Predicting the Developmental Trajectories of Externalizing and Internalizing Behaviors from Parenting Quality and Children's Respiratory Sinus Arrhythmia

by

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ABSTRACT

The current study delineated the developmental trajectories of early childhood externalizing and internalizing symptoms reported by mothers and fathers, and examined the role of the 18-month observed parenting quality × Respiratory Sinus Arrhythmia (RSA) interaction in predicting these trajectories. Child sex was tested as a covariate and moderator. It was found that children's low baseline RSA or high RSA reactivity, in comparison to high baseline RSA or low RSA reactivity, was more reactive as a function of early parenting quality when predicting the development of early childhood problem symptoms. Differential patterns of the interaction between parenting quality and RSA were detected for mothers' and fathers' reports. Mother-reported models showed a diathesis-stress pattern, whereas the father-reported model showed a vantage-sensitivity pattern, especially for internalizing symptoms. This may imply the potential benefit of fathers' active engagement in children's early development. In addition, the effect of the parenting quality × RSA interaction in predicting the mother-reported models was found to be further moderated by child sex. Specifically, the parenting quality × baseline RSA interaction was significantly predictive of girls' 54-month internalizing, and the parenting quality × RSA reactivity interaction significantly predicted boys' internalizing slope. Girls with low baseline RSA or boys with high RSA reactivity were vulnerable to the less positive parenting, exhibiting high levels of 54-month internalizing symptoms or slow decline in internalizing over time, respectively. Future research directions were discussed in terms of integrating the measures of SNS and PNS in psychopathology study, exploring the mechanisms underlying the sex difference in parenting quality × RSA interaction, and comparing the findings of children's typical and atypical development.

Keywords: Observed Parenting Quality, RSA, Sex, Externalizing Symptoms, Internalizing Symptoms

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INTRODUCTION

Externalizing is regarded as a general category of problem behaviors including aggressive, oppositional defiant, anti-social behaviors, delinquency, and hyperactive behaviors, whereas internalizing refers to the category of problem behaviors to which depression, anxiety, social withdrawal, fearfulness and psychosomatic symptoms belong. Starting early in life and persisting into adolescence and early adulthood, both externalizing and internalizing behaviors in childhood are reported as precursors of various forms of continued maladaptation, such as academic difficulties (Hinshaw, 1992; Valiente, Swanson, & Eisenberg, 2012), peer rejection (Brendgen, Vitaro, Bukowski, Doyle, & Markiewicz, 2001; Laird et al., 2001), and poor adulthood physical health (Slopen, Kubzansky, & Koenen, 2014).

The developmental trajectory of externalizing behaviors has consistently been found to decline after approximately three years old across early childhood (Alink et al., 2006). Internalizing behaviors have been reported to slightly decrease during preschool years (Sterba, Prinstein, & Cox, 2007), and then to increase throughout childhood into adolescence for both boys and girls (Leve, Kim, & Pears, 2005; Keiley, Bates, Dodge, & Pettit, 2000). However, externalizing and internalizing behaviors are also subject to great individual differences as children grow, and biological/physiological (e.g., genetic predisposition and physiological changes), social (e.g., cultural norms, family/socialization process, neighborhood, school context and peer relationships), and psychological factors (e.g., temperament) have been reported as diverse predictors of the development of externalizing and internalizing symptoms (more reviews below). The current study investigated the growth curves of externalizing and internalizing behaviors

across 24 months to 54 months, and explored the role of 18-month parenting, 18-month children's RSA (respiratory sinus arrhythmia), and their interactions as predictors of these growth curves.

Traditional Definitions of Parenting

Literature and research on parenting and its effect in child development are somewhat difficult to integrate, partially because of the different definitions and measures used in different studies. Some developmental theorists define parenting as a whole and organized construct (i.e., parenting style; Baumrind, 1971, 1991), of which the potential effects cover across a wide range of aspects of children's adjustment. However, other theorists stress the importance of unpacking parenting into individual components in order to pinpoint the specific effects of parenting on the specific outcomes of interest (i.e., specificity of parenting; Caron, Weiss, Harris, & Catron, 2006; O'Conner, 2002). In addition, related terms depicting parent-child relationships are also used interchangeably with parenting (e.g., mother-infant attachment as a measure of maternal parenting). I should first acknowledge that it is not possible for researchers to draw a clear boundary between the concept of parenting style and specific parenting practices. This is because parenting styles can be viewed as different combinations of the specific parenting behaviors (Baumrind, 1991). Yet, to some extent, this distinction is useful, especially when it comes to the description, explanation, prediction, and intervention in parenting dynamics (Grusec, Goodnow, & Kuczynski, 2000), and to the understanding of how parenting influences child behavioral development as a proximal environmental factor.

