UNLOCKING THE HETEROGENEITY OF EXTERNALIZING PROBLEMS ACROSS EARLY CHILDHOOD:
UNITING NEUROBIOLOGICAL, PARENTING, AND DEVELOPMENTAL PERSPECTIVES

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ABSTRACT

Heterogeneity in externalizing behaviors such as conduct problems impedes the elucidation of underlying developmental mechanisms. Recent studies suggest that individuals exhibiting conduct problems may show entirely discrepant patterns of biological vulnerabilities depending on whether they have comorbid internalizing problems or not. However, no research to date has examined this from a developmental perspective. The current study examines theoretically derived hypotheses regarding the developmental risk pathways that differentiate children with conduct problems who do and do not manifest comorbid anxiety. Biological vulnerability was examined with respect to blunted or exacerbated stress responsivity. Salivary cortisol was collected in response to a stressful task at 7, 15, and 24 months of age. Additionally, home visitors reported children’s behaviors at each time point, and parenting styles were observed in a structured interaction with their child.

Results showed that children with conduct problems only at 5 years of ages were characterized by the exacerbated stress responsivity at 7 months of age followed by blunted stress responsivity at 24 months old. In contrast, children with comorbid internalizing problems were typified by the interrelationship of heightened stress responsivity and high behavioral approach tendencies at 15 months of age. While both children with and without comorbid anxiety displayed exacerbated stress responsivity with greater exposure to intrusive parenting, the developmental time points that this biological sensitivity to context became apparent was different.

The present study provides a better understand of the developmental mechanisms in that undergird distinct externalizing problems phenotypes with and without comorbid internalizing problems in early childhood. Overall, these findings suggest that it may be more efficacious for intervention programs to target biological, behavioral, and parenting domains of risk at key development time points to prevent the later development of distinct conduct problem phenotypes.
## TABLE OF CONTENTS

LIST OF FIGURES .............................................................................................................. v

LIST OF TABLES ................................................................................................................ vi

ACKNOWLEDGEMENTS ................................................................................................... vii

Chapter 1  Introduction .................................................................................................1

Chapter 2  Methods ........................................................................................................23

Chapter 3  Results ..........................................................................................................35

Chapter 4  Discussion ....................................................................................................62

REFERENCES ..................................................................................................................78
LIST OF FIGURES

Figure 1: Changes in Children’s Irritability across Early Childhood within each Conduct Problem Subgroups .............................................................................................................................42

Figure 2: Changes in Children’s Exposure to Intrusive Parenting Across Early Childhood within each Conduct Problem Subgroups .............................................................................................................................42

Figure 3: Interrelationships between HPA Activity and Intrusive Parenting at 7 Months of Age Predicting CP Subgroup Membership ..............................................................................................................46

Figure 4: Interrelationships between Children’s HPA Activity and Approach Tendencies at 15 Months of Age Predicting COMORBID Subgroup Membership .......................................................................................53

Figure 5: Interrelationships between Children’s HPA Activity and Intrusive Parenting at 15 Months of Age Predicting COMORBID Subgroup Membership .......................................................................................53

Figure 6: Interrelationships between Approach Tendencies and Sensitive Parenting at 15 Months of Age Predicting CP Subgroup Membership ............................................................................................................54

Figure 7: Interrelationships among Children’s HPA Activity, Approach Tendencies, and Sensitive Parenting at 24 Months of Age Predicting CP Subgroup Membership ............................................................................................................60

Figure 8: Interrelationships among Children’s HPA Activity, Irritability, and Intrusive Parenting at 24 Months of Age Predicting COMORBID Subgroup Membership ............................................................................................................60
LIST OF TABLES

Table 1: Percentages and Means (Standard Deviations) of Demographic Variables for Conduct Problem Subgroups..................................................................................................................26

Table 2: Means (SD) Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles across Early Childhood by Conduct Problems Subgroups..............................38

Table 3: Likelihood Ratio Tests for Models Relating Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 7 Months of Age to Conduct Problem Subgroups........................................................................44

Table 4: Parameter Estimates for the Multinomial Logistic Regression Associating Children’s Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 7 Months of Age to Conduct Problem Subgroups..................................................................................45

Table 5: Likelihood Ratio Tests for Models Relating Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 15 Months of Age to Conduct Problem Subgroups..........................................................................................48

Table 6: Parameter Estimates for the Multinomial Logistic Regression Associating Children’s Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 15 Months of Age to Conduct Problem Subgroups..................................................................................50

Table 7: Likelihood Ratio Tests for Models Relating Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 24 Months of Age to Conduct Problem Subgroups..................................................................................................................56

Table 8: Parameter Estimates for the Multinomial Logistic Regression Associating Children’s Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 24 Months of Age to Conduct Problem Subgroups..................................................................................57

Table 9: Summary of Risk Factors across Early Childhood that Characterize the Development of Conduct Problem Subgroups at 5 Years of Age..................................................................................61
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Chapter 1

Introduction
The inherent heterogeneity of externalizing problems in early childhood makes it difficult to determine the antecedents, correlates, and developmental pathways underlying these problems (e.g., Beauchaine et al., 2008; Dodge, Coie, & Lynam, 2006; Raine, 2002). To date, most attempts to identify the heterogeneity of externalizing problems have focused on parsing the various forms of problem behavior, such as whether or not externalizing problems are reactive responses to stimuli or proactively enacted toward instrumental goals (e.g., Berkowitz, 1993; Dodge & Coie, 1987; Gendreau & Archer, 2005; Vitaro, Gendreau, Tremblay, & Olligny, 1998). Unfortunately, reliable identification of externalizing subgroups based on these behavioral factors alone has been difficult to achieve (Tremblay & Nagin, 2005; Vitaro, Brendgen, & Tremblay, 2002), with many studies revealing high correlations between the reactive and proactive classifications (e.g., Dodge & Coie, 1987; Poulin & Boivin, 2000; Price & Dodge, 1989). Due to the lack of success of parsing classifications solely based on externalizing behaviors, researchers have combined clinical and biological perspectives to propose that it may be beneficial to determine subgroups based on patterns of comorbidity involving externalizing and internalizing symptoms (review, Granic & Lamey, 2002; Hinshaw & Anderson, 1996; Hodgins, de Brito, Siminoff, Vloet, & Viding, 2009; Lahey & Loeber, 1994; Raine, 2002; Steiben et al., 2007). While this is a potentially promising theory, the study of externalizing and internalizing comorbidity as an effective way to elucidate the inherent heterogeneity of externalizing problems is still in its nascent phase, highlighting a need for further empirical investigation.

Externalizing and internalizing problems during childhood may appear to be opposing phenomena, but the co-occurrence of these problems has been demonstrated in many epidemiological and longitudinal studies (Caron & Rutter, 1991; Keiley, Lofthouse, Bates, Dodge, & Kenneth, 2003; Rutter & Sroufe, 2000). What has yet to be well-established is whether children with comorbid
externalizing and internalizing problems have different antecedents and developmental pathways than children who exhibit externalizing behaviors without internalizing problems. Different domains of risk are thought to provide unique contributions to the development of internalizing and externalizing problems including both biological and behavioral aspects of the child as well as exposure to various parenting styles (e.g., Deater-Deckard, Dodge, Bates, & Pettit, 1998; Hodgings et al., 2009). For example, a longitudinal investigation of children from 5 to 14 years old revealed that child temperamental characteristics such as resistance to control and harsh parental discipline were related to externalizing symptoms only, while maternal reports of difficult temperament and overall family stress were related to comorbid externalizing and internalizing symptoms (Keiley et al., 2003).

Yet, there is still a large research gap in determining the interrelationships among individual and contextual domains of risk during infancy and toddlerhood that may provide the most explanatory power for the early emergence of externalizing problems and comorbid internalizing symptoms. Understanding the association between specific risk factors and psychological outcomes becomes increasingly complicated when examined over developmental time (Bronfenbrenner & Morris, 1998; Cicchetti & Tucker, 1994; Rutter, 1990; Sameroff & Chandler, 1975; Sroufe & Rutter, 1984). Some researchers have argued that risk factors that are transient do not function in the same way as those that are chronic, and that single time-point assessments capture noise by failing to distinguish between qualities that are unique to this assessment from those that are truly characteristic of the individual. This hypothesis suggests that repeated measurements should be employed and only individuals who repeatedly demonstrate the presence of a particular risk factor should be examined. However, because of the normative developmental changes that occur from infancy to school entry, the predictive validity of one risk factor may be more apparent at certain developmental time points. This is likely a function of the fact that psychological constructs such as threat identification and regulatory ability have different meaning across developmental time.
For instance, it may be highly adaptive for an infant to demonstrate high behavioral reactivity and low biological reactivity to stress (e.g., Gunnar & Davis, 2003; Gunnar & Donzella, 2002). Presumably the high behavioral reactivity serves to signal to the caregiver the need for attention, and the biological system need only be taxed if the caregiver’s response to the behavioral signal does not sufficiently reduce the stress. However, as infants transition into independent movement, it may be more appropriate for them to engage their own biological and behavioral resources to resolve challenges independently, without necessarily signaling distress to an adult unless they find they are unable to resolve the challenge alone (e.g., Gottlieb, 1992; Sameroff, 1990; Shaw, Keenan, & Vondra, 1994). It is possible that no absolute association between biological and contextual risk characterizes children across development, but rather profiles that are developmentally inappropriate for the particular measurement time have greatest explanatory value. Thus, it is proposed here that the predictive validity of interactions between risk domains changes across developmental time, and that these interactions over time may be more significant than the change or stability of any one risk factor alone.

Thus, the overarching objective of the current study is to integrate neurobiological, behavioral, parenting, and developmental research perspectives to explore the risk pathways in infancy and toddlerhood that may result in distinct subgroups of externalizing problems, specifically those with and without comorbid internalizing symptoms, at 5 years of age. In the current investigation, the externalizing behaviors consist of conduct problems ranging from temper tantrums to physical aggression, while the internalizing problems primarily entail symptoms of anxiety. Overall, this study proposes that the complex interplay among biological stress responsivity, children’s behavioral reactivity and regulation, and parenting styles at 7, 15, and 24 months of age will contribute to children’s membership in behavioral subgroups characterized by high levels of conduct problems without anxiety.
(CP), comorbid conduct problems and anxiety (COMORBID), or normal development (COMPARISON) (see Figure 1). In general, knowledge regarding these early risk factors may be necessary to designing more effective early prevention programs, especially when considering that externalizing problems have been shown to become more resistant to treatment over time (Kazdin, 1985, 1987; Loeber, Green, Lahey, & Stouthamer-Loeber, 1991).

Chapter Overview. In this first chapter, the background and rationale for the current analyses will be presented and framed around Figure 1. The sections will proceed from the individual to the contextual levels of risk (the bottom to top left of Figure 1). Each of the three sections includes how interrelationships among children’s biological stress responsivity, behavioral reactivity and regulation, and parenting styles at key developmental time points (7, 15, and 24 months of age) may be different when predicting development of CP, COMORBID, and COMPARISON subgroups at 5 years of age.
Initial discussions in first section further elucidate the differences in biological stress responsivity that may undergird the three theoretically-distinct subgroups associated with conduct problems.

Consistent with the literature on the hypothalamic-pituitary-adrenal (HPA) as an indicator of biological stress responsivity, this study focuses on cortisol response to challenge—the end product of the neuroendocrine cascade involved in HPA functioning—as an indicator of susceptibility. The second and third sections discuss how children's behavioral reactivity and regulation as well as children’s exposure to different parenting styles may moderate (and be moderated by) HPA activity in infancy and toddlerhood to result in different developmental trajectories underlying distinct behavioral outcomes. Lastly, the overall aims and hypotheses of the current study are identified.

**Biological Stress Responsivity**

As seen in Figure 1, the most basic domain of risk (depicted as the lowest risk level) to potentially differentiate the development of CP, COMORBID, and COMPARISON subgroups is biological stress responsivity. Scientists propose that two distinct biological pathways to externalizing behavior exist: elevated activation of biological responses to perceived threat that makes appropriate behavioral control challenging (*heightened* response), or a deficit in reactivity (*blunted* response) that may result in lowered sensitivity to punishment and ultimately poor socialization (Hodgins, 2007; Hodgins et al., 2009; Raine, 2002; Steiben et al., 2007). Therefore, it is hypothesized that children who develop comorbid symptoms are likely to be characterized by elevated stress responsivity (a reduced threshold for threat identification and a rapid and *heightened* biological response to stress), whereas those with CP are likely to be characterized by a reduction in or absence of stress responsivity (a high threshold for threat identification and *blunted* biological stress responsivity)(review, Granic & Lamey, 2002; e.g., Hinshaw & Anderson, 1996; Lahey & Loeber, 1994).

**Heightened HPA Activity.** In general, children who have low thresholds for threat detection, and consequently heightened biological reactivity to contextual challenges, are believed to have a higher
propensity for displaying comorbid anxiety (e.g., Hubbard et al., 2002; McBurnett et al., 1991). Biological reactivity to threat consists of a sequence of responses across multiple physiological systems. The autonomic nervous system is engaged in immediate response to threat, preparing the body for fight-or-flight reactivity. Activation in this system is a shared correlate of both anxiety (flight) and reactive aggression (fight) in children (Hubbard et al., 2002; Tyson, 1998; van Goozen et al., 1998). If the threat is perceived as severe, or engagement of the autonomic system does not result in rapid diffusion of threat, HPA responsivity is engaged resulting in a systemic release of cortisol to minimize catabolic activity as well as reproductive and immune system functioning in order to maximize resources to sustain efforts to escape or resolve the threat (e.g., Chrousos, 1998; Chrousos & Gold, 1992; Porges, 2001; Ulrich-Lai & Herman, 2009). Because of the sequential nature of this process, measurement of cortisol is conducted both before the task and approximately 20 minutes after the onset of the experimental challenge, and cortisol reactivity is proposed to be evidence of subjective assessment of the challenge as being extremely stressful and difficult to cope with. In other words, failure to exhibit a cortisol response is considered to indicate that the individual did not perceive the experience as stressful, or felt sufficiently capable of coping with the stressor. Consistent with this model, evidence suggests that children with externalizing problems comorbid with anxiety exhibit higher salivary cortisol responsivity following an interpersonal frustration task than children with conduct problems only (van Goozen et al., 1998). Additionally, children with conduct disorder exhibit higher cortisol baseline levels if they have comorbid anxiety symptoms (McBurnett et al., 1991; 2000).

HPA axis activation results in a systemic release of cortisol which not only induces changes in peripheral physiology, but modulates activity in a wide range of brain regions. In particular, a heightened cortisol concentration results in down-regulation of higher-order prefrontal systems (e.g., Teicher et al., 2003). This effect of cortisol is considered to be adaptive, as the higher-order cognitive processing of the prefrontal system is computationally slower than limbic systems and may be
detritmental under acute and immediate threat. However, activation of this system that is disproportionate to the actual contextual challenge will disrupt children’s ability to enact self-regulation skills and executive processing (e.g., McBurnett et al., 1991), leading to greater disruption of behavior in both internalizing and externalizing domains. This context-specific profile of cognitive disruption explains why children who are capable of identifying appropriate and prosocial responses to challenging experiences presented hypothetically often fail to enact those responses in ecologically-valid contexts when physiological arousal is more likely to be engaged (Dearing et al., 2002).

**Blunted HPA Activity.** Evidence from research on autonomic arousal as well as HPA responsivity suggests that children characterized by blunted baseline cortisol levels and reactivity to challenge have greater amounts of externalizing problems in later childhood and adulthood (reviews, Fowles, 2000; Raine, 2002), as well as lower than average levels of anxiety (Hodgins et al., 2009; Loney et al., 2006). However, blunted HPA activity appears in only a small percentage of the population (Del Giudice, Ellis, & Shirtcliff, 2010; Hawes, Brennan, & Dadds, 2009; Raine, 2002; Raine, Venables, & Mednick, 1997). It has been proposed that blunted HPA reactivity represents a biological adaptation to protect the individual from the physiological consequences of prolonged cortisol exposure that may occur in environments of chronic threat (Del Giudice et al., 2010; Gunnar & Vazquez, 2006; Gustafsson, Janlert, Theorell, & Hammarstrom, 2010; Susman, 2006; Tarullo & Gunnar, 2006). To date, however, very little research has tested the developmental progression of the predicted relationship between blunted HPA activity and psychopathology.

Biologically blunted stress responsivity may be associated with a decreased sensitivity to punishment (Beauchaine, 2001; Corr, 2004; Gray, 1987; review, van Goozen, Fairchild, Snoek, & Harold, 2007). Decreased sensitivity to punishment makes it difficult to internalize consequences and may lead to greater avoidance conditioning and deficits in moral socialization that enhance risk for developing conduct problem symptoms (Frick & White, 2008; Hodgins et al., 2009). At the extreme end of the
spectrum, blunted stress reactivity across the full spectrum of challenge severity may be associated with a child's likelihood to display callous and unemotional traits.

