Examining the Interplay of Vagal Tone and Parenting as Predictors of Child Psychopathology

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Abstract

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Emotion dysregulation is a critical factor in the development of psychopathology (Cicchetti, Ackerman, & Izard, 1995), and deficits in emotion regulation are a core component of many psychiatric disorders (DelCarmen-Wiggins, 2008). Given its importance, understanding factors that contribute to the development of emotion dysregulation is essential for identifying mechanisms underlying the development of psychopathology. Rearing behaviors have been identified as key predictors of children's emotional reactivity and regulation strategies (Eisenberg, Cumberland, & Spinrad, 1998) and potential targets of intervention. This study tested bidirectional relations between vagal tone, a physiological indicator of emotional reactivity, and parenting behaviors. More specifically, we examined how initial levels and changes in vagal tone predicted anxiety, depression, and disruptive behavior problems in young children, and how parenting shapes children's vagal tone over time. In addition, children's cognitive self-regulation was purported to interact with emotional reactivity, serving to amplify or mitigate the effects of emotional dysregulation. Thus, we considered how these processes might vary across levels of children's self-regulation, identifying how executive control may moderate the relation of emotional reactivity to parenting and psychopathology.

These hypotheses were tested using a sample of 305 3-year-old children and their mothers assessed across four time points, each separated by nine months. Children's physiologic

emotional reactivity and observations of maternal parenting were measured when children were 36-, 45-, and 54-months old and were examined as predictors of psychopathology at 63-months of age. The results indicate that children's vagal tone, as measured by Respiratory Sinus Arrhythmia, did not predict the emergence of psychopathology. Instead, symptoms of anxiety, depression, and disruptive behavior problems persisted from early through late preschool. Further, initial levels and changes in vagal tone were predicted by children's anxiety and depression symptoms at the start of the study. In addition, children's executive control moderated the effect of initial vagal tone in shaping parenting. Specifically, children low in self-control elicited less maternal responsiveness, which persisted across time. Overall, these findings suggest that the mechanisms of developmental psychopathology are dynamic and complex. It is therefore important to account for dual components of regulation, emotional and executive, in shaping maternal behavior across time.

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Dedication

To my grandfather, Richard, for his lifelong love of learning and tenacious passion for improving society so that future generations may thrive. To my husband, Jeffrey, for his steadfast and unyielding support of my dreams.

Introduction

Emotional reactivity and regulation are important for understanding the etiology of psychopathology, as difficulties with emotion are not only risk factors for, but also core characteristics of some forms of psychopathology (DelCarmen-Wiggins, 2008). Understanding the contribution of reactivity to psychopathology may be particularly important in children growing up in high-risk contexts, such as those living in low income households, where exposure to stress contributes to emotion dysregulation. Moreover, emotional reactivity may be shaped or constrained by parenting behaviors (Propper et al., 2008) and children's self-regulation (Carver et al., 2008), which may in turn predict psychopathology. Examining these factors across early childhood will highlight important mechanisms in the development of mental health problems.

Emotional Reactivity

Emotional reactivity is genetically based responsiveness to stimuli and reflects a proclivity to exhibit heightened levels of activation in emotions, thoughts, and actions (Rothbart & Bates, 2006). A physiological marker of emotional reactivity is vagal tone, which reflects parasympathetic influence on heart rate variability via the vagus nerve (Bernston, Quigley, & Lozano, 2007). A noninvasively obtained index of vagal tone is respiratory sinus arrhythmia (RSA), which reflects parasympathetic influence on cardiac variability (Porges, 1995) by assessing the variability in heart rate across the respiratory cycle attributable to the 10th cranial nerve (Porges et al. 2007)[1]. Levels of RSA have been linked to emotional reactivity such that low basal RSA indicates increased emotional lability, inflexibility, dysregulation, and physiological stress (Porges, 1995). Conversely, RSA regulation indicates vagal regulation, which quantifies changes RSA in response to environmental stimuli (Beauchaine, 2001). Thus,

[1] The terms vagal tone and basal RSA will be used interchangeably in the theoretical discussions of this manuscript. This stems from the use of RSA as an indirect indicator of vagal tone and reflects variability in the literature in authors' reference to vagal tone and RSA as an index of the underlying construct of physiologic reactivity.

basal RSA is thought to indicate trait-level emotional reactivity, while regulation reflects state-dependent changes in emotional regulation (Beauchaine, 2001). Therefore, this study examined basal RSA given the interest in examining a constitutionally-based indicator emotional dysregulation.

Polyvagal theory, put forth by Porges (1995), indexes parasympathetic activity based on variation in heart rate as a result of innervations of the sino-atrial (SA) node by the right branch of the vagus nerve, also called the smart vagus. The smart vagus originates in the nucleus ambiguus and contains efferent and afferent fibers projecting to multiple organs in the body including the heart (Porges, 1995). Vagal efference serves to inhibit cardiac activity by input from the right nucleus ambiguus on firing in the SA node. Firing serves to slow the heart rate during exhalation and increase heart rate during inhalation. Decreased efferent outflow has an immediate effect on heart rate and serves to increase measures of RSA (Beauchaine, 2001; Porges, 1995). Alternatively, vagal afference originates in the heart and projects to the nucleus tractus solitarius. The origination of the vagus nerve in the brainstem and its composition of afferent and efferent fibers allow integration of neurophysiological pathways with cardiac activity through this neurochemical feedback loop (Porges, 2007). Importantly, the nucleus ambiguus directly communicates with the amgydala, linking efferent activity from the brainstem to changes in cardiac activity (LeDoux, 2000; Porges, 1995).

Changes in RSA can be observed across development as myelination of vagal fibers increases. Research examining genetic and environmental contributions to RSA suggests modest heritability (25-31%) of baseline measures and moderate heritability (50%) of RSA regulation (Snieder, Boomsma, VanDoornen, & DeGeus, 1997). Porges (1995) notes that transitory vagal withdrawal (or regulation) is common in response to internal or external stressors. However, exposure to chronic adversity or stress may result in long-term depression of RSA. This may be particularly important during the preschool period, a developmental stage marked by rapid neurological, physiological, and behavioral development.

Vagal Tone and Psychopathology

As noted above, emotional lability and dysregulation are central to expressions of psychopathology. Thus, not surprisingly low basal RSA has been widely linked to psychopathology in children and adults. Previous research suggests that RSA may represent a general vulnerability for psychopathology (Beauchaine, 2001). However, emerging studies suggest that low baseline RSA may serve as a differential indicator among psychiatric disorders. In particular, research examining the role of RSA in internalizing disorders has been equivocal. Studies have identified that low basal RSA or vagal tone is related to internalizing problems or precursors of internalizing disorders (e.g., inhibition) in young children (Forbes, Fox, Cohn, Galles, Kovacs, 2006; Rubin, Hastings, Stewart, Henerson, & Chen, 1997). However, less attention has been directed toward identifying specific pathways implicated in the development of anxiety and depression in young children. Importantly, researchers have been able to distinctly classify children as anxious or depressed, suggesting differences in the etiology of these disorders. Moreover, there are differences in the core emotional components of each disorder, including sadness for depression and fear for anxiety (Brandy & Kendall, 1992), as well as differences in relation to low positive affect which is related to depression (Watson, Clark, & Carey, 1998). Extending this to the physiological substrates of psychopathology, emerging evidence suggests potentially different associations between RSA and types of internalizing disorders. Particularly, autonomic hyperarousal as indexed by high sympathetic activation and low vagal activity has been linked with anxiety disorders (Kagan, Reznick, & Snidman, 1987; Mezzacappa, Tremblay, Kindlon, & Saul, 1997). Moreover, robust associations have been found between panic disorder and impaired vagal reactivity in adults (Friedman, 2007). In children, Monk et al. (2001) demonstrated that children with anxiety disorders exhibited reduced heart rate variability to novelty. With regard to depression, a recent meta-analysis demonstrated a small effect size for the relation of basal RSA and depression in adults (Rottenberg, 2007). However, this analysis only included published manuscripts, the

majority of which did not control for the presence of related disorders (e.g., anxiety). Recently, one study examined the differential relation of depression and anxiety in pre-adolescent children (Greaves-Lord et al., 2007). The results were such that basal RSA was related to concurrent anxiety but not depression symptoms, providing a robust test of differential relations between RSA and types of internalizing disorders. The current study aims to replicate and extend these findings by examining the relative association of basal RSA with anxiety and depression in a sample of preschool children.

Externalizing symptoms, particularly disruptive behavior problems (DBP), are among the most common psychiatric problems in young children (Beauchaine, Strassberg, Kees, & Drabick, 2002). Disruptive behavior problems are characterized by high emotional lability and are behaviorally expressed as aggression, tantrums, and defiance. Moreover, these problems show a normative decline beginning at age two (Calkins, Blandon, Williford, & Keane, 2007). Disruptive behavior problems and related disorders, including oppositional defiant disorder (ODD), often co-occur with anxiety and depression in young children (Lahey, 2008). Low basal RSA has been linked to conduct problems and ODD in pre-adolescent and adolescent males (Beauchaine et al., 2007). However, the relation between RSA and disruptive behavior problems has received mixed support in samples of preschool children, with some studies finding that lower RSA relates to more externalizing symptoms (Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996) and other studies finding no relation (e.g., Beauchaine et al., 2007). This study will help to clarify the relation between basal RSA and disruptive behavior problems. Specifically, we will examine initial levels and changes in baseline RSA as a predictor of anxiety, depression and DBP. Moreover, we will parse out correlations among mental health problems by controlling for co-occurring disorders (i.e., depression will be entered as a covariate in analyses examining anxiety and vice versa), allowing unique pathways to be identified.

Parenting

Parenting is an important predictor of children's social and emotional adjustment (Maccoby, 2000). Researchers tend to view parenting behaviors as falling along dimensions, including behaviors that convey parents' affect toward the child and behaviors that reflect control (e.g., Frick, 1994; Loeber & Stouthamer-Loeber, 1986; Maccoby, 2000; McLeod, Weisz & Wood, 2007). However, recent research highlights increasing attention to parental responses to children's emotions, including emotion responsiveness and emotion coaching (e.g., Gottman, 1997).

The affective quality of the parent-child relationship has been typically described along the dimension of warm and accepting versus critical and rejecting. This dimension reflects parents' positive affect, appreciation, affection, and involvement with their children and warm, supportive, and open family environments foster well-being in children. Conversely, family environments laden with emotional expressions of negativity, rejection, and diminished warmth foster maladjustment and the development of internalizing and externalizing problems (Downey & Coyne, 1990; Herman & McHale, 1993; Siqueland et al., 1996). For example, parental acceptance is related to fewer emotional and behavioral problems (e.g., Papp, Cummings & Goeke-Morey, 2005), and parental warmth is related to empathy and prosocial behaviors (Bornstein, 1989; Davidov & Grusec, 2006). Parental negative affect, or rejection, predicts higher levels of internalizing and externalizing problems (e.g., Burge & Hammen, 1991; McLeod et al., 2007; Mezulis, Hyde, & Abramson, 2006; Muris, Schmidt, Lambrichs, & Meesters, 2001; Stormshak et al., 2000).

Parental control behaviors have been further delineated in the literature, including behavioral control strategies (e.g., discipline and monitoring) and psychological control strategies (e.g., autonomy granting, overcontrol, and intrusive). These distinctions reflect the target of the parenting behavior (Barber, 1996). Behavioral control focuses on efforts to restrict and manage children's behaviors by monitoring children's activities, conveying rules or standards for

appropriate or desirable behaviors, employing reinforcement for appropriate or desirable behaviors and consequences for inappropriate behaviors, as well as engaging in these behaviors with reasonable consistency. These rearing patterns predict children's emotional and behavioral problems, particularly when parenting strategies are inconsistently applied (e.g., Barber, 1996; Chamberlain & Patterson, 1995; Hill, Bush, & Roosa, 2003), harsh or coercive (e.g., Nix et al., 1999), or include the use of physical punishment (e.g., Deater-Deckard, Dodge, Bates, & Pettit, 1996; Stormshak et al., 2000).

Parental psychological control has been defined as attempts that intrude into a child's psychological and emotional development by stifling independent thinking and self-expression (Barber, 1996; Barber & Harmon, 2002; Stone, Buehler, & Barber, 2002). Such control behaviors may be particularly apparent or detrimental in situations where children are able or expected to function independently (Rubin, Cheah, & Fox, 2001). This type of control has been operationalized in various ways, including low autonomy granting, intrusiveness, negative control, and overcontrol (e.g., Barber, Bean, & Erickson, 2002; McLeod et al., 2007; Rubin, Burgess, & Hastings, 2002; Rubin et al., 2001; Silk, Morris, Kanaya, & Steinberg, 2003). Psychological control negatively impacts child development (Barber, 1996), and may engender risk for internalizing disorders (Barber, Stolz, & Olsen, 2005; Eccles, Early, Fraiser, Belansky & McCarthy, 1997; Siqueland et al., 1996; Stark, Humphrey, Crook & Lewis, 1990; Whaley et al., 1999), although some studies have demonstrated an association with children's externalizing problems (Morris, Steinberg, et al., 2002).

Increasing attention is being focused on the dynamic nature of parent-child interactions by considering rearing behaviors exhibited in response to children's emotions, including acknowledging, supporting, and guiding children's emotional responses. These types of behaviors have included maternal responsiveness, empathic awareness, synchrony, and sensitivity. Responsiveness is thought to capture the presence (or absence) as well as fit of the maternal response, thus encompassing synchrony and sensitivity. In addition, responsiveness

is a multi-dimensional construct that includes responses to children's specific emotional expressions (Davidof & Grusec, 2006) and general needs (Bornstein, Tamis-LeMonda, Hahn, & Haynes, 2008). Emotion-focused parenting behaviors are contingent on children's emotional expressions and may promote children's emotional awareness as well as extend the emotional experience into an opportunity for learning and problem solving (Gottman, 1997). Research has demonstrated that maternal responsiveness to children's negative emotions (e.g., anger) reduces expressions of anger and encourages positive affect (Denham, 1993). More generally, responsive and sensitive parenting predicts children's prosocial adjustment (Bornstein et al., 2008; Davidof & Grusec, 2006). Less research has examined the relation of responsiveness to specific adjustment outcomes, as much of the research examining maternal responsiveness has focused on the parenting of infants and toddlers. Recently, it was shown that maternal responsiveness during infancy predicted children's conduct problems later in childhood, however these results were moderated by children's fear (Lahey et al., 2008).

Thus, research has supported that negative parenting behaviors present a risk factor for the development of adjustment problems in children. However, in drawing general conclusions, it is important to acknowledge the lack of consistency across studies in the labeling and operationalization of parenting behaviors. For example, responsiveness is a term that might capture a range of behaviors that include parental responses to negative affect, sensitivity to infant or child cues, or scaffolding behaviors. Additionally, many variables reported in the literature combine several parenting behaviors including those that cross dimensions (e.g., measures of overprotection may combine measures of control and warmth; Rubin et al., 2001) making it difficult to distinguish the unique effects of specific parenting variables. This lack of standard conceptualization and operationalization of parenting behaviors complicates comparison of the findings from this research with prior studies. The parenting variables for this study were selected to reflect the dimensions of maternal positivity, negativity, responsiveness, and overcontrol, as these rearing behaviors have been examined previously as potentially

relating to children's emotional reactivity and/or psychopathology symptoms.

