baseline PEP).*

Multiple regression analyses examining the relations among CD symptoms, CU traits, and baseline PEP were conducted. After controlling for child age, sex and household income, results indicated that CD symptom severity, CU trait severity, and the CD × CU interaction term were not significantly associated with baseline PEP (all  $p$ 's > .21) (Table 5).

Table 5

*Multiple Regression Analysis Summary for Variables Predicting Baseline PEP*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.10	.10	.11
Age	.93	1.11	.10			
Sex	-4.23	2.76	-.18			
Household Income	-1.63	.88	-.22			
Step 2				.17	.07	.20
CD symptoms	2.32	1.86	.20			
CU traits	.39	1.59	.04			
CD symptoms $\times$ CU traits	-.25	1.04	-.04			

*Note.* CD = conduct disorder, CU = callous-unemotional.

**Hypothesis 2b:** It was predicted that among children with higher CD symptom severity, those with high CU symptom severity would show attenuated PEP reactivity (less PEP shortening), whereas those with low CU symptom severity would show heightened PEP reactivity (more PEP shortening).

Contrary to prediction, CD symptom severity, CU traits, and the CD×CU interaction term were not significantly associated with PEP reactivity (all  $p$ 's > .71) (Table 6).

Table 6

*Multiple Regression Analysis Summary for Variables Predicting PEP Reactivity*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.01	.01	.01
Age	.24	.37	.08			
Sex	-.24	.93	-.03			
Household Income	.23	.30	.09			
Step 2				.03	.02	.03
CD symptoms	.05	.65	.01			
CU traits	.21	.56	.06			
CD symptoms × CU traits	-.00	.37	-.00			

*Note.* CD = conduct disorder, CU = callous-unemotional.

***Aim 3:** To examine whether (a) harsh parenting behaviors and (b) parental warmth/involvement moderate the relations between CD symptoms and ANS functioning and between CU traits and ANS functioning.*

A set of analyses was conducted examining whether parenting factors (i.e., harsh discipline or warmth/involvement) moderated the relation between either (a) CD symptoms and/or (b) CU traits and child autonomic functioning. These moderational analyses were conducted only predicting baseline RSA and RSA reactivity as these were the only ANS variables for which CD and/or CU symptoms were significantly associated based on the results of the first two aims.

***Hypothesis 3a:** It was predicted that harsh parenting behaviors would moderate relations between CD symptoms and ANS functioning, but not moderate relations between CU traits and ANS functioning.*

To examine whether harsh parental discipline moderated the relations between CD or CU symptoms and baseline RSA or RSA reactivity, a series of regression equations were conducted for which RSA (baseline or reactivity) was the outcome variable (Aiken & West, 1991). Each multiple regression analysis included the following steps: (a) child age, sex, and household income and (b) CD or CU symptoms, harsh parental discipline, and the CD or CU  $\times$  harsh parental discipline cross-product interaction term. To better capture the CD-only vs. CD+CU distinction in these analyses, I controlled for CU traits in the analyses involving the CD  $\times$  harsh parental discipline interaction term, but did not control for CD in the analyses involving the CU  $\times$  harsh parental discipline interaction. Results revealed that neither the CD  $\times$  harsh parental discipline nor the CU  $\times$  harsh parental discipline interaction terms significantly predicted RSA baseline or reactivity. However, there was a marginally significant main effect ( $\beta = 0.25, p = .06$ )

of harsh parental discipline on baseline RSA in the analyses involving the CD  $\times$  harsh parental discipline interaction. Results are presented in Tables 7, 8, 9, and 10.

Table 7

*Multiple Regression Analysis Summary for the CD  $\times$  Harsh Parental Discipline Interaction*

*Term Predicting Baseline RSA*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.05	.05	.05
Age	-.01	.05	-.02			
Sex	.05	.12	.06			
Household Income	.07	.04	.22			
Step 2				.15	.10	.18
CD symptoms	-.02	.07	-.04			
CU traits	.01	.07	.02			
Harsh Parental Discipline	.12	.06	.25 <sup>†</sup>			
CD $\times$ Harsh Parental Discipline	.10	.08	.19			

*Note.* CD = conduct disorder, CU = callous-unemotional. <sup>†</sup> $p < .10$ .

Table 8

*Multiple Regression Analysis Summary for the CD × Harsh Parental Discipline Interaction**Term Predicting RSA Reactivity*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.06	.06	.06
Age	-.03	.02	-.15			
Sex	.07	.05	.16			
Household Income	-.02	.02	-.14			
Step 2				.12	.06	.14
CD symptoms	.00	.03	.00			
CU traits	-.00	.03	-.01			
Harsh Parental Discipline	.00	.03	.01			
CD × Harsh Parental Discipline	-.02	.03	-.08			

*Note.* CD = conduct disorder, CU = callous-unemotional.