Stability and Change over Time. Although there is substantial plasticity in children's state HPA responsivity in infancy (Davidson, Jackson, & Kalin, 2000), the theoretical trait of biological sensitivity that HPA responsivity is believed to reflect is likely to become canalized and more resistant to change over time (Turkheimer & Gottesman, 1991). One possibility is that children characterized by consistently blunted responsivity are at much greater risk for developing conduct problems without comorbidity (e.g., Hawes et al., 2009; Raine et al., 1997), and that those with consistently heightened responsivity are at greater risk for developing comorbid conduct problems (e.g., McBurnett et al., 1991; van Goozen et al., 1998). Alternatively, changes in HPA reactivity patterns over time may be more predictive. Specifically, the down-regulation of biological stress responsivity over time as an adaptation to early heightened activity (the attenuation hypothesis; Susman, 2006) may be more indicative of long term changes in physiological and psychological systems that reflect greater canalization of risk (Gunnar & Vazquez, 2006; Gustafsson et al., 2010; Tarullo & Gunnar, 2006). This is especially likely to predict the presence of conduct problems without comorbid anxiety.

Interrelationships of Risk Domains at 7 Months of Age. In infancy, two main interrelationships are expected to significantly predict differences in the development of CP, COMORBID, and COMPARISON subgroups: 1) the reciprocal interplay between behavioral reactivity and adverse parenting styles and 2) adverse parenting exacerbating the influences of vulnerabilities in biological stress responsivity. These relationships are depicted in the dark blue box on the far left side of Figure 1.

Before children have acquired and mastered various self-regulation skills (e.g., Gunnar & Donzella, 2002), how behavioral aspects of overall negative reactivity and irritability reciprocally interact with adverse parenting styles may have significant explanatory power when determining whether children will become members of CP, COMORBID, versus COMPARISON subgroups at 5 years of age.
Many empirical investigations have demonstrated that behavioral-level risk factors and adverse parenting styles reciprocally interact in early childhood to influence the later development of conduct problems (Bates & Bayle, 1988; Bates, Pettit, Dodge, & Ridge, 1998; review, Putnam et al., 2002; Rubin, Burgess, Dwyer, & Hastings, 2003). As such, adverse parenting style may be a critical factor affecting children’s developing behavioral reactivity and regulation during early childhood, and vice versa (see Figure 1).

That being said, biological stress responsivity is the most plastic in early infancy as a means of “appropriately” adapting to the amount and severity of stressors a child is (and will likely be) exposed to in a particular environment (e.g., Joseph, 1999; Nelson & Carver, 1998). At very early ages (before 12 months of age), children have a very limited behavioral repertoire and have not yet developed the motivation or ability to regulate emotions such as frustration (e.g., Gunnar & Donzella, 2002; Rothbart, Sheese, & Posner, 2007). Therefore at this stage, it is hypothesized that biological measures of stress reactivity may be a better predictor of how children respond to adverse parenting. This is especially likely given recent theories of stress reactivity as moderating the extent of sensitivity to contextual factors and explaining individual differences in risk and resilience to environmental stress (Boyce & Ellis, 2005; Del Giudice et al., 2010). Therefore, it may be that the interactions between adverse parenting styles and biological stress responsivity at 7 months of age to distinguish the development of CP, COMORBID, versus COMPARISON subgroups.

Behaviors of Reactivity and Regulation

Behavioral reactivity and regulation, especially after the first year of life, is the key intermediate domain of risk for early development of CP, COMORBID, and COMPARISON subgroups (see Figure 1). In general, behavioral reactivity and regulation risk is hypothesized as having moderating effects on the relationship between biological stress responsivity as well as parenting styles and conduct-related symptoms at 5 years of age. Specific behavioral risks empirically associated with early conduct problems
include behavioral reactivity qualities such as irritability, as well as indices of low behavioral regulation such as high approach tendencies and difficulties with sustained attention (e.g., Eisenberg et al., 2009; Farmer, Bierman, & CPPRG, 2002; Keiley et al., 2003; Nigg, 2006).

**Irritability.** Behavioral reactivity such as irritability has been associated with conduct problems both with and without anxiety (e.g., Eisenberg et al., 2001, 2009; Farmer et al., 2002; Milich & Landau, 1984). However, negative reactivity and irritability in the early school years appears to be more characteristic of children comorbid for externalizing and internalizing problems versus externalizing alone (Eisenberg et al., 2009; Keiley et al., 2003; Khan et al., 2005). Therefore, it is unclear how the sensitivity and specificity of this trait changes over developmental time. It is possible that in infancy, negative reactivity is a primary behavior of a very limited repertoire, and thus, is broadly predictive of risk for psychopathology. As children age, those who persist in high levels of irritability at developmentally inappropriate time points may be more likely to develop high levels of pathology including comorbidity. Alternatively, for a large majority of normally developing children with early negativity will successfully develop coping skills that diminish the presence of irritability at later time points.

**Attention Regulation.** Deficits in emotion regulation during stressful situations may also result from impaired control over lower-order brain regions involved in emotion processing by higher-order brain regions in the frontal lobes (MacDonald, 2008). Children with conduct problems, on average, have been shown to have difficulties attending to environmental feedback when trying to extinguish ineffective behaviors (Giancola & Tartar, 1999; Sagvolden, Johansen, Aase, & Russell, 2005). For instance, an empirical investigation of a subset of the low income, nonmetropolitan population utilized in the current analyses demonstrated that attention problems (lack of persistent attention, low levels of behavioral constancy on tasks of difficult levels, and limitations in sustained interests in objects/test materials) in the toddler years, among children exposed to high interparental conflict, was associated
with conduct problems at 3 years of age (Towe-Goodman, Stifter, Coccia, Cox, & The FLP Key Investigators, 2011).

In general, the ability to focus attention is linked to the ability to plan behaviors that can be used to mitigate stressful contexts (e.g., NICHD, 2005). Sustained attention and the ability to shift attention have been associated with both a reduction in internalizing symptoms of anxiety and overall anger and defiance. This makes it difficult to determine whether the degree of attention regulation problems significantly varies for children who develop externalizing problems with and without internalizing symptoms. Some studies have revealed that children within comorbid externalizing-internalizing problem subgroups had greater parent- and teacher-reported problems with attention, including difficulties with concentrating and sustaining interest in tasks and high levels of impulsivity in kindergarten (Farmer et al., 2002) and grade school (Boivin et al., 1994; Kellam et al., 1991) as compared to children with externalizing problems alone. In contrast, other investigations have demonstrated that attention problems, such as difficulties maintaining focus on tasks or appropriately shifting focus to manage task-related demands, are similar for both externalizing problem subtypes (Eisenberg et al., 2009; Steinberg et al., 2009). Therefore, the only difference that is clearly expected in the current study is that both CP and COMORBID subgroups will be characterized by greater precursors of attention regulation difficulties than children in the COMPARISON subgroup.

High Approach Tendencies. Unbridled approach tendencies are hypothesized as being characteristic of individuals with externalizing behaviors such as CP. Extreme or context inappropriate approach behavior has been associated with blunted arousal across multiple physiological systems, and it is possible that failure to recognize risk negates normal processes that are designed to increase cautiousness when faced with novelty or potential threat (Beauchaine, Katkin, Strassberg, & Snarr, 2001; Quay, 1993). Some researchers have postulated that blunted arousal is perceived as subjectively aversive, ultimately motivating sensation seeking behavior in attempt to increase physiological arousal...
Importantly, high levels of approach behavior in infancy have also been associated with the development of higher IQ in childhood (Guerin, Gottfried, Oliver, & Thomas, 2003), so it is important to consider how additional risk factors moderate the relationship between early behavioral disposition and later symptoms of externalizing behavior. In addition, the presumption that approach behaviors are consistent with externalizing symptoms lends to the hypothesis that this trait should differentially predict conduct problems without anxiety from the comorbid presentation. Many studies have reported this pattern (Johnson, Turner, & Iwata, 2003; Oldenhinkel, Hartman, Dewinter, Veenstra, & Ormel, 2004), although others have reported a positive association with early approach behavior and comorbid externalizing and internalizing symptoms (Keiley et al., 2003).

It is important to note that HPA reactivity and behavioral reactivity and regulation represent distinct components of individual-level risk and are not necessarily coordinated (Gunnar & Davis, 2003). Some theories posit that the ability of biological and behavioral systems to respond to context at least partially independently fosters adaptability across development (Glassman, 1973; Quas, Hong, Alkon, & Boyce, 2000), but it is important to note that adaption in one domain may confer consequences in other domains (Crespi & Denver, 2005). For instance, blunted HPA activity may transpire as a means of adapting to a chronically adverse context to protect the body against physiological damage from repeated acute stress reactivity (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Susman, 2006). To date, no empirical research has been performed on how the dissociation between biological and behavioral reactivity and regulation in infancy and toddlerhood may differentially predict later patterns of conduct problems with and without anxiety symptoms.

**Stability and Change over Time.** The stability and change of affective behaviors over time may also be central to determining which children will develop externalizing problems with and without internalizing problems. While most empirical findings have indicated that children with externalizing
behaviors have more attention regulation difficulties than their same age counterparts (Boivin et al., 1994; Eisenberg et al., 2009; Farmer et al., 2002), there are inconsistencies in the data as to whether children who show co-occurring internalizing and externalizing symptoms have greater deficits in attention than do those who have externalizing symptoms only.

It is possible that the developmental period during which these issues arise, and/or the stability of these problems across early childhood, may be at the root of these inconsistencies. For instance, high approach tendencies that are stable across early childhood in a community sample were significantly associated with the development of comorbid externalizing and internalizing symptoms versus no symptoms or only conduct problems at 4.5 years of age (Stifter, Putnam, & Jahromi, 2008). The authors speculated that this relationship may have occurred because children with high approach tendencies are also likely to become easily frustrated or depressed over time if their goals are blocked or do not produce adequate progress, increasing their risk for comorbidity. While a promising study, more investigations are necessary to determine if this relationship between stable high approach tendencies and comorbid outcomes still holds in high risk samples.

**Interrelationships of Risk Domains at 15 Months of Age.** On average, as children enter later infancy (beginning of their second year of life), they have more control over their behavioral reactivity and utilize necessary self-regulatory behaviors as an initial way to mitigate responses to stress (Gottlieb, 1992; Rothbart et al., 2007). For instance, children with warm and sensitive parents may have formed an internal working model that caregivers will come to their aid when faced with stressors (e.g., Ainsworth, Blehar, & Wall, 1979; Bowlby, 1969). This allows children to express emotional distress and elicit help without unnecessarily triggering a “fight or flight” response from the sympathetic branch of the ANS or the negative feedback response of HPA system—biological and behavioral reactivity in this instance are uncoupled. Therefore, it is only when behavioral reactivity and regulation are unsuccessful in mitigating stress that biological systems are activated (Gottlieb, 2002). Given this theoretical premise, the most
significant and prevalent mechanisms undergirding the development of conduct problem subgroups may involve the interrelationships between behavioral reactivity and regulation and parenting style as well as children's biological stress responsivity and behavioral reactivity and regulation (see Figure 1).

Increased mobility in late infancy allows for the inverse relationship between social approach tendencies and biological stress responsivity to potentially become more evident at 15 months of age. This relationship between blunted HPA activity and high approach tendencies in later infancy may be related to the development of children's membership into CP versus COMORBID subgroups. What is uncertain is whether high approach tendencies may moderate the relationship between heightened biological stress responsivity and COMORBID subgroup membership at 5 years of age. Empirical research has demonstrated that individuals with only internalizing problems in childhood and adolescence tend to have heightened HPA activity and low approach tendencies (e.g., Granger, Weisz, & Kaunekis, 1994; Granger, Weisz, McCracken, Ikeda, & Douglas, 1996), but no known empirical investigations have determined, on average, how HPA activity relates to approach tendencies in children with comorbid externalizing and internalizing symptoms.

Since children are able to exert increased effortful control, such as sustained attention, by the second year of life (Derryberry & Rothbart, 1997; Kochanska, Murray, & Coy, 1997), reciprocal interactions of attention difficulties with biological stress responsivity and with parenting styles in early childhood are believed to contribute to the unique and disparate development of externalizing problems with and without anxiety (MacDonald, 2008). Yet, there is a significant knowledge gap in the literature regarding the explanatory power and exact interrelationships involving these attention regulation difficulties as the predictor of these conduct problems subgroups.

Parenting Styles

The review of the interrelationships among risk domains thus far has already begun to highlight how parenting style (depicted at the top Figure 1) is central in determining the pathways to the
development of CP, COMORBID, and COMPARISON subgroups at 5 years of age. While parental insensitivity and intrusiveness are believed to play key roles in the development of conduct problems (e.g., Frick et al., 1990; Patterson, Reid, & Dishion, 1992), there is a research gap as to whether parenting styles differentially relate to the likelihood of developing comorbid anxiety. Maternal sensitivity—responding to children's needs in a timely, warm, and responsive fashion—appears to be associated with reduced risk for heightened HPA reactivity (e.g., reviews, Adam, Klimes-Dougan, & Gunnar, 2006; Gunnar & Donzella, 2002), and potentially later mental health problems (e.g., Blair et al., 2008). Yet, the relationship among these variables is unclear. Some researchers hypothesize that maternal sensitivity allows children to face stressors with a greater sense of efficacy and safety, thus not requiring the engagement of the HPA system. Conversely, maternal insensitivity may impede the child’s development of effective coping skills, leading to a greater likelihood of heightened biological reactivity to stress. This is likely to create a transactional process whereby increased and prolonged stress reactivity may result in more parenting challenges such as difficulties in calming a child once s/he becomes aroused, which may exacerbate a parent’s tendency to withdraw.

Stress reactivity is also proposed to enhance engagement with, and attention to, environmental factors, leading to greater susceptibility to both positive and negative contextual influences (e.g., Belsky, 1997, 2005; Boyce & Ellis, 2005; Del Giudice et al., 2010). Therefore, it is possible that only when biological and parenting risks are both present is the child’s likelihood of developing symptoms of psychopathology increased. This model suggests that reduced stress responsivity may actually serve a protective effect by “tuning out” the presence of adverse or insensitive parenting. However, it is alternatively possible that the interaction between proximal child behaviors and parenting styles are stronger predictors of psychopathological outcomes because these behaviors define the dyadic interactions that shape child development. Clearly, a greater understanding of how stress reactivity interacts with parenting style is needed.
Maternal intrusiveness—imposing one’s own agenda, ignoring the child’s needs, and expressing harsh feelings towards the child (NICHD Early Child Care Research Network, 1999)—provides an additional source of unpredictability and stress for children (Adam et al., 2007; Gunnar & Donzella, 2002; Teicher et al., 2003), and may be particularly detrimental for those with heightened HPA reactivity who are hypothesized to be especially sensitive to their context (Del Giudice et al., 2010; Hudson & Rapee, 2001; Rogosch & Cicchetti, 1994). As suggested above, children with heightened reactivity to stress may evoke increases in maternal intrusiveness during parent-child interactions as a result of parents’ efforts to control and alter the child’s emotionally labile responsivity. In turn, children may, over time, become hypervigilant to hostile cues, further exacerbating their tendency to overidentify threat, and ultimately increasing risk for overreacting in mild or ambiguous social interactions (Dodge, 1991). These reciprocal relationships will theoretically result in later externalizing problems with comorbid internalizing symptoms.

As suggested above, blunted HPA responsivity may buffer the effects of intrusive parenting, as children with reduced reactivity may be more likely to engage in avoidance conditioning and to circumvent interactions with parents once they initiate intrusive behaviors (Blair, 1995; Blair, Colledge, Murray, & Mitchell, 2001; McBurnett, Swanson, Pfiffner, & Tamm, 1997). This may result in protection against the development of anxiety symptoms. However, children with blunted HPA responsivity may be adversely affected by inconsistent parenting because of their purported deficiencies in socialization through punishment. For these children, high levels of positive reinforcement for appropriate behavior, rather than a reliance on punishment for inappropriate behavior, may be especially effective in decreasing risk for the development of later externalizing behaviors (Blair, 2004; Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Kochanska, 1997). Therefore, although adverse parenting is likely to interact with biological stress responsivity to enhance risk for psychopathology, it is proposed that the specific profile of biological vulnerability (heightened or blunted HPA activity) differentially interacts
with different types of adverse parenting, and that these combinations predict diverse patterns of comorbidity in the development of conduct problems.