Parenting styles. Among the perspectives on parenting, parenting styles have been of primary interest to developmental researchers for a long time. Parenting style is

an organized and stable pattern derived from the parental attitudes, values, and beliefs about children's development combined with their actual parenting practices (Baumrind, 1971, 1991). Any influence of specific parenting practices should be considered as depending on the organization and integration of all other aspects. Based on the idea of interactions between warmth/support and control/demandingness (Baumrind, 1991), parenting style has been classified into four prototypes: *authoritative* parenting, which combines high warmth and firm control by parents implementing reasoning and structuring; *authoritarian* parenting, which is characterized by parental high demandingness and power assertion but with low warmth; *permissive* parenting, which features high parental support and responsiveness but without controlling or monitoring of children's misbehaviors; and *disengaged* parenting, characterized by parents who are neither demanding nor warm, but rather, actively neglect their responsibility as a caregiver (Baumrind, 1991).

Throughout the years of research after Baumrind proposed the parenting style, authoritative parenting has been identified as the most beneficial style of parenting (Baumrind, 1996) and has been related to children's successful socio-emotional development (O'Reilly & Peterson, in press), better ego-resiliency (Dubas, Gerris, Janssens, & Vermulst, 2002), and increased self-esteem and competence (Farris, Lefever, Borkowski, & Whitman, 2013), as well as general mental health (Joussemet, Mageau, & Koestner, 2013). Authoritarian parents who are restrictive and punitive, on the other hand, require high child compliance and obedience, and they tend to have children with compromised outcomes (Spera, 2005).

From the approach of Baumrind's typology of parenting, it is tempting to conclude that the differential developmental outcomes of children from families with authoritative parents versus authoritarian parents are due to the different levels of parental warmth/support because the demandingness/control are supposed to be high in both families. However, this is not entirely correct. As noted in the early work of Maccoby and Martin (1983), differences in the quality and quantity of "demandingness/control" between these two styles of parenting cannot be captured by the model depending on the two-dimensional measures containing merely warmth and demandingness. Instead, it should distinguish important characteristics such as restrictive control versus authoritative control. Acknowledging this point, Baumrind et al. (2010) explicitly differentiated two types of demandingness/control—coercive control (i.e., restrictive control and power assertion) and confrontive control (i.e., firm and consistent discipline). They refer the coercive control as *authoritarian-distinctive* controlling behaviors, while confrontive control is a part of authoritative parenting. This distinction is important in terms of two aspects: first, it helps to solve the inconsistency in the study findings using different operationalizations of parental control; second, it implies the potential to explore individual parenting behaviors/practices rather than parenting style as a whole. This work brought up the potential idea of untangling the effects of parenting style by exploring individual parenting practices.

Another criticism of defining parenting style from this configuration approach is that it is not useful for interpreting individual or cultural variability in parenting behaviors. For example, why was the authoritarian style of parenting that may seem disadvantageous in Western countries actually predictive of higher academic achievement

within Eastern Asian cultures (Chao, 1994)? In Chao's early work, she proposed that there are some unique concepts/dimensions in Chinese parenting that are not captured by the authoritarian style of parenting from Baumrind theory, such as emphasizing children's obedience in reaching the socialization goals for filial piety (Chao, 2000). She further argued that it is a culturally specific parenting practice that is associated with different outcomes of Western versus Eastern Asian children. Thus, cross-cultural work on parenting also promoted the developmental research on specific parenting practices.

Specific parenting practices. In fact, back to the 90s, Darling and Steinberg (1993) conceptualized parenting style as a context in which the specific parenting practices exert influences during the socialization processes. It is argued that research questions regarding parental socialization cannot be accurately addressed unless developmentalists acknowledge, and thus demonstrate the distinctions between parenting style and specific parenting behaviors in their studies. Caron and colleagues (2006) also highlighted the investigation of specific parenting behaviors as a critical issue relevant to understanding the development or maintenance of child psychopathology.