Parenting as a Predictor of Child Psychopathology

Researchers suggest that warm, supportive, and open family environments foster well-being in children. Conversely, family environments laden with negativity, rejection, and diminished warmth foster maladjustment and predict the development of anxiety (Siqueland Kendall, & Steinberg, 1996; McLeod et al., 2007), depression (Burge & Hammen, 1991; Mezulis et al., 2006; Muris, Schmidt, Lambrichs, & Meesters, 2001), and externalizing symptoms (Muris, Meesters, & van den Berg, 2003). In addition, high, consistent levels of behavioral control denotes setting appropriate limits and monitoring children's behavior, and have been linked to lower levels of externalizing symptoms (Barber, 1996; Caron, Weiss, Harris, & Catron, 2006). Furthermore, low behavioral control, specifically poor supervision, has been linked with oppositional defiant disorder and conduct problems (Frick et al., 1992). Parental control behaviors targeting children's autonomous expressions, thoughts and decision-making have also been linked to the development and presence of anxiety disorders (Messer & Beidel, 1994; McLeod et al., 2007), depression (Barber, 1996; Stark, Humphrey, Crook, & Lewis, 1990), and externalizing symptoms (Aunola & Nurmi, 2005).

Overall, each of these dimensions of parental rearing behaviors has been link with child adjustment problems. Further, some research suggests that there may be specificity in the types of adjustment problems children develop across specific types of parent rearing patterns. For example, Rapee (1997) noted several parenting behaviors were related to children's internalizing problems, including negative and hostile feelings toward the child (rejection and lack of warmth) and overprotection and overcontrol. This supposition has been supported with research linking parental negativity and overcontrol with children's depression and anxiety symptoms (Kiff, Lengua, & Bush, 2011; McLeod et al., 2007). In addition, research on the development of children's disruptive behavior problems highlights the importance of parental monitoring and involvement, consistent discipline strategies, and behavioral control (Burke,

Pardini, & Loeber, 2008) in shaping the emergence of externalizing symptoms.

Although the research summarized above suggests that emotional dysregulation and parenting are both important factors in the development of psychopathology, research investigating the direct effects of each of these variables does not adequately capture the dynamic interplay of individual and familial factors that may contribute to the development and stability of mental health problems.

Vagal Tone and Parenting

Models investigating reciprocal relations of parenting or emotional dysregulation on psychopathology may capture dynamic relations between children and their environment and may elucidate processes by which psychopathology develops. Unfolding the interplay between children's characteristics and parenting behaviors may be important to address in personalizing interventions to maximize effectiveness. In order to understand how individuals shape and are shaped by their environment, transactional models of the effect of parenting and emotional reactivity on psychopathology must be considered. As noted above, parenting is an important predictor of child psychopathology (Maccoby, 2000). Moreover, as suggested by Belsky (1984), children's emotionality is an important determinant of parenting. Variations in emotional reactivity may systematically elicit different parenting behaviors (Karraker & Coleman, 2005), which may in turn reinforce or decrease the child's emotional characteristics (Hinshaw, 2008).

Although bidirectional relations between children and parent socialization strategies have been considered in the literature for quite some time (e.g., Bell, 1968), the majority of research examining the association between parenting behaviors and children's characteristics to the development of psychopathology continues to examine the unidirectional effect of parents on children. However, recent research suggests that children have an evocative effect on parents' behaviors, indicating that models examining socialization strategies need to consider reciprocal relations between parenting behavior and children's characteristics in the development of psychopathology. Such a need was addressed in a special section of the *Journal of Abnormal*

Child Psychology (July 2008), which examined bidirectional associations between parenting and children's psychopathology. The findings support bidirectional models of influence and suggest symmetric effects of children's behavior on parenting and vice versa. That is, children's psychopathology and parenting were both important predictors of each other and of later psychopathology (Pettit & Arsiwalla, 2008). However, several studies have begun to examine bidirectional relations between children's physiological reactivity and parenting (Propper & Moore, 2006). Most of the studies examining parenting and children's RSA examine unidirectional effects, thereby constraining our understanding of how parenting may shape the development of psychopathology. Thus, the proposed study aimed to examine bidirectional relations in order to clarify developmental psychopathology models and increase our understanding of specific pathways to the development of anxiety, depression, and DBP. Below is a review of studies examining relations between parenting and children's RSA. Because few studies have examined relations of RSA to parenting, this review includes studies examining both basal RSA and measures of RSA regulation.

Parent Effects. Examination of parenting in relation to RSA has begun to demonstrate the importance of extending models of parenting in predicting developmental pathways beyond observational measures of child behavior and psychopathology. In infants, observations of maternal sensitivity in children at genetic risk for developing psychopathology were related to increases in vagal regulation across infancy. Specifically, maternal sensitivity predicted better regulation across 9 months, with higher sensitivity in mothers of infants at genetic risk predicting levels of vagal regulation similar to those observed in the control infants (Propper et al., 2008). Similarly, mother-child affective synchrony has been shown to relate to vagal tone in infants such that lower synchrony was related to more dysregulation (Moore & Calkins, 2004; Porter, 2003). However, these studies were cross-sectional and could not tease apart the direction of effects.

Several studies examined the relation of parenting to RSA in toddlers. One study

demonstrated that maternal negative control, but not maternal positive guidance, predicted less vagal regulation in 2-year-old children during emotion eliciting tasks. Thus, higher maternal negative control during parent-child interactions predicted reduced physiological regulation in response to emotionally excitable stimuli (Calkins, Smith, Gill, & Johnson, 1998). In addition, maternal support was shown to relate to RSA regulation, such that higher ratings of maternal support during a challenging laboratory task were concurrently related to more vagal withdrawal and less dysregulation (Calkins & Keane, 2004). A global measure of parenting encompassing hostility, stress, and positive tone measured at age 2 was shown to predict children's vagal regulation at age 5, such that more negative parenting was related to less regulation three years later (Calkins, Graziano, Berdan, Keane, & Degnan, 2008). Few studies have examined these relations beyond infancy and toddlerhood. In a sample of preschool children, maternal negative control predicted vagal regulation and in turn externalizing symptoms (Hastings et al., 2008). The results were such that higher negative control elicited less vagal regulation, and in turn more externalizing symptoms. These studies demonstrate that parenting predicts vagal regulation in infants and young children, although only one known study has examined these relations in preschool. However, the extent to which parenting shapes vagal tone is not as well understood. Few studies have examined the role of parenting in shaping RSA, despite the established hypothesis that physiological measures of reactivity are shaped by environmental factors in early childhood (Propper & Moore, 2006). Further, these associations have rarely been studied beyond infancy, and the pattern of findings is somewhat inconsistent. These variations may be related to the variety of parenting dimensions examined across studies or to variations in contexts in which RSA was measured. The proposed study addressed several of these gaps in the literature by examining longitudinal bidirectional relations of RSA and parenting across a range of parenting behaviors during the preschool period.

Child Effects. Studies examining the relation of RSA to parenting have demonstrated mixed support for the hypothesis that RSA predicts current and future parenting. One study

failed to demonstrate an interaction between RSA and observed frustration in 18-month-old children in predicting maternal positive guidance, negative control, and intrusiveness (Calkins & Johnson, 1998). Only one known study examined bidirectional relations between parenting and basal RSA. Kennedy and colleagues (Kennedy, Rubin, Hastings, & Maisel, 2004) examined cross-lagged panel models in which the effect of parenting and RSA on later levels of each variable could be compared. The results were such that lower vagal tone at age 2 predicted more restrictive maternal parenting two years later. Conversely, restrictive maternal parenting at age 2 did not predict basal RSA at age 4. Similarly, the same study found that high basal RSA at age 2 predicted supportive parenting at age 4, but not vice versa. Examination of relations among maternal overprotection and RSA revealed that, while both remained stable across the study, neither variable predicted changes in one another (Kennedy et al., 2004). This study presents the most rigorous test known to date of bidirectional relations between RSA and parenting. The findings suggest that physiological markers of emotional dysregulation may predict changes in maternal parenting. However, because this study only measured two time points, it could not address whether this pattern of findings predicted future influences of parenting on RSA and vice versa. Moreover, this study looked at whether RSA and parenting predicted future levels of the other. However, despite stability in levels across time, cross-lagged models do not allow modeling of individual variation or growth. The current study used latent growth modeling to assess slope differences in RSA and parenting, and variability in change within individuals.

Executive Control

Executive Control (EC) is a core mechanism of self-regulation and is characterized by processes that inhibit a dominant response in order to perform a subdominant response, allowing behavioral regulation in the service of current and future needs (Rueda, Posner, & Rothbart, 2005). The specific mechanisms underlying EC are associated with activation of the anterior cingulate cortex (ACC), which has been implicated in attention focusing or regulation,

and the lateral prefrontal cortex, which is associated with inhibitory control (Posner & Rothbart, 2000; Rueda et al., 2005). Attention regulation serves to adjust reactivity, and is thought to be important in the modulation of emotion-related processes and states through the regulation of one's awareness by attention shifting or focusing, and regulating exposure to stimuli (Derryberry & Reed, 1996).

Importantly, the ACC is housed within the limbic system, which links it to brain structures associated with emotional processing (Rueda et al., 2005). Imaging studies have demonstrated dorsal ACC activation during Stroop-like tasks (Bush, Luu, & Posner, 2000), which has been linked with concurrent activation of the dorsolateral prefrontal cortex during tasks requiring attention control (Carver et al., 2008). Conversely, the rostral and ventral areas of the ACC represent the affective subdivision, which is involved in the appraisal of emotional information and regulation of responses (Bush et al., 2000). Imaging studies suggest reciprocal activation of these structures such that activation of the affective ACC results in deactivation of the cognitive ACC (Bush et al., 2000). In particular, the rostral ACC appears particularly important in the cognitive control of emotional stimuli as it represents the pathway by which attentional processing and the amgydala converge (Carver et al., 2008). Moreover, the cognitive and affective regulatory areas of the ACC exert effects on lower structures, including those related to autonomic arousal. However, subcortical structures also exert bottom-up activation, creating a feedback loop in which regulatory structures influence and constrain reactivity structures and vice versa (Thompson, Lewis, & Calkins, 2008). This feedback pathway identifies a potential mechanism whereby EC may interact with RSA to predict psychopathology. In particular, decreased regulation of cognitive and emotional information may serve to amplify the effects of emotion dysregulation. Preliminary research suggests that deficits in attention have been linked to psychopathology such that depression (Carver et al., 2008) and anxiety (Muris, Meesters, & Rompelberg, 2006) are related to a negative bias to attend to threatening information. Moreover, deficits in executive control may prevent children from inhibiting dominant responses

that may intensify the negative effects of high emotional reactivity (Muris & Ollendick, 2005). Thus, this study aims to examine whether executive control interacts with basal RSA and parenting in predicting psychopathology in children.

This Study

Person by environment interactions are increasingly important in understanding the development of psychopathology given that pathways to mental health problems are not linear and immutable, but rather occur through reciprocal transactions between children's current functioning and environmental factors (Hinshaw, 2008). Individual factors identified as vulnerabilities for psychopathology include emotional dysregulation and poor cognitive self-regulation (Eisenberg et al., 2001). Important environmental factors include parenting, which has been shown to relate to the development of psychopathology and has been identified as a fruitful target of intervention (Hibbs & Jensen, 2002), particularly with young children.

Emotional reactivity or dysregulation is consistently linked to psychopathology (Beauchaine, 2001). An important physiological substrate of emotional dysregulation, respiratory sinus arrhythmia (RSA), can facilitate our understanding of the emergence of psychopathology. RSA is an indicator of parasympathetic arousal, and more specifically, basal RSA has been regarded as a marker of emotional inflexibility and dysregulation (Beauchaine, 2001) and has been linked to the development of anxiety disorders, depression, and disruptive behavior problems (Beauchaine, Gatzke-Kopp, & Mead, 2007; Monk et al., 2001; Rottenberg, 2007).

It is unclear to what degree RSA is influenced by environmental factors and whether it may be shaped by parenting. Changes in tonic RSA in response to parenting across time would identify one manner in which parents affect the development of children's neurobiological systems, which in turn, might be an important pathway for the development of psychopathology. In addition, links between parenting and RSA may identify a potential target for intervention, as this would suggest that changes in parenting could alter the trajectory of physiological underpinnings of psychopathology. The preschool period presents a period of

rapid development and has been linked to the onset of psychopathology (Keenan, Shaw, Delliquadri, Giovanneli, & Walsh, 1998); thus, presenting an important opportunity for intervention.

Importantly, emotional reactivity varies according to an individual's cognitive self-regulation. A core mechanism of self-regulation is executive control (EC), which reflects executive-based individual differences in attention and inhibitory control that constrain the degree of emotional reactivity and expression (Rueda, Posner, & Rothbart, 2005). Thus, there is a potential for low EC to amplify the effect of emotional reactivity in relation to psychopathology (Carver, Johnson, & Joormann, 2008). This study aimed to examine whether low executive control amplified the relation between RSA, parenting, and psychopathology. Variations in the association between RSA and psychopathology across levels of EC would identify an important person by environment interaction in the emergence of mental health problems in young children.

Further, the effects of emotional reactivity and parenting on adjustment might be particularly important for children growing up in low income households. These children are at elevated risk for developing problems such as internalizing (Gilman, Kawachi, Fitzmaurice, & Buka, 2002) and externalizing (Dearing, McCartney, & Taylor 2006). Parents of low income families also show parenting practices that are related to child psychopathology (McLoyd, 1998). One mechanism of the effects of low income, and the parenting problems associated with low income, on children's symptomatology might be emotion dysregulation. Therefore, examination of person by environment interactions including RSA, EC, and parenting might elucidate one pathway through which low income predicts mental health problems in children.

Thus, this study examined the bidirectional relation between parenting and children's emotion dysregulation in predicting the early emergence of psychopathology in a high-risk sample of preschool children using latent trajectory modeling. Further, we considered whether executive control served to amplify or attenuate the role of RSA in the development of psychopathology. In this study, we tested the following aims.

Aim I: Examine whether initial status and changes in emotional dysregulation differentially predict depression, anxiety, and disruptive behavior problems in young children. In this aim, we expected that initial status and changes in basal RSA would differentially predict child psychopathology. Specifically, we predicted that low baseline RSA would more strongly predict anxiety and disruptive behavior problems than depression.

Aim II: Examine whether children's emotional dysregulation and parenting predict changes in one another over time, and in turn, predict children's emerging psychopathology.

Aim IIa: It was expected that mothers' rearing behaviors (positive affect, negativity, responsiveness, autonomy granting, and intrusiveness) would predict changes in children's emotional dysregulation as measured by RSA. Maternal negativity and negative control would predict decreases in baseline RSA, whereas positive affect, responsiveness, and autonomy granting would predict increases in baseline RSA.

Aim IIb: We predicted that initial levels in parents' behavior would in part predict child psychopathology through changes in children's emotional dysregulation.

Aim IIc: We also predicted that children's emotional dysregulation, or low baseline RSA, would have an evocative association with maternal parenting, predicting higher rates of maternal negativity and intrusiveness and lower rates of positive affect, responsiveness, and autonomy granting, and in turn shape RSA.

Aim IId: We further predicted that initial levels in baseline RSA would, in part, predict child psychopathology through changes in parents' behavior.

Aim III: Examine person x environment interactions to determine whether the relations among emotional dysregulation, parenting, and psychopathology vary across levels of self-regulation.

Aim IIIa: We expected that lower executive control would amplify the relation between low baseline RSA and negative parenting behaviors.

Aim IIIb: We further expected that lower executive control would amplify the relation of low baseline RSA and changes in RSA with later psychopathology.