*Stability and Change over Time.* Although parental warmth and sensitivity tend to be stable across early childhood, adverse parenting styles such as intrusiveness, detachment, and hostility are more likely to be inconsistent (Dallaire & Weinrab, 2005). Moreover, inconsistent parenting styles may be prevalent in children characterized by conduct problems without anxiety (Blair, 2004; Blair et al., 2006; Kochanska, 1997). While this has been empirically demonstrated in later childhood and adolescence with regard to parental coerciveness and hostility (e.g., review, Bugental, Johnson, New, & Sylvester, 1998; Patterson et al., 1992), there is a paucity of investigations on how change or stability in parenting styles in early childhood may predispose children to the development of conduct problems alone or in combination with internalizing symptoms. Determining whether changes or stability in adverse parenting styles across development put children more at risk for developing conduct problems with and without internalizing problems could be helpful in designing family-based interventions that target particular techniques to increase the likelihood of an immediate response or improvement from the child at an early age.

*Interrelationships of Risk Domains at 24 Months of Age.* Between 24 and 36 months of age, it becomes imperative to view the development of externalizing behaviors via an entirely transactional approach, whereby multiple and reciprocal exchanges between risk factors predict deleterious outcomes (Sameroff, 1990; Shaw, Keenan, & Vondra, 1994). The acquisition of increased mobility allows for greater opportunity to encounter potentially novel environments and stressors. Additionally, the child’s increasing ability to proactively influence his or her environment allows for more complex interactions with parenting styles. Thus, it follows that behavioral reactivity and regulation will moderate the reciprocal interactions between biological stress responsivity and parenting style to distinguish children’s development into CP, COMORBID, versus COMPARISON subgroups (see Figure 1).
Although it is clear theoretically that the interrelationship between these three domains of risk will have the most explanatory power for predicting the development of CP, COMORBID, versus COMPARISON subgroups at 5 years of age, the lack of empirical investigations make it difficult to determine a priori the exact nature of these interrelationships.

**Aims and Hypotheses**

The overarching objective of the current study is to determine how children’s biological stress responsivity, children’s behavioral reactivity and regulation, and parents’ parenting styles in early childhood differentially influence the development of CP, COMORBID, and COMPARISON subgroups at 5 years of age. To accomplish this objective, the following research aims will be addressed:

**Aim 1.** To perform an exploratory investigation regarding the stability or instability of each potential risk indicator—children’s biological stress responsivity, children's behavioral reactivity and regulation, and parenting styles—across infancy and toddlerhood separately by behaviorally-identified subgroups at 5 years of age.

In theoretical alignment with the reviewed literature, early childhood precursors of the COMORBID subgroups versus the CP and COMPARISON subgroups may include the following:

- consistent heightened biological stress responsivity (hyperactivity of HPA axis);
- stable and high levels of irritability;
- deficits in attention;
- high initial displays of social approach followed by decreased approach tendencies during toddlerhood as a result of increased negative parent-child interactions;
- stable exposure to high levels of intrusive parenting styles.

In contrast, early risk factors for the CP subgroup versus the COMORBID and COMPARISON subgroups are proposed to include the following:
- blunted stress responsivity (consistently blunted HPA activity or decreases in HPA activity over time based on the attenuation hypothesis; Susman, 2006);
- deficits in sustained attention;
- stable and consistently high social approach tendencies;
- inconsistent exposure to intrusive parenting and lack of maternal warmth/sensitivity.

In general, however, it was hypothesized interactions between multiple risk domains at key time points in infancy and toddlerhood, as opposed to stability or change in any one domain of risk, will have the most explanatory power in the development of these distinct subgroups.

**Aim 2.** To determine how the interplay among children’s biological stress responsivity, children’s behavioral reactivity and regulation, and parenting styles at key time points (7, 15, and 24 months of age) differentially predicts the development of CP, COMORBID, and COMPARISON subgroups at 5 years of age.

In accordance with the reviewed theoretical constructs regarding biological-behavioral-contextual interrelationships (Boyce & Ellis, 2005; Bronfenbrenner & Morris, 1998; Del Giudice et al., 2010; Gottlieb, 1992), it was hypothesized that the reciprocal interactions among children’s biological stress responsivity, children’s behavioral reactivity and regulation, and parenting styles will become more intertwined at 15 and 24 months of age relative to earlier time points. In fact, it is proposed that by toddlerhood (24 months of age) only these complex interactions among all three domains will significantly and differentially predict CP, COMORBID, and COMPARISON subgroups at 5 years of age.

In summary, the major differences expected among children’s biological stress responsivity, children’s behavioral reactivity and regulation, and parenting styles at 7, 15, and 24 months of age for the three subgroups are as follows:

**Aim 2a. 7 Months of Age.** Based on numerous empirical findings (review, Hodgins et al., 2009), children’s exposure to adverse parenting styles may foster and/or exacerbate a pattern of heightened
HPA activity in early infancy (e.g., Boyce & Ellis, 2005; Gunnar & Donzella, 2002), and may ultimately be a precursor for membership in the later COMORBID (Hudson & Rapee, 2001) and CP (Del Giudice et al., 2010; Susman, 2006) versus the COMPARISON subgroup. Moreover, in accordance with prior empirical work (Boivin et al., 1994; Eisenberg et al., 2001, 2009; Farmer et al., 2002; Milich & Dodge, 1984), the reciprocal interaction between behavioral negative reactivity, such as irritability, and adverse parenting style may also contribute to the development of later conduct problem subgroups at 5 years of age.

Examination of all other interrelationships of children’s biological stress responsivity, children’s behavioral reactivity and regulation, and parenting style indices at 7 months of age and their relation to development of the three subgroups will be exploratory in nature due to the lack of extant empirical research. Due to limited mobility and the incomplete maturation of attention skills in early infancy, behavioral indices of approach tendencies and sustained attention would be unlikely to be significantly correlated with the differential development of the three groups; however, exploratory analyses of approach tendencies as well as sustained attention behaviors and their relation to CP, COMORBID, and COMPARISON subgroups were conducted.

Aim 2b. 15 Months of Age. In line with theoretical premises and empirical research indicating that children learn adaptive behavioral reactivity and regulation skills through interaction with their environment (Gottlieb, 1992; Rothbart et al., 2007), by 15 months of age affective behavioral indices will be involved in almost all of the significant interrelationships that predict membership in CP, COMORBID, and COMPARISON subgroups at 5 years of age.

In general, it is expected that demonstrations of high irritability and/or overall negative reactivity from children may continue to evoke a lack of maternal warmth and sensitivity as well as maternal intrusiveness (e.g., Rutter et al., 1997), which may in turn reciprocally solidify the patterns of behavioral irritability and overall negative reactivity in children. It is clear that such interactions would be significantly more likely to aid in the development of CP and COMORBID subgroups. Additionally,
since children with COMORBID symptoms have a higher propensity for negative behavioral interactions with family and peers (Boivin, Poulin, & Vitaro, 1994; Farmer et al., 2002; Milich & Landau, 1984), it is postulated that children’s high levels of irritability paired with adverse parenting styles at 15 months of age would be more prevalent with COMORBID versus CP subgroups.

Regarding attention regulation, maternal warmth and responsivity have been shown to be associated with emotional and social regulation competencies such as attention (Campbell & von Stauffenberg, 2008; Greenberg, Kusche, & Speltz, 1991), whereas a lack of maternal warmth as well as maternal intrusiveness may result in difficulties in these self-regulation skills. Therefore, while high levels of maternal sensitivity along with high levels of sustained attention would be likely precursors to COMPARISON subgroup membership, children exposed to adverse parenting styles would exhibit greater deficits in attention and be more likely to develop membership in the CP and COMORBID subgroups. Exploratory analyses will be performed to determine if the sustained attention and parenting associations at 15 months of age differentially predict CP versus COMORBID subgroups.

Furthermore, based on theoretical premises (Eysenck, 1977; Raine, 2002) and empirical research (Stifter et al., 2008) discussed earlier, children at risk for CP and COMORBID subgroup membership tend to have high approach tendencies. It may be that the “loose coupling” of high approach tendencies with biological stress responsivity in infancy differentially predicts the development of these distinct subgroups. Specifically, it is proposed that blunted HPA activity in conjunction with high approach tendencies at 15 months of age may confer risk for becoming part of the CP subgroup in the early school years, while heightened HPA activity along with high approach tendencies may be more likely to be significant precursors of the COMORBID subgroup.

Lastly, it should also be noted that the relationship between high maternal intrusiveness and heightened HPA activity may be pervasive across infancy and toddlerhood (e.g., Boyce & Ellis, 2005; Del
Giudice et al., 2010). This relationship at 15 months of age is also expected to be a precursor for later COMORBID as opposed to CP and COMPARISON subgroups.

Aim 3c. 24 Months of Age. In toddlerhood, only interrelationships among all three factors—children’s biological stress responsivity, children's behavioral reactivity and regulation, and parenting styles — are expected to differentially predict the development of CP, COMORBID, versus COMPARISON subgroups at 5 years of age. Due to the lack of investigations into how both biological and behavioral indices moderate parenting styles, most interrelationships that are tested are exploratory. Overall, it is proposed that blunted HPA activity along with high approach tendencies or deficits in attention in toddlerhood would predict CP versus the other subgroups in contexts with less exposure to maternal warmth and sensitivity. Alternatively, heightened HPA activity along with high irritability, deficits in attention, or high approach tendencies in toddlerhood may predict later COMORBID subgroup membership versus membership in the other subgroups when exposed to greater levels of maternal intrusiveness.
Chapter 2

Methods

Study Design and Participants

The Family Life Project. Participants for the current study were drawn from the Family Life Project, an ongoing longitudinal investigation of child development and family functioning in predominately low-income, nonmetropolitan communities in Pennsylvania and North Carolina.

Families were recruited from local hospitals shortly after the birth of a child, over-sampling for low-income and African American families. For detailed information regarding the sampling plan and recruitment procedures in the Family Life Project (see Vernon-Feagans et al., 2008).

Of the 1,292 families who participated in the first wave of data collection when the child was 2-months of age, 1,204 (93%) families participated when the child was approximately 7 months of age, 1,169 (90%) at 15 months of age, and 1144 (89%) at 24 months of age, and 1081 (83%) at 5 years of age. Approximately half of the children were female (47%), and 42% were identified as black (58% identified as white). The majority of families were less than 200% of the poverty line at the 7-month visit.

Due to cancellations and difficulties in scheduling family assessments during the particular age range of interest, the analyses were restricted to the following ages at each assessment: 1) between 5 and 9 months of age at the early infancy assessment ($M_{age}=7.31$ and $SD=1.00$ at 7-month visit); 2) between 13.5 and 19 months of age at the late infancy assessment ($M_{age}=15.47$ and $SD=.90$ at 15-month visit); and 3) between 23 and 31 months of age at the toddler assessment ($M_{age}=24.70$ and $SD=1.47$ at 24-month visit). This resulted in the exclusion of 137 participants at the 7-month visit, 49 participants at the 15-month visit, and 63 participants from the 24-month visit. At the 5 years visit age ranged from 57 to 75 months old ($M_{age}=60.50$ and $SD=3.21$).

From this larger sample of FLP participants, a subset of children were identified and selected for the present study who met criteria for the behaviorally-based subtypes of interest. Specifically, children
the current analysis included subgroups of children who exhibited high levels of symptoms in conduct problems \( (n=72, 7.0\% \text{ of the restricted sample}) \), both conduct problems and anxiety \( (n=64, 6.1\% \text{ of the restricted sample}) \), or low levels of difficulties on both conduct problems and anxiety \( (n=370, 35.5\% \text{ of the restricted sample}) \) at 5 years of age. Unlike the larger epidemiological population of diverse, impoverished, and non-metropolitan families, this subset of children \( (\text{total } N=506) \) only represents the clinically-significant subset of children with conduct problems and comorbid anxiety as well as the extremely low-risk subset of children concerning these problems.

**Identification of Conduct Problem Subgroups.** Within the current sample, subgroups with behaviorally-based differences in conduct problems were identified at 5 years of age. These subgroups were formed based on findings from epidemiological investigation of mental health outcomes in over ten thousand children \( (4 \text{ to } 17 \text{ year olds}) \) in the U.S., which utilized the parent-reported data from the Strengths and Difficulties Questionnaire \( (\text{SDQ}; \text{Bourdon, Goodman, Rae, Simpson, & Koretz, 2005}) \).

In particular, the **Conduct Problem** and **Emotional Symptoms** (assessment of internalizing problems) subscales from parent-reported SDQ were utilized to form the behaviorally-based conduct problem subgroups. Previously, the SDQ with multiple informants—parents, teachers, and older children—successfully identified 5 to 15 year olds with a psychiatric diagnosis based on criteria from the Diagnostic and Statistical Manual of Mental Disorders \( \text{(DSM IV)} \) in 70% of the individuals with conduct problems as well as internalizing disorders including generalized anxiety and depression \( (\text{Goodman, Ford, Simmons, Gatward, & Meltzer, 2000}) \). Out of over 8000 participants, the SDQ identified childhood psychopathology with both a high specificity \( (94.6\%; \text{ range } 94.1-95.1\%) \) and moderate to high sensitivity \( (63.3\%, \text{ range } 59.7-66.9\%) \). Generally, both parent- and teacher-reported problems on the SDQ were equally successful at diagnosing psychopathology, although the use of both informants predicted the most specificity and sensitivity. \( (\text{Details of Conduct Problem and Emotional Symptoms subscales are described in the Measures section.}) \)
A slightly more conservative subscale range than reported by Bourdon and colleagues (2005) were used to identify the subgroups at the 5 year assessment. The range of each of the subscales is from 0 (no symptoms) to 10 (maximum number of symptoms). In the epidemiological population, 10% of the children had scores from 4 to 10 on the conduct problems subscale, while 8% of the children had scores of 5 to 10 on the internalizing subscale. In the current study, the subgroups were comprised accordingly:

a) **COMPARISON** subgroup was defined as children who ranged from 0 to 1 on the conduct problem subscale and 0 to 2 on the emotional symptom subscale;

b) **CP** subgroup was comprised of children who ranged from 4 to 10 conduct problem subscale and 0 to 2 on the emotional symptom subscale; and

c) **COMORBID** subgroup was defined as children who ranged from 4 to 10 on the conduct problem subscale and 5 to 10 on the emotional problems subscale.

Table 1 contains the demographic information of the sample by subgroup. In general, the CP subgroup members were more likely to be males. Alternatively, the COMORBID subgroup members were more likely to be black and to be low income. Both CP and COMORBID subgroups had children whose mothers were single-status and had significantly lower educational achievement than the COMPARISON subgroup.
Table 1 Percentages and Means (Standard Deviations) of Demographic Variables for Conduct Problem Subgroups.

<table>
<thead>
<tr>
<th>Demographics</th>
<th>COMPARISON (n=370)</th>
<th>CP (n=72)</th>
<th>COMORBID (n=64)</th>
<th>p-value&lt;sup&gt;1&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>47.84%&lt;sub&gt;f&lt;/sub&gt;</td>
<td>62.50%</td>
<td>54.69%&lt;sub&gt;f&lt;/sub&gt;</td>
<td>†</td>
</tr>
<tr>
<td>Race (Black)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>34.59%&lt;sub&gt;f&lt;/sub&gt;</td>
<td>41.67%&lt;sub&gt;f&lt;/sub&gt;</td>
<td>57.81%</td>
<td>**</td>
</tr>
<tr>
<td>Living in NC&lt;sup&gt;b&lt;/sup&gt;</td>
<td>55.68%</td>
<td>54.17%</td>
<td>65.63%</td>
<td>***</td>
</tr>
<tr>
<td>Low-Income&lt;sup&gt;c&lt;/sup&gt;</td>
<td>52.26%</td>
<td>66.67%</td>
<td>78.13%</td>
<td>***</td>
</tr>
<tr>
<td>Single-Parent Status</td>
<td>41.74%</td>
<td>62.50%&lt;sub&gt;f&lt;/sub&gt;</td>
<td>64.06%&lt;sub&gt;f&lt;/sub&gt;</td>
<td>***</td>
</tr>
<tr>
<td>Maternal Education Achievement&lt;sup&gt;d&lt;/sup&gt;</td>
<td>15.43 (2.49)</td>
<td>13.99 (2.73)&lt;sub&gt;f&lt;/sub&gt;</td>
<td>13.23 (2.95)&lt;sub&gt;f&lt;/sub&gt;</td>
<td>***</td>
</tr>
</tbody>
</table>

Notes: <sup>a</sup> White=0 and Black=1; <sup>b</sup> NC=1 and PA=0; <sup>c</sup> Income-to-Needs of 200% below government-defined poverty level; <sup>d</sup> Years of school completed. <sup>1</sup>p-value based on chi-square tests or (for maternal education achievement) ANOVA to determine independence between each demographic variable and subgroups. Percentages and means (SD) in the same row that share “f” subscripts do not differ at p < .05 according to chi-square test of independence pair-wise comparisons or (for maternal education achievement) post-hoc Tukey analyses.<br><sup>†</sup> p<.08, <sup>*</sup>p<.05, <sup>**</sup>p<.01, <sup>***</sup>p<.001.