Table 9

*Multiple Regression Analysis Summary for the CU × Harsh Parental Discipline Interaction**Term Predicting Baseline RSA*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.05	.05	.01
Age	-.01	.05	-.02			
Sex	.05	.12	.06			
Household Income	.07	.04	.22 <sup>†</sup>			
Step 2				.15 <sup>†</sup>	.02	.03
CU traits	.02	.06	.04			
Harsh Parental Discipline	.06	.06	.13			
CU × Harsh Parental Discipline	-.11	.07	-.21			

*Note.* CU = callous-unemotional.

<sup>†</sup> $p < .10$ .

Table 10

*Multiple Regression Analysis Summary for the CU × Harsh Parental Discipline Interaction**Term Predicting RSA Reactivity*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.06	.06	.06
Age	-.03	.02	-.15			
Sex	.07	.05	.16			
Household Income	-.02	.02	-.14			
Step 2				.11	.05	.12
CU traits	-.00	.03	-.02			
Harsh Parental Discipline	.01	.03	.05			
CU × Harsh Parental Discipline	-.01	.03	-.04			

*Note.* CU = callous-unemotional.

***Hypothesis 3b:*** *It was predicted that parental warmth/involvement would moderate relations between CU traits and ANS functioning, but not moderate relations between CD symptoms and ANS functioning.*

To examine whether parental warmth/involvement moderated the relations between CD or CU symptoms and baseline RSA or RSA reactivity, a series of regression equations were conducted for which RSA (baseline or reactivity) was the outcome variable (Aiken & West, 1991). Each multiple regression analysis included the following steps: (a) child age, sex and household income, and (b) CD or CU symptoms, parental warmth/involvement, and the CD or CU  $\times$  parental warmth/involvement cross-product interaction term. Again, to better capture the CD-only vs. CD+CU distinction in these analyses, I controlled for CU traits in the analyses involving the CD  $\times$  parental warmth/involvement interaction term, but did not control for CD in the analyses involving the CU  $\times$  parental warmth/involvement interaction. Results indicated that neither the CD  $\times$  parental warmth/involvement nor the CU  $\times$  parental warmth/involvement interaction terms significantly predicted baseline RSA or RSA reactivity. There were however, non-significant trends with respect to the main effects. Specifically, parental warmth/involvement tended to be associated with baseline RSA ( $\beta = -0.23, p = .09$ ) as well as RSA reactivity ( $\beta = -0.23, p = .08$ ) in the analyses examining the CD  $\times$  parental warmth/involvement interaction. Furthermore, parental warmth/involvement tended to be associated with RSA reactivity ( $\beta = -0.23, p = .09$ ) in the analyses examining the CU  $\times$  parental warmth/involvement interaction. Results are presented in Tables 11, 12, 13 and 14.

Table 11

*Multiple Regression Analysis Summary for the CD × Parental Warmth/Involvement Interaction**Term Predicting Baseline RSA*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.05	.05	.05
Age	-.00	.05	-.02			
Sex	.05	.12	.06			
Household Income	.07	.04	.22			
Step 2				.16 <sup>†</sup>	.11	.19
CD symptoms	.02	.07	.04			
CU traits	-.02	.08	-.04			
Parental Warmth/Involvement	-.11	.07	-.23 <sup>†</sup>			
CD × Parental Warmth/Involvement	-.06	.06	-.14			

*Note.* CD = conduct disorder, CU = callous-unemotional.

<sup>†</sup> $p < .10$ .

Table 12

*Multiple Regression Analysis Summary for the CD × Parental Warmth/Involvement Interaction**Term Predicting RSA Reactivity*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.06	.06	.06
Age	-.03	.02	-.15			
Sex	.07	.05	.16			
Household Income	-.02	.02	-.14			
Step 2				.16 <sup>†</sup>	.10	.19
CD symptoms	.00	.03	.01			
CU traits	-.02	.03	-.09			
Parental Warmth/Involvement	-.05	.03	-.23 <sup>†</sup>			
CD × Parental Warmth/Involvement	.01	.02	.06			

*Note.* CD = conduct disorder, CU = callous-unemotional.

<sup>†</sup> $p < .10$ .

Table 13

*Multiple Regression Analysis Summary for the CU × Parental Warmth/Involvement Interaction**Term Predicting Baseline RSA*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.05	.05	.05
Age	-.01	.05	-.02			
Sex	.05	.12	.06			
Household Income	.07	.04	.22			
Step 2				.14 <sup>†</sup>	.09	.16
CU traits	.00	.08	.00			
Parental Warmth/Involvement	-.11	.07	-.22			
CU × Parental Warmth/Involvement	-.05	.07	-.10			

*Note.* CU = callous-unemotional.

<sup>†</sup> $p < .10$ .