Method

Participants

Participants were a community-based sample of 305 children and their mothers who were assessed during laboratory and/or in-home interviews at four time points, each separated by nine months. Time 1 interviews began when children were 36-months (*M* = 36.67 months, *SD* = .89, Range = 35.52 - 40.34). At Time 2 children were 45-months (*M* = 46.02 months, *SD* = 1.28, Range = 43.56 – 51.29), at Time 3 children were 54-months of age (*M* = 55.08 months, *SD* = 1.13, Range = 51.71 – 59.56), and at Time 4 children were 63-months of age (*M* = 63.92 months, *SD* = 1.49, Range = 54.70 – 76.19). Sixty eight percent of study participants were recruited from the University of Washington Subject Pool, a voluntary database of mothers who consented to be contacted for participation in future research studies. Mothers were approached by university staff after giving birth at the University of Washington Medical Center. Access to this database of these potential participants is then made available to researchers at the University of Washington for a fee. The remaining thirty two percent of families were recruited from various public- and privately-funded sources including daycares, preschools, libraries, health clinics, charitable agencies, and organizations serving low income families (e.g., county-sponsored “play and learn” groups, food banks, Catholic Community Services). Community-based recruitment sites (i.e., not the subject pool) were initially approached by informational letter outlining the study goals and providing contact information for the study coordinator. The project director followed-up each letter by contacting the agency organization to provide additional information and answer questions. Interested sites were provided with Family Contact Information forms to distribute to mothers of 3-year-old children. Completed forms were returned to the project and interested families were phoned by the project coordinator. The study staff tracked response rates for each agency and organization, and sites were provided an honorarium for referring families. Sites received an honorarium of $100 when 90% or more of the families returned the forms. Lower response rates were proportionally

compensated (e.g., $75 for a 75% response rate or $50 for a 50% response rate.

Children with developmental disabilities (except learning disabilities), and families not fluent in English were excluded from the study to ensure adequate comprehension of the questionnaire measures and behavioral tasks. In addition, children taking anticonvulsant or blood pressure medications were excluded to ensure the accuracy of physiological measures. A female primary caregiver was required to participate. Overall, 1,459 families were contacted for potential participation. Three hundred and eighteen families were scheduled for initial visits and 305 Time 1 assessments were completed. Of the remaining 1,141 families, 655 were ineligible due to the child's age, non-English speaking status, presence of developmental delays, or living outside of King County. In addition, recruitment was stratified across income categories, and families were turned away from the study if a sufficient number of participants from an income category were already assessed.

Annual family income was diversely represented with approximately 12% of families earning less than 21,200 per year, 8% between 21,201 and 28,400, 13% between 28,401 and 39,200, 24% between 39,201 and 58,400, 16% between 58,401 and 75,000, 9% between 75,001 and 100,000, 15% between 100,000 and 150,000, and 3% above 150,000. Further, 16% met the 2009 federal poverty guidelines, as assessed by an income to needs (mother-reported family size) ratio. The distribution of family income category by recruitment source is outlined in Table 1. As noted, there were few differences in recruitment methods across income categories. The sample included 8% African American children, 6% Asian American children, 10% Latino or Hispanic children, 3% children from Indigenous cultures or Pacific Islanders, 63% European American children, and 9% multiple ethnic or racial backgrounds. Mothers declined to provide information on children's ethnic or racial background in four cases. Mothers were generally well educated with the modal level of educational attainment at a college or university graduate. Eighty percent of families consisted of two-parent households.

Attrition was low with 96% of participants remaining in the study at Time 4. Few

assessments were missed across time points (missing assessments: Time 2 = 3; Time 3 = 14). At the time of this writing partial data was available for Time 4, with 246 completed responses. Assessments were missing for 44 families because the final time point was not yet scheduled. Therefore, these data points can be considered missing by design. In addition, six families were contacted but were not scheduled for Time 4 assessments. Additional missing data points across assessments was in part due to technical problems, and therefore can be considered missing at random: time 1 (n = 18), time 2 (n = 14), and time 3 (n = 12). Participants with missing data on any variable in the first three assessments were compared to those with complete data on all variables: demographics (child sex and age, family income, maternal education, single parent status), parenting (positive affect, negativity, responsiveness, autonomy granting, and intrusiveness), RSA, executive control, and outcomes (anxiety, depression, and disruptive behavior problems). The *t* tests indicated that participants with any missing data (n = 186)[2] differed from those with no missing data (n = 120) only on family income and Time 4 anxiety symptoms. Families without any missing data reported higher incomes at the start of the study (missing, $M = 8.28$, $SD = 4.11$; no missing, $M = 9.47$, $SD = 3.52$, $t(280) = 2.69$, $p < .001$). However, the relation of income to missingness was a small effect ($r = -.15$, $p = .01$) and was considered as a covariate in all analyses, limiting potential bias related to income differences. Further, families with missing data were more likely to have children with higher anxiety symptoms at the end of the study (missing, $M = 3.31$, $SD = 3.05$; no missing, $M = 2.01$, $SD = 2.31$, $t(231) = -2.38$, $p = .02$). These differences were related to family income ($r = -.36$, $p < .001$) and maternal education ($r = -.22$, $p < .001$), which were included as covariates in all subsequent analyses. Further, the strength of association between familial demographics and Time 4 anxiety symptoms did not reach previously cited thresholds for introducing substantial bias (e.g., $r > .40$; see Collins, Schafer, & Kam, 2001) suggesting little

[2] The high number of missing data was due to 61 families missing data for Time 4, as well missing data for RSA (n = 127). RSA was most commonly missing for only one time point (n = 86) with few children (n = 9) missing all three assessments.

bias was introduced due to missing data.

Procedure

Structured 2-hour interviews were conducted at the University of Washington and were attended by the participating child and his/her female primary caregiver. After obtaining maternal consent and child assent, child participants completed a series of neurocognitive and behavioral tasks by trained interviewers. During the assessment, a neutral baseline for children's physiologic emotional dysregulation was collected. In an adjacent room joined by a one-way mirror, mothers were administered a series of questionnaire measures. Trained interviewers read all instructions and items allowed in order to minimize errors in interpretation and address potential problems with literacy in parents. After conclusion of the child tasks and the parent interview, mothers rejoined their children for the parent-child interaction tasks. Dyads participated in a series of interaction tasks, which were video-recorded for later observational coding. Dyads were scheduled for subsequent assessments approximately 9- (M = 9.40, SD = .66), 18-months (M = 9.04, SD = .90), and 27-months (M = 8.87, SD = .81) after the initial assessment. Assessment procedures were identical across time points. Families received $70 compensation for participating at Time 1, with the compensation increasing by $20 each additional year the families participated.

To facilitate participant retention, assessments were conducted in families' homes if requested by the mother. Home-based assessments were minimal and often completed when family circumstances (e.g., recent birth of a new child) made it difficult for the family to travel to the laboratory. Few in-home assessments were conducted (Time 1 = 2, Time 2 = 10, Time 3 = 14, Time 4 = 11). Aside from the setting, all procedures and tasks were administered in a manner equivalent to that of the laboratory assessment.

Measures

Respiratory Sinus Arrhythmia (RSA). RSA was measured using a 2-lead electrocardiograph (ECG) and respiratory band. These materials were purchased from Biopac

PRO Lab 3.7.1 (Goleta, CA). Electrodes were placed on the child's right clavicle and lower left abdomen. A ground electrode was placed on the left, upper chest. The respiratory band fit approximately under the child's ribcage. To collect the baseline measure electrodes and the respiratory band was placed securely and comfortably on the child. Following system calibration, a 2-minute rest was conducted during which the experimenter read the child a neutral story. A team of trained undergraduate research assistants (RAs) processed each file to edit and clean the acquired ECG and respiration data. RAs received specialized training in ECG data cleaning and editing procedures prior to working independently. In addition, they received ongoing supervision during weekly meetings with project staff.

Respiratory Sinus Arrhythmia (RSA) was calculated using Porges' (1985) method of spectral analysis, which involves decomposing the ECG R-R time series into component heart rate variability frequencies using fast Fourier transformations. The resulting components are expressed in a spectral density function, specifying the amount of spectral power within given frequency bands. Spectral power was divided into low- to mid-frequency variability (< .24 Hz) and high-frequency variability (>.24 Hz). The high-frequency range is primarily where parasympathetic influences on heart rate variability are observed (Akselrod et al., 1981) for young children (Berntson et al., 2007). In this study, ECG R-waves were detected and time stamped using Acqknowledge software (Goleta, CA) and then exported along with the original ECG recordings. Custom-purpose MatLab software was used to overlay inter-beat interval (IBI) time series on the ECG wave forms allowing the accuracy of the R-wave detection to be verified and providing a method for detecting abnormalities in the R-spikes. Epochs contaminated by large mechanical artifacts or electrical interferences that distorted or obscured the ECG waveforms were excluded from analyses. Artifact-free segment of the corrected IBI sequence were resampled at 2.8 Hz. Each re-sampled segment was filtered with a 21-point, third order polynomial derived from Litvack et al. (1995; see Figure 3A in Litvack et al., 1995) and subtracted from the unfiltered segment to obtain a high-pass filtered segment. The derived

segment was then converted into a sequence of Fourier coefficients using Fast-Fourier transformation. Respiratory Sinus Arrhythmia (RSA) was defined as the log of the average power of Fourier coefficients falling in the frequency range between 0.24 Hz and 1.04 Hz (reported in units of $\ln(ms^2)$). The age-specific frequency pass-band used for RSA was selected to match developmentally normed respiration rates for young children (Berntson et al., 2007 Calkins & Keane, 2004; Hastings et al., 2008).

Parenting. At each time point, mothers engaged in semi-structured interactions with their children. Dyads were observed in a series of activities (7 minutes restricted play/mother-directed play, 7 minutes free play/child-directed play, 7 minutes instructional activity, 3 minutes cleanup; see Kerig & Lindahl, 2001) totaling approximately 25 minutes. At Time 1, dyads engaged in a restricted and then free play activity. During restricted play, the mother was instructed to allow the child to play with all the toys in the room, except for those located in a designated area, a freely accessible book case of highly desirable toys. This allowed the observational rating of parental control strategies. After 7 minutes, the dyad was informed that they could now play with all of the previously restricted toys, allowing observation of maternal positive affect and responsiveness to the child's cues. At Time 2 and 3, dyads engaged in a child-directed play task followed by a mother-directed play task. In child-directed play, mothers were instructed to follow the activity and/or game established by the child. Further, explicit instructions not to introduce new activities or change the game were provided. After 7-minutes the dyad was instructed to shift to mother-directed play, in which mothers were asked to change the activity to a game of their choice. At all time points, the play activities were followed by a figure-building task. During the block task, mothers were instructed to help the child build a Lego (Time 1 and 2) or pattern block (Time 3) figure that is challenging for the child, allowing observation of maternal autonomy granting, guidance, responsiveness, and intrusive control. The decision to change the instructional activity was based on developmental changes in children's abilities to solve puzzles and follow instructions. Finally, a cleanup segment allowed

additional coding of control strategies.

Parenting behaviors were coded from videotapes of mother-child interactions by undergraduates using a coding system adapted from existing, well-established systems: the *System for Coding Interactions and Family Functioning* (SCIFF: Lindahl & Malik, 2000), the *Parenting Style Ratings Manual* (Cowan & Cowan, 1992), and the *Parental Warmth and Control Scale* (Rubin & Cheah, 2000). Parenting behaviors of interest were selected from each coding system and adapted, as necessary, for use with this age group. Codes for maternal positive affect, negativity, responsiveness, autonomy granting, and intrusiveness were rated on 6-point Likert scales with 0 indicating the absence of the behavior and 5 indicating the highest level of behavior. Tasks were coded in 1-minute segments, with each segment receiving a score on each code. A mean score was then achieved by calculating the average for each task. Inter-rater reliability was calculated by double coding 20% of tapes at Time 1 and 10% of tapes at remaining time points. Reliability was calculated using intra-class correlations (reported below).

Positive Affect (Time 1: ICC = .77, Time 2: ICC = .78, Time 3: ICC = .80) captured the frequency and level of behavioral and verbal expressions of happiness, comfort, and connection in the interaction and warmth toward the child. *Negativity* (Time 1: ICC = .75, Time 2: ICC = .80, Time 3: ICC = .80) assessed the negative tone or level of tension expressed by the mother and includes verbal and non-verbal expressions of irritation or frustration with the child that are critical, rejecting, or invalidating. *Responsiveness* (Time 1: ICC = .67, Time 2: ICC = .77, Time 3: ICC = .86) to children's expressions of negative affect, difficulty with the task, or general needs was rated. Effective responsiveness refers to the parent's ability to intervene when the child needs and disengage when the child was functioning independently again. *Autonomy Granting* (Time 1: ICC = .71, Time 2: ICC = .48, Time 3: ICC = .74) was coded based on whether or not the mother allowed and encouraged a range of autonomous behaviors, giving the child room to explore his/her surroundings, assert his/her needs and

desires, take credit for accomplishments, and express ideas freely. *Intrusiveness* (Time 1: ICC = .81, Time 2: ICC = .85, Time 3: ICC = .86) was the frequency of unsolicited interventions made by the mother during the interaction tasks, including interruptions of the child's autonomous behavior in the absence of cues for assistance. Parenting behaviors were correlated within dimension and across task. Relations across tasks, within each parenting behavior, and across time points were significant and in the expected directions (rs ranged from .22 to .72). Therefore, in an effort to reduce the number of variables and increase observations of maternal behavior across contexts (tasks), maternal behaviors were averaged within each code across tasks. This allowed for the use of one indicator of each observed parenting behavior.

Additionally, maternal parenting behaviors were moderately correlated across dimensions (see Tables 2 through 4); and the pattern of association did not differ greatly across time as positive parenting dimensions (e.g., positive affect, responsiveness, and autonomy granting) were positively related to one another and inversely related to parenting variables reflecting maternal negativity or control (i.e., negativity and intrusiveness). Similarly, the pattern of relations was in the expected direction for variables capturing maternal negativity and control behaviors. The consistency of relations suggested that collapsing across dimensions to create variables reflecting parenting domains might be useful. However, given the broader study goal to consider specific parenting behaviors, individual parenting variables were retained as originally measured.

Executive Control (EC). The neurocognitive assessment of executive control included a comprehensive battery of neuropsychological and laboratory tasks, measuring multiple components of EC including attention regulation and cognitive and behavioral inhibitory control. In order to create an index of executive control, aspects of self-regulation were measured from the executive function battery of the NEPSY-II, a developmental neuropsychological assessment battery (Korkman, Kirk, & Kemp, 2007), as well as tasks developed by Murray &

Kochanska (2002).

Executive control was assessed using five tasks. The *Inhibition* and *Auditory Attention* subscales of the NEPSY-II were designed for use with children 5 and older. However, in this study the scales were administered to the children younger than five because of the broader study aim to assess the development of executive control. Thus, these tasks were understandably difficult for children in this sample. The *Inhibition* subtest assesses a child's ability to inhibit a dominant response in order to enact a novel response. Specifically, children were shown an array of circles and squares and then asked to label each shape in an opposite manner (e.g. say circle when they see square) while being timed. The *Auditory Attention* subtest is a continuous performance test that assesses the ability to be vigilant and to maintain and shift selective auditory set. Children were required to listen to a series of words and respond only when they heard a specific target word, while refraining from response to all other words. Total scores for both scales were the proportion correct responses across the task. Average scores at Time 1 were 0.09 (*SD* = 0.24, *Range* = 0.17 – 0.89) and 0.14 (*SD* = 0.28, *Range* = 0.00 – 1.00) for *Auditory Attention* and *Inhibition* respectively.

Behavioral inhibitory control was assessed using the *Monkey-Dragon* task (Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996), which required children to perform actions when a directive is given by a monkey puppet, but not when given by a dragon puppet. Children's actions were scored as performing no movement, a wrong movement, a partial movement, or a complete movement, with scores ranging from 0-3. Total scores were the proportion of the score across both monkey and dragon items to the total possible score. The average score at Time 1 was 0.62 (*SD* = 0.20, *Range* = 0.33 – 1.00).