Procedures

Families were visited in their home by trained research assistants when the target child was approximately 7, 15, and 24 months of age in order to conduct in-home interviews, and to collect observational and biological assessments of the target child. Mothers completed questionnaires via laptop computer and reported demographic information on all household members. At 5 years old, parent-reported questionnaires on child mental health were collected.

At 7-, 15-, and 24-month assessments, children’s salivary cortisol samples were collected around a series of emotionally-eliciting challenge tasks that were developmentally salient. Additionally, home visitors independently completed post-visit ratings of the child’s behavior over the course of the entire visit in early childhood. Finally, parent-child interactions were video-taped during a set of free play activities, and later coded for parenting styles of maternal sensitivity and intrusiveness.

Challenge Tasks. At each visit, children participated in a series of developmentally appropriate challenging tasks designed to elicit emotional reactivity and self-regulatory behavior, which were
videotaped for later coding (e.g., Buss & Goldsmith, 1998; Kochanska, Tjebkes, & Forman, 1998; Stifter & Braungart, 1995). At the 7-month visit, infants were presented with series of three tasks while seated in a walker: a mask presentation, followed by a barrier challenge, and finally an arm restraint procedure. For the mask presentation, mothers were seated beside their child while the child was presented with a succession of four unusual masks for 10-second intervals. While wearing each of the masks, the research assistant leaned towards the seated child, turned their head from side to side, and repeated the child’s name. Mothers were asked not to interfere or distract their child, but to respond as they normally would if their child looked to them. For the barrier challenge, mothers were asked to step outside of the infant’s line of sight, but to remain within hearing distance of their child. Infants were given an attractive toy to play with for 30 seconds, which was then removed just beyond the child’s reach by the research assistant and placed behind a clear, plastic barrier for 30 seconds. The toy was returned to the child, and the procedure was repeated twice for a total of three cycles. For the arm restraint procedure, the mother remained outside of the child’s line of sight, and a research assistant crouched behind the infant and gently restrained the child’s arms for 2 minutes.

At the 15-month and 24-month visit, children were presented with two tasks while seated in a booster seat: a toy-removal procedure (replacing the barrier task), followed by the mask presentation. For the 15-month toy removal task, the mother was given an attractive toy, and asked to play with the child and the toy for one minute. The mother was then instructed to remove the toy and place it outside of the child’s reach, at which point the home visitor and the mother engaged in conversation while the child remained in the booster seat. After two minutes, the mother returned the toy to the child, but did not play or engage the child with the toy in any way. For the 24-month toy-removal a similar procedure was followed. After playing together with the toy for one minute, the mother was asked to remove the toy and place it inside a clear container with the lid screwed on tightly. The container was then given to child while the mother and research assistant conversed for two minutes. The mother then returned the
toy to the child and soothed or engaged the child in play for one minute. The mask presentation followed the same protocol as in the infancy assessment. For all of the tasks at each time point, mothers were informed that they could stop the procedure at any time, and the research assistant terminated the tasks if the child engaged in 20-seconds of hard crying.

Salivary Cortisol Collection. To assess overall levels of HPA activity as well as cortisol reactivity in response to the emotionally arousing challenge tasks, three saliva samples were collected from children: 1) a baseline sample collected prior to the challenge tasks, 2) a sample collected 20-minutes after the child’s peak arousal to the tasks, and 3) a sample collected 40-minutes after the child’s peak arousal. Peak arousal was clearly outlined for home visitors in the protocol, with a substantial portion of the children reaching peak arousal during the final task in the series at each time point. Saliva samples were collected using cotton or hydrocellulose absorbent material and expressed into cryogenic storage vials (cotton) or by centrifugation upon arrival at the laboratory (hydrocellulose) (Granger et al., 2007). Prior studies have found no differences in cortisol levels depending on the use of cotton or hydrocellulose collection techniques (Granger, et al., 2007; Harmon, Granger, Hibel, & Rumyantseva, 2007). After collection, samples were placed on ice, temporarily stored in a -20°C freezer, shipped overnight in batches to the Behavioral Endocrinology Laboratory at the Pennsylvania State University, and then stored in -80°C freezers until they were assayed.

All samples were assayed in duplicate for cortisol using a highly sensitive enzyme immunoassay (510k) designed to measure adrenal function from saliva samples (Salimetrics). The tests utilized 25 µL of saliva, had average intra- and inter-assay coefficients of variation less than 10 and 15%, and had a lower limit of sensitivity of .007 µg/dL (range of sensitivity from .007 to 3.0 µg/dL).

Measures

Conduct Problem Subgroups. Children’s behavioral outcomes of conduct problems and anxiety were assessed at the 5 year assessment through parent ratings on the Strengths and Difficulties
Questionnaire (SDQ; Goodman, 1997). The current proposal utilizes items from the conduct problems and emotional problems subscales. Items are rated on a 3-pt Likert scale from ‘not true’=0 to ‘certainly true’=2, with 5-items per subscale. The items on the Conduct Problems subscale included “often has temper tantrums or hot tempers,” “generally obedient, usually does what adults request,” “often fights with other children or bullies them,” “often lies or cheats,” and “steals from home, school, or elsewhere.” The items on the Emotional Symptoms subscale included “often complains of headaches, stomachache, or sickness,” “many worries, often seems worried,” “often unhappy, down-hearted, or tearful,” “nervous or clingy in new situations, easily loses confidence,” and “many fears, easily scared.” The internal consistencies are adequate for the Conduct Problem subscale (α ranges from .46 to .76) and Emotional Symptoms subscale (α ranges from .60 to .76; review, Stone, Otten, Engels, Vermulst, & Janssens, 2010). Mean test-retest reliability correlations across studies are .66 for both subscales. On average, the concurrent validity with the Child Behavior Checklists (CBCL) revealed weighted correlations of .71 Conduct Problem subscale and Emotional Symptoms subscale .64.

These two subscales were used to determine the CP, COMORBID, and COMPARISON subgroups (see previous Identification of Conduct Problem Subgroups subsection for details).

Biological Stress Responsivity. Pre-task baselines are believed to represent “trait”-like or acute anticipatory stress patterns of HPA activity; therefore, pre-task cortisol levels (ug/dL) were assessed at 7-, 15-, and 24-month visits as separate indicators of stress than cortisol reactivity. To assess children’s cortisol reactivity to the challenge tasks, 20-minute post-task cortisol values were subtracted from the pre-task cortisol values. The level of pre-task cortisol involves a negative association between the initial pre-task level and the magnitude of change (e.g., higher initial value of cortisol restricts the range of reactivity to be a smaller change), this phenomenon is called the Law of Initial Value (LIV; Lewis & Ramsay, 1999; Lewis & Thomas, 1990). Adding pre-task cortisol levels to each of the analyses accounted
for the variance due to this initial value when determining whether cortisol reactivity is a significant predictor of the developmental outcomes of interest.

**Behavioral Reactivity and Regulation.** At each of the assessments in infancy and toddlerhood, behavioral reactivity and regulation that may be associated with later conduct problem outcomes were assessed via observation by home visitors. Home visitors independently rated the child’s behavior using an adaptation of the Infant Behavior Record (IBR; Bayley, 1969). In the present study, the IBR was applied globally to behavior observed across the entire home visit (see Stifter & Corey, 2001). Four IBR items/scales of interest in the present study were overall reactivity, irritability, sustained attention, and approach tendencies. Overall reactivity is comprised of one item assessing sensitivity and excitability to stimuli in general, which is scored from 1='Unreactive; responds only to strong or repeated stimulation' to 9='Very reactive; startles, reacts quickly, seems keenly sensitive to things going on around him/her.' Irritability is also comprised of one 9-point item (1='No irritability; infant passively responds to all stimulation' to 9='Irritable to all degrees encountered throughout the home visit'). Sustained attention is a superordinate scale comprised of the mean across three 9-point items assessing tendency towards persistent attention, behavioral constancy on tasks of difficult levels, and level of sustained interests in objects/test materials. Approach tendencies forms a superordinate scale comprised of a mean across 3 items. The first 9-point item asked about the responsiveness to persons (1='Behavior towards persons is not different from behavior towards objects' to 9='Behaviors seems to be continuously affected by awareness of persons present'). Next were two items included 5-point Likert scales on the responsiveness to the examiner and to their mother/caregiver (1='Avoiding or withdrawn ' to 5 = 'Inviting (initiating, demanding)'). The mean of the home visitors’ ratings were used; cross-rater correlations ranged from .56 to .59.
Parenting Styles. Mothers were given four standard, age-appropriate toys, and instructed to play with their infants as they normally would for 10 minutes while seated on a baby blanket on the floor. Interactions were later coded to assess levels of intrusive behavior or warm engagement.

Coders were trained to reliably assess maternal interaction behaviors during this parent-child free-play episode (Cox, Paley, Burchinal, & Payne, 1999), and on a random selection of approximately 30% of the total sample to prevent coder drift. Maternal sensitivity, detachment, intrusiveness, positive regard, negative regard, developmental stimulation, and animation were globally coded on a 1 to 5 scale, ranging from **not at all characteristic** to **highly characteristic**. Based on the results of factor analyses conducted with an oblique rotation (i.e., Promax), maternal sensitivity (ICC=.87) was defined as the mean of mother’s scores on four characteristics: detachment (reversed; degree of emotional disengagement), positive regard (expressed level of positive feelings towards the child), animation (energy level), and developmental stimulation (scaffolding activities with the child appropriately).

Maternal intrusiveness/negativity (ICC=.80) was defined as the mean of mothers’ scores for three characteristics: sensitivity (reversed; level of responsiveness to the needs, gestures, and expressions of the child), intrusiveness (amount the mother imposed her own agenda on the interaction, and ignored the baby’s needs), and negative regard (degree of harsh, negative feelings expressed toward child). The composite scores for maternal sensitivity and intrusiveness were negatively correlated (r=.35; p < .01). Reliability was assessed on a random subset of the sample (~30% of the recordings), each videotape was double-coded (κ range=.62-.70).

Covariates. There are a wide range of sociocultural risk factors that have been linked to externalizing behaviors throughout childhood, including poverty (e.g., McLoyd, 1990) and adverse family characteristics (e.g., single-mother home; Achenbach, Howell, Quay, & Conners, 1991), regardless of phenotype. Poverty and family structure are among the most pervasive sociocultural risk factors regarding the onset and development of conduct problems (e.g., Capaldi, DeGarmo, Patterson, &
Forgatch, 2002; Mcloyd, 1990). Therefore, factors associated with these sociocultural risk factors were included in the study as covariates.

Mothers reported on demographic information on all household members, including age, gender, ethnicity/race, marital status, and income from all sources. In order to calculate the income-to-needs ratio of the family, the total household income from all sources was divided by the federal poverty threshold for that year, adjusted for the number and types of individuals in the household. An income-to-need ratio of 1.00 indicates the family income is at the poverty line.

Due to the variable time in which saliva samples were collected and the distinct diurnal rhythm that occurs throughout the day (Blair et al., 2008), time of day for sample collection was entered as a covariate in all of the analyses. In addition, since the use of medications including acetaminophen by infants has also been shown to account for variance in cortisol levels and reactivity (Hibel, Granger, Kivlighan, Blair, & the FLP Investigators, 2006), medication use was also tested.

Overview of the Analytical Plan

The analytical plan for the present study is as follows: First, the conduct problem subgroups (CP, COMORBID, and COMPARISON subgroups) were identified. Then descriptive statics were explored for all demographic, biological stress responsivity, behavioral reactivity and regulation, and parenting style variables of interest by the three conduct problem subgroups. Chi-square tests were performed to assess subgroup differences in all categorical (non-continuous) demographic variables; while, analysis of variances (ANOVAs) were used to assess subgroup difference in continuous variable. Followed up post-hoc chi-square test of pairwise comparisons and Tukey tests were performed when significant omnibus subgroup differences were found. ANOVAs—controlling for demographic risk factors—assessed subgroup differences in biological stress responsivity, behavioral reactivity and regulation, and parenting style variables at each developmental time point (7-, 15-, and 24-months). Post-hoc Tukey analyses were used to identify mean differences in these indices of interest across subgroups.
Second, linear mixed model (LMM) were run for each factor of interest—HPA activity, affective behaviors of reactivity and regulation, as well as parenting style—using PROC MIXED (SAS Institute 1990) to identify mean changes (or stability) in these variables across early childhood (7, 15, and 24 months of age) within each subgroup. This statistical technique was conducted instead of repeated measures ANOVA because it uses maximum likelihood instead of listwise deletion when handling missing data (Collins, Schafer, & Kam, 2001). Additionally, LMMs biological stress responsivity, behavioral reactivity and regulation, and parenting style accommodate static and dynamic covariates of change as well as more accurately model random effects of variability within individual than repeated measure ANOVAs.

Lastly, three separate multinomial logistic regressions (MLRs) utilizing imputed datasets (discussed below) assessed how biological stress responsivity, behavioral reactivity and regulation, and parenting styles at each developmental time point (7, 15, or 24 months of age) significantly influences differences in the development of conduct problem subgroups. MLRs were performed because this regression technique can estimate the relation between a nominal dependent variable with multiple groups (the three subgroups) and independent variables that are categorical as well as continuous (Demaris, 1992; Hosmer & Lemeshow, 1989). In MLRs, the Wald chi-square statistic tests the unique contribution of each independent variable over and above other predictors in the model. Additionally, the odds ratio represents a probability ratio with respect to the reference group, whereby the closer the odds ratio is to 1.00, the lower the likelihood that the covariate, independent variable, and/or interaction can predict subgroup membership.

Significant demographic variables were entered into the logistic regression model as covariates in all of the MLRs. Independent variables included pretask cortisol levels, cortisol reactivity to emotional tasks, approach tendencies, sustained attention behaviors, overall behavioral reactivity, behaviors of irritability, sensitive parenting, as well as intrusive parenting. Interactions hypothesized a priori as well as exploratory interactions among independent variables were entered separately into the multinomial
logistic regression; these were only retained in the model if they explained a significant amount of the variance in the dependent conduct problem subgroups.

**Multiple Imputations of Missing Data.** To account for missing data and avoid estimation bias using listwise deletion when running the multiple logistic regressions, data on the subset of FLP participants included in the present study was multiply imputed using PROC MI command in SAS version 9.1. In accordance with the multiple imputation approach (Rubin, 1987; Schafer, 1997; Schafer & Graham, 2002), 30 imputed datasets were estimated with the assumption that data was primarily missing at random. The variables utilized for the imputation included all demographic information and variables contained in each of the models at all time points (7-month, 15-month, 24-month, and 5 year assessments). Data from measures that were not included in the models but would theoretically aid in the accurate estimating these 30 datasets (auxiliary variables) were also used in the imputation process. Measures of these auxiliary variables included: Infant Behavior Questionnaire (IBQ) at the 7-month assessment, micro-coded behaviors during the stress task at all assessments, as well as the ADHD Questionnaire (Report of DSM-IV ADHD Symptoms) and Child Behavior Questionnaire at the 5 year assessment.

The development of appropriate Wald $\chi^2$ statistic that summarize multinomial logistic regression estimates across multiple imputations has yet to be created; thus, ranges across the 30 imputed data sets were reported. The statistical significance given is based on the mean Wald $\chi^2$. PROC MIANALYZE (SAS Institute, 2002) was used to summarize the parameter and covariance estimates as well as calculate the appropriate standard error estimates across the 30 imputed datasets for each of the multinomial logistic regression models. The specific contrasts between subgroups do not list a Wald $\chi^2$ statistic but do provide the betas, standard errors, and significance values to assess post-hoc differences between subgroups. Parameters and covariance estimates were used to calculate the odds ratios and confidence intervals for each of the contrasts.
Chapter 3

Results

Descriptive Statistics

The percentages or means (standard deviations) for gender, race, income-to-needs ratio, marital status, and maternal educations are shown in Table 1 by behaviorally-derived conduct problem subgroups at 5 years of age. Chi-square tests revealed significant differences in race ($\chi^2(2, N=506)=12.77, p=.002$), low-income ($\chi^2(4, N=506)=24.47, p<.001$), marital status($\chi^2( 2, N=506)=19.14 , p<.001$), and maternal education achievement ($F(2, 506)=25.54, p<.001$) among the subgroups. Post-hoc chi-square independence tests and Tukey analyses showed that both CP and COMORBID subgroups were comprised of a significantly higher proportion of children with demographic risk than the COMPARISON subgroups. Specifically, COMORBID and CP subgroups had higher proportion of low income-to-needs (65.63% and 54.17%, respectively) and single-parent households (64.06% and 62.50%, respectively), as well as mothers with lower educational achievement versus the COMPARISON subgroup (52.26% were low-income and 41.74% were from single-parent families). In addition, the COMORBID subgroup included a greater proportion of Black children than CP or COMPARISON subgroups; while, the CP subgroup revealed a trend toward a significantly higher proportion of males compared to the COMPARISON subgroup.