Table 14

*Multiple Regression Analysis Summary for the CU × Parental Warmth/Involvement Interaction**Term Predicting RSA Reactivity*

Step and Variable	<i>B</i>	<i>SE B</i>	$\beta$	$R^2$	$\Delta R^2$	$f^2$
Step 1				.06	.06	.06
Age	-.03	.02	-.15			
Sex	.07	.05	.16			
Household Income	-.02	.02	-.14			
Step 2				.14 <sup>†</sup>	.08	.16
CU traits	-.02	.03	-.12			
Parental Warmth/Involvement	-.05	.03	-.23 <sup>†</sup>			
CU × Parental Warmth/Involvement	.02	.03	.07			

*Note.* CU = callous-unemotional.

<sup>†</sup> $p < .10$ .

## CHAPTER 4 DISCUSSION

Given the detrimental outcomes associated with childhood-onset CD for both society and the individual, it is essential to improve our understanding of distinct etiologies and trajectories of subtypes of CD, as well as to tailor intervention efforts. One strategy for addressing these goals and for decreasing heterogeneity within the CD category is to consider CU traits among youth with CD. In particular, the examination of CU traits in conjunction with childhood-onset CD is crucial given that children with CD and high levels of CU traits demonstrate a particularly pernicious and stable pattern of aggression and antisocial behavior compared to other youth with CD (Frick, 2012; McMahon et al., 2010; Rowe et al., 2009). However, despite the important implications for etiological and intervention models, little research has examined CD with and without CU traits in childhood, and there is a dearth of research examining risk factors and/or mechanisms that may underpin the development of these particular subtypes of childhood-onset CD.

The primary goal of this study was to examine the relations among CD, CU, and child-specific and contextual risk factors. Specifically, in the present study, I investigated whether CU traits moderated the relations between CD symptoms and (a) PNS functioning at baseline and PNS reactivity and (b) SNS functioning at baseline and SNS reactivity. I also examined whether particular parenting behaviors (i.e., harsh discipline and warmth/involvement) moderated the relations between CD and CU and children's ANS functioning. Findings indicate that the relations between CD symptoms and PNS functioning (baseline RSA and RSA reactivity) do in fact differ based on levels of CU traits. Thus, among children with high CD symptoms, those with high levels of CU traits evidenced different patterns of PNS functioning compared to those

with low levels of CU traits. The results of the present analyses provide support for the assertion that there are distinct patterns of ANS functioning for childhood-onset CD with and without high levels of CU traits, and that the ability to regulate emotions is likely a key factor in distinguishing CD+CU from CD-only (Frick, 2006, 2012). Furthermore, the present study extends the existing literature by providing the first empirical investigation of the associations among CD symptoms, CU traits, and ANS functioning using separate and concurrent assessment of PNS and SNS functioning among a low income, ethnic minority sample of children in middle childhood.

***Aim 1:** To examine differences in the relation between CD symptoms and PNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.*

I hypothesized that higher CD symptom severity would be associated with low baseline RSA among children high and low on CU traits. Instead, findings demonstrated that CU traits moderated the relation between CD symptoms and baseline RSA. Among children with higher CD symptom severity, high CU symptom severity was associated with low baseline RSA, consistent with the predicted associations, but low CU symptom severity was actually associated with higher baseline RSA. This finding is contrary to prediction and somewhat contrary to previous research. As low baseline RSA is associated with emotion dysregulation whereas high baseline RSA is associated with social competence, empathy, and emotion regulation (Beauchaine, 2001, 2012; Beauchaine et al., 2007; Eisenberg et al., 1995; Fabes, Eisenberg, & Eisenbud, 1993; Fox & Field, 1989), I expected that high CD symptom severity would be associated with low baseline RSA. Much of the extant research examining relations between CD and RSA has found an association between high CD symptoms and low baseline RSA; however,

some community samples have not found this relation (Calkins et al., 2007; El-Sheikh et al., 2001; El-Sheikh & Whitson, 2006).

This particular finding may be a function of the current sample in two ways. First, this is a community sample drawn from low income, contextually disadvantaged neighborhoods characterized by high crime and violence. It is possible that some of the behaviors that characterize CD are actually adaptive and allow individuals to more effectively navigate living in this type of environment. If this were the case, exhibiting higher levels of CD symptoms, albeit lower levels than what would be seen in a clinical sample, would not necessarily preclude a child from also demonstrating social competence as CD behaviors might be considered socially competent within low income, urban environments. Thus, given that the mean level of CD symptoms for this sample is lower than what we would expect among a clinically diagnosed (but higher than the normative) sample, it may be that engaging in CD behaviors is not necessarily maladaptive within this context. This is a prime example of why it is crucial to consider the role of context in investigations of behavior as the developmental psychopathology perspective advocates.