Cognitive inhibitory control was assessed using the *Day-Night* task (Gerstadt, Hong, & Diamond, 1994), which required the child to say "day" when shown a picture of moon and stars and "night" when shown a picture of the sun. Children's actions were scored 1 for correctly providing the non-dominant response or 0 for providing the dominant response. Total scores

were the proportion of correct responses. The average total score at Time 1 was 0.44 (*SD* = 0.33, *Range* = 0.00 – 1.00).

The *Head, Toes, Knees, Shoulders* (HTKS) task integrates attention and inhibitory control (Cameron, McClelland, Jewkes, Connor, Farris & Morrison, 2008). Children were asked to follow the instructions of the experimenter, but to enact the opposite of what the experimenter directs (e.g. touch toes when asked to touch head). Behaviors were coded as 0 points if the child touched the directed body part, 1 point if the child self-corrected his/her behavior, and 2 points if the child only touched the opposite body part. Total scores were the proportion of the score across items to the total possible score. The average score for Time 1 was 0.03 (*SD* = 0.09, *Range* = 0.00 – 0.65). Twenty percent of all executive control tasks were independently re-scored. ICC's on all tasks ranged from 0.72 to 0.98.

An overall executive control score was computed as the mean of the proportion scores of the individual tasks. Executive control scores were considered missing if > 50% of the component scores were missing. At time 1, internal consistency of the composite executive control measure was 0.67, and the inter-rater reliability was 0.83.

Child Psychopathology. Children's mental health outcomes were assessed using parent reports at the first and last time points. Parents reported on children's symptoms using the Child Behavior Checklist (CBCL, 4-18 years, version; Achenbach, 1991). The scale was augmented with problem behavior items from the preschool version (ages 2-3) that do not overlap with the items on the 4-18 version (34 items) to allow for the administration of identical measures across all time points. This is critical for the analysis of change across assessments, as earlier levels of symptoms were controlled for in predicting future problems. The CBCL has a strong empirical foundation and been shown to be a valid and reliable measure of children's psychopathology (see Achenbach, 1991). Maternal responses on the CBCL scales of Anxiety (12 items; Time 4 α = .73), Depression (12 items; Time 4 α = .66), and disruptive behavior problems (Aggression and Delinquency scales, 21 items; Time 4 α = .83) were used to assess

the three mental health outcomes of interest (Lengua, Sadowski, Friedrich, & Fisher, 2001).

Analytic Plan

Before testing the study hypotheses, potential covariates were identified. Subsequently, each of the three study aims were tested systematically, first by modeling unconditioned latent growth factors of RSA and each parenting variable. Parallel growth models in which initial levels of RSA and parenting were estimated as predictors of changes in one another were then tested as predictors of the emergence of child psychopathology across 27-months. Lastly, the role of children's executive control in moderating the relation of initial basal RSA on changes in RSA and parenting was examined. An outline of the analytic plan is delineated below.

Covariates. Child and family characteristics known to be related to children's psychopathology and parenting were examined as potential covariates. Evaluated covariates included child sex, family income, maternal education, and single parent status (Brooks-Gunn & Duncan, 1997). Potential covariates with significant zero-order correlations with outcome variables were retained for subsequent analyses.

Tests of study hypotheses. Analyses to test the proposed aims included the application of latent growth curve (LGC) models, tests of mediation, and a test of moderation. Missing data across time points was addressed using full information maximum likelihood estimation (FIMLE) when possible. FIMLE requires estimation of means, intercepts, covariances, and path coefficients, and uses all available data simultaneously to calculate parameter estimates (Kline, 1998). Analyses using FIMLE have been found to be less biased and more efficient than other techniques to handle missing data such as pairwise and listwise deletion (Arbuckle, 1996). As noted above, missing data was related to family income. Therefore, this was included as covariate to reduce potential systematic bias related to missingness.

Aim I. To test the first aim, whether initial levels or changes in baseline RSA differentially predict anxiety, depression, and DBP an unconditioned LGC was specified such that the intercept factor is the average initial level of RSA and that the slope is the linear rate of change

across 18 months (in 9-month intervals). Examination of unconditioned growth in RSA determined whether linear growth fit the data, and whether individuals significantly differed in their initial levels (intercept) and rate of change (slope). Significant means are indicative of non-zero levels of RSA, while a significant slope indicates that RSA, on average, changes over time. Moreover, significant (i.e. non-zero) variances for each of factor would indicate between individual differences in initial levels and rate of change. After successful specification of the unconditioned LGC, the intercept and slope factor of RSA was tested as predictors of anxiety, depression, and disruptive behavior problems. In addition, symptom levels at time 1 were entered as covariates, allowing identification of how RSA predicts unique changes in the emergence of psychopathology symptoms.

Aim II. To test whether children's emotionality and parenting predicted changes in one another over time (Aims IIa and c), parallel process growth models in which initial levels of RSA predicted changes (slope) in parenting, and vice versa, were specified. Separate unconditioned growth models of RSA and parenting were first examined for variability in the intercept and slopes as described above (see Aim I). For parenting, separate growth models were specified for each parenting variable (positive affect, negativity, responsiveness, autonomy granting, and intrusiveness); totally five parallel models. After successful specification of each unconditioned LGC, parallel process growth curves in which initial levels in RSA and parenting predicted changes in one another were estimated (Cheong et al., 2003). In addition, child sex, family characteristics, and symptom levels at time 1 were entered as covariates. Further, we examined whether the effects of initial baseline RSA and parenting on psychopathology were mediated by changes in one another across time (Aims IIb and d). The full model is depicted in Figure 1. We expected that the effect of initial levels of RSA on later psychopathology (anxiety, depression, disruptive behavior problems) would be explained partly through changes in maternal behavior. We also expected that the reverse model would be supported, such that the effect of initial parenting on later psychopathology would be partly mediated by its effect on

changes in children's RSA. Significant mediated paths would be tested according to the guidelines for testing mediation in parallel process growth models put forth by Cheong et al. (2003) using the product of coefficients method ($\alpha\beta$; see Cheong et al., 2003). Evidence for a mediated effect of either child or parent effects would suggest that initial levels in parenting (or RSA) predicts changes in the trajectory of RSA (or parenting) and in turn the emergence of child psychopathology.

Aim III. The third aim tested whether initial levels (intercept) of basal RSA were moderated by executive control at Time 1 to predict changes in parenting and psychopathology. In order to test this aim, we followed guidelines put forth by Muthén and Asparouhov (2003) for modeling interactions between latent and observed continuous variables. Mplus allows for an interaction term comprised of the multiplicative of the latent factor and the observed variable, thereby allowing interactions of observed variables with growth parameters to be estimated. Modeling the interaction of basal RSA by time 1 executive control allowed examination of the hypothesis that low executive control would serve to amplify the relation of RSA to psychopathology (see Figure 2).

Table 1 *Family Income Category Distributed by Recruitment Source*

	Poverty (n = 50)	150% Poverty (n = 44)	200% Poverty (n = 36)	Low Income (n = 56)	Middle and High Income (n = 120)
UW Subject Pool	31	31	29	47	72
Community Center	6	3	--	--	3
Daycares and Schools	7	6	5	5	25
Print Advertisements and Referrals	6	4	2	4	20

Note. UW = University of Washington.

Table 2 *Time 1 Correlations among Parenting Variables*

	1	2	3	4	5
1. Positive Affect	--	-.34**	.20**	.45**	-.44**
2. Negativity		--	-.36**	-.44**	.48**
3. Responsiveness			--	.20**	-.32**
4. Autonomy Granting				1	-.63**
5. Intrusiveness				--	--

Table 3 *Time 2 Correlations among Parenting Variables*

	1	2	3	4	5
1. Positive Affect	--	-.45**	.34**	.58**	-.33**
2. Negativity		--	-.52**	-.53**	.67**
3. Responsiveness			--	.27**	-.40**
4. Autonomy Granting				--	-.53**
5. Intrusiveness					--

Table 4 *Time 3 Correlations among Parenting Variables*

	1	2	3	4	5
1. Positive Affect	--	-.39**	.32**	.55**	-.19**
2. Negativity		--	-.48**	-.60**	.64**
3. Responsiveness			--	.38**	-.45**
4. Autonomy Granting				--	-.58**
5. Intrusiveness					--

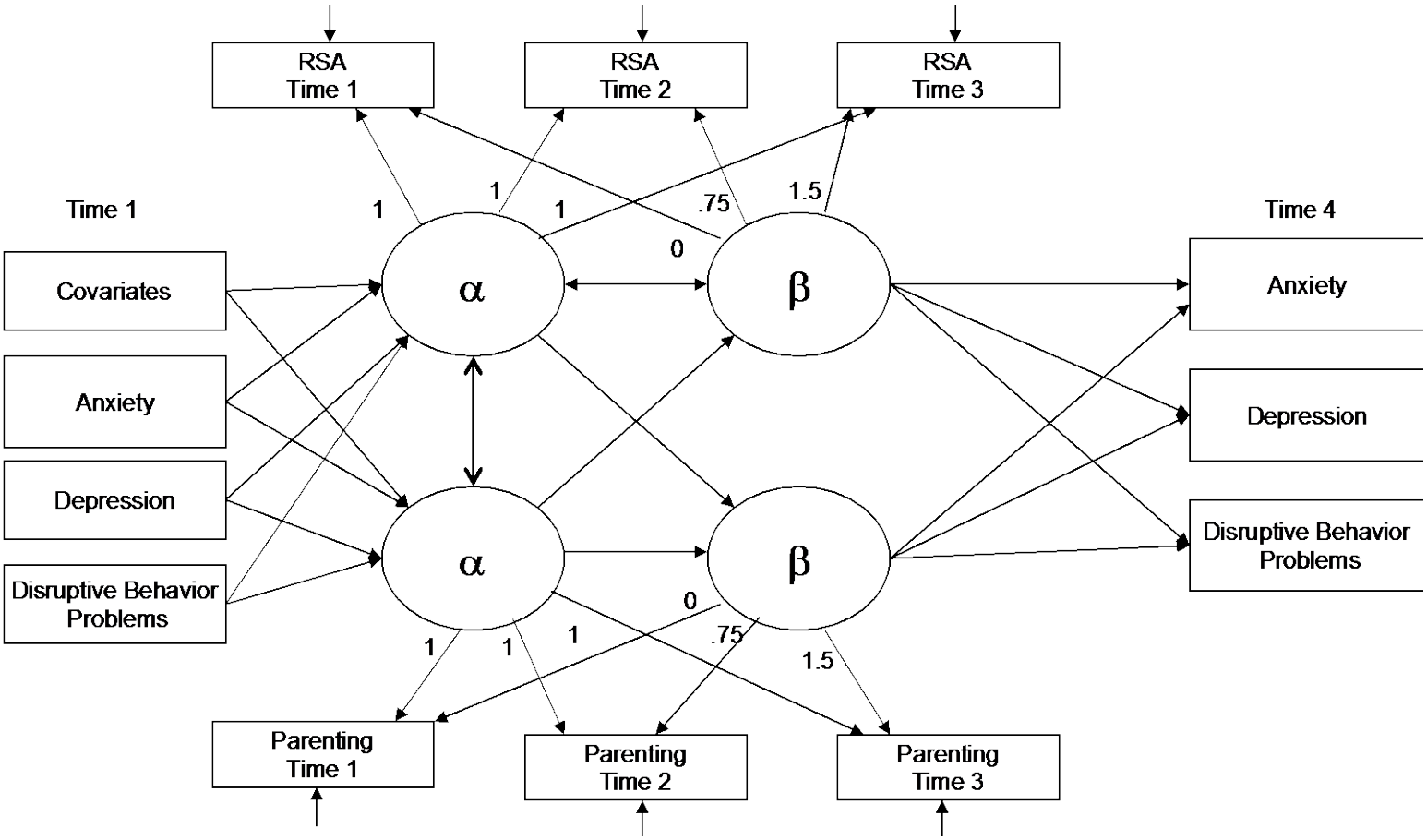

Figure 1. Proposed parallel process latent growth model examining the bidirectional relations between RSA and parenting in predicting child psychopathology. Although not depicted (to facilitate figure readability), each covariate was estimated as conditioned predictors of the slope of RSA and each parenting variable.

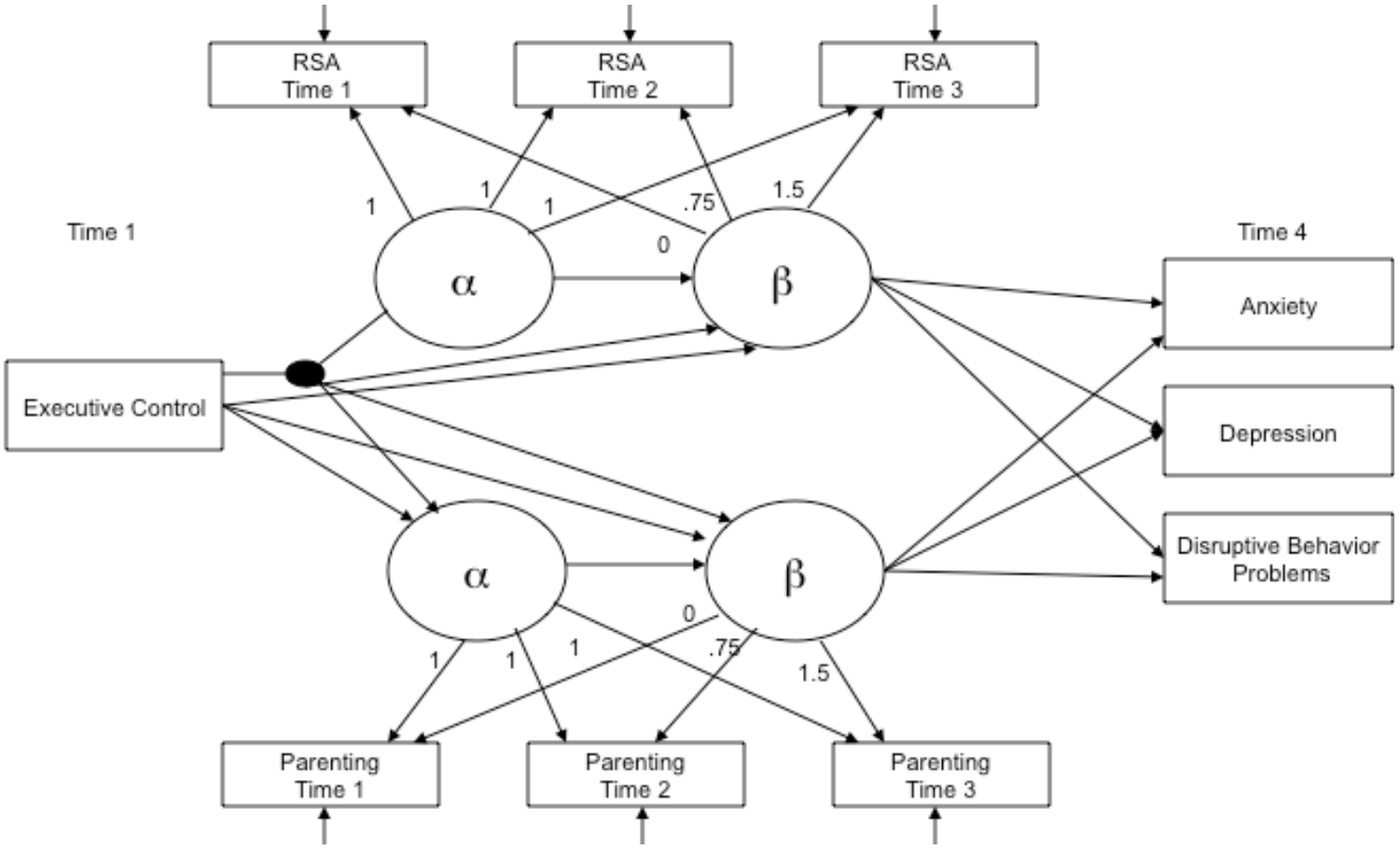

Figure 2. Proposed latent-by-continuous variable interaction predicting change in parenting and RSA Family income and child symptoms at Time 1 were included as covariate predictors of Time 4 outcomes.