All demographic variables that demonstrated significant differences between the subgroups were included in the subsequent analyses: gender, race, income-to-needs ratio, marital status, and maternal education level. Other potential covariates were tested in each of the models—time of saliva collection, teen mother status, state of residence, and acetaminophen use in early childhood. These potential covariates were tested in each of the models in the present analyses. However, because they did not explain a significant amount of the variance predicting the subgroups of interest, they were removed from all analyses.
Table 2 depicts the means and standard deviations for biological stress responsivity, behavioral reactivity and regulation, and parenting styles by developmental time point (7, 15, and 24 months of age) for each conduct problem subgroup at 5 years of age. One-way ANOVAs—controlling for race, gender, maternal education achievement, marital status and income-to-needs ratio—revealed significant mean differences in pretask cortisol levels ($F(2, 506)=3.60, p=.03$), cortisol reactivity ($F(2, 506)=3.45, p=.03$), and sensitive parenting style ($F(2, 506)=5.72, p=.01$) among the three groups.

Post-hoc Tukey analyses revealed that CP subgroup had higher levels of pretask cortisol ($M=.28$, $SD=.44$) at 7 months of age compared to both the COMPARISON ($M=.21$, $SD=.27$) and the COMORBID ($M=.19$, $SD=.21$) subgroups. Additionally, the CP subgroups during the 7-month assessment showed significant decreases in cortisol (blunted HPA activity) during the stress exposure ($M=-.05$, $SD=.44$); in contrast, the COMPARISON subgroup showed significantly greater increases in cortisol (heightened HPA activity) during the emotion-eliciting task ($M=.06$, $SD=.35$).

In terms of behavioral reactivity and regulation, contrary to the current studies prediction, the COMPARISON subgroup demonstrated a trend toward higher observed overall reactivity ($M=6.12$, $SD=.85$) than the COMORBID subgroup ($M=5.82$, $SD=1.23$) at toddlerhood (24-month assessment).

Finally, across all of early childhood, the COMPARISON subgroup was exposed to significantly greater amounts of observed maternal sensitivity ($M=3.05$, $SD=.76$ at 7 months old; $M=3.00$, $SD=.76$ at 15 months old; $M=3.11$, $SD=.83$ at 24 months old) than children with CP ($M=2.68$, $SD=.91$ at 7 months old; $M=2.61$, $SD=.70$ at 15 months old; $M=2.62$, $SD=.87$ at 24 months old) and COMORBID ($M=2.57$, $SD=.83$ at 7 months old; $M=2.37$, $SD=.95$ at 15 months old; $M=2.53$, $SD=.85$ at 24 months old).

In sum, a greater proportion of kindergarteners in both CP and COMORBID subtypes were exposed to more demographic risk factors (poverty, divorced and single-parent households, and low maternal education) than the COMPARISON subgroup during infancy and toddlerhood. Additionally, the CP subgroup had the greatest proportion of males; whereas, the COMORBID subgroup had the highest
proportions of Black children. Significant biological differences in stress level and stress responsivity between the subgroups were apparent in early infancy, wherein the CP subgroup (on average) has the significantly higher pretask cortisol levels and blunted cortisol reactivity in early infancy in comparison to COMORBID subgroup and the COMPARISON subgroups. Significant mean differences in behaviors did not occur until toddlerhood, revealing that the COMPARISON subgroup demonstrated greater overall behavioral reactivity. Alternatively, (on average) both CP and COMORBID subtypes were exposed to lower levels of maternal sensitivity than the COMPARISON subgroup.
Table 2. Means (SD) Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles across Early Childhood by Conduct Problem Subgroups.

<table>
<thead>
<tr>
<th></th>
<th>COMPARISON Subgroup (n=370)</th>
<th>CP Subgroup (n=72)</th>
<th>COMORBID Subgroup (n=64)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7 mos old</td>
<td>15 mos old</td>
<td>24 mos old</td>
</tr>
<tr>
<td><strong>Biological Stress Responsivity</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretask Cortisol Levels (ug/dL)</td>
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<td>.24</td>
<td>.19</td>
</tr>
<tr>
<td></td>
<td>(.27)</td>
<td>(.46)</td>
<td>(.28)</td>
</tr>
<tr>
<td>Cortisol Reactivity across Emotion Task</td>
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<td>.03</td>
<td>.02</td>
</tr>
<tr>
<td></td>
<td>(.35)</td>
<td>(.37)</td>
<td>(.21)</td>
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<tr>
<td><strong>Behavioral Reactivity and Regulation</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Approach Tendencies</td>
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<td>13.94</td>
<td>13.95</td>
</tr>
<tr>
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<td>(1.56)</td>
<td>(1.77)</td>
<td>(1.99)</td>
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<td>18.01</td>
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<tr>
<td></td>
<td>(.85)</td>
<td>(.95)</td>
<td>(.85)</td>
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<tr>
<td>Irritability</td>
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<td>3.28</td>
<td>3.00</td>
</tr>
<tr>
<td></td>
<td>(1.01)</td>
<td>(0.91)</td>
<td>(1.24)</td>
</tr>
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<td><strong>Parenting Styles</strong></td>
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<td></td>
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<td>Sensitive/Responsive</td>
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<td>3.00***</td>
<td>3.11***</td>
</tr>
<tr>
<td></td>
<td>(.76)</td>
<td>(.76)</td>
<td>(.83)</td>
</tr>
<tr>
<td>Intrusive/Negative</td>
<td>2.33</td>
<td>2.15*</td>
<td>2.25</td>
</tr>
<tr>
<td></td>
<td>(.72)</td>
<td>(.63)</td>
<td>(.82)</td>
</tr>
</tbody>
</table>

Notes: *Cortisol Reactivity=(20 min posttask cortisol levels – pretask cortisol levels); †Home visitor reported on children’s observed behaviors across the entire visit; ‡Micro-behavioral coding of parent-child interactions during play tasks. *p-values are based on analyses of variance (ANOVAs) and indicate significant omnibus mean differences among subgroups for each variable of interest within each developmental time point. Means (SD) in the same row and developmental time point (e.g., “7 mos old”) column that share subscripts do NOT differ at \( p < .05 \) according to post-hoc Tukey analyses.

\( ^{p<.08} \), \( *p<.05 \), \( **p<.01 \), \( ***p<.001 \)
Aim 1. *Descriptions of Stability and Change of Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles across Early Childhood with Conduct Problem Subgroups.*

A 3 (developmental time point) X 3 (conduct problem subgroups) linear mixed models (LMM) was run for each risk factor of interest—biological stress responsivity, behavioral reactivity and regulation, and parenting styles—to determine mean changes within the behaviorally-derived conduct problem subgroup across early childhood (7, 15, and 24 months of age). These LMMs controlled for race, gender, maternal education level, income-to-needs ratio, and marital status. Each model was tested with an unstructured, autoregressive, and toeplitz covariance structure. Based on the AIC and BIC goodness of fit statistics, all models were best fit using an unstructured covariance matrix.

Only indices of irritability (AIC=4312.06, BIC=4400.23) and intrusive parenting (AIC=2753.69, BIC=2841.85) demonstrated mean changes within the conduct problem subgroup across early childhood (7, 15, and 24 months of age). The LLM for with behaviors of irritability as the dependent factor revealed a trend towards a significant fixed effect for the interactions between subgroup and developmental time point ($F(2, 584)=2.21, p=.06$). Simple effect comparisons were performed to determine which contrasts were significant within each subgroup, and least-squared means (LS-Means)—adjusted for covariance model effects—were plotted (see Figure 1). In the COMPARISON subgroup, children’s displays of irritability behaviors were significantly greater at 15 months of age versus 7 months of age ($\beta=.21, SE=.07, t(490)=3.07, p=.002$) and 24 months of age ($\beta=-.29, SE=.08, t(488)=3.98, p<.0001$). In the COMORBID subgroup, children’s irritability was significantly lower at 7 months of age versus 15 months of age ($\beta=-.55, SE=.17, t(494)=3.31, p=.001$) and 24 months of age ($\beta=.44, SE=.19, t(484)=2.33, p=.02$). There was no significant difference in children’s irritability across early childhood for the CP subgroup.

The LLM for with exposure to intrusive parenting as the dependent factor revealed a trend towards a significant fixed effect for the interactions between subgroup and developmental time point ($F(2, 554)=2.11, p=.07$). As seen in Figure 2, for the COMPARISON subgroup, children’s exposure to
intrusive parenting was significantly less at 15 months of age versus 7 months of age ($\beta=-.18$, $SE=.04$, $t(460)=4.60$, $p<.0001$) and 24 months of age ($\beta=.11$, $SE=.05$, $t(464)=2.35$, $p=.02$). In the COMORBID subgroup, children at 15-month assessment revealed significantly less exposure to intrusive parenting compared to the 24-month assessment ($\beta=-.34$, $SE=.11$, $t(478)=3.02$, $p=.002$), but no significant within subgroup differences were revealed regarding intrusive parenting levels at 7 months of age. Similarly, in the CP subgroup, children at 15-month assessment revealed significantly less exposure to intrusive parenting compared to the 24-month assessment ($\beta=-.30$, $SE=.11$, $t(464)=2.79$, $p=.005$), but no significant within subgroup differences were revealed for intrusive parenting levels at 7 months of age.

In contrast, two behavioral reactivity and regulation variables showed differences in main effects between developmental time points with no significant interactions effects: social approach tendencies (AIC=5825.01, BIC=5913.18) and overall reactivity (AIC=3878.14, BIC=3966.31). Children’s displays of social approach demonstrated significant differences across developmental time points ($F(2, 484)=9.83$, $p<.0001$). On average and regardless of subgroup, children demonstrated significantly lower approach tendencies at 7 months of age ($M=13.35$) than at 15 months ($M=13.93$) and 24 months of age ($M=13.71$), ($t$s(484)=4.48 and 2.00, $p$s=.0001 and .05, respectively). Additionally, children’s overall behavioral reactivity demonstrated significant differences across developmental time points ($F(2, 482)=9.83$, $p<.0001$). In particular, children demonstrated significantly lower overall reactivity behaviors at 7 months old ($M=5.70$) than at 15 ($M=5.99$) and 24 months of age ($M=6.03$), ($t$s (494)=2.19 and 2.22, $p$s=.03, respectively).

Alternatively, maternal sensitivity was only significantly associated with main effect differences in subgroup status at 5 years of age (AIC=2511.11, BIC=2639.28; $F(2, 486)=4.10$, $p=.02$), with no significant interactions effects. Children exposed to higher levels of maternal sensitivity were significantly associated with the COMPARISON subgroup ($M=2.89$) versus children within CP ($M=2.72$) and COMORBID ($M=2.74$) subgroups, ($t$s(486)=2.52 and 2.53, $p$s =.01, respectively). Finally, LMMs (AIC
ranges=620.26-7058.16, BIC ranges=708.43-7146.35) with pre-task cortisol level, cortisol reactivity, and sustained attention behaviors as the dependent variables showed no significant differences across subgroups ($F$s (2, 492) ranged from .68 to 2.50, $ns$), developmental time points ($F$s (2, 492) ranged from .43 to .97, $ns$), and subgroup by developmental time point interaction ($F$s (4, 492) ranged from 1.43 to 1.70, $ns$).
Figure 1 Changes in Children’s Irritability across Early Childhood within each Conduct Problem Subgroups. Children in the COMPARISON subgroup had significant increased irritability from 7 to 15 months of age followed by a significant decrease in irritability demonstration by 24 months. In contrast, the COMORBID subgroup had increased irritability from 7 to 15 months of age that stayed higher at 24 months of age. No significant differences were found for the CP subgroup.

Figure 2 Changes in Children’s Exposure to Intrusive Parenting Across Early Childhood within each Conduct Problem Subgroups. Children in the COMPARISON subgroup had significant decreased exposure to intrusive parenting from 7 to 15 months of age that stayed low from 15 to 24 months of age. Alternatively, both CP and COMORBID subgroups had lower level of intrusive exposure at 15 versus 7 months of age with an increase in levels by 24 months of age.
Aim 2. Early Childhood Indices of CP, COMORBID, and COMPARISON Subgroup Development

Aim 2a. 7 months of age. MLR for Model 1 utilized 7-month assessment indices of biological stress responsivity, behavioral reactivity and regulation, and parenting styles to predict children’s membership in three behavioral-derived conduct problem subgroups at 5 years of age. The overall model was significant across the 30 imputed data sets, $Wald \chi^2(28, 506)=73.46-83.91, p<.001$. The likelihood ratio tests revealed that maternal education achievement ($Wald \chi^2(28, 506)=12.42-18.21, Wald \chi^2$ was $p<.001$), pretask cortisol levels ($Wald \chi^2(28, 506)=2.35-9.38, p<.05$), cortisol reactivity ($Wald \chi^2(28, 506)=1.40-10.42, p<.05$), as well as interactions between cortisol reactivity and intrusive parenting ($Wald \chi^2(28, 506)=2.83-8.75, p<.05$) significantly predicted children’s membership into the three conduct problem subgroups (see Table 3). As shown in Table 4, contrasts of parameter estimates demonstrated that infants who mothers had the lowest levels of maternal achievement were more likely be in the CP ($\beta=-.15, SE=.06, p=.04, OR=.86, CI=.76-.97$) and COMORBID ($\beta=-.23, SE=.06, p<.001, OR=.80, CI=.70-90$) subgroups versus the COMPARISON subgroup. Moreover, the infants with higher pretask cortisol levels were 1.49 times more likely to be in the CP versus COMORBID subgroup ($\beta=.41, SE=.21, p=.02, OR=1.49, CI=1.05-2.18$).
Table 3 Likelihood Ratio Tests for Models Relating Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 7 Months of Age to Conduct Problem Subgroups.

<table>
<thead>
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<th>AIC</th>
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<td>73.46-83.91***</td>
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<td><strong>Covariates</strong></td>
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<td>Low-Income$^b$</td>
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<td>Married</td>
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<td>Maternal Education Achievement$^c$</td>
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<tr>
<td>Pretask Cortisol Levels (ug/dL)</td>
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<td>Cortisol Reactivity across Emotion Tasks$^d$</td>
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<td>Cortisol Reactivity X Intrusive Parenting</td>
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</table>

*Note: Multiple imputations were performed, resulting in a range of Wald $\chi^2$ across 30 imputed datasets.

$^a$ White=1 and Black=2;$^b$ Income-to-Needs of 200% below government-defined poverty level; $^c$ Years of school completed; $^d$ Cortisol Reactivity= ((20 min posttask cortisol levels – pretask cortisol levels); $^e$ Home visitor reported cortisol levels across the entire visit; $^f$ Micro-behavioral coding of parent-child interactions during play tasks.
Table 4 Parameter Estimates for the Multinomial Logistic Regression Associating Children’s Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 7 Months of Age to Conduct Problem Subgroups.

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<th>OR</th>
<th>CI</th>
</tr>
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<tbody>
<tr>
<td><strong>COMPARISON vs CP</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal Education Achievement&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.15*</td>
<td>.06</td>
<td>.86</td>
<td>.76 - .97</td>
</tr>
<tr>
<td>Cortisol Reactivity&lt;sup&gt;b&lt;/sup&gt; X Intrusive Parenting</td>
<td>.31*</td>
<td>.14</td>
<td>1.36</td>
<td>1.06-1.75</td>
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<td><strong>COMPARISON vs COMORBID</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Maternal Education Achievement&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.23***</td>
<td>.06</td>
<td>.80</td>
<td>.70 - .90</td>
</tr>
<tr>
<td><strong>COMORBID vs CP</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pretask Cortisol Levels (ug/dL)</td>
<td>.41*</td>
<td>.21</td>
<td>1.49</td>
<td>1.05-2.18</td>
</tr>
</tbody>
</table>

<sup>a</sup> Years of school completed; <sup>b</sup> Cortisol Reactivity = (20 min posttask cortisol levels – pretask cortisol levels)

Note: The COMPARISON subgroup is the referent group in the ‘COMPARISON vs CP’ and ‘COMPARISON vs COMORBID’ contrasts. The COMORBID subgroup is the referent group in the ‘COMORBID vs CP’ contrast.