Second, this unexpected finding may be a consequence of the age of the current sample. As discussed previously, prior research has indicated that parasympathetic deficiencies associated with externalizing symptoms come "online" during middle childhood (Beauchaine et al., 2007), and RSA evidences developmental changes until late childhood or early adolescence (El-Sheikh, 2005; Hinnant & El-Sheikh, 2009; Hinnant et al., 2011). Therefore, had RSA been assessed in this sample at a later age, relations may have been different. In addition, the current sample had a mean age of almost 10 years old, with some participants over the age of 11. Although the age range of the current sample qualifies as middle childhood, the current DSM

criteria pertaining to CD specifies a diagnosis of childhood-onset CD as the onset of CD symptoms prior to age 10, and a diagnosis of adolescent-onset CD as the absence of CD symptoms prior to age 10. In the current study, parents reported on current symptoms, and we did not gather information on age of onset.

Moreover, evidence shows that the age of onset of puberty has decreased significantly over time in the US, and the age of pubertal development has been found to be even lower among ethnic minority children (Herman-Giddens, et al. 1997; Wu, Mendola, & Buck, 2002). This suggests that some of the children in this sample may be starting puberty. Therefore, because some models link adolescent-onset CD to puberty, it is possible that some of the children in this sample may actually be exhibiting CD symptoms that are time-limited, consistent with adolescent-onset CD. Youth with adolescent-onset CD are less likely to demonstrate problems with emotional reactivity and regulation compared to youth with childhood-onset CD (Frick, 2012; Frick & Viding, 2009; Moffitt, 2003, 2006; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Moffitt, Caspi, Harrington, & Milne, 2002). As high baseline RSA is associated with better emotion regulation, this discrepancy in the current findings may stem from the assessment of children who are actually displaying time-limited CD (consistent with adolescent-onset CD), rather than childhood-onset and potentially life-course-persistent CD (Moffitt, 1993). Though speculative but in line with this explanation, it is possible that some of the behaviors that the parents reported as CD symptoms are actually the beginnings of the normative developmental increase in risk-taking and sensation-seeking behavior that occurs during adolescence (Steinberg, 2008; Steinberg et al., 2008; Stephenson, Hoyle, Palmgreen & Slater, 2003). Moreover, the high crime and violence to which children are exposed when living in low income, urban neighborhoods could exacerbate normative adolescent rebellion and/or

adolescent-onset CD (Frick, 2012). Ultimately, these are empirical questions that need to be examined in similar samples of children of varying ages with attention to pubertal development and using prospective designs to consider the developmental pathways of CD and CU among at-risk youth. If the current findings are accurate, results would suggest that these patterns of findings are characteristic of community samples of children with high CD symptom severity who differ on levels of CU traits and that there may be meaningful differences between clinical and community samples of children in terms of baseline PNS functioning. If the current results do not generalize to similar samples, this may suggest that contextual factors and age exert an important influence on relations among CD, CU, and ANS functioning.

In addition, among children with lower CD symptom severity, low CU symptom severity was associated with lower baseline RSA. This finding is contrary to expectations, as previous research indicates that low baseline RSA is associated with emotion dysregulation whereas high baseline RSA is associated with social competence, empathy, and emotion regulation. Thus, I expected that among children with low CD symptom severity, those with low CU symptom severity would exhibit higher baseline RSA. However, the current finding is not completely inconsistent with previous literature. Low baseline RSA is a non-specific marker of emotion dysregulation and is associated with internalizing symptoms (Dietrich et al., 2007; Forbes, Fox, Cohn, Galles, & Kovacs, 2006; Hinnant & El-Sheikh, 2009). Furthermore, low income, urban youth are at elevated risk for internalizing symptoms (Singer, Anglin, Song, & Longhofer, 1995). Thus, it could be that this group of youth with low CD, low CU, and low baseline RSA actually has elevated levels of internalizing symptoms. Given the high comorbidity between internalizing and externalizing disorders in childhood (Bubier & Drabick, 2009; Connor, Ford, Albert, & Doerfler, 2007; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Drabick, Gadow, & Loney,

2008; Drabick, Gadow, & Sprafkin, 2006; Drabick, Ollendick, & Bubier, 2010; Wolff & Ollendick, 2006), future research with CD should consider the potential role of internalizing symptoms.

As suggested, this finding may also be related to the context in which these children live. Lower baseline RSA is indicative of higher arousal at rest (Hinnant & El-Sheikh, 2009), which may be manifested behaviorally among low income, urban children as over-vigilance to potential dangers in their environment. This increased vigilance may be adaptive for effective functioning, given that these children reside in high crime and violence neighborhoods (Taylor & Seeman, 1999). Thus, this finding may be indicative of the influence of context on biological functioning (Boyce, 2006, 2007; Boyce & Ellis, 2005; Obradovic et al., 2010). However, future studies are necessary in order to clarify the bi-directional influences of context and children's ANS functioning.