Results

Descriptive statistics for all study variables are outlined in Table 5.

Correlations among Study Variables

The correlations among maternal parenting behaviors, children's physiologic emotionality (i.e., RSA), and maternal reports of children's anxiety, depression, and disruptive behavior problems are reported in Table 6. The correlations among the study variables indicated that children's basal RSA demonstrated modest stability across time but was not related to observations of maternal parenting or children's psychopathology symptom. For maternal parenting, observations of behavior were moderately intercorrelated. In addition, maternal parenting behaviors were related to children's anxiety and disruptive behavior problems, but not depressive symptoms. More specifically, higher maternal positive affect, lower negativity, higher responsiveness, and greater autonomy granting were related to lower anxiety and disruptive behavior problems. However, this pattern was not consistent across observations of parenting with stronger correlations between Time 2 parenting and Time 4 outcomes. The other parenting behaviors were not related to later psychopathology symptoms.

Unconditioned Growth in RSA and Parenting

Latent growth curve models were used to examine the trajectories of change for RSA and parenting behaviors over time. Using FIMLE in Mplus 6.11 (Muthén & Muthén, 1998-2010), models were specified in which factor loadings were set to define the intercept as levels of the variable at time 1 and the slope as linear change in the variable across 18 months of the study. RSA and each parenting behavior were first examined as unconditioned models (i.e., without predictors) to estimate the degree of variability in initial levels and growth across time.

RSA. Examination of the unconditioned model revealed that initial levels of RSA were significantly greater than zer (see Table 7). Further, there was significantly variability around the intercept indicating a lack of uniformity in initial values of RSA within the sample. The significant variability for the intercept factor suggested that exogenous predictors might be useful for

explaining variability in initial basal RSA. Further, the significant slope factor suggests that basal RSA values increased across time (see Table 7). However, the pattern of change appeared to be largely uniform within the sample; that is, there was not significant variability around the slope of RSA.

Parenting. Unconditioned growth models were also specified for each parenting variable. For maternal positive affect, the unconditioned model specifying a latent intercept and slope fit the data poorly, χ^2 (1) = 25.98, $p < .001$, RMSEA = .29, $p < .001$, CFI = .72. Given the poor model fit, attempts to improve fit by freeing and constraining parameters were made in a systematic manner following the guidelines put forth by Kline (2005). These steps included (a) trimming the error covariances to zero for each time point, (b) constraining error covariances across time points, and (c) estimating the error covariance for each time point to be free and unique. None of these efforts resulted in an improved model fit for linear change in maternal positive affect across 18-months. In addition, the obtained findings indicated a significant intercept (b = 3.32, SE = .03, $p < .001$) indicating that the degree of maternal positivity observed at the start of the study was significantly greater than zero. Further, mothers demonstrated significant variability in their initial levels of positive affect suggesting that positivity at the start of the study was not uniform across the sample and may be explained by exogenous predictors (b = .08, $p < .05$). However, maternal positive affect did not change across time as indicated by a nonsignificant slope factor (b = .02, $p > .05$). Thus, in an final attempt to improve model fit, an intercept only model of maternal positive affect was specified and yielded a modest advancement in model fit with a similar pattern in the intercept and estimated variance (see Table 7). Thus, the intercept only model of maternal positive affect was retained for subsequent analyses.

With regard to the remaining parenting variables, as shown in Table 7, obtained fit estimates were largely adequate or good for maternal negativity, intrusiveness, and autonomy granting. However, the unconditioned growth model for maternal responsiveness did not fit the

data well. Therefore, similar attempts to improve model fit, as outlined by Kline (2005), were followed with minimal benefit. However, the estimated model fit for linear change in maternal responsiveness significantly improved once conditioned parameters were estimated as predictors of growth in maternal responsiveness (see below). This may suggest that the unconditioned growth model was not sufficiently powered, as only one degree of freedom remained after the intercept and slope factors were estimated. Therefore, the a priori intercept and slope factor model estimating linear change across 18-months was retained for subsequent analyses.

With regard to the patterns of unconditioned linear change in maternal parenting, estimates for the intercept and slope each maternal parenting behavior (negativity, responsiveness, autonomy granting, and intrusiveness) changed across time (see Table 7). More specifically, maternal negativity and intrusiveness increased across time, while maternal responsiveness and autonomy granting generally decreased across the study. Moreover, for most of the parenting variables measured, the pattern of change was not uniform within the sample; that is, there was significant variability around the rate of change for maternal negativity, responsiveness, and intrusiveness. Conversely, the pattern of change was largely uniform for maternal autonomy granting as mothers were observed to grant less autonomy across time. The significant variability for these factors suggests that exogenous predictors may be useful for explaining variability in the rate of change for maternal rearing behaviors.

Covariate Model

Next, a covariate model was tested to determine whether child sex, family income, maternal education, and single parent status predicted the emergence of psychopathology across the study. The correlation of each potential covariate with child psychopathology symptoms is reported in Table 8. Examination of the zero order correlations among these potential covariates and Time 4 outcomes (anxiety, depression, and disruptive behavior problems) revealed that higher family income at the start of the study was related to less disruptive behavior problems at

Time 4. None of the other potential covariates was related to outcomes.

To further evaluate the contribution of these covariates to the proposed study hypotheses the zero order correlations of each potential covariate (child sex, family income, maternal education, and single parent status) with children's RSA and each maternal parenting behavior (positive affect, negativity, responsiveness, autonomy granting, and intrusiveness) were examined (see Table 9). The results indicate that mothers of boys were observed as less responsive and more intrusive. In addition, familial characteristics including income, maternal education, and marital status were related to observations of parenting. More specifically, higher maternal positivity, responsiveness, and autonomy granting as well as lower maternal negativity and intrusiveness were related to higher family income, higher maternal education, and two-parent family status.

Subsequently, conditioned latent growth models in which all potential covariates (child sex, family income, maternal education, and single parent status) were specified as simultaneous predictors of growth in RSA and each parenting variable (positive affect, negativity, responsiveness, autonomy granting, and intrusiveness). The results are shown in Table 10. With regard to RSA, there was a trend for boys to exhibit lower RSA at the start of the study. None of the familial characteristics (income, education, marital status) was related to initial levels or changes in RSA. Thus, child sex was retained as a covariate for RSA in all subsequent analyses.

When observations of parenting were considered, again higher familial income, higher maternal education, and mothers in two-parent families were observed to exhibit more positive and less controlling parenting behaviors. Although the pattern of significance differed somewhat across parenting variables, all evaluated covariates were retained given their relation to the variables of interest. Thus, child sex, family income, maternal education, and single parent status were retained as covariates for each parenting variable in all subsequent analyses.

Aim I: Growth in RSA as a Predictor of Psychopathology Symptoms

To test the proposed hypothesis that children's physiological emotionality, as indicated by RSA, would differentially predict the emergence of psychopathology symptoms, latent growth models were specified in which initial levels of RSA and changes across 18-months were examined as predictors of symptomatology at age 63-months. Parallel models were run for each outcome of interest (anxiety symptoms, depressive symptoms, and disruptive behavior problems). Given that symptoms often co-occur in normative samples and were correlated in this study, comorbid symptoms at Time 1 were included as covariates. Thus, when anxiety was considered co-occurring depressive symptoms and disruptive behavior problems were parsed out to facilitate the unique emergence of anxiety symptoms. In addition, child sex and family income were included as covariates given their association with (1) missing data within the sample and (2) children's symptomatology.

For anxiety symptoms (see Figure 3), the model fit the data well, χ^2 (7) = 3.89, p = .79, CFI = 1.00, RMSEA < .001, p = .96, and indicated that anxiety symptoms were relatively stable across the study (b = .47, p < .001, b = .39). Initial levels and growth in RSA did not predict the emergence of anxiety symptoms (intercept: b = -.14, p = .81, β = -.02; slope b = .36, p = .82, β = .03). However, there was a modest trend toward initial anxiety symptoms predicting changes in RSA (b = -.02, p = .08, β = -.25), although the indirect effect on later symptoms was not significant (indirect effect = -.01, p = .82).

The model predicting the emergence of depressive symptoms (see Figure 4) also fit the data well, χ^2 (7) = 7.67, p = .36, CFI = 1.00, RMSEA = .02, p = .76. Again, initial levels and growth in RSA did not predict depressive symptoms at the end of the study (intercept: b = .35, p = .44, β = .06; slope b = .58, p = .62, β = .06). Further, higher depressive symptoms at 36-months of age predicted lower initial basal RSA (b = -.05, p = .02, β = -.24), but did not predict depressive symptoms across time (indirect effect = -.02, p = .46). Instead, depressive symptoms

remained stable across the study (*b* = .45, *p* < .001, β = .37).

Lastly, a parallel model was specified to examine the emergence of disruptive behavior problems (see Figure 5). The specified model again fit the data well, χ^2 (7) = 6.40, *p* = .49, CFI = 1.00, RMSEA < .001, *p* = .85. Similar to the findings for internalizing symptoms, disruptive behavior problems were not predicted by initial levels or changes in children's basal RSA (intercept: *b* = .19, *p* = .80, β = .02; slope: *b* = -.30, *p* < .88, β = -.02). Instead, symptoms persisted from 36- to 63-months of age (*b* = .57, *p* < .001, β = .50). Further, unlike the previous models, disruptive behavior problems at the start of the study did not predict initial levels or trajectories of RSA (intercept: *b* = .01, *p* = .17, β = .05; slope: *b* = .02, *p* = .81, β = .03).

Aim II: Parallel Growth in RSA and Parenting Behaviors

Parallel process growth models were specified in which initial levels and changes in RSA and each parenting behavior to test the hypothesis that emotionality and parenting would mutually influence one another and predict the emergence of children's psychopathology symptoms. Steps to test this hypothesis were followed in accordance of recommendations put forth by Muthén & Muthén (1998-2010), such that first unconditioned parallel growth models of RSA with each parenting variable were estimated. Once adequate fit was achieved, covariates including child sex, family demographics, and psychopathology symptoms at the start of the study were included as covariates in each model. Each of the specified models demonstrated adequate fit to the data (see Table 11). Overall, the findings indicate that children's emotional dysregulation and maternal rearing behaviors were not related to one another at the start of the study (see Table 11). Further, children's RSA and parenting did not predict changes in one another across time. When the emergence of psychopathology was considered, RSA and parenting were not significant predictors of changes in symptoms across 27-months, with one exception. Increases in maternal intrusiveness across the study predicted fewer anxiety symptoms when children were 63-months old. Further, this relation was above the effect of

earlier symptoms of anxiety.

Aim III: Executive Control as a Moderator of Initial Emotional Dysregulation

To test the third study aim, whether the effect of initial levels (intercept) of basal RSA on initial levels and changes in parenting and the emergence of psychopathology were moderated by children's executive control at Time 1, a latent-by-observed variable interaction was specified in accordance with guidelines but forth by Muthén and Asparouhov (2003). Specifically, the direct effect of children's executive control, the intercept of basal RSA, and an interaction term of the multiplicative of these variables were specified as predictors of the intercept and slope of each parenting behavior across the study, as well as children's psychopathology symptoms at 63-months of age. The results are presented in Table 12. Notably, children's executive control at 36-months of age conditioned the relation of initial emotional dysregulation on initial levels, but not changes, in maternal responsiveness (see Figure 6). The pattern of relations was such that at high initial levels of basal RSA, children observed to be at or above the mean of executive control elicited more maternal responsiveness at the start of the study; that is, higher basal and physiologic regulation facilitated more responsive maternal parenting behaviors. Conversely, when high basal RSA was coupled with low executive relation, lower maternal responsiveness was observed. However, the interaction of executive control and basal RSA did not predict trajectories of responsiveness, other maternal behaviors (negativity, autonomy granting, or intrusiveness), the trajectory of RSA, or the emergence of psychopathology symptoms.

Table 5 *Descriptive statistics for Study Variables*

	Mean	Standard Deviation	Minimum	Maximum	Skew	Kurtosis
			Time 1			
Executive Control	.29	.15	.00	.77	.66	.18
RSA	4.82	.53	3.12	7.14	.13	1.30
Positive Affect	3.37	.50	1.87	4.79	-.16	.06
Negativity	.36	.38	.00	2.42	2.04	5.50
Responsiveness	4.40	.74	.75	5.00	-2.17	5.48
Autonomy Granting	3.14	.55	.79	4.56	-.30	1.05
Intrusiveness	.97	.79	.00	3.38	.93	.37
Anxiety	2.57	2.28	.00	13.09	1.27	1.98
Depression	2.10	1.86	.00	9.00	.95	.55
Disruptive Behavior Problems	5.87	3.49	.00	19.00	.95	1.56

Table 5 continued. *Descriptive statistics for Study Variables*

	Mean	Standard Deviation	Minimum	Maximum	Skew	Kurtosis
			Time 2			
RSA	4.95	.54	3.11	6.26	-.26	.64
Positive Affect	3.25	.49	1.55	4.62	-.15	.84
Negativity	.46	.48	.00	2.64	1.47	2.42
Responsiveness	4.44	.50	2.08	5.00	-1.39	3.07
Autonomy Granting	3.07	.43	1.58	4.48	-.40	1.67
Intrusiveness	.96	.70	.00	3.45	.72	-.01
			Time 3			
RSA	5.07	.52	3.51	6.97	-.03	.83
Positive Affect	3.38	.48	1.17	4.88	-.37	2.46
Negativity	.50	.43	.00	2.48	1.73	3.81
Responsiveness	4.22	.55	2.00	5.00	-1.03	1.07
Autonomy Granting	3.06	.50	1.29	4.77	-.37	1.77
Intrusiveness	1.13	.63	.00	3.57	.85	.69

Table 5 continued. *Descriptive statistics for Study Variables*

	Mean	Standard Deviation	Minimum	Maximum	Skew	Kurtosis
Anxiety	2.91	2.74	.00	17.00	1.69	4.57
Depression	2.14	2.25	.00	14.00	1.89	4.95
Disruptive Behavior Problems	5.08	3.99	.00	23.00	1.39	2.93