There are several specific parenting dimensions unpacked from the Baumrind's parenting styles that have been substantially examined in child psychopathology literatures. The first dimension is parental warmth/responsiveness. It denotes parental expression of positive emotions, affect, admiration and encouragement toward the child. It also involves parental fondness and enjoyment of interacting with children and parent-child closeness (e.g., Roberts & Strayer, 1987). Second, parental sensitivity, based on the tenet of attachment theory, refers to the extent to which parents accurately perceive and interpret the signals of child behaviors, and to if parents respond to them promptly,

appropriately, and consistently (e.g., Ainsworth, 1979; Lohaus, Keller, Ball, Voelker, & Elben, 2004). Third, authoritative control, which is similar to confrontive control associated with Baumrind's authoritative style of parenting, refers to the parenting behaviors wherein rules and limits are imposed on children by parental emphasis of discussion, clear explanation, and communication (Baumrind, 1966).

On the other hand, parental intrusiveness has been regarded as parent-centered caregiving behaviors; it is a coercive control that features parents' prohibiting unwanted child behaviors by verbal demanding and physical interference (Baumrind, 2012). Despite the slightly different terms used in different studies (e.g., authoritative control is also labelled as firm supervision or behavioral control; intrusiveness is also labeled as harsh control, or psychological control which is viewed as the opposite of parental autonomy support), the aforementioned parenting dimensions are believed to represent basic structures of the parenting process, and they individually or jointly function as predictors of the development of children's psychopathology. For example, Gray and Steinberg (1999) found that higher parental autonomy granting along with firm supervision/monitoring was associated with lower internalizing symptoms among adolescents (more review of studies is presented in the following section).

The concept of parenting quality. The definition of parenting (i.e., parenting quality) and its measurement in the current study reflected an integration of the traditional parenting style and specific parenting practices; that is, it was conceptualized as a global parenting construct derived from the linear combination of parental warmth, sensitivity, authoritative control, and intrusiveness (see the method section below). Although it is a global parenting measure that appears to be similar to Baumrind's

typology of parenting styles, the parenting quality variable used is different in that warmth and intrusiveness are not considered as independent or orthogonal dimensions as in Baumrind's theory. I expected their effects to be intertwined such that the linear combination of high warmth and low intrusiveness represents positive parenting quality (Deater-Deckard et al., 2011; Wright & Cullen, 2001). Also different from Baumrind's parenting typologies, parenting quality in the current study took into consideration the parent-child relationship/harmony during their interactions.

Furthermore, instead of examining specific parenting behaviors individually, the index of parenting quality used in this study reflected a parenting strategy that combines the individually coded parenting behaviors. By doing this, the focus was not on one or two parenting dimensions, which has been viewed as a potential limitation in parenting research (Caron et al., 2006). Therefore, based on above definition/theoretical considerations of the parenting variable and the current parenting quality variable with integrated nature, the following section reviews previous studies relating parenting (both parenting style and specific parenting practices aforementioned) to childhood problem behaviors (i.e., externalizing and internalizing).

Parenting and Children's Problem Behaviors

Parenting styles and problem behaviors. Child psychopathology researchers studying parenting styles as a predictor have reported the association of authoritative parenting style with not only less concurrent behavior problems during childhood (Querido et al., 2002), but also a more dramatic decline in externalizing and a smaller increase in internalizing through the transition years of childhood to adolescence compared to other types of parenting style (Williams et al., 2009). Authoritative parenting

was also found to deter affiliation with deviant peers and involvement in juvenile delinquency (Simons, Simons, Burt, Brody, & Curtona, 2005). In addition, adolescent criminal offenders with authoritative parents, relative to those from families with other types of parenting style, were more psychologically mature (i.e., higher in personal responsibility, aggression suppression, empathy and resistance to peer influence), and less prone to internalizing symptoms (Steinberg, Blatt-Eisengart, & Cauffman, 2006). This study also found that juvenile offenders with neglectful parents demonstrated the least mature characteristics, and highest level of internalizing symptoms, whereas the associations for authoritarian and permissive parenting styles were in the middle.