In terms of RSA reactivity, I hypothesized that among children with higher CD symptom severity, lower CU traits would be associated with lower RSA reactivity scores. Nevertheless, in the present sample, among children with higher CD symptom severity, those with lower CU symptom severity exhibited higher RSA reactivity (PNS activation). Although this finding is contrary to prediction, the literature regarding relations between RSA reactivity and CD symptoms (or externalizing behavior problems more broadly) is mixed. Both Obradovic et al. (2010) and Boyce et al. (2001) have reported relations between PNS activation in response to challenge (high RSA reactivity in the current study) and externalizing behavior problems in community samples of children, yet other studies report the opposite relation or no relation at all. Furthermore, several studies have reported a relation between PNS activation in response to challenge and child maladjustment in the context of adversity (El-Sheikh et al., 2001; El-Sheikh

& Whitson, 2006; Katz & Gottman, 1995, 1997). Thus, the current finding may be a reflection of both the use of a community sample in the present study, as well as the environment in which these children live.

Among children with higher CD symptom severity relative to youth in the present sample, higher CU symptom severity was associated with low RSA reactivity, or PNS withdrawal relative to baseline, which is consistent with the temperamental profile of children who are high on both CD and CU symptoms. PNS withdrawal in response to challenges is the typical and adaptive response to stressors, as this process allows a person to effectively utilize attentional and social resources to engage or disengage with the environment (Bornstein & Suess, 2000). The antisocial behavior of children with elevated levels of both CD symptoms and CU traits is thought to stem from deficits in moral development rather than deficits in emotion regulation (Frick, 2006, 2012; Frick & White, 2008), and PNS withdrawal in response to stressors is associated with better emotion and behavioral regulation (Calkins, 1997; El-Sheikh et al., 2001; El-Sheikh & Whitson, 2006; Porges, 2007). Thus, based on the literature, it is reasonable to expect that children with higher levels of both CD and CU symptoms would display "normative" emotion regulation abilities that would therefore be reflected in "normative" RSA reactivity, or RSA reactivity similar to that for children with low symptom levels. These normative levels of RSA reactivity, and therefore emotion regulation, afford children who are elevated on both CD and CU the ability to control their emotional response in the face of a threat or stressor, and to thus be more planful and less impulsive with their use of aggression, which is reflected in the high levels of proactive aggression exhibited by this group with childhood-onset CD. In addition, among children with low levels of CD symptom severity, lower levels of CU symptom severity were also associated with low RSA reactivity. Although both of these groups

exhibited PNS withdrawal that was lower than the mean, it is unclear from the literature what level of PNS withdrawal is adaptive. Future research will be necessary to better understand the relations among PNS withdrawal, CD, and CU traits among children who experience different contextual influences.

Interestingly, among children with lower CD symptom severity, those with high CU symptom severity demonstrated high RSA reactivity (PNS activation in response to stressors). Based on the literature linking PNS withdrawal with better emotion regulation abilities, I would expect that children who are lower on CD symptoms, but higher on CU symptoms only would exhibit low RSA reactivity. However, this finding may reflect the general link between autonomic underarousal and psychopathic traits. It is also possible that among children with higher CU traits in the current sample, the stressors used in the present study to elicit reactivity were not found to be stressful. Thus, if these children did not experience the challenge as a stressor, their PNS response may not have been an accurate reflection of their typical PNS response in the face of “real world” stressors. Future research examining these relations should consider utilizing different challenges that may prove to be more stressful such as peer provocation or a frustration-inducing task or use manipulation checks to determine whether the participants experienced the stressors as challenging. It is also the case that there have been no studies to date that have assessed RSA separately in examinations of children with CD in combination with high and low levels of CU traits. Thus, the findings from the current study must be considered preliminary until future research can assess the generalizability of the present results.

*Aim 2: To examine differences in the relation between CD and SNS functioning based on whether children exhibit high or low levels of CU traits relative to other youth in the present sample.*

Contrary to hypotheses, neither CD nor CU symptom severity was significantly associated with PEP variables (baseline PEP and PEP reactivity). These results are surprising given that individual differences in SNS functioning become apparent prior to those related to PNS functioning. There is also the possibility that other variables not considered in the current analyses may better account for these relations. For example, bivariate correlations indicate that household income was negatively associated with PEP at baseline; in other words, higher household income was associated with decreased sympathetic arousal at baseline. Thus, other variables such as neighborhood factors (i.e., neighborhood danger, neighborhood cohesion) may be important to consider in examinations of the relations among CD, CU, and PEP indices. For example, among first to third grade children drawn from the same neighborhoods as the current sample, Bubier et al. (2009) found that child SNS functioning moderated the relations between neighborhood cohesion and externalizing behavior problems. More specifically, the authors found that among children with attenuated baseline PEP, higher levels of neighborhood cohesion were related to lower levels of externalizing behavior problems, whereas among children with heightened baseline PEP, higher neighborhood cohesion was associated with higher levels of externalizing problems. These results suggest that contextual factors may be especially important to consider in addition to child-specific variables when examining relations between behavior problems and SNS functioning among ethnic minority children residing in the inner city.