Table 6 Correlations among Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29
1. Child Sex	--	-.05	-.08	.01	-.09	-.11	-.08	.05	.00	.01	.01	.03	.06	.04	-.12*	-.03	.03	-.07	-.06	-.06	.17**	.06	.09	.01	.00	.03	-.04	.13*	.11
2. Family Income		--	.47**	-.50**	.19**	-.01	-.01	-.12*	.27**	.34**	.24**	-.26**	-.39**	-.31**	.07	.21**	.25**	.40**	.37**	.22**	-.30**	-.28**	-.21**	.05	-.11	.05	-.06	-.02	-.17**
3. Maternal Education			--	-.25**	.21**	-.09	.01	-.07	.26**	.29**	.23**	-.31**	-.38**	-.31**	.17**	.24**	.22**	.41**	.37**	.28**	-.40**	-.36**	-.32**	-.05	.02	.12*	.09	-.02	.01
4. Single Parent Status				--	-.17**	.03	.03	.04	-.20**	-.25**	-.18**	.29**	.37**	.34**	-.08	-.19**	-.26**	-.21**	-.35**	-.30**	.18**	.24**	.26**	-.03	.03	-.02	.04	.01	.11
5. Executive Control					--	.11	-.05	-.02	.26**	.06	-.01	-.24**	-.19**	-.24**	.21**	.10	.09	.27**	.17**	.26**	-.31**	-.22**	-.20**	.01	.09	-.02	.08	-.08	-.02
6. RSA T1						--	.45**	.39**	-.01	-.04	-.07	.07	.07	-.01	-.02	-.08	-.05	-.05	.05	.00	.01	.05	.04	.00	-.03	-.10	.04	-.05	.01
7. RSA T2							--	.53**	.03	.04	.00	.06	.07	.00	-.07	.07	-.01	-.02	.02	-.03	.03	.03	.06	.02	.00	-.08	-.06	.03	-.04
8. RSA T3								--	-.01	-.04	-.04	.05	.08	.03	.01	.00	-.08	-.03	.02	.03	.04	.05	.06	-.09	-.04	-.08	.03	.03	.03
9. Maternal Positive Affect T1									--	.34**	.31**	-.34**	-.23**	-.23**	.20**	.28**	.20**	.45**	.23**	.23**	-.44**	-.14*	-.12*	-.03	-.01	.03	.08	-.14*	-.12
10. Maternal Positive Affect T2										--	.44**	-.13*	-.45**	-.26**	.07	.34**	.28**	.25**	.58**	.28**	-.20**	-.33**	-.19**	-.05	-.17*	.00	-.06	-.06	-.19**
11. Maternal Positive Affect T3											--	-.14*	-.29**	-.39**	.07	.22**	.32**	.25**	.25**	.55**	-.14*	-.17**	-.19**	-.11	.03	.03	.00	-.10	-.03
12. Maternal Negativity T1												--	.41**	.38**	-.36**	-.29**	-.17**	-.44**	-.27**	-.29**	.48**	.29**	.29**	-.02	.00	-.01	-.03	.08	.09
13. Maternal Negativity T2													--	.50**	-.12	-.52**	-.35**	-.40**	-.53**	-.34**	.38**	.67**	.33**	-.01	.19**	-.01	.00	.06	.19**
14. Maternal Negativity T3														--	-.14*	-.27**	-.48**	-.20**	-.39**	-.60**	.20**	.39**	.64**	.06	.00	-.04	-.04	.08	.14*
15. Maternal Responsiveness T1															--	.20**	.00	.20**	.09	.12	-.32**	-.13*	-.11	-.04	-.05	-.05	.01	-.12*	-.11
16. Maternal Responsiveness T2																--	.19**	.33**	.27**	.22**	-.39**	-.40**	-.09	-.03	-.07	-.02	.02	-.09	-.08
17. Maternal Responsiveness T3																	--	.11	.32**	.38**	-.10	-.25**	-.45**	.02	.05	.04	.03	-.10	-.08
18. Maternal Autonomy Granting T1																		--	.33**	.25**	-.63**	-.39**	-.16*	.04	-.03	.09	.04	-.12*	-.08
19. Maternal Autonomy Granting T2																			--	.32**	-.27**	-.54**	-.35**	-.07	-.18**	-.04	-.08	-.07	-.23**
20. Maternal Autonomy Granting T3																				--	-.31**	-.35**	-.58**	-.08	.03	-.05	.05	-.10	-.10
21. Maternal Intrusiveness T1																					--	.41**	.25**	.00	.09	-.05	-.04	.12*	.13*
22. Maternal Intrusiveness T2																						--	.38**	-.02	.07	-.04	-.09	.00	.14*
23. Maternal Intrusiveness T3																							--	.02	-.07	.04	-.05	.12	.12
24. Anxiety Symptoms T1																								--	.44**	.46**	.36**	.24**	.19**
25. Anxiety Symptoms T4																									--	.31**	.51**	.22**	.45**
26. Depression Symptoms T1																										--	.48**	.35**	.24**
27. Depression Symptoms T4																											--	.27**	.51**
28. Disruptive Behavior Problems T1																												--	.53**
29. Disruptive Behavior Problems T4																													--

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

Table 7 *Unconditioned Growth in RSA and Parenting*

	Model Fit			Intercept Factor				Slope Factor			
	χ^2 (1)	RMSEA	CFI	M	SE	Variance	SE	M	SE	Variance	SE
RSA (n = 297)	.05	.00	1.00	4.82***	.03	.14**	.04	.17***	.03	.04	.03
Parenting (n = 305)											
Positive Affect	27.39***[3]	.14**	.74	3.32***	.02	.08***	.01	--	--	--	--
Negativity	3.37	.09	.98	.37***	.02	.08***	.02	.09***	.02	.04*	.02
Responsiveness	12.48**	.19**	.24	4.48***	.04	.14**	.05	-.14***	.04	.10*	.05
Autonomy Granting	1.95	.06	.98	3.12***	.03	.09**	.03	-.05*	.03	.01	.03
Intrusiveness	3.90*	.10	.97	.94***	.05	.34***	.07	.12**	.04	.13*	.05

Note. Reported means and variances are unstandardized values.
*p < .05, **p < .01, ***p < .001

[3] Degrees of freedom for the unconditioned model of maternal positive affect were four given that only an intercept factor was specified.

Table 8 *Correlations of Potential Covariates with Psychopathology Symptoms at 63-Months of Age*

	Time 4 Symptoms		
	Anxiety	Depression	Disruptive Behavior Problems
Child Sex	.00	-.04	.11
Family Income	-.11	-.06	-.17**
Maternal Education	.02	.09	.01
Single Parent Status	.03	.04	.11
Time 1 Anxiety	.44**	.36**	.19**
Time 1 Depression	.31**	.48**	.24**
Time 1 Disruptive Behavior Problems	.22**	.27**	.53**

Table 9 *Correlations among Study Variables and Potential Covariates*

	RSA			Positive Affect			Negativity			Responsiveness			Autonomy Granting			Intrusiveness		
	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3	T1	T2	T3
Child Sex	-.11	-.08	.05	.00	.01	.01	.03	.06	.04	-.12*	-.03	.03	-.07	-.06	-.06	.17*	.06	.09
Family Income	-.01	-.01	-.12*	.27*	.34*	.24*	-.26*	-.39*	-.31*	.07	.21*	.25*	.40*	.37*	.22*	-.30*	-.28*	-.21*
Maternal Education	-.09	.01	-.07	.26**	.29*	.23*	-.31*	-.38*	-.30*	.17*	.24*	.22*	.41**	.37*	.28*	-.40*	-.36*	-.32*
Single Parent Status	.03	.03	.04	-.20*	-.25*	-.18*	.29*	.37*	.34*	-.08	-.19*	-.26*	-.21*	-.35*	-.30*	.18*	.24*	.26*
Executive Control	.11	-.05	-.02	.26*	.06	-.01	-.24*	-.19*	-.24*	.21*	.10	.09	.27*	.17*	.26*	-.31*	-.22*	-.20*
Anxiety T1	.00	.02	-.09	-.03	-.05	-.11	-.02	-.01	.06	-.04	-.03	.02	.04	-.07	-.08	.00	-.02	.02
Anxiety T4	-.03	.00	-.04	-.01	-.17*	.03	.00	.19*	.00	-.05	-.07	.05	-.03	-.18*	.03	.09	.07	-.07
Depression T1	-.10	-.08	-.08	.03	.00	.03	-.01	-.01	-.04	-.05	-.02	.04	.09	-.04	-.05	-.05	-.04	.04
Depression T4	.04	-.06	.03	.08	-.06	.00	-.03	.00	-.04	.01	.02	.03	.04	-.08	.05	-.04	-.09	-.05
DBP T1	-.05	.03	.03	-.14*	-.06	-.10	.08	.06	.08	-.12*	-.09	-.10	-.12*	-.07	-.10	.12*	.00	.12
DBP T4	.01	-.04	.03	-.12	-.19*	-.03	.09	.19*	.14*	-.11	-.08	-.08	-.08	-.23*	-.10	.13*	.14*	.12

Note. DBP = Disruptive Behavior Problems. To conserve space, the degree of significance is reported at the .05 level although many correlations fell well below this threshold.
*$p < .05$

Table 10 *Relation of Potential Covariates and Growth Parameters*

	RSA		Positive Affect		Negativity		Responsiveness		Autonomy Granting		Intrusiveness	
	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope	Intercept	Slope
	b (SE) β	b (SE) β	b (SE) β	b (SE) β	b (SE) β	B (SE) β	b (SE) β	b (SE) β	b (SE) β	B (SE) β	b (SE) β	b (SE) β
Child Sex	-.11^{t}	.09^{t}	.03		.00	.01	-.13^{t}	.13^{t}	-.02	.00	.17*	-.07
	(.07)	(.05)	(.04)	--	(.04)	(.04)	(.08)	(.07)	(.05)	(.05)	(.08)	(.07)
	-.31	.45	.12		-.01	.07	-.35	.40	-.06	-.01	.29	-.19
T1 Family Income	.01	-.01^{t}	.02*		.00	-.01	-.01	.02	.04***	-.03**	-.03*	.02
	(.01)	(.01)	(.01)	--	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)
	.09	-.26	.22		-.07	-.16	-.06	.18	.46	-.80	-.17	.19
T1 Maternal	-.02	.02	.05***		-.05***	.00	.07**	-.02	.08***	-.02	-.14***	.03
Education	(.02)	(.02)	(.01)	--	(.01)	(.01)	(.03)	(.02)	(.02)	(.02)	(.03)	(.02)
	-.11	.16	.31		-.38	.03	.33	-.11	.47	-.27	-.43	.15
T1 Single Parent	.04	-.01	-.13*		.21***	.04	-.10	-.10	-.05	-.17*	.08	.16
Status	(.10)	(.08)	(.06)	--	(.06)	(.05)	(.12)	(.11)	(.08)	(.07)	(.12)	(.10)
	.04	-.02	-.18		.34	.10	-.11	-.12	-.07	-.53	.05	.17

Note. Model fit information for each growth model is RSA: n = 293, χ^2 (5) = 2.99, p = .70, CFI = 1.00, RMSEA < .001, p = .91; Maternal Positive Affect: n = 300, χ^2 (12) = 39.71, p < .001, CFI = .80, RMSEA = .09, p = .02; Maternal Negativity: n = 300, χ^2 (5) = 18.63, p = .002, CFI = .94, RMSEA = .10, p = .05; Maternal Responsiveness: n = 300, χ^2 (5) = 13.15, p = .02, CFI = .86, RMSEA = .07, p = .17; Maternal Autonomy Granting: n = 300, χ^2 (5) = 6.12, p = .30 , CFI = .99, RMSEA = .03, p = .66; Maternal Intrusiveness: n = 300, χ^2 (5) = 6.00, p = .31, CFI = .99, RMSEA = .03, p = .67.

^{t}p < .10, *p < .05, **p < .01, ***p < .001

Table 11 *Unstandardized and Standardized Parameters from Parallel Process Growth Models for RSA and Maternal Parenting*

	Model Fit			Growth Relations			Time 4 Adjustment	
	χ^2 (df)	RMSEA	CFI	Corr. btw. Intercepts	RSA int. → Par. slope	Par. int. → RSA slope	RSA slope → Symptoms	Par. slope → Symptoms
Outcome: T4 Anxiety Symptoms								
RSA and								
Positive Affect	66.46**	.04	.92	-.01	--	-.04	.30	-.39[4]
	(42)			(.03)		(.11)	(1.73)	(1.04)
				-.20		-.06	.02	-.04
Negativity	45.84	.04	.97	.01	-.08	.05	.08	.71
	(32)			(.01)	(.10)	(.12)	(1.65)	(4.13)
				.14	-.24	.06	.01	.03
Responsiveness	30.44	.00	1.00	-.01	-.03	.09	.12	.61
	(32)			(.02)	(.07)	(.11)	(1.57)	(1.09)
				-.10	-.04	.16	.01	.08
Autonomy	44.59	.02	.99	.01	.01	-.05	.28	16.54
Granting	(39)			(.01)	(.03)	(.08)	(1.62)	(12.03)
				.13	.03	-.07	.02	.67[t]
Intrusiveness	25.95	.00	1.00	.00	.09	.01	.11	-1.75[t]
	(32)			(.02)	(.08)	(.05)	(1.54)	(.97)
				.00	.09	.03	.01	-.24*[5]

[4] Denotes intercept of maternal positive affect predicting Time 4 symptoms

[5] The difference in significance level between the unstandardized and standardized coefficients is due to differences in the sampling distributions used by Mplus.

Table 11 continued. *Unstandardized and Standardized Parameters from Parallel Process Growth Models for RSA and Maternal Parenting*

	Model Fit			Growth Relations			Time 4 Adjustment	
	χ^2 (df)	RMSEA	CFI	Corr. btw. Intercepts	RSA int. → Par. slope	Par. int. → RSA slope	RSA slope → Symptoms	Par. slope → Symptoms
Outcome: T4 Depression Symptoms								
RSA and								
Positive Affect	62.55*	.04	.94	.01	--	-.04	.50	.36[3]
	(42)			(.01)		(.11)	(1.28)	(.83)
				-.21		-.06	.05	.05
Negativity	41.73	.02	.99	-.01	-.07	.07	.86	-3.44
	(39)			(.03)	(.07)	(.11)	(1.34)	(8.00)
				-.19	-.27	.07	.08	-.14
Responsiveness	33.45	.01	1.00	-.01	-.03	.09	-.81	.13
	(32)			(.02)	(.07)	(.11)	(2.17)	(1.52)
				-.10	-.04	.16	-.04	.01
Autonomy	43.95	.02	.99	.00	.06	-.05	.74	4.65
Granting	(39)			(.01)	(.06)	(.08)	(1.48)	(4.36)
				.04	.16	-.08	.07	.29
Intrusiveness	32.20	.01	1.00	-.01	.10	.01	1.07	-.14
	(32)			(.03)	(.09)	(.05)	(1.65)	(.59)
				-.15	.09	.02	.10	-.02

Table 11 continued. *Unstandardized and Standardized Parameters from Parallel Process Growth Models for RSA and Maternal Parenting*

	Model Fit			Growth Relations			Time 4 Adjustment	
	χ^2 (df)	RMSEA	CFI	Corr. btw. Intercepts	RSA int. → Par. slope	Par. int. → RSA slope	RSA slope → Symptoms	Par. slope → Symptoms
Outcome: T4 Disruptive Behavior Problems								
RSA and								
Positive Affect	63.29*	.04	.94	.01	--	-.04	.82	-1.43
	(42)			(.01)		(.11)	(2.31)	(1.43)
				.07		-.05	-.04	-.10
Negativity	37.49	.02	.99	.01	-.07	.05	-.59	6.63
	(32)			(.01)	(.09)	(.12)	(2.31)	(6.15)
				.13	-.25	.06	-.03	.17
Responsiveness	33.45	.01	1.00	-.02	-.03	.09	-.81	.13
	(32)			(.03)	(.07)	(.11)	(2.17)	(1.52)
				-.10	-.04	.16	-.04	.01
Autonomy	49.92	.03	.97	.01	.04	-.05	-.13	-5.00
Granting	(39)			(.01)	(.07)	(.08)	(2.56)	(4.84)
				.09	.09	-.09	-.01	-.18
Intrusiveness	34.21	.02	.99	-.02	.09	.01	-.93	-.29
	(32)			(.03)	(.09)	(.05)	(2.18)	(1.12)
				-.24	.10	.01	-.05	-.03

Note. Values are the unstandardized coefficients (standard errors) and standardized coefficients. Child sex, family income, maternal education, single parent status, and time 1 symptoms were controlled in each model. Child sex and initial symptoms were specified

as covariates of RSA, while all covariates were included for the parenting variables. Df=Degrees of freedom; RMSEA=Root Mean Square Error of Approximation; CFI=Comparative Fit Index; Corr.=Correlation; btw=between; int.=intercept; par.=parenting.
$^{t}p < .10$, * $p < .05$

Table 12 *Parameters for Executive Control as a Moderator of Children's RSA in Shaping Maternal Rearing Behaviors*