In fact, neglectful parenting style, which often involves parents' chronically failing to provide proper supervision and affection, has been consistently identified as predicting severe cognitive and academic deficits, social inhibition, and internalizing problems among children and adolescents (see Hildyard & Wolfe, 2002, for a review). However, the permissive parenting style appears to have mixed relations to different child outcomes. For example, children from permissive families tend to perform poorly in school and tend to score high in externalizing problems (such as drug and alcohol use), yet demonstrate adjustment in terms of social competence and self-confidence, and lower internalizing problems (Darling, 1999; Steinberg, Lamborn, Darling, Mounts, & Dornbusch, 1994).

In regard to the authoritarian parenting style, although adolescents from authoritarian families also exhibited a mix of positive (i.e., better academic performance; Darling, 1999) and negative outcomes, the relation between authoritarian parenting and higher externalizing behaviors is relatively clear. For example, early empirical work on

authoritarian parenting reported that maternal authoritarian parenting during preschool years significantly predicted mother-, and teacher-reported externalizing in first grade after controlling the stability of children's externalizing (Heller, Baker, Henker, & Hinshaw, 1996). Zhou and colleagues (2004) found similar results among Chinese school-aged children, such that authoritarian parenting was associated with lower level of children's effortful control, which in turn, predicted higher levels of externalizing problems. Furthermore, children's internalizing behaviors have also been positively predicted from authoritarian parenting (Lee et al., 2013). A recent study on the Chinese early-school-aged children detected an interaction between authoritarian parenting and children's effortful control in predicting children's internalizing behaviors 3.8 years later (Muhtadie, Zhou, Eisenberg, & Wang, 2013). Specifically, relative to their counterparts with low effortful control, children with high effortful control displayed a decline in their internalizing behaviors when exposed to an increased level of authoritarian parenting, suggesting a moderating effect of child characteristics.

From the previous literature discussed above, it appears that authoritative parenting is an optimal parenting style that is beneficial to lower levels of problem behaviors in children and authoritarian parenting is partially detrimental (i.e., depending on the potential moderators) across different cultures and contexts. Amato and Fowler (2002) also reported similar direct relations of parenting styles regardless of parents' race, ethnicity, and family structure.

Another interesting issue is differences in the parenting styles of mothers and fathers, as well as their potential differences in the effects in children's development. Nevertheless, investigators have highlighted the importance of the coherence of maternal

and paternal parenting, such that having two authoritative parents is predictive of the most positive outcomes among adolescents (i.e., lower delinquency, and depression; Rudy & Grusec, 2001). However, having one authoritative parent may buffer children from negative consequences associated with the absence of two authoritative caregivers.

Neither cross-cultural issues nor the effect of parent gender was explored in the current study. Thus, the subsequent literature review is not focused on these factors.

Specific parenting practices and problem behaviors. As noted previously, parental warmth, sensitivity and authoritative control, along with parental intrusiveness, which are unpacked from the parenting styles, have been the focus of research on specific parenting behaviors. Because these parenting behaviors are the primary interest of the present investigation, theoretical rationales and empirical studies linking these parenting practices and children's problem behaviors are reviewed.

Theoretical rationales. Theoretically, there are several ways that parental warmth/sensitivity and intrusiveness might influence children's externalizing and internalizing behaviors, especially via emotion and behavioral regulation processes. First, parents who are higher in warmth and sensitivity are generally lower in psychopathological symptoms themselves (Kendler, Sham, & MacLean, 1997; Adam, Gunnar, & Tanaka, 2004). They are more capable of managing their own emotions and offer rapid responses to children's needs during parent-child interactions. Thus, those parents are more likely to model effective behavioral regulation strategies for children to learn and use in the situations in which aggressive reactions (such as punching at an annoying peer) or ruminating on a depressing event must be controlled (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Moreover, warm and sensitive parents also tend to

have higher SES and education (Davis-Kean, 2005), and they are more likely to create a harmonious family climate by demonstrating low levels of marital conflicts and expressing more positive emotions toward family members (Linver, Brooks-Gunn, & Kohen, 2002). All of these beneficial parenting practices have been proposed to influence children's behavior problems through promoting children's emotion understanding and interpretation of emotion clues (Eisenberg, Cumberland, & Spinrad, 1998), as well as through fostering a secure attachment (Buist, Deković, Meeus, & Aken, 2004).