In addition, only one significant interaction distinguished the COMPARISON from the CP subgroup, the interrelationship between cortisol reactivity and intrusive parenting ($\beta=.31$, $SE=.14$, $p=.03$, $OR=1.36$, $CI=1.06-1.75$; see Table 4). The plot of infant cortisol reactivity moderating the relationship between exposure to intrusive parenting at 7 months of age and the probability of developing CP is shown in Figure 3. This plot revealed that infants with high cortisol reactivity (top 25%) had an almost 25% increase in probability of being in the CP subgroup (compared to COMPARISON subgroup) as the level of exposure to intrusive parenting increased. There was no significant difference in the infants that had the lowest cortisol reactivity (bottom 25%)—no change or decreases in cortisol levels during an emotional eliciting task—for predicting the probability of being in the CP subgroup.
Figure 3 *Interrelationships between HPA Activity and Intrusive Parenting at 7 Months of Age Predicting CP Subgroup Membership.* Children with high cortisol reactivity to stressors (n=111) had greater probabilities of becoming part of the CP versus COMPARISON subgroup as exposure to intrusive parenting increased.

**Aim 2b. 15 months of age.** MLR for Model 2 utilized 15-month assessment indices of biological stress responsivity, behavioral reactivity and regulation, and parenting styles to predict children’s membership in the conduct problem subgroups. The overall model was significant across the 30 imputed data sets, \(\text{Wald } \chi^2(36, 506)=74.48-87.26, p<.001\). As in early infancy, the likelihood ratio test revealed that maternal education achievement in early infancy significantly predicted children’s membership into the three subgroups, \(\text{Wald } \chi^2(28, 506)=6.92-10.80, p=.008\) (see Table 5). Again, contrasts of parameter estimates demonstrated that infants whose mothers had the lowest levels of maternal achievement were much more likely to be categorized into CP (\(\beta=-.14, SE=.07, p=.04, OR=.87, CI=.77-1.00\)) and COMORBID (\(\beta=-.15, SE=.07, p=.04, OR=.85, CI=.75-.98\)) subgroups versus the
COMPARISON subgroup (see Table 6). Moreover, there was a trend (Wald $\chi^2$(28, 506)=2.34-7.41, $p=.06$), towards children in families with the lowest income-to-needs ratios being more likely to obtain membership in the COMORBID versus COMPARISON subgroups ($\beta=-.44$, $SE=.23$, $p=.07$, $OR=.63$, $CI=.40$-1.00). All other main effects cannot be interpreted because they are involved in interactions that were significantly associated in the three subgroups at 5 years of age.
Table 5 Likelihood Ratio Tests for Models Relating Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 15 Months of Age to Conduct Problem Subgroups.

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<tr>
<td>Married</td>
<td>.74-2.65</td>
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<tr>
<td>Maternal Education Achievement$^c$</td>
<td>6.92-10.80**</td>
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<td>Approach Tendencies</td>
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<td>Overall Reactivity</td>
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<td><strong>Parenting Styles</strong>$^f$</td>
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<td>Intrusive/Negative</td>
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<td><strong>Interactions</strong></td>
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<tr>
<td>Pretask Cortisol X Intrusive Parenting</td>
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<tr>
<td>Cortisol Reactivity X Behavioral Approach</td>
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<td>Behavioral Attention X Sensitive Parenting</td>
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<tr>
<td>Behavioral Approach X Sensitive Parenting</td>
<td>3.01-10.01*</td>
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<tr>
<td>Behavioral Irritability X Sensitive Parenting</td>
<td>5.54-10.76*</td>
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</table>

Note: Multiple imputations were performed, resulting in a range of Wald $\chi^2$ across 30 imputed datasets.

$^a$ White=1 and Black=2; $^b$ Income-To-Needs of 200% below government-defined poverty level; $^c$ Years of school completed; $^d$ Cortisol Reactivity= ((20 min posttask cortisol levels – pretask cortisol levels); $^e$ Home visitor reported on children's observed behaviors across the entire visit; $^f$ Micro-behavioral coding of parent-child interactions during play tasks.
A majority of the interactions at 15 months of age significantly predicting subgroup differences in mental health involved behavioral demonstrations of reactivity and regulation (Table 4). Specifically, contrast analyses shown in Table 5 revealed that all but one interaction predicted differences in group membership between COMORBID and COMPARISON subgroups.

The interactions between children’s sustained attention and sensitive parenting ($Wald \chi^2(36, 506)=4.41-11.45, p=.03$) as well as children’s irritability and sensitive parenting ($Wald \chi^2(36, 506)=5.54-10.76, p=.03$) in later infancy significantly predicted group differences among conduct problems subgroups. Contrasts revealed that the significant differences in interactions of sustained attention and sensitive parenting ($\beta=-.36, SE=.15, p=.02, OR=.70, CI=.53-.92$) as well as irritability and sensitive parenting ($\beta=-.36, SE=.14, p=.02, OR=.69, CI=.50-.93$) were between the COMPARISON and COMORBID subgroups (Table 6).

Probing of sustained attention and sensitive parenting as well as irritability and sensitive parenting revealed similar results (Aiken & West, 1991). All individuals began at approximately a 50% probability for co-occurring conduct problems and anxiety versus no-to-low symptom subgroup membership when maternal sensitivity was at its minimum. Yet, children with low levels of observed irritability (bottom 25th percentile, $n=80$) and high levels of sustained attention (top 25th percentile, $N=129$) had a much steeper and abrupt decrease in the probability of being part of the COMORBID versus COMPARISON subgroup. That is, at moderate level of exposure to sensitive parenting (2.5 out of 5 behavioral coding), these individuals had a less than 10% chance of being in the COMORBID subgroup and a 0% chance at the maximum level of sensitive parenting. Alternatively, children with high levels of observed irritability (top 25th percentile, $n=100$) and low levels of sustained attention (top 25th percentile, $n=114$) had gradual and steep decrease that never reached below a 9% probability of being part of the COMORBID versus the COMPARISON subgroup.
Table 6 Parameter Estimates for the Multinomial Logistic Regression Associating Children’s Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 15 Months of Age to Conduct Problem Subgroups.

<table>
<thead>
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<th>OR</th>
<th>CI</th>
</tr>
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<td><strong>COMPARISON vs CP</strong></td>
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</tr>
<tr>
<td>Maternal Education Achievement(^a)</td>
<td>-.14*</td>
<td>.07</td>
<td>.87</td>
<td>.77-1.00</td>
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<td><strong>COMPARISON vs COMORBID</strong></td>
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<td></td>
</tr>
<tr>
<td>Low-Income(^b)</td>
<td>-.44†</td>
<td>.23</td>
<td>.63</td>
<td>.40-1.00</td>
</tr>
<tr>
<td>Maternal Education Achievement(^a)</td>
<td>-.15*</td>
<td>.07</td>
<td>.85</td>
<td>.75-98</td>
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<tr>
<td>Pretask Cortisol ( \times ) Intrusive Parenting</td>
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<td>.17</td>
<td>1.40</td>
<td>1.05-1.90</td>
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<td>Cortisol Reactivity(^c) ( \times ) Behavioral Approach</td>
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<td>1.72</td>
<td>1.12-2.63</td>
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<tr>
<td>Behavioral Attention ( \times ) Sensitive Parenting</td>
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<td>.70</td>
<td>.53-92</td>
</tr>
<tr>
<td>Behavioral Irritability ( \times ) Sensitive Parenting</td>
<td>-.36*</td>
<td>.14</td>
<td>.69</td>
<td>.50-93</td>
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<td><strong>COMORBID vs CP</strong></td>
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<tr>
<td>Behavioral Approach ( \times ) Sensitive Parenting</td>
<td>.40*</td>
<td>.19</td>
<td>1.53</td>
<td>1.09-2.12</td>
</tr>
</tbody>
</table>

\(^{p<.08, *p<.05, **p<.01, ***p<.001. \(^a\) Years of school completed; \(^b\) Income-to-Needs of 200\% below government-defined poverty level; \(^c\) Cortisol Reactivity= (20 min posttask cortisol levels – pretask cortisol levels)

Note: The COMPARISON subgroup is the referent group in the ‘COMPARISON vs CP’ and ‘COMPARISON vs COMORBID’ contrasts. The COMORBID subgroup is the referent group in the ‘COMORBID vs CP’ contrast.

The interactions between children’s cortisol reactivity and approach tendencies (Wald \( \chi^2 \)(36, 506)=3.02-13.48, \( p=.04 \)) in later infancy significantly predicted group differences among the conduct problems subgroups. Contrasts revealed that the significant differences in interactions of cortisol reactivity and approach tendencies (\( \beta=.51, SE=.27, p=.01, OR=1.72, CI=1.12-2.63 \)) were between the COMPARISON and COMORBID subgroups (Table 6).

Figure 4 shows the plot of children’s cortisol reactivity moderating the relationship between social approach tendencies and the probability of developing membership into the COMORBID versus COMPARISON subgroup. For children with high cortisol reactivity (top 25\(^{\text{th}}\) percentile; heightened HPA
activity) at 15 months of age, there was a significant increase in the probability (~30%) of developing COMORBID status at 5 years old as their tendencies toward social approach increased. Alternatively, children with low cortisol reactivity (bottom 25th percentile; blunted HPA activity), there was a significant decrease in the probability (~20%) of displaying later COMORBID status as their tendencies toward social approach increased.

The interactions between children’s pretask cortisol levels and intrusive parenting (Wald $\chi^2(36, 506)=3.25-10.20$, $p=.04$) in later infancy significantly predicted group differences among the conduct problems subgroups. Contrasts revealed that the significant differences in interactions of pretask cortisol levels and intrusive parenting ($\beta=.33$, $SE=.17$, $p=.02$, $OR=1.40$, $CI=1.05-1.90$) were between the COMPARISON and COMORBID subgroups (Table 6).

Figure 5 depicts the plot of pretask cortisol levels the relationship between observed intrusive parenting at 15 months of age and the probability of developing CP versus COMORBID subgroup status. Interaction probing revealed that children with high pretask cortisol levels (top 25%) had over a 10% increase in probability of being in the CP subgroup as the levels of exposure to intrusive parenting increased (versus the COMORBID subgroup). There was no significant difference in the infants that had the lowest cortisol reactivity (bottom 25%)—no change or decreases in cortisol levels during an emotional eliciting task—for predicting the probability of being in the CP subgroup.

The interactions between children’s social approach tendencies and sensitive parenting (Wald $\chi^2(36, 506)=3.01-10.01$, $p=.04$) in later infancy significantly predicted group differences among the behaviorally-derived conduct problems subgroups. Contrasts revealed that the significant differences in interactions of approach tendencies and sensitive parenting ($\beta=.40$, $SE=.19$, $p=.03$, $OR=1.53$, $CI=1.09-2.12$) were between the COMORBID and CP subgroups (Table 6).

Figure 6 shows the plot of children’s social approach moderating the relationship between maternal sensitive parenting exposure and the probability of developing CP versus COMORBID subgroup
status. For children with high social approach tendencies (top 25\textsuperscript{th} percentile) at 15 months of age, there was a significant increase in the probability (from 65\% to 95\% likelihood) of developing CP subgroup status as exposure to sensitive parenting increased. Alternatively, children with low social approach tendencies (bottom 25\textsuperscript{th} percentile), there was a significant increase in the probability (~10\%) of displaying later COMORBID subgroup status as exposure to sensitive parenting increased.
Figure 4 Interrelationships between Children’s HPA Activity and Approach Tendencies at 15 Months of Age Predicting COMORBID Subgroup Membership. Children with high cortisol reactivity to stressors had a significantly higher probability of developing COMORBID versus COMPARISON subgroup status as the tendencies for exhibiting social approach increased. In contrast, children with low cortisol reactivity to stressors were significantly less likely to developing COMORBID status as the tendencies for exhibiting social approach increased.

Figure 5 Interrelationships between Children’s HPA Activity and Intrusive Parenting at 15 Months of Age Predicting COMORBID Subgroup Membership. Children with high pretask cortisol levels had a significantly higher probability of developing COMORBID versus COMPARISON subgroup status as their exposure to intrusive parenting increased. No significant differences in infants with low cortisol levels in relation to intrusive parenting exposure were revealed.
Figure 6 Interrelationships between Approach Tendencies and Sensitive Parenting at 15 Months of Age Predicting CP Subgroup Membership. Children with high demonstrations of approach tendencies ($n=49$) had a significantly higher probability of developing CP subgroup status the greater their exposure to maternal sensitivity versus the COMORBID subgroup. In contrast, children with low approach tendencies ($n=68$) were significantly more likely to developing COMORBID versus CP subgroup status with increased maternal sensitivity exposure.

**Aim 2c. 24 months of age.** MLR for Model 3 utilized 24-month assessment indices of biological stress responsivity, behavioral reactivity and regulation, and parenting styles to predict children’s membership in the conduct problems subgroups. The overall model was significant across the 30 imputed data sets, $Wald \chi^2(42, 506)=75.19-93.89, p<.001$. Similar to 7 and 15 months of age, the likelihood ratio test revealed that maternal education achievement ($Wald \chi^2(42, 506)=.91-15.24, p<.001$) significantly predicted children’s membership into the conduct problems subgroups (see Table 7). In particular, mothers had the lowest levels of maternal achievement were much more likely to be categorized as CP ($\beta=-.14, SE=.06, p=.04, OR=.87, CI=.78-.98$) and COMORBID ($\beta=-.22, SE=.07, p=.007, OR=.80, CI=.74-.92$) subgroups versus the COMPARISON subgroup (see Table 7). Moreover, cortisol
reactivity to emotion eliciting tasks significantly predicted differences among the conduct problem subgroups ($Wald \chi^2(42, 506)=1.83-10.82, p=.04$). To explicate, children with decreased cortisol reactivity were more likely to be members of the CP subgroup (see Table 9); whereas, increased cortisol reactivity was more likely to be associated with the COMPARISON subgroup ($\beta=-.27, SE=.13, p=.03, OR=.83, CI=.76-1.12$).
Table 7 Likelihood Ratio Tests for Models Relating Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 24 Months of Age to Conduct Problem Subgroups.

<table>
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<td><strong>Behavioral Reactivity and Regulation$^e$</strong></td>
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<td>Approach Tendencies</td>
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Note: Multiple imputations were performed, resulting in a range of Wald $\chi^2$ across 30 imputed datasets.

$^a$ White=1 and Black=2; $^b$ Income-to-Needs of 200% below government-defined poverty level; $^c$ Years of school completed; $^d$ Cortisol Reactivity= ((20 min posttask cortisol levels – pretask cortisol levels); $^e$ Home visitor reported on children’s observed behaviors across the entire visit; $^f$ Micro-behavioral coding of parent-child interactions during play tasks.
Table 8 Parameter Estimates for the Multinomial Logistic Regression Associating Children’s Biological Stress Responsivity, Behavioral Reactivity and Regulation, and Parenting Styles at 24 Months of Age to Conduct Problem Subgroups.

<table>
<thead>
<tr>
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<th>OR</th>
<th>CI</th>
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</tr>
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<td>.87</td>
<td>.78-.98</td>
</tr>
<tr>
<td>Cortisol Reactivity across Emotion Tasks$^b$</td>
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<td>.13</td>
<td>.83</td>
<td>.76-1.12</td>
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<tr>
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<td>.16</td>
<td>1.64</td>
<td>1.21-2.19</td>
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<tr>
<td>Maternal Education Achievement$^a$</td>
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<td>.74-.92</td>
</tr>
<tr>
<td>Pretask Cortisol X Behavioral Irritability X Intrusive Parenting</td>
<td>-.49**</td>
<td>.19</td>
<td>.68</td>
<td>.52-.94</td>
</tr>
<tr>
<td><strong>COMORBID vs COMPARISON</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No significant differences</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

$p<.08$, *$p<.05$, **$p<.01$, ***$p<.001$. $^a$Years of school completed.

Note: The COMPARISON subgroup is the referent group in the ‘COMPARISON vs CP’ and ‘COMPARISON vs COMORBID’ contrasts. The COMORBID subgroup is the referent group in the ‘COMORBID vs CP’ contrast.

At 24 months of age, primarily three-way interactions among pretask cortisol levels, affective behavioral reactivity and regulation, and parenting styles were associated with significant differences in developing CP versus COMPARISON subgroup status (see Table 7). Specifically, interrelationships among pretask cortisol levels, social approach behaviors, and sensitive parenting significantly predicted conduct problem subgroup differences ($Wald \chi^2 (42, 506)=7.91-19.28, p<.001$). The moderating effects of pretask cortisol levels and approach tendencies on sensitive parenting at 24 months of age were associated with differences in developing CP and COMPARISON subgroup status ($\beta=.48, SE=.16, p=.005, OR=1.64, CI=1.21-2.19$; see Table 7).