	Growth Relations			Time 4 Adjustment					
	RSA x EC → RSA slope	RSA x EC → Par. int.	RSA x EC → Par. slope	RSA x EC → Anxiety	Par. slope → Anxiety	RSA x EC → Dep.	Par. Slope → Dep.	RSA x EC → DBP	Par. slope → DBP
RSA x EC									
Negativity	.38	-.69	.39	.33	-.30	.20	-.30	.31	1.84
	(.43)	(.85)	(.58)	(.26)	(1.23)	(.21)	(1.00)	(.43)	(1.78)
Responsiveness	.47	1.57*	-.07	.32	.88	.16	.27	.26	-.76
	(.44)	(.70)	(.59)	(.26)	(1.55)	(.23)	(1.33)	(.43)	(2.20)
Autonomy	.38	-.15	-.11	-.35	8.74	.18	4.48	.50	-4.65
Granting	(.42)	(.79)	(.72)	(1.40)	(16.13)	(.26)	(5.72)	(.43)	(7.69)
Intrusiveness	.37	-.51	-.18	.32	-1.35	.19	-.60	.44	1.28
	(.42)	(1.44)	(.81)	(.26)	(1.02)	(.22)	(.83)	(.42)	(1.19)

Note. Values are the unstandardized coefficients (standard errors). Standardized estimates are not available for random effects models. The direct effect of executive control and the intercept of RSA were controlled for in predicting the intercept of parenting and the slope of RSA and parenting. Child sex, family income, maternal education, single parent status, and time 1 symptoms were specified as covariates for Time 4 outcomes. Maternal positive affect was not included in this aim, as a slope parameter was not estimated. EC=executive control; par.=parenting; int.=intercept; dep.=depression; DBP=disruptive behavior problems.
$^{t}p < .10$, * $p < .05$ ** $p < .05$

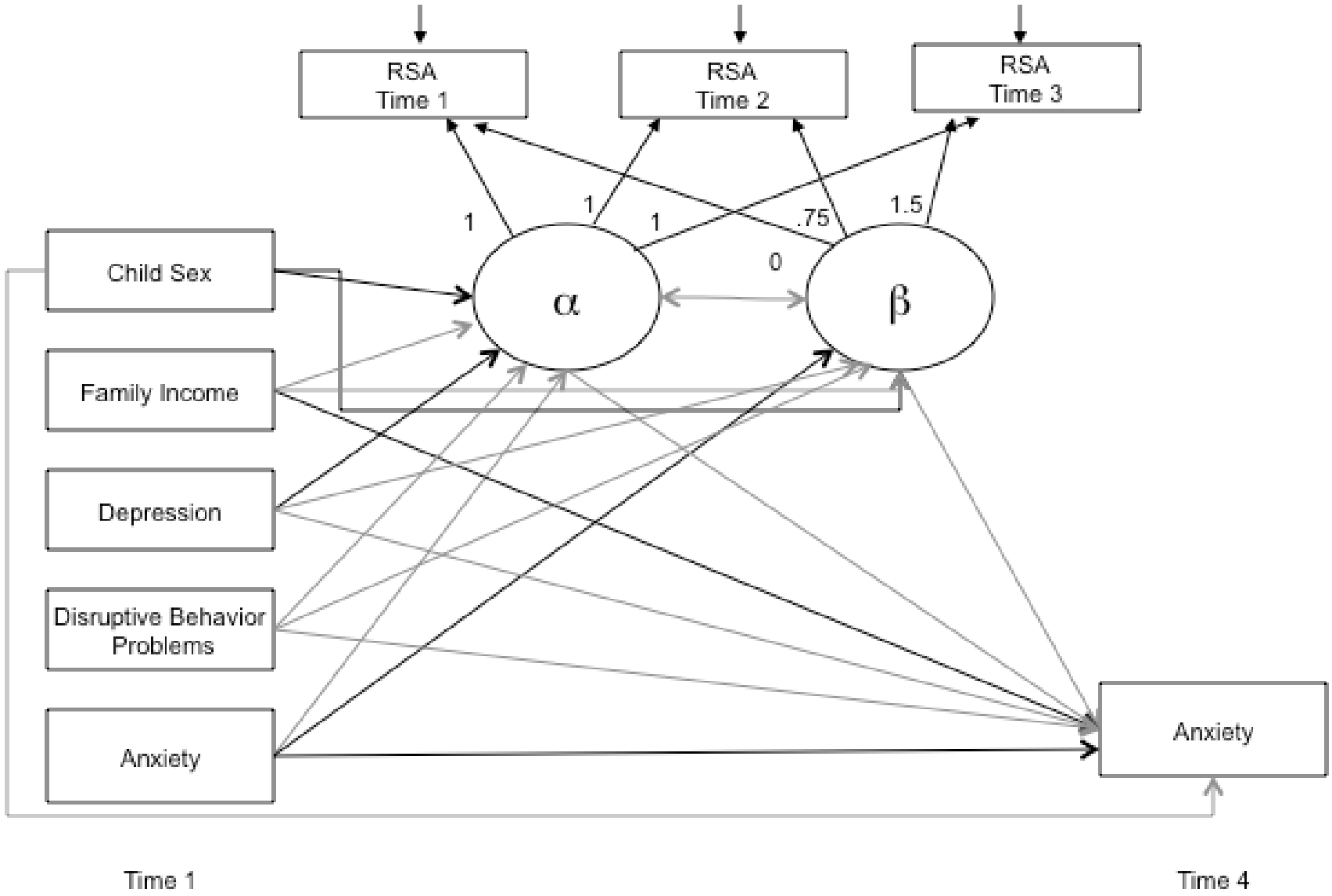

Figure 3. RSA predicting the emergence of anxiety symptoms across the study. Significant paths are presented in black and nonsignificant paths in gray.

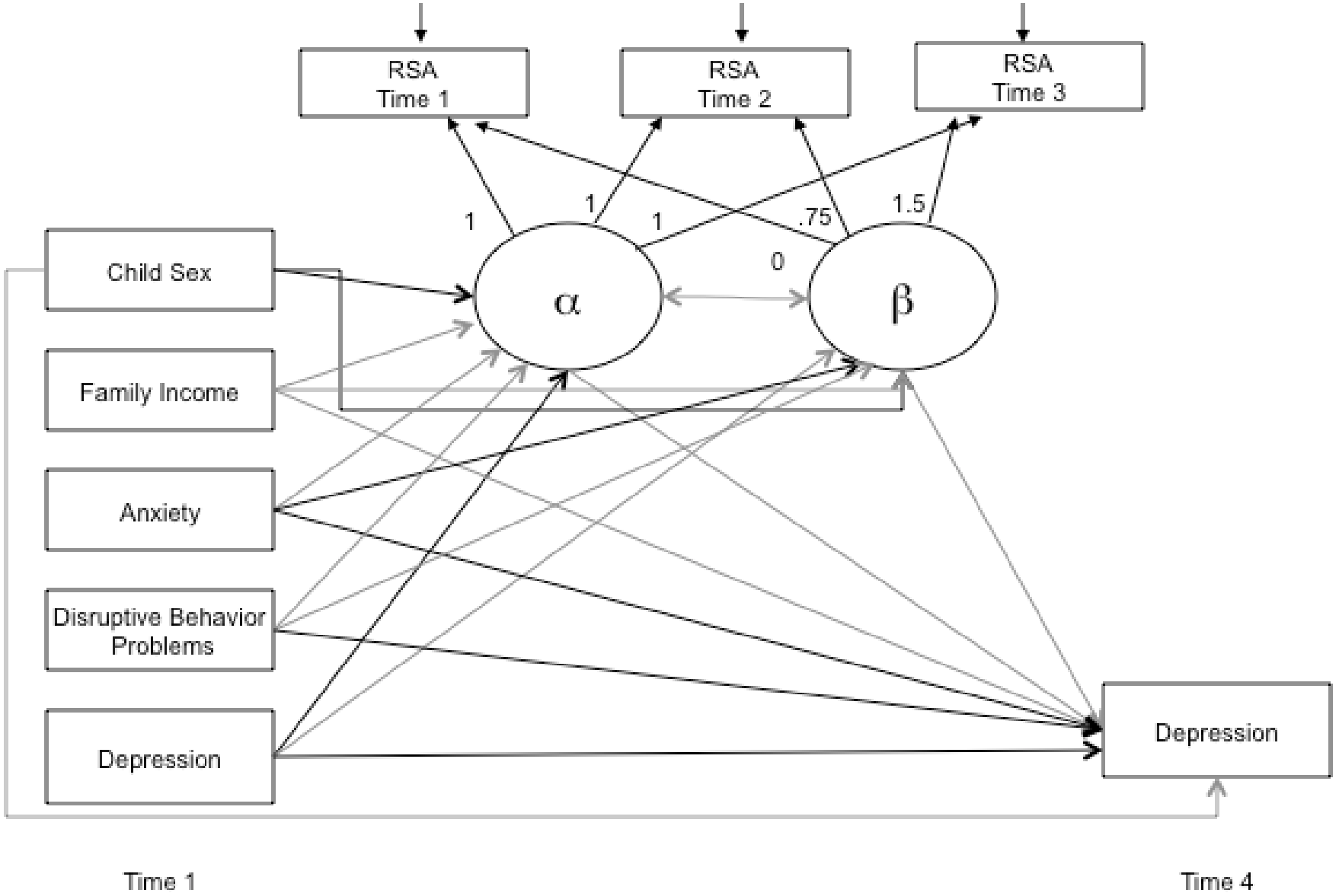

Figure 4. RSA predicting the emergence of depression symptoms across the study. Significant paths are presented in black and nonsignificant paths in gray.

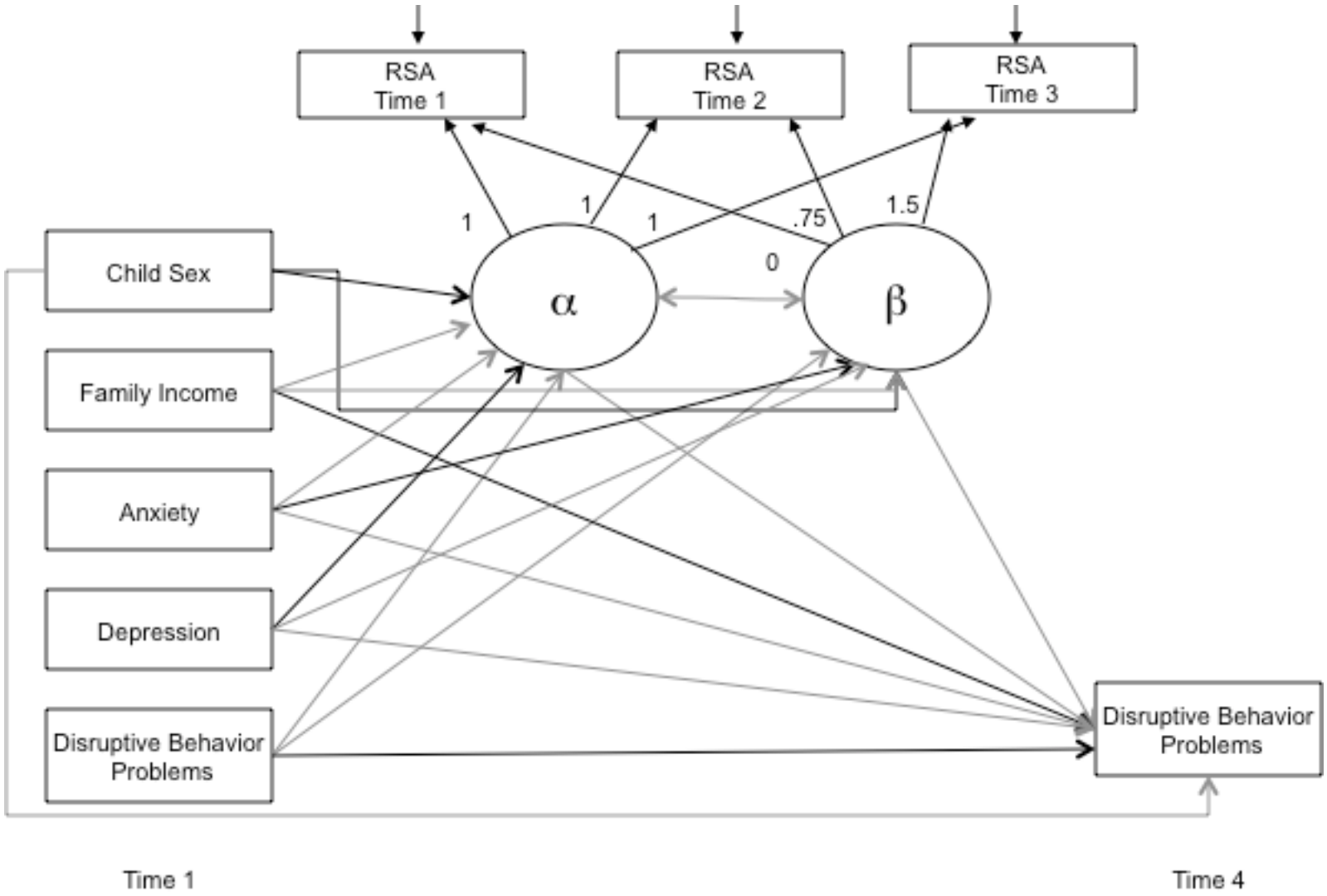

Figure 5. RSA predicting the emergence of disruptive behavior problems across the study. Significant paths are presented in black and nonsignificant paths in gray.

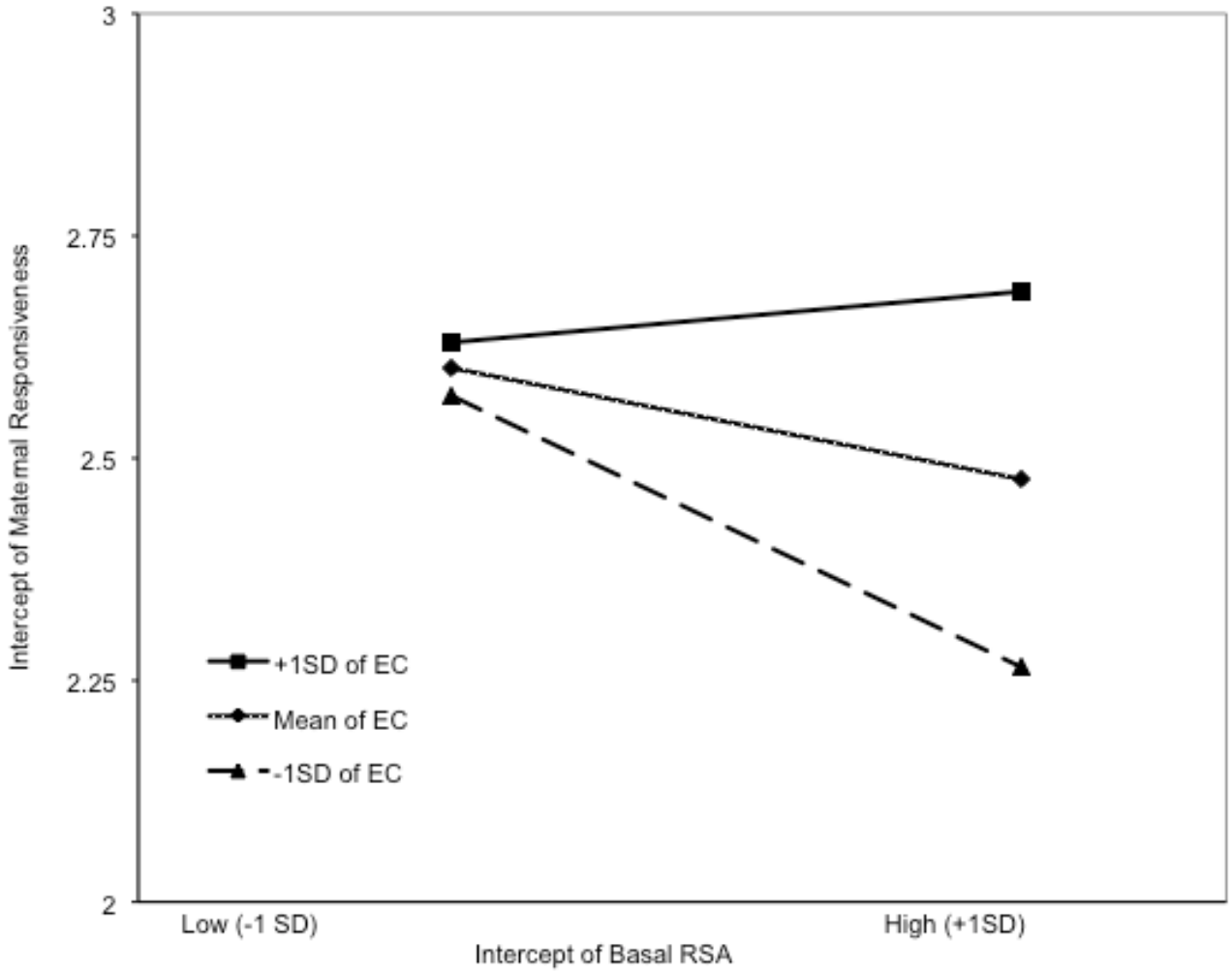

Figure 6. The interaction of basal RSA and executive control (EC) predicting the intercept of maternal responsiveness. Simple slope values are plotted at one standard deviation above and below the mean of basal RSA.