Second, parents high in warmth and sensitivity tend to encourage their children's expression of negative emotions, whereas controlling and intrusive parents are more likely to suppress, neglect, or even disapprove children's negative emotions (Bariola, Gullone, & Hughes, 2011). These different parental reactions may be associated with children's distinct behavioral responses to external clues. For example, while experiencing increased physiological arousal, children who hide negative emotions due to the consistent exposure to parental coercive control of emotion expressivity may feel depressed or anxious in potentially insecure contexts (Robert & Strayer, 1987).

Third, active guidance and support from parents high in warmth and sensitivity are optimal for the development of young children's behavioral control/regulation ability. Through the effective parental socialization, children have more opportunities to internalize their parents' regulation strategies (Choe, Olson, & Sameroff, 2013). This is argued to be critical in the development of children's independent emotional, attentional, and behavioral regulation (Sameroff, 2009).

In contrast, parenting that characterized by higher intrusiveness and demandingness is believed to impair children's self-control ability and, thus, is expected

to be associated with elevated externalizing (Eisenberg et al., 2003; Ramsden & Hubbard, 2002) and internalizing problems (Muhtadie et al., 2013). However, authoritative control, which, as aforementioned, pertains to parental close monitoring and regulating children's behaviors by using age-appropriate reasoning and explanations, allows parents to serve as supportive external regulators of children's behaviors by means of scaffolding them to refrain from disruptive behaviors in lieu of more regulated alternatives (Choe et al., 2013; Pettit, Bates, & Dodge, 1997). Therefore, having warm and supportive parents is believed to enhance children's willingness to internalize parental socialization rules and efforts and, thus, their adjustment. Conversely, parenting behaviors that feature low warmth but high power assertive controlling and discipline strategies (such as spanking or verbal harsh controlling) are believed to heighten children's negative arousal (Cummings, Keller, & Davies, 2005), and may compromise children's intentions to follow parental socialization instructions (Eisenberg et al., 2005). These parenting practices might further hinder children's learning in the disciplinary context and the development of self-regulatory ability in controlling externalizing symptoms and regulating internalizing symptoms.

Empirical studies. In addition to the aforementioned theoretical rationale, empirical research also has linked the discussed parenting practices with the development of children's behavior problems. For example, McKee and colleagues (2008) reported an association between parental warmth and decreased externalizing symptoms throughout late childhood to early adolescence. Similar results were also found in a sample of at-risk African Americans (Jone et al., 2008). Another longitudinal study with 5-year-old children reported that children from families with higher parental emotion responsiveness

and emotion coaching demonstrated fewer teacher-reported behavioral problems 3 years later (Hooven, Gottman, & Katz, 1995). The positive relation between parental warmth and a low level externalizing behavior was also supported when aggression during peer play among children with and without conduct problems were examined (Katz & Windecker-Nelson, 2004).

Parental sensitivity, often as a combined measure with parental warmth, has been related to relatively low levels of externalizing symptoms in a 5-year longitudinal NICHD study (Bradley & Corwyn, 2007). By creating a composite from parental support, autonomy granting, and low hostility, Belsky and colleagues (2007) also found that higher positive maternal parenting at 54 months predicted children's attentional control two years later, which in turn predicted lower teacher-reported externalizing problems one year later.

Although the negative associations between warm/sensitive parenting and internalizing problems were not substantially documented (instead, findings are more about the positive relations between intrusive or low warm parenting and high internalizing), several intervention studies demonstrated the effectiveness of intervention programs in reducing children's internalizing. For instance, children who participated in an attachment-based program targeted at improving caregivers' sensitivity showed lower level of internalizing after the 10-week intervention (Moss et al., 2011). Similarly, Van Zeijl et al. (2006) found that an intervention program promoting parents' sensitive discipline was effective in reducing children's externalizing (overactive) problems, especially in the families with more daily hassles and home chaos. Another intervention study revealed the beneficial effects of parenting improvement (e.g., higher warmth and

positive involvement) for decreasing children's externalizing and internalizing problems (DeGarmo, Patterson, & Forgatch, 2004).