Non-significant results related to PEP variables also may stem from the particular tasks utilized in the psychophysiological protocol administered in the present study. PEP is an

indicator of SNS response specifically to reward (Beauchaine, 2009, 2012; Beauchaine et al., 2008; Brenner & Beauchaine, 2011; Brenner et al., 2005); thus, it is possible that the tasks used in the current protocol were not considered rewarding by the participants and therefore did not adequately elicit PEP reactivity. However, prior work from an earlier phase of the Child Health and Behavior Study (CHBS) utilizing the same psychophysiological protocol demonstrated significant associations between PEP indices and externalizing behavior problems (e.g., Bubier & Drabick, 2008; Bubier et al., 2009). The sample from this earlier phase of CHBS data collection consisted of children in first through third grade drawn from the same neighborhoods as the current sample. Among this sample, Bubier and Drabick found a significant association between attenuated PEP reactivity and hyperactive-impulsive symptoms. However, this relation was specific to PEP reactivity during the emotion-inducing task of the psychophysiological protocol. Furthermore, among this same sample, Bubier et al. reported a significant association between baseline PEP and externalizing behaviors, which consisted of both symptoms of ODD and CD, and that baseline PEP moderated the relation between neighborhood cohesion and externalizing behaviors. Therefore, there are a couple of potential reasons for the lack of significant findings related to PEP variables in the present investigation. First, the children in the current study were older than those in the earlier phase of CHBS. Patterns of SNS functioning stabilize by middle childhood, earlier than patterns of PNS functioning (Bornstein & Suess, 2000; Calkins & Keane, 2004; Doussard-Roosevelt, Montgomery, & Porges, 2003; El-Sheikh, 2005, 2007; El-Sheikh et al., 2009; Hinnant & El-Sheikh, 2009; Hinnant, Elmore-Staton, & El-Sheikh, 2011). Furthermore, in a longitudinal investigation of the development of RSA and PEP, Hinnant et al. (2011) found a significant increase in children's PEP across ages 8 to 10. More specifically, the authors reported that although there was a great deal of variability in PEP levels

at age 8, PEP changed in the same way (i.e., increased) for all of the children over time. It is therefore possible that compared to the earlier phase of CHBS, there is reduced variability in PEP among the current sample, making it more difficult to detect inter-individual differences. Thus, the lack of significant findings in the current study may reflect the developmental course of PEP.

Second, prior work in CHBS examined relations among PEP variables and ADHD and externalizing behaviors more broadly, whereas the current study examined specifically CD symptoms and CU traits. Thus, it may be that among this sample, use of the more narrowly defined constructs of CD and CU, which also have lower base rates than externalizing behaviors, led to reduced variability and power to predict outcomes. However, given that this is the first study to my knowledge to address relations among PEP functioning, CD, and CU, future research will be necessary to determine which of these explanations can account for these discrepant findings. In addition, future research examining relations among CD, CU, and PEP should consider the use of alternative tasks that may better approximate conditions of reward and include contextual factors as predictors to determine whether the current findings are specific to the sample. If the current findings were replicated in future research, it would suggest that differences in PEP between children who are high on CD symptom severity but differ in level of CU symptom severity may be specific to samples of children with clinical levels of CD symptoms, as opposed to the lower levels of CD symptoms identified in community-based samples of youth.

*Aim 3: To examine whether (a) harsh parenting behaviors and (b) parental warmth/involvement moderate the relations between CD symptoms and ANS functioning and between CU traits and ANS functioning.*

Multiple regression analyses examining child  $\times$  context interactions allowed for a more in depth examination of the relations between child-specific and contextual risk factors and child ANS functioning. Multiple regression analyses were limited to RSA variables, as RSA was the only outcome for which significant prediction was evidenced from the first two aims.

#### *Harsh Parental Discipline*

Harsh parental discipline did not moderate the relations between CD and RSA variables, or between CU and RSA variables. The lack of prediction from CD was not hypothesized, though the lack of prediction from CU was expected. There are several explanations to account for the lack of significant findings. First, given the small sample size, I may have been underpowered to detect effects. Second, it is possible that the scale used to assess harsh parental discipline in the present study did not adequately capture children's experiences of harsh parental discipline. Measurement of harsh parental discipline relied on open-ended responses by parents; given the potentially sensitive nature of the subject, parents may have underreported their use of harsh discipline. Furthermore, the presence of harsh discipline is based on parental responses to hypothetical scenarios and it is unclear whether these situations adequately captured situations that would elicit harsh parenting behaviors. Future research would benefit from the examination of parenting behavior as reported from different sources and utilizing different methods. Lastly, the lack of significant findings related to harsh parental discipline may be due to the nature of the sample. The sample was 98% African- American, and the literature suggests that the use of harsh parental discipline may be more normative among African Americans compared to other ethnic

groups (Deater-Deckard & Dodge, 1997). Furthermore, evidence indicates that there is a weaker relation between harsh parental behaviors and conduct problems among ethnic minority (particularly African-American), as compared to European-American, samples (Deater-Deckard & Dodge, 1997; Hill & Bush, 2001). This evidence suggests that ethnic minority children may differ in their sensitivity to the effects of harsh parental discipline, which could partially account for the lack of significant findings in the present study.