Discussion

The study targeted gaps in the existing literature by examining the role of children's emotional dysregulation, as measured by RSA, in the development of anxiety, depression, and DBP in early childhood. Moreover, we aimed to clarify the mechanisms by which RSA and parenting predict psychopathology by testing bidirectional associations between emotional dysregulation and parenting in the development of psychiatric symptoms. Lastly, this study considered the role of executive control, a core component of self-regulation, in constraining or amplifying the relations between RSA and parenting, and in turn the emergence psychopathology. Although existing literature has begun to consider the potentially complex patterns of inter-relations between children's physiological emotional reactivity and parenting in shaping development, only one known study has tested bidirectional relations between these variables (Kennedy et al., 2004). Moreover, no known study has extended bidirectional relations between vagal tone and parenting to examine potential pathways for predicting psychopathology. Thus, we aimed to elucidate processes driving patterns of dynamic and mutual influence between children's constitutionally-based characteristics and parenting behaviors.

Overall, the results provided limited support for reciprocal models between children's physiologic emotional lability and parenting behaviors. Instead, child and parental factors appeared to operate independent of one another to influence development, with one exception. The findings suggest that a potential mechanism whereby children evoke different parenting behaviors may be contingent on multiple aspects of children's characteristics, including emotional reactivity and neurocognitive regulation. This highlights the increasingly complexity of models that reflect children's development. Further, there was some support for the role of RSA and parenting behaviors in predicting child psychopathology symptoms. However, the overall findings suggest that additional factors, including intermediate levels of child characteristics (e.g., temperament), may reflect behavioral precursors of psychopathology and should be

considered in models of mutual influence.

Vagal Tone and Parenting as Predictors of Child Psychopathology Symptoms

Prior research has yielded mixed support for the relation of vagal tone and child psychopathology symptoms in preschool children. While research in older children, adolescents, and adults indicates that tonic RSA as a marker of physiological reactivity relates to expressions of psychopathology and higher levels of symptoms, few studies have considered whether these patterns extend into early childhood. Of the studies examining the preschool period, there has been some support for tonic vagal tone predicting precursors of internalizing disorders (Forbes et al., 2006; Rubin et al., 1997) and externalizing symptoms (Cole et al., 2007). However, consistent with the majority of published studies to date (e.g., Beauchaine et al., 2007), our findings suggest that initial levels and changes in basal RSA across the preschool period do not predict the emergence of anxiety symptoms, depressive symptoms, or disruptive behavior problems.

Thus, this study adds to a growing body of literature suggesting that basal RSA does not predict increases in internalizing or externalizing symptoms across the preschool period. Instead, models from older developmental periods may not extend downward to early childhood and instead indicate that alternative models linking physiologic reactivity and child mental health outcomes should be considered. One possibility is that for preschool children, we need to include behavioral precursors of psychopathology. For example, prior research has demonstrated that inhibition (e.g., Rubin et al., 2007) and is related to both basal vagal tone and early precursors for internalizing symptoms, indicating that behavioral measures of temperament may be important mediators that were not included in the current study. Further, this research may benefit from extending the developmental period measured into school age or early adolescence, thereby including periods of developmental transition. By examining multiple developmental transitions, research may clarify sensitive periods of development important for understanding how RSA emerges as a risk factor for psychopathology.

Additionally, it is interesting to note that there was some variability in the pattern of relations between initial levels of anxiety, depression, and externalizing problems with basal RSA. For example, anxiety symptoms and disruptive behavior problems at the start of the study remained stable across time. That is, the co-occurrence of other symptoms (i.e., depression and DBP when predicting anxiety) was not related to symptom levels at 63-months of age (see Figures 3 and 5). Conversely, in predicting depressive symptoms, earlier levels of co-occurring symptoms (i.e., anxiety and DBP) as well as early levels of depression predicted adjustment at 63-months. This highlights the potential for different pathways to predict the development of psychopathology and suggests that when measuring depression, the co-occurrence of other symptoms may represent nonspecific risk for psychopathology.

When parenting behaviors were considered, few relations with child psychopathology symptoms emerged. This was in contrast to prior research, which demonstrated the protective role of maternal positive parenting behaviors and the deleterious effect of maternal overcontrol and negativity (Frick, 1994; Loeber & Stouthamer-Loeber, 1986; McLeod et al. 2007). However, it is interesting to note that faster increases in maternal intrusiveness, or uninvited and potentially stifling control behavior, predicted lower anxiety symptoms at 63-months of age. This is in contrast to prior research demonstrating the deleterious effect of maternal intrusive control behaviors on the emergence and/or maintenance of child anxiety symptoms (McLeod et al., 2007). However, much of this research has been conducted in clinical samples with older children, indicating that perhaps developmental differences may account for the findings in the current study.

The Interplay of Children's Vagal Tone and Maternal Parenting

This study adds to a growing body of literature, which suggests little relation between children's physiologic reactivity and parenting behaviors. Although bioecological models, whereby children shape and are shaped by their environment (Maccoby, 2000; Propper & Moore, 2006) have been extensively advocated, few studies provide rigorous tests of such

models. Of the existing evidence for bidirectional relations of parenting and children's characteristics, most studies demonstrating support have used behavioral indicators of children's emotional dysregulation (e.g., Kiff, Lengua, & Zalewski, 2011; Pettit & Arsiwalla, 2008). Still, some emerging research suggests that maternal negative control, or intrusive and hostile parenting, predicts decreases in young children's vagal regulation (Calkins et al., 1998; Hastings et al., 2008). However, these researchers examined how parenting may shape changes in how children's RSA responds to experiences of external stress. Alternatively, the current study examined how basal RSA, a constitutional indicator of emotional reactivity, may relate to parenting across time. The lack of relations between parenting and RSA in this sample may be in part related to our measurement of tonic RSA. More specifically, Porges (1995) suggests that RSA changes across development such that patterns of vagal regulation in response to repeated environmental stress become codified into lower basal RSA across early development. Given that the current sample assessed RSA between 36- and 54-months of age, it may be that relations between parenting and vagal regulation (instead of vagal tone) would better capture reciprocal relations between parenting and children's physiologic characteristics. This would align with findings from previous research demonstrating that parenting shapes young children's vagal regulation and underlines the need to extend these models across a wider age range to best capture how development unfolds.

Alternatively, it may be that children have an evocative effect on parenting, and in turn that rearing behaviors shape children's behavior and characteristics. This has been widely theorized in the current literature and most support stems from studies examining children's behavioral characteristics (Propper & Moore, 2006). Thus, it may be that support for relations between parenting and children's emotional dysregulation are limited to more behavioral indicators of reactivity and that perhaps additional mediators (e.g., children's temperament characteristics) should be considered in these models.

Executive Control as a Modifier of Children's Vagal Tone

Although basal RSA was not shown to shape parents' behavior across this study, it may be that additional child characteristics must be considered in order to understand the evocative effect of children on parenting behaviors. More specifically, we found that it is important to consider children's executive control, a core component of self-regulation, in moderating the effect of basal RSA on parenting. Thus, when RSA and executive control were considered simultaneously, self-regulation was shown to attenuate the relation of children's physiologic regulation (as indicated by high basal RSA), and in turn reduce maternal responsiveness. This suggests that a when high physiologic regulation is combined with better executive regulation, children elicit more responsive parenting. However, when children demonstrate difficulty inhibiting automatic and potentially impulsive behaviors, less responsive parenting is observed despite lower emotional lability. Notably, self-regulation did not moderate basal RSA to shape other maternal behaviors (negativity, autonomy granting, or intrusiveness) or the emergence of child psychopathology symptoms.

Summary and Conclusions

In sum, this study provided initial support for the role of constitutionally-based physiological characteristics in shaping parental rearing behaviors. Specifically, when children's physiologic reactivity and self-regulatory characteristics are considered simultaneously we may begin to understand the processes whereby children shape parents' rearing behaviors. However, there were few significant findings, and as a result, little can be said about the reciprocal relations between children's characteristics and parenting. In part, our findings were a departure from previous research, which has supported the role of mutual influence between children's behavioral characteristics and parenting or considered more reactive components of children's physiologic responses (e.g., Calkins et al., 1998; Hastings et al., 2008). Further, the current study did not replicate and extend previous findings linking parenting and children's emotional dysregulation with the development of anxiety symptoms, depressive symptoms, or

externalizing problems. This suggests that perhaps models linking physiologic indicators and parenting with child psychopathology are not supported in preschool samples. Instead, it may be that additional mediators such as children's observed behavioral characteristics (e.g., temperament) may need to be accounted for in these models. Alternatively, it may be that models capturing children's vagal regulation in response to stimuli may best shape parents' behaviors. This would suggest that children's reactions to external stimuli, rather than constitutional reactivity, interrelate with parenting to shape adjustment. Overall, although a growing and extensive body of literature supports the dynamic interplay of children and parenting in shaping one another to influence development (Propper & Moore, 2006), additional research providing rigorous tests of these models are needed.

Strengths and Limitations

This study included several, notable strengths. The use of longitudinal methods allowed predictive hypotheses to be tested, and the application of multimethod measurement (physiological measures, neurocognitive assessments, observational ratings, and parent report). Further, this study is one of the first to test developmental trajectories of RSA and how environmental characteristics may shape this developmental course. In addition, this study employed a high risk sample and demonstrated an impressive retention rate across time. Thus, few biases in the data were related to missingness. Overall, this study was able to provide initial tests of developmental psychopathology models examining the potential interplay of children's vagal tone and maternal parenting.

However, several limitations must be considered. First, this study is limited to considering bidirectional relations between maternal parenting and children's physiological reactivity. Fathers were not included as participants in part because of the broader study effort to recruit a low income sample, which increases the likelihood that families will be single parent households (Brooks-Gunn & Duncan, 1997). In the current study, 20% of children were from single parent families reducing the availability of a second caregiver as a participant of this study. Second,

this study employed a community sample rather than a clinical sample. As a result, levels of anxiety, depression, and DBP were low. However, the sample was of relatively high socioeconomic risk, as participants from low-income families accounted for over half of participants and therefore increased the likelihood that children would exhibit increased psychopathology. Nevertheless, developmental models utilizing community samples may capture important mechanisms preceding the development and emergence of psychopathology. In addition, use of a high-risk community sample, rather than a clinical sample, eliminated potential biases introduced when participants are clinic-referred (see Caron et al., 2006). Thirdly, the current study did not account for the potential role of shared genetic effects in the development of psychopathology. Thus, mothers and children share genetic information that may contribute to basal RSA and parenting behaviors. However, this study examined how initial status and changes in RSA and parenting predicted changes in the one another, thereby examining changes in physiological and behavioral expression over time. This longitudinal analysis helps to clarify the role of environmental and genetic effects on development.

Fourth, several analytic considerations should be addressed in interpreting the study findings. These include difficulty with obtaining adequate or good model for some of the maternal parenting variables, particularly the unconditioned growth models for maternal positive affect and responsiveness. While some efforts were made to address these analytic obstacles, significant improvements in model fit were not always possible (e.g., maternal positive affect). Therefore, the general study findings should be interpreted with the limitation that the estimated unconditioned models of maternal parenting were less than ideal and therefore the study findings require replication. In addition, the conditioned models of RSA and parenting included a large number of significant covariates, which may have taxed already complicated models by reducing power. However, these variables were demonstrated to be important family characteristics that may account for maternal parenting and/or the emergence of children's behavior across time. Thus, these broader analytic concerns suggest that future studies

employing larger samples may be better suited to fully address the complex nature of how maternal parenting and children's emotional reactivity may shape one another across time.

Lastly, the level of psychopathology reported by mothers was quite low. This in part may be related to the age of the sample, as children were quite young across all time points of the study. Thus, future research should examine these models (1) across a longer developmental period and/or (2) in adolescent samples, where levels of psychopathology are expected to be higher. Lastly, the use of physiological and observational measures is a general strength of this study, allowing the objective assessment of children's emotional reactivity and parenting behaviors. However, parenting behaviors observed in structured interactions may not reflect actual interaction patterns, but rather dyads' "best behavior". Thus, the measures of parenting might be regarded as a lower bound, and therefore effects may be underestimated (Morris et al., 2002). Further, the lack of association between parenting and children's physiology suggest that intermediate levels of analysis at the individual level need to be considered as mediators of physiologic reactivity on children's behavior. Thus, future research may consider the dynamic interplay between children's observed emotional reactivity and parental rearing behaviors. Consideration of how parents' and children's behavior shape one another may elucidate mechanisms in children's development.

Implications and Future Directions

Researchers have long suggested that children influence and are influenced by their environment (e.g., Bell, 1968). Further, a key mechanism of this purported dynamic process of developmental influence is thought to operate through parents' rearing behaviors. In this study, we targeted gaps in a growing, but small body of literature, attempting to uncover the mechanisms whereby precursors such as emotional dysregulation develop into mental health outcomes.

The findings of this study provide limited support for models of bidirectional relations between parenting and children's vagal tone. Overall, the complex patterns whereby children

and parents mutually influence on another require large-scale, longitudinal studies. This research provides an initial investigation and helps clarify the developmental periods and child factors that should be examined in our attempt to explain the processes underlying the development of child psychopathology. By clarifying how constitutionally-based factors transact with the environment to predict abnormal development, we can begin to identify developmental mechanisms and targets for interventions. Moreover, consideration of individuals' emotionality allows tailoring of treatment methods, producing more efficacious and effective interventions (Sheeber & McDevitt, 1997).

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Vita

Cara Kiff was born in Menomonee Falls, Wisconsin but spent most of her youth in Southern California. She completed her undergraduate degree in Psychology at UCLA, where she graduated magna cum laude and with highest departmental honors. Her time at UCLA sparked her interest in the role of parenting in children's anxiety symptoms. This drive inspired her to earn her doctorate at the University of Washington where she developed a research program examining the interaction between individual and contextual risk factors in the development of psychopathology. In 2012 she earned her Doctorate in Child Clinical Psychology with a minor in Quantitative Psychology at the University of Washington. She completed a one-year clinical internship at the University of Washington School of Medicine.