Plots of interactions revealed that toddlers who exhibit high pre-task cortisol levels as well as high approach behaviors (top 25th percentile for both variable; n=29) had approximately a 7% increase in
their probability of becoming a member of the CP versus COMPARISON (see Figure 7). Children who were low on both pretask cortisol levels and social approach behaviors (bottom 25th percentile for both variables; \( n = 24 \)) or had uncoordinated pretask cortisol levels and approach tendencies (high or low on either) showed a decreased probability of developing later CP with increased maternal sensitivity exposure. For instance, children with low pretask cortisol levels and high approach tendencies (bottom 25th percentile for both variable; \( n = 38 \)) had nearly a 30% decrease in risk for developing membership into the CP subgroup as the maternal sensitivity increased from its lowest to highest levels.

Additionally, at 24-months, the interrelationships among pretask cortisol levels, irritability, and intrusive parenting significantly predicted conduct problem subgroup differences (\( \text{Wald } \chi^2(42, 506) = 4.77-13.25, p = .009 \)). The moderating effects of children’s pretask cortisol levels and irritability on intrusive parenting at 24 months of age was associated with differences in developing COMORBID versus COMPARISON subgroup status (\( \beta = -.49, SE = .16, p = .007, OR = .68, CI = .52-.94; \) see Table 8).

Figure 8 demonstrates that children (\( n = 20 \)) with low pretask cortisol levels (bottom 25th percentile) and high behavioral displays of irritability (top 25th percentile) had a significant increase in the probability (from 80% to nearly 100%) of having COMORBID versus COMPARISON subgroup status when maternal intrusive behaviors increased from minimum to maximum exposure levels. In contrast, children low on both pretask cortisol levels and behavioral display of irritability (both are the bottom 25th percentile, \( n = 35 \)) had significant decreases in probability (from 48% to nearly 20%) of developing COMORBIS subgroup status as maternal intrusive behaviors increased. There was no to little change in children who showed high levels of pretask cortisol with high or low levels of irritability in the probability of developing COMORBID subgroup status in relation to changes in maternal sensitivity exposure.

**Brief Summary of Findings for Aim 2**

Table 10 depicts a summary of all main and interaction effects by children’s age (7, 15, and 24 months of age) and by subgroup membership (CP, COMORBID, versus COMPARISON subgroups). Within
the table “↑” indicates that high levels of the given risk variable are characteristic of this subgroup; whereas, “↓” indicates that low levels of the given risk factor are characteristic of this risk factor. When the term “with” is seen after a given risk factor this means that there was an interaction with the subsequent risk factor. For example, this table shows that in late infancy children with low levels of observed irritability are more likely to be members of the COMPARISON Subgroup as exposure to sensitive parenting increased.
Figure 7 Interrelationships among Children’s HPA Activity, Approach Tendencies, and Sensitive Parenting at 24 Months of Age Predicting CP Subgroup Membership. Children with high pre-task cortisol levels and high approach tendencies (n=29) had a greater probability of developing CP versus COMPARISON subgroup status as exposure levels of maternal sensitivity increased. In contrast, children with low-low or mixed levels of pretask cortisol levels were significantly less likely to develop CP subgroup status as maternal sensitivity level increased.

Figure 8 Interrelationships among Children’s HPA Activity, Irritability, and Intrusive Parenting at 24 Months of Age Predicting COMORBID Subgroup Membership. Children with low pretask cortisol levels and high irritability (n=20) had a greater probability of developing COMORBID versus COMPARISON subgroup status as their exposure to intrusive parenting increased. Alternatively, children with low pretask cortisol levels and low irritability (n=35) had a significantly lower probability of developing COMORBID subgroup status as the levels of intrusive parenting increased.

*Note: High and low cutoffs of the moderating variables (pre-task cortisol levels, social approach, and irritability) were formed at +/- 1SD above the mean.
Table 10 Summary of Risk Factors across Early Childhood that Characterize the Development of Conduct Problem Subgroups at 5 Years of Age.

<table>
<thead>
<tr>
<th></th>
<th>COMPARISON Subgroup (n=370)</th>
<th>CP Subgroup (n=72)</th>
<th>COMORBID Subgroup (n=64)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7 months of age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ Pretask Cortisol Levels</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ Cortisol Reactivity with ↑ Intrusive Parenting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 months of age</td>
<td>↓ Irritability with ↑ Sensitive Parenting</td>
<td>↑ Approach Tendencies with ↑ Sensitive Parenting</td>
<td>↑ Cortisol Reactivity with ↑ Approach Tendencies</td>
</tr>
<tr>
<td></td>
<td>↑ Attention with ↑ Sensitive Parenting</td>
<td></td>
<td>↑ Pretask Cortisol Levels with ↑ Intrusive Parenting</td>
</tr>
<tr>
<td>24 months of age</td>
<td>↑ Cortisol Reactivity during Emotional Tasks</td>
<td>↓ Cortisol Reactivity during Emotional Tasks</td>
<td>↓ Pretask Cortisol Levels with ↑ Irritability with ↑ Intrusive Parenting</td>
</tr>
<tr>
<td></td>
<td>↑ Pretask Cortisol Levels with ↑ Approach Tendencies with ↑ Sensitive Parenting</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: &uparrow; indicates high levels of the given risk variable are characteristic of this subgroup; whereas, &downarrow; indicated low levels of the given risk factor. When the term “with” is in the bullet point this means that there was an interaction with the subsequent risk factor.
Chapter 4

Discussion

In an attempt to unlock the heterogeneity of externalizing problems, the present study explored how children’s biological stress responsivity, behavioral reactivity and regulation, and exposure to different parenting styles in early childhood contribute to the development of CP, COMORBID, and COMPARISON subgroups. In general, findings demonstrated that the interrelationships among these three domains of risk at key developmental time points in early childhood had greater explanatory power than the change or stability of any one risk factor alone. In fact, out of the eight major risk factors assessed, only changes of maternal intrusiveness and behavioral irritability explained children’s membership into conduct problem subgroups at 5 years of age.

Among the many complex interrelationships among risk domains that significantly predicted the conduct problem subgroups (see Table 10 for summary), some lent evidentiary support to, while others refuted, the initial conceptual model proposed in Figure 1. To explicate, as broadly theorized (see left side of the blue box in Figure 1), the interplay between heightened biological stress responsivity and adverse parenting styles at 7 months of age did significantly discern whether children would develop CP, COMORBID, or COMPARISON subgroup membership. However, counter to expectations and findings from other empirical investigations (e.g., Bates & Bayle, 1988; Bates et al., 1998; review, Putnam, Sanson, & Rothbart, 2002), behavioral reactivity did not reciprocally interact with parenting styles to significantly predict conduct problems subgroups. By 15 months of age, as speculated (see center of the blue box in Figure 1), the behavioral reactivity and regulation domain of risk was involved in the majority of interrelationships associated with the development of conduct problem subgroups. Finally, findings at 24 months of age (see right side of the blue box in Figure 1) were in-line with theoretical expectations that multiple and reciprocal exchanges among risk factors by the toddlerhood and pre-school years...
becomes the primary predictors of deleterious outcomes (Sameroff, 1990; Shaw et al., 1994). In particular, the current study revealed that behavioral reactivity and regulation moderated the reciprocal interactions between HPA activity (heightened and blunted) and parenting styles at 24 months of age to distinguish the development of CP, COMORBID, and COMPARISON subgroups.

**Stability and Change in Developmental Risk Predicting Conduct Problem Subgroups**

Patterns of stability and change in maternal sensitivity and behavioral displays of irritability across 7, 15, and 24 months distinguished the development of conduct problem subgroups in the present study. While all subgroups began with similar levels of observed intrusive parenting at 7 months of age, higher instability in intrusive parenting was observed from 15 to 24 months of age among children with conduct problems (with and without anxiety). In fact, children who were members of the CP and COMORBID subgroups demonstrated significant increases in their levels of intrusive parenting exposure, on average, versus the COMPARISON subgroup from infancy to toddlerhood. As previously reviewed, negative parenting styles such as detachment and hostility tend to show instability over the first 6 years of life (e.g., Dallaire & Weinrab, 2005). The inconsistency in this type of adverse parenting style has been linked to aggressive and antisocial behaviors in middle childhood and adolescence (e.g., Dodge & McCourt, 2010). Yet, this study is unique because it is the first to demonstrate that increased exposure to intrusive parenting styles even in early childhood is characteristic of children who have conduct problems with or without comorbid anxiety.

Additionally, significant between-group differences revealed themselves in the level of irritability displayed across early childhood. Children who were members of the CP subgroup at age 5 revealed consistently lower levels of observed irritability across infancy and toddlerhood, while children in the COMORBID subgroup showed higher levels of irritability in late infancy and those levels remained high at 24 months of age. This finding lends credence to the hypothesis that, in early infancy, observed irritability is similar for children who later develop conduct problems with and without anxiety, but
children with comorbid anxiety may encounter increased negative social interactions with families and peers as well as higher deficits in self-regulation skills, resulting in increases in observed irritability over time (e.g., Boivin et al., 1994; Farmer et al., 2002; Milich & Landau, 1984).

**Interrelationships of Risk Domains Underlying the Development of Conduct Problem Subgroups**

As an initial note, findings from this unique sample further support the extant research by demonstrating that low socioeconomic status and parent educational achievement may increase the risk of early-onset conduct problems both with and without co-occurring anxiety (McLoyd, 1990; Offord, Boyle, & Racine, 1991; West & Farrington, 1973). Moreover, males were more likely to exhibit conduct problems without comorbid anxiety symptoms (see Zahn-Waxler, Shirtcliff, & Marceau, 2008). The current study also provides empirical substantiation that individual to contextual domains of risk influence the development of externalizing problems above and beyond sociocultural risk (e.g., Bronfenbrenner & Morris, 1998; McCloyd, 1990; Reid, Patterson, & Snyder, 2002).

More notably, this investigation indicated that the interrelationships of risk domains—biological stress responsivity, behavioral reactivity and regulation, and parenting styles—with the most predictive validity varies at different developmental time points in infancy and toddlerhood when distinguishing membership into conduct problems subgroups. This suggests that repeated displays of risk factors may not be the only or most important way to examine risk factors when predicting later behavioral problems, and examining risk domains that have different meanings and importance at key developmental times in infancy and toddlerhood may help to discern the mechanisms underlying externalizing problems with and without comorbid anxiety.

**Biological Stress Responsivity and Parenting Styles.** Ultimately, the findings from this empirical investigation does not lend empirical support to theories that elicit a straightforward biological stress responsivity distinction between children with conduct problems only having blunted biological stress activity and those with comorbid anxiety having heightened biological stress reactivity (e.g., Hodgins et
Counter to expectations, heightened biological stress responsivity interacted with intrusive parenting to characterize both the CP and COMORBID subgroups versus the COMPARISON subgroup. The subset of children with heightened HPA reactivity to the emotion-eliciting tasks at 7 months increased their likelihood of becoming a member of the CP subgroup as their exposure to intrusive parenting levels increased. On the other hand, children with heightened pretask HPA levels (which could represent "trait"-like or anticipatory stress, as discussed in the limitation section below) at 15 months of age had a greater likelihood of developing COMORBID subgroup membership as their exposure to intrusive parenting increased. Jointly, these results imply that children with conduct problems with and without comorbid anxiety are likely to be biologically sensitive to contextual stressors during infancy, particularly adverse parenting styles. Since heightened HPA responsivity are speculated as being especially sensitive to their contexts (Del Giudice et al., 2010; Hudson & Rapee, 2001; Rogosch & Cicchetti, 1994), exposure to the addition source of stress such as intrusive parenting may further exacerbate their overidentification of hostile cues and overreactions to mild or ambiguous stressors (Dodge, 1991). Over time, such reciprocal interactions between children's biological stress responsivity and maternal intrusiveness may result in more externalizing behaviors when trying to cope with environments that are perceived as challenging and/or threatening.

Further, biological stress responsivity alone was able to distinguish the development of CP, COMORBID, versus COMPARISON subgroups. At 7 months of age, on average, greater HPA pretask levels significantly predicted CP versus COMORBID subgroup membership at 5 years of age. Conversely, at children's 24 month assessment, on average, blunted HPA reactivity to emotion-eliciting tasks was associated with CP membership, while heightened HPA reactivity predicted COMPARISON subgroup
membership. This was counter to what was expected—the COMORBID subgroup did not have heightened stress responsivity versus the CP and COMPARISONS subgroups, further reinforcing that mechanisms underlying these subgroups are comprised of various domains of risk that may manifest themselves in particular ways depending on the developmental time frame.

As previously discussed, two developmental pathways are speculated as leading to the development of conduct problems without anxiety (review, Del Giudice et al., 2010). The more recently theorized pathway consists of an initial propensity for heightened biological stress responsivity in response to threat and challenge with developmental shifts towards blunted biological stress responsivity when environmental contexts are plagued by chronic stressors (Del Giudice et al., 2010; Gunnar & Vazquez, 2006; Gustafsson et al., 2010; Susman, 2006; Tarullo & Gunnar, 2006). This attenuation from heightened to blunted arousal is thought to occur in middle childhood or adolescence, but the exact timing remains unknown. Although not specifically tested within each individual in the current study, the findings highlight that the downregulation of HPA reactivity for the subgroup of children who develop early conduct problems may occur as early as toddlerhood. Specifically, heightened HPA reactivity to challenges with increased exposure to intrusive parenting for children at 7 months had the most explanatory power for differentiating whether children will develop membership into the CP versus COMPARISON subgroup, but blunted HPA reactivity alone at 24 months predicted CP versus COMPARISON subgroup membership. However, this trend may be specific to this high-risk population, since intrusive parenting also demonstrated significant increases from infancy to toddlerhood in the CP and COMORBID subgroups. This increased exposure to adverse parenting styles may be indicative of severe and chronic stressors in the familial context, resulting in a faster attenuation in stress system functioning from primarily heightened to blunted biological stress responsivity.

The alternative pathway that researchers theorize for the CP subgroup is thought to occur in all contexts regardless of the level and severity of chronic stressors and involves a robust genetic
predisposition to blunted biological stress responsivity even in early infancy and continuing into adulthood (Del Giudice et al., 2010; Hawes et al., 2009; Raine, 2002; Raine et al., 1997). Although the children in the CP subgroup did not demonstrate this biological stress responsivity profile on average, a post-hoc investigation revealed that there is a very small subset of children within this subgroup who exhibited consistently blunted cortisol levels across 7, 15, and 24 months of age. It should also be noted that these consistently blunted HPA levels did not exist in the COMORBID or COMPARISON subgroups.

Taken together, this analysis and the post-hoc investigation calls for a within person differences investigation of the stability of blunted or downregulation in biological stress responsivity to help further explain the existence of within CP subgroup heterogeneity. Moreover, future analysis should examine whether subgroups of children with conduct problems defined according to these distinct biological stress responsivity patterns—attenuated HPA responsivity versus consistently blunted HPA responsivity—may display disparate interrelationships between “loosely” coupled behaviors and parenting styles in predicting later mental health outcomes. This is especially important because researchers believe these two divergent stress responsivity pathways may have very different implications for later prevention strategies (Del Giudice et al., 2010; O’Neal et al., 2010). A detailed discussion of these prevention implications is presented later.

**Behavioral Reactivity and Regulation as a Key Risk Domain.** The findings from the current investigations highlight that behavioral reactivity and regulation at 15 and 24 months of age becomes the key intermediate domain of risk for distinguishing membership of children into the early subgroups of CP, COMORBID, or COMPARISON subgroups. These results are consistent with the view that the “loose” coupling of children’s biological and behavioral manifestations of vulnerabilities, as well as the reciprocal interactions of risky children’s behaviors and parenting styles, in early childhood are essential in determining whether children will develop later conduct problems (Bates & Bayle, 1988; Bates et al., 1998; Putnam et al., 2002; Rubin et al., 2003). Overall, these findings underscore that after the first year
of life, risky behaviors are a key factor in the developmental mechanisms underlying conduct problem outcomes.

For example, high levels of approach tendencies—a potential precursor of elevated sensation-seeking tendencies (Beauchaine et al., 2001; Quay, 1993)—were associated with CP and COMORBID subgroups. It is not surprising that this relation emerged after the first year of life when children have increased mobility and therefore greater opportunity to display approach behaviors. Unexpectedly, however, this relation was most evident in children exposed to responsive and sensitive parenting for the CP subgroup. Previous research has shown that the propensity to approach novel and potentially stressful situations is more evident in securely attached children whose mothers sensitively respond to their children’s needs (e.g., Ainsworth et al., 1979; Bowlby, 1969). It may be that sensitive parenting style fosters children’s approach tendencies, allowing them to have greater opportunities to engage in aggressive and other externalizing behaviors associated with later conduct problems. Alternatively, children who display high levels of approach to potentially stressful and novel situations may evoke increases in maternal engagement to help ensure their safety.