Finally, relations between parental authoritative control and lower level of externalizing problems have been well-documented (Grolnick & Pomerantz, 2009). For example, parental monitoring was directly predictive of low levels of adolescents' substance use. This association was also indirectly mediated by parental knowledge about adolescents' activities and friendships (Fletcher, Steinberg, & Williams-Wheeler, 2004). Parental monitoring and warmth were positively correlated with parental knowledge, which in turn, predicted lower substance use among adolescents. Researchers further pointed out that there are various sources for parents to get the information of their children's whereabouts, such as child disclosure, and parent surveillance and solicitation (Fletcher et al., 2004). In fact, results from Stattin and Kerr's (2000) study suggested that children's disclosure as the source of knowledge explained the largest variance in parental monitoring, and was linked to children's lower delinquency (such as norm-breaking and police contact). According to Stattin and colleagues, a child's own voluntary disclosure might be a sign of a good parent-child relationship (e.g., a secure attachment), and secure attachment status is positively correlated with parental warmth and sensitivity.

On the other hand, a considerable amount of evidence indicates that frequent and intensely negative mother-child interactions is a sign of parenting dysfunction, which tends to be detrimental to children's adjustment. For instance, it has been reported that low maternal warmth/sensitivity toward 2-year-old children was predictive of lower self-regulation a year later, which in turn, predicted more externalizing behaviors in kindergarten (Eiden, Edwards, & Leonard, 2007). Low levels of parental warmth and

high levels of rejection were also related to increased externalizing and internalizing symptoms among adolescents (Muris, Meesters, & van den Berg, 2003). Parental intrusiveness and hostility at the 7th, 8th, and 9th grade were reported to predict children's depressive symptoms as well as conduct problems at the 10th grade (Ge, Best, Conger, & Simons, 1996). Low warm-engaged parenting was associated with higher externalizing behaviors (i.e., oppositional defiant, ADHD, and conduct symptoms), and combined symptoms of externalizing and anxiety disorder (Bayer, Sanson, & Hemphill, 2006; Gere et al., 2012).

Furthermore, Stocker and colleagues (2007) reported a positive relation between parental negative emotion expressivity in the family (i.e., one indicator of lower parental warmth that may cause children's feeling of being rejected) and self-reported internalizing symptoms among adolescents. Similar findings were documented in another study with young adults such that perceptions of parental punishing and neglecting responses to negative emotion display (i.e., lower warmth and sensitivity, more intrusiveness) were linked to higher psychological distress in males and females (Garside & Klimes-Dougan, 2002). The same findings were obtained for adolescents (O'Neal & Magai, 2005) and for female teenagers with high risk for clinically-diagnosed alcoholism (Haller & Chassin, 2011). Additionally, in comparison to parents of children with no evident behavior problems, significantly more intrusive socializing behaviors (i.e., the requirement of obedience) were detected among parents of children with the co-occurrence of externalizing and internalizing symptoms (Klimes-Dougan et al., 2007).

To summarize, externalizing and/or internalizing behaviors during childhood and adolescence have been fairly consistently negatively related to different measures of

parental warmth and sensitivity, such as high quality of attachment in infancy (Buist et al., 2004; Guttmann-Steinmetz & Crowell, 2006; Lyons-Ruth, Ann, & Cibelli, 1997), high positive expressivity (Eisenberg et al., 2005), parental acceptance (Scott, Scott, & McCabe, 1991; Wood et al., 2003) or involvement (Beyers, Bates, Pettit, & Dodge, 2003; Reitz, Deković, & Meijer, 2006), firm but noncoercive behavioral control (Galambos, Barker, & Almeida, 2003), and effective and constructive communications and interactions (Boeldt et al., 2012) . Conversely, intrusiveness and high psychological control (Aunola & Nurmi, 2005; Pettit, Laird, Dodge, Bates, & Criss, 2001), perceived parental strictness (Reitz et al., 2006) and hostility (Morris et al., 2002), and a high level of parental physical/corporal discipline (Deater-Deckard & Dodge, 1997; Deater-Deckard, Ivy, & Petrill, 2006) have all predicted a higher level of externalizing and/or internalizing behaviors among children.