#### *Parental Warmth/Involvement*

Consistent with hypotheses, parental warmth/involvement did not moderate the relation between CD symptoms and RSA variables. However, contrary to prediction, parental warmth/involvement did not moderate the relations between CU traits and RSA variables. As with harsh parental discipline, non-significant findings may be due to the way in which parental warmth/involvement was measured in the present study. Specifically, there may be various ways in which parents express warmth to their children that were not captured in the scale utilized in the present study (e.g., physical displays of affection). Furthermore, the current scale did not include parental verbal expressions of warmth or observation of positive affect expressed toward the child, both of which have been utilized to measure warmth in other studies with similar samples (e.g., Caspi et al., 2004; Davis-Kean, 2005; Mistry, Vandewater, Huston, & McLoyd, 2002; Odgers et al., 2012; Shaw, Winslow, Owens, Vondra, Cohn, & Bell, 1998). Moreover, as parenting practices differ across ethnic and socioeconomic groups (Deater-Deckard & Dodge, 1997; Harrison, Wilson, Pine, Chan, & Buriel, 1990; Hill & Bush, 2001), the construct of warmth/involvement may similarly vary. In particular, parents residing in low SES environments have been found to demonstrate less warmth and responsiveness, and poverty has been found to diminish mothers' ability to adequately respond to her child (Evans, Boxhill, & Pinkaya, 2008;

Grant, Compas, Stuhlmacher, Thurm, McMahon, & Halpert, 2003; Magnuson & Duncan, 2002; McCloyd, 1990). Future research that considers alternative approaches for operationalizing warmth/involvement that may be more relevant to the present sample's characteristics would be useful for determining whether the lack of significant findings is a function of the interview used, sample characteristics, or other factors not addressed in the present study.

### Strengths, Limitations and Future Directions

#### *Strengths*

The current study has several strengths. First, I examined relations among ANS functioning and CD and CU in an urban, low income, and thus, at-risk, sample. Elucidating the ways in which different risk factors for behavioral maladjustment interact with each other is of particular importance among this population, given increased risk of CD among youth residing in urban, low income neighborhoods (Aneshensel & Sucoff, 1996; Attar, Guerra, & Tolan, 1994; Farrington, 1998; Farrington, Ullrich, & Salekin, 2010; Kimonis et al., 2008a; Leventhal & Brooks-Gunn, 2000; Schonberg & Shaw, 2007; Thornberry, Huizinga, & Loeber, 1995).

Another strength of the present study is the consideration of CU traits within the context of childhood-onset CD symptoms, given the potentially negative course and correlates but dearth of research that considers CU traits in examinations of childhood-onset CD. In addition, the present study utilized objective physiological measures of emotional and behavioral states rather than self-report, and the use of different reporters and indices reduces concerns regarding mono-rater and mono-method biases. Furthermore, the present study contributes to the extant literature by including RSA and PEP indices, which allows for the examination of the independent contributions of the PNS and the SNS. It is clear that these two branches of the ANS do not influence behavior in the same way (Beauchaine, 2009); thus, this approach enhances our

understanding of human behavior by delineating the contributions of each branch and the sympathetic and parasympathetic patterns that ultimately result in particular types of behavior, including CD with and without CU.

### *Limitations and Future Directions*

In spite of its strengths, the present study has several limitations. First, the sample size was relatively small, particularly for tests of moderation. Although a power analysis indicated sufficient power for examining these relations, the small sample size suggests that I may have been underpowered to detect effects and to consider CD, CU, and parenting variables concurrently in the ancillary analyses. Furthermore, the sample size limited the ability to utilize more complex statistical analyses such as structural equation modeling, which allows for the examination of multiple outcomes concurrently, as well as inter-relations among predictors, and therefore may have been better able to capture the nature of the relations between CD and CU. However, findings provide preliminary evidence for distinctions and potential developmental pathways for childhood-onset CD with and without higher levels of CU traits that can be further investigated and validated in larger, prospective studies using varied statistical methodology.

Furthermore, although the examination of these processes in an inner city sample is a strength of the current investigation, the sampling method (i.e., self-selection) presents potential sampling biases. For instance, it is possible that the self-selection method is more conducive to the recruitment of families with higher levels of functioning (i.e., families that have more resources or are better able to access available resources, or are experiencing fewer stressors, etc.) compared to other families of similar background and SES. Thus, it is possible that the children who participated may demonstrate lower levels of behavioral and psychological symptoms compared to others experiencing similar levels of contextual disadvantage. However,

children in this sample demonstrated higher mean levels of CD symptoms compared to children in the normative sample. Therefore, it is also a possibility that families who elected to participate in the present study may have done so because their children manifest significant symptoms of psychopathology. In light of these competing possibilities, future research is necessary to assess the generalizability of the current findings.