In contrast, for the children who became members of the COMORBID subgroup the interrelationships of biological stress responsivity and social approach tendencies had the most predictive validity at 15 months of age. In particular, children with heightened HPA reactivity to stressors showed an increased probability for developing COMORBID subgroup membership as their observed social approach tendencies increased. This finding may suggest that high approach tendencies in these children increase their likelihood of encountering social interactions wherein they become too physiologically aroused to enact effortful inhibition or executive processing, resulting in behaviors associated with reactive aggression and other externalizing behaviors (Hubbard et al., 2002; Tyson, 1998; van Goozen et al., 1998). With increased negative experiences when socially interacting, these children may become more anxious about engaging with other family members and/or peers (e.g.,
Eisenburg et al., 2009; Farmer et al., 2002; Keiley et al., 2003; Nigg, 2006). In general, the moderating effect of biological stress responsivity may explain why there are inconsistencies in the literature on the relationship between approach tendencies and comorbid internalizing and externalizing problems in children (Johnson et al., 2003; Keiley et al., 2003; Oldenhinkel et al., 2004).

Moreover, at the 15 month assessment, certain behavioral reactivity and regulation factors significantly enhanced the relationship between positive parenting styles and membership into the COMPARISON subgroup at 5 years old. Specifically, children with low irritability and high levels of sustained attention had greater than 90% probabilities of being in the COMPARISON subgroup when exposed to only moderate levels of sensitive parenting. These findings highlight that mastery of behaviors of reactivity and regulation in infancy as a means of coping with environmental stressors is crucial for mitigating risk for developing conduct problems with comorbid anxiety (e.g., Calkins, 2007).

Additionally, similar to prior empirical investigations (Eisenberg et al., 2009; Steinberg et al., 2009), the degree of children's attention regulation difficulties in early childhood was similar between the COMORBID and CP subgroups within this high-risk population.

Complex Interplay of All Domains of Risk by Toddlerhood. In alignment with multiple theoretical models of developmental processes (e.g., Bronfenbrenner & Morris, 1998; Cicchetti & Tucker, 1994; Rutter, 1990; Sameroff & Chandler, 1975; Sroufe & Rutter, 1984), these findings suggest that as children gain more experiences with proximal contexts and acquire behavioral skills to manipulate their environmental interactions, the interrelationships among multiple domains of risk will have increasing predictive value for the development of negative mental health outcomes. In fact, by 24 months of age, it was primarily the interrelationships among all three domains of risk that was associated with the development of CP and COMORBID subgroups versus the COMPARISON subgroup. Specifically, children with high pretask HPA activity along with greater demonstrations of approach tendencies were more likely to develop CP subgroup membership as exposure levels of sensitive and responsive parenting
increased. This combination of high approach tendencies and heightened HPA activity may result in more opportunities to engage in problem behaviors of aggression and rule breaking. That is, heightened activation of the HPA makes it more difficult for children to enact self-regulation skills and executive processing (e.g., McBurnett et al., 1998). If this biological propensity is compounded with a tendency to approach more situations which may require these regulation skills in interacting with others, then the probability using aggression or antisocial behaviors to cope with the situation may be amplified.

Ultimately, the coupling of these two biological and behavioral risk factors may require more maternal engagement and sensitivity as an attempt to maintain their children’s well being and mitigate behavior problems (e.g., Rutter, 2007; Rutter & Silberg, 2002; Rutter et al., 1997; Hershberger, 1994).

Alternatively, children with low pretask HPA levels and high displays of irritability in toddlerhood increased children’s probabilities for developing COMORBID subgroup membership as exposure levels to intrusive parenting increased. There are many potential explanations for this unexpected finding; two possibilities are discussed here. First, the blunted biological stress levels may indicate that these children have reduced sensitivity to punishment (Beauchaine, 2001; Corr, 2004; Gray, 1987; review, van Goozen et al., 2007); therefore, they are unable to learn that their behaviors of irritability may evoke increases in intrusive parenting (Damasio, 2000). The increased negative interactions over time may lead to increased bouts of aggression and specific forms of anxiety problems that may be more likely to be associated with blunted HPA activity including post traumatic stress disorder (review, Elhert, Gaab, & Heinrichs, 2001).

Second, and a more likely explanation in this author’s opinion, is that stress reactivity may become more difficult to elicit as children get older due to increased behavioral coping mechanisms, habituation to similar stress, and/or a potential stress hyporesponsive period in early childhood (review, Lupien, McEwen, Gunnar, & Heim, 2009). In the current study, on average, children did not exhibit a HPA response to the emotional tasks during the 24-month assessment. Together with the notion that
children with low HPA activity have a greater propensity to exhibit a cortisol response to a subsequent stressor via the law of initial values—i.e., the existence of a negative correlation between initial level and the magnitude of change in reaction to a stressor (e.g., Ramsay & Lewis, 2003) – this implies that children with these low pretask cortisol levels may be prone to heightened biological stress reactivity. This heightened HPA activity may be associated with high irritability, and there is strong theoretical and empirical support for intrusive parenting being part of the developmental processes underlying conduct problems and comorbid anxiety in early childhood (Boivin et al., 1994; Farmer et al., 2002; Milich & Landau, 1984). Therefore, with a more effective stressor, one may speculate that these children would have displayed heightened stress reactivity in toddlerhood, which would have interacted with high levels of irritability and intrusive parenting to influence the later development of co-occurring conduct problems and anxiety. However, to verify or negate this conjecture, future studies with more effective stressors in toddlerhood should be performed.

**Prevention Implications**

Many prevention and intervention programs that focus on mitigating risk for the development of externalizing behaviors in childhood target multiple domains of risk, including adverse parenting styles and risky behaviors (e.g., CPPRG, 1999; Olds, 2006; Webster-Stratton, 2000, 2005; Webster-Stratton, Reid, & Hammond, 2001). Efficacy trials from a number of caregiver-focused interventions that promote warm and consistent parental involvement, as well as children’s acquisition of appropriate affective behavioral skills, have demonstrated decreases in aggressive behaviors during childhood and adolescence (e.g., Dishion & Patterson, 1996; Old, 2002; Webster-Stratton, 1998), even in families with high levels of accumulated risk (Hart & Risley, 1995; Lengua, 2002). Yet, in all studies, a significant portion of children with conduct problems do not demonstrate therapeutic responses, and many improvements are not maintained over long intervals (e.g., Forehand & Long, 1986; Webster-Stratton, 1990).
Researchers who currently design and implement prevention programs for conduct problems may desire a continued focus on improving all components of parenting practices and children’s emotional reactivity and self-regulation skills. Yet, findings from the current study suggest that it may be more efficacious to target the specific parenting and behavioral risk factors at key development time points that possess the most explanatory power regarding the later development of conduct problems with and without comorbid anxiety in early childhood. Additionally, the current study provides initial support for the suggestion that incorporating the assessment of biological vulnerabilities to stress responsivity along with the behavioral and parenting domains of risk may be critical for designing more efficacious interventions in early childhood to meet the unique needs of children who may develop only conduct problems compared to those who develop comorbid anxiety (e.g., Beauchaine et al., 2008; Hodgins, 2007; van Goozen & Fairchild, 2008).

In this high-risk sample, results indicate that prevention programs in early infancy may benefit from determining whether children have heightened biological stress responsivity. Heightened HPA stress responsivity in infancy was associated with the later development of CP subgroup membership. To prevent the attenuation of HPA reactivity that is proposed to transpire as a result of chronic exposure to stress in the CP subgroup (e.g., Del Giudice et al., 2010; Susman, 2006), the current study advocates that prevention programs focus on the consistent reduction of intrusive parenting styles starting in early infancy. Researchers have speculated that, because of the heightened biological sensitivity to context in these infants, changes to environmental conditions over prolonged periods of time (e.g., through the reduction of exposure to intrusive parenting styles; Adam et al., 2007; Gunnar & Donzella, 2002; Teicher et al., 2003) may prevent blunted HPA stress responsivity and/or allow their systems to revert back to more moderate or heightened HPA functioning (Del Giudice et al., 2010). The possibility that this reversion of biological stress responsivity may result in a reduction in aggressive behaviors was clearly demonstrated in a recent experimental intervention (O’Neal et al., 2010). Specifically, children who
changed from blunted to heightened cortisol responsivity in anticipation of social stressors showed lower levels of aggressive behaviors during middle childhood, while those children who showed blunted patterns pre- and post-intervention continued to demonstrate high levels of externalizing behaviors.

As children progress into later infancy, these results suggest sensitive and responsive parenting may reciprocally exacerbate childrens’ propensity to engage in high social approach (sensation-seeking behaviors), ultimately leading to CP subgroup membership at age 5. It does not seem reasonable to decrease parenting styles of warmth and sensitivity. However, these findings may suggest that prevention programs after the second year of life may need to focus on consistent limit-setting parenting practices to decrease the amount of approach behaviors potentially related to greater displays of externalizing behaviors (e.g., Bates et al., 1998). Future research that explores how parenting practices interrelate with approach tendencies in early childhood to influence later conduct problems, with and without comorbid anxiety, is warranted to determine the validity of such speculations.

Disparate from children who develop CP subgroup membership, the present study’s findings indicate that preventative interventions for early childhood displays of co-occurring conduct problems and anxiety would be most beneficial when targeting key developmental time points in later infancy and toddlerhood. As previously discussed, children who display heightened biological stress responsivity along with high approach tendencies may possess deficits in effortful inhibitory control or executive processing (Hubbard et al., 2002; Tyson, 1998; van Goozen et al., 1998). Therefore, preventative interventions may need to focus on enhancing children’s self-regulation skills and parents’ modeling of appropriate emotional reactivity and regulation. In later infancy and toddlerhood, as with children who are in the CP subgroup, infants who develop COMORBID subgroup membership display heightened biological stress responsivity to intrusive parenting styles. Prevention programs should focus on the reduction of intrusive parenting to decrease contextual stressors, which may help avert the further up-regulation of their biological stress responsivity across development. This may ultimately aid in reducing
the limbic system’s dominance over higher-order prefrontal systems under perceived threat, and decreasing associated externalizing behavior problems.

*Concluding Remarks on Prevention Implications.* High attrition rates of families are thought to partially explain the lack of intervention efficacy (Kaminski, Stormshak, Good, & Goodman, 2002; Reid & Eddy, 2002). Focusing on selective biological vulnerabilities, affective behavioral risks, and adverse parenting styles targeting key developmental time points may 1) ensure that parents and children who do not complete the entire intervention get the information that targets their children’s specific vulnerabilities and related risk factors, and 2) increase the likelihood of an immediate response (or improvement) from the child, thereby reinforcing both the parents’ and children’s utilization of specific techniques and their continued participation in the prevention program.

*Limitations and Future Analyses*

The inferences of the present study must be tempered by limitations associated with the sample, subgroup identification, and other study procedures and measures. First, the non-urban, predominantly low-income, White and Black children in the sample are an underrepresented population in the developmental literature (Dill, 2001; Morris & Monroe, 2009). Although utilizing this population adds to the developmental field’s knowledge base regarding this often neglected set of children and families, there is limited generalizability beyond this population in the conclusions drawn from the current analyses. Therefore, future analyses are needed with a more nationally representative sample of both urban and non-urban children of different ethnicities from larger geographical regions to determine if similar findings are revealed.

While conduct problem subgroups were identified utilizing a reliable and validated parent-report measure of conduct problems and anxiety at 5 years of age (Goodman et al., 2000), there were several limitations to the identification procedure. First, even though parent reports have been successful in determining whether children show behavior symptoms associated with DSM IV criteria,
the use of both parent and teacher reports of children’s behavioral outcomes at 5 years of age could have reduced informant bias and led to more accurate assessments of these problems. Additionally, the longitudinal design of the study, which allowed for the investigation of precursors in infancy and toddlerhood for the identification of subgroups of children three years later, is an exceptional contribution to the literature. However, children who demonstrate conduct problems with and without anxiety may not demonstrate these patterns by early adolescence (Broidy et al., 2003; Loeber et al., 1991; Moffitt, 1993; Patterson et al., 1992). In fact, a small portion of children (4-11%) demonstrated this stability in externalizing problems from early childhood to adolescence. Thus, in order to identify subgroups with more homogenous underlying biological vulnerabilities, these groups may need to be formed in adolescence. Future investigations should identify children who have conduct problems with and without comorbid anxiety using data from multiple informants during adolescence to determine whether similar biological vulnerabilities, risky behaviors, and adverse parenting styles in early childhood (and their interrelationships) predict the development of these subgroups.

Furthermore, the findings from this study demonstrate that there may still be a great deal of within- and between-subgroup heterogeneity regarding precursors of these conduct problem subgroups. For example, heightened biological stress responsivity and intrusive parenting interact to predict the development of conduct problems with and without comorbid anxiety. Additionally, there are potentially two distinct biological phenotypes – attenuation of HPA reactivity and consistently blunted HPA activity – underlying the CP subgroup (Del Giudice et al., 2010; Susman, 2006). Thus, the utilization of a bottom-up approach, wherein neurophysiological markers of stress responsivity in infancy and toddlerhood informs the creation of distinct conduct problem subgroups (Gatzke-Kopp et al., 2010), seems warranted in future analyses.

Pretask cortisol levels may represent a more “trait”-like indicator of heightened biological stress responsivity or, as an alternative explanation, an anticipatory stress response (review, Kudielka,
Hellhammer, & Wust, 2009). To determine whether the response was a “true” baseline or simply anticipatory stress reactivity, children’s habituation to the same (or similar) stressors over multiple days or weeks would have to be assessed. Determining whether the pretask level of HPA activity was “trait”-like could be helpful when interpreting whether the interaction between heightened biological stress reactivity and parenting styles differed for children in the CP subgroup (whose heightened HPA activity may reflect HPA reactivity to the stressor) compared to those in the COMORBID subgroup (whose heightened HPA activity may reflect high levels of anticipatory stress). Unfortunately, this is beyond the scope of these analyses. Future research determining trait-like versus state-like biological vulnerabilities across early childhood may be helpful in disentangling the developmental mechanisms underlying conduct problems with and without comorbid anxiety.

As mentioned previously, the emotion-inducing tasks given to children at the 24-month assessment failed to, on average, successfully elicit a biological stress response. This also led to decreased variability in HPA reactivity in the population. Thus, the current study should be replicated with a stress task that elicits greater variability in HPA reactivity in order to more accurately determine how biological vulnerabilities in stress response relate to other domains of risk to influence the development of conduct problem subgroups.

Finally, poor parenting practices such as inconsistent reinforcement and application of consequences (e.g., Beauchaine & Neuhaus, 2008); limited modeling of empathy (Fowles & Dindo, 2006; Hawes & Dadds, 2005); as well as deficits in cognitive functioning such as poor inhibitory control (e.g., Reid & Eddy, 2002) have been associated with biological vulnerabilities and later development of conduct problems. Thus, additional risk factors in early childhood such as other parenting practices, maternal depression, and cognitive functioning, should also be assessed as potential moderators of later conduct problem development in this sample.

Conclusions and Contributions
Researchers often call for an integrative approach, involving the assessment of interrelationships among biological, behavioral, and parental domains of risk across early childhood, to determine the developmental mechanisms underlying externalizing problems (Bronfenbrenner & Morris, 1998; Cicchetti & Tucker, 1994; Rutter, 1990; Sameroff & Chandler, 1975; Shaw, Keenan, & Vondra, 1994; Sroufe & Rutter, 1984). However, a dearth of empirical investigations utilizing this approach remains in the developmental literature. Moreover, investigations of how interactions among these domains of risk at key developmental time points in early childhood predict distinct phenotypes of children who have conduct problems with and without comorbid anxiety are even more scarce.

The current investigation addressed this research gap and made three contributions to the extant literature. First, in infancy, this study revealed different developmental pathways related to children who have conduct problems with and without comorbid anxiety at 5 years of age. Specifically, children who were characterized by heightened biological stress responsivity and exposed to intrusive parenting are more likely to develop conduct problems without comorbid anxiety. Second, after the first year of life, children's behavioral reactivity and regulation are important in distinguishing the underlying mechanisms associated with the development of CP, COMORBID, and COMPARISON subgroups at 5 years of age. Lastly, during toddlerhood, interrelationships among all three domains of risk—biological stress responsivity, behavioral reactivity and regulation, and parenting styles—have the most explanatory value when predicting the development of conduct problem subgroups. Overall, the current study is a preliminary step toward explicating how the dynamic and complex interrelationships among children's biological stress responsivity, children's behavioral reactivity and regulation, and exposure to parenting styles in infancy and toddlerhood may lead to the development of conduct problems with and without comorbid anxiety in early childhood. Taken together, these findings provide a preliminary framework for the design of more targeted and efficacious preventative programs.
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