In addition, SES and ethnicity were confounded in this sample. However, the sample is representative of the schools and neighborhoods from which it was drawn. Future research that disentangles SES and ethnicity will be necessary to address this confound, though I chose to focus on this sample of children given elevated risk associated with CD among contextually disadvantaged children, as well as the importance of prevention and early intervention efforts among at-risk youth. Furthermore, PEP and RSA were assessed at the same time point as CD symptoms and CU traits; therefore, I could not evaluate prospective relations between ANS functioning and CD symptoms and CU traits. Future research using a prospective design is necessary to establish whether specific patterns of ANS functioning are correlates or contribute to the development of CD symptoms with and without high levels of CU traits. Such prospective research also could aid in elucidating factors that may protect or buffer risk among children with maladaptive patterns of ANS functioning from detrimental outcomes.

Additionally, although the present study examined parenting behaviors as moderators of the relations between CD symptoms or CU traits and children's ANS functioning, it is likely that parent-child influences are bidirectional and transactional. The present study, however, was cross-sectional and therefore did not account for such bidirectional effects. Thus, future research should include longitudinal and cross-lagged designs that can disentangle the direction of effects between parents and children.

Furthermore, future research should examine other potential moderators of these relations, including other contextual factors that are expected to influence youth during this developmental period (e.g., neighborhood factors, peer influence). In addition to the more proximal, stage-relevant contextual influences examined in the current study (i.e., parent-child relations), it is important to consider the influence of other contextual factors whose influence may change over time and that may influence relations among childhood-onset CD, CU traits, and ANS functioning (Bubier et al., 2009; Lynam, Caspi, Moffitt, Wikstrom, & Loeber, 2000; Meier, Slutske, Arndt, & Cadoret, 2008; Scarpa, Tanaka, & Haden, 2008). Nevertheless, although contextual risk factors differentially predict behavior problems depending on youth's ANS functioning (e.g., Bubier et al., 2009; Scarpa et al., 2008), there is a dearth of research examining moderation between contextual factors and ANS functioning in the development of CD with and without CU traits. Thus, future research would benefit from examination of such moderators, as consideration of interactions between contextual and child-specific variables can help to determine the utility and roles of these factors in conferring or buffering risk for childhood-onset CD with and without CU traits.

Another important challenge for future research in this area would be to consider how these processes might differ in terms of child sex. Given the small sample size in the current study, sex differences were not examined. Although there are sex differences in prevalence rates, developmental trajectories, correlates, and outcomes of CD and CU traits (Costello et al., 2003; Dadds et al., 2009; Fanti et al., 2009; Fontaine, Rijdsdijk, McCrory, & Viding, 2010; Silverthorn & Frick, 1999; Vitale et al., 2005; Xie, Drabick, & Chen, 2011), and ANS-behavior relations often differ for boys and girls (Beauchaine, 2009; Beauchaine et al., 2008; Wang et al., 2012),

much more research is needed regarding sex differences in the links between CD with and without CU traits and ANS functioning.

Lastly, CD symptoms were operationalized using a dimensional rating scale, rather than a diagnostic interview. Thus, results pertaining to CD symptoms are not equivalent to the diagnostic category of CD. However, the CASI-4 is a *DSM*-referenced instrument and has been used in many studies of CD (Beauchaine et al., 2008; Drabick, Gadow, & Sprafkin, 2006; Frick et al., 2000b; Kochanska, Barry, Aksan, & Boldt, 2008; Kochanska, Barry, Stellern, & O'Bleness, 2009). Furthermore, even at levels below that which is required for a clinical diagnosis, CD symptoms can cause significant impairment and a dimensional approach is more useful for identifying patterns of maladaptation that can inform etiological and intervention models (Angold et al., 1999; Drabick, 2009).

In sum, results of the present study suggest that patterns of PNS functioning differ between children with high levels of CD symptoms with and without high levels of CU traits. These findings further support the existence of distinct subtypes of childhood-onset CD and highlight the need to distinguish these subtypes in future research. The findings from the present investigation provide preliminary evidence for unique relations among ANS functioning and childhood-onset CD with and without higher levels of CU traits and point to potentially distinct etiological mechanisms underlying the development of these subtypes. Ultimately, the goal of elucidating factors that are associated with and may contribute to distinctions between childhood-onset CD with and without CU traits is to inform etiological models of psychopathy and subsequently develop strategies to intervene before symptoms become more pernicious and intractable to treatment. Further examination of CD, CU, and ANS relations in various developmental periods and with developmentally relevant contextual factors could help to clarify

the developmental course of psychopathy and etiological models of CD+CU. In addition, findings from developmentally sensitive research designs could inform more individualized prevention and intervention strategies that could mitigate or prevent CD+CU and the associated negative course and sequelae.

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