In addition, to understand the established relations between parenting and child problem behaviors, developmental psychopathologists have also been concerned with the emergence and maintenance of disorders at the level of pinpointing the specific vulnerabilities (biologically, physiologically and psychologically) that predispose children to different behavior problems. Among these "vulnerabilities," a physiological indicator—cardiac RSA—was the focus of the present study.

RSA (respiratory sinus arrhythmia)

The human autonomic nervous system (ANS) is comprised of the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) branches, the effects of which are generally antagonistic. The presence of external threats will trigger the "flight-or-fight" response mode (Cannon, 1929) in which SNS activity is increased and

PNS activity is withdrawn to optimize the organism's metabolism for confronting the challenges. Physiologically, these ANS activities are reflected in the symptoms of increased heart rate and blood pressure. Contrary to the "activation" function of SNS, PNS, reflected in cardiac vagal tone, promotes restoration and conservation from these challenge-triggered responses by slowing heart rate and decreasing blood pressure, and is proposed to be an index of human regulatory mechanism (Porges, 1992).

Among the measures of human PNS, baseline RSA (i.e., an index of baseline vagal tone) and RSA reactivity (i.e., an index of vagal reactivity) are most widely used in psychological research. Respiratory sinus arrhythmia (RSA) is defined as *a cardiorespiratory phenomenon characterized in mammals by heart rate or R-R-interval (RRI) fluctuations that are in phase with inhalation and exhalation cycles* (Grossman & Taylor, 2007, p. 263). It has been shown to reflect rhythmic waxing and waning effects of the cardiac efferent fibers, which regulate the cardiac vagal activity through consistent feedback to the brain. Specifically, efferent fibers, serving as the cardiac pacemaker, are inhibitory in nature, and thus their activation slows heart rate and reflects RSA suppression/withdrawal.

Baseline RSA (i.e., the measure of RSA during relative quiescence periods) reflects the functioning of PNS at rest and may be related to temperament, emotionality, and the ability to initiate reaction (Porges, 2007). RSA reactivity (i.e., estimated by the RSA shift in response to external demands) reflects PNS responding to the environmental challenges where coping is required. Depending on the activity of the vagus efferent fibers, RSA reactivity includes RSA suppression (assessed by RSA decrease from the baseline) and RSA augmentation (assessed by RSA increase from the baseline).

Baseline RSA. In terms of the functions of baseline RSA, some researchers have found relations between baseline RSA and children's temperamental reactivity (Blandon, Calkins, Keane, & O'Brien, 2008; Calkins, 1997), both of which has been linked to children's behavior problems. However, the nature of these associations is somewhat inconsistent.

During infancy, studies have linked higher baseline RSA to infants' greater behavioral reactivity (Porter, Porges, & Marshal, 1988) and more mother-reported temperamental difficulties (Porges, Doussard-Roosevelt, Portales, & Suess, 1994). In two additional studies, infants with higher baseline RSA also demonstrated higher regulatory disorders at 8 months of age (Degangi, Dipietro, Greenspan, & Porges, 1991) and more negative emotion expressions during arm-restraint procedure at 5 months of age (Stifter & Fox, 1990). However, other researchers found opposite results such that newborns with higher baseline RSA exhibited larger cortisol responses (i.e., greater stress reactivity) toward an aversive stimulus, implying better neurobehavioral organization (Gunnar, Porter, Wolf, Rigatuso, & Larson, 1995). Also, positive relations between higher baseline RSA and infants' lower negative emotionality (Huffman et al., 1998), more active engagement (Stifter, Fox, & Porges, 1989), and greater attentional ability indexed by shorter period of attention to novel stimuli (Richards, 1985) have been documented. The discrepancy among different findings may imply that RSA in infancy is a physiological marker of behavioral reactivity to external challenges, both in positive and negative way (Beauchaine, 2001).

In contrast to the period of infancy, researchers have reported a relatively consistent pattern between higher baseline RSA and better developmental adjustment and

higher sleep efficiency (Elmore-Staton, El-Sheikh, Vaughn, & Arsiwalla, 2012) during childhood and preschool years. In a series of analysis, Eisenberg and colleagues found that high baseline RSA, especially in boys, predicted more sympathetic responses (Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994; similar findings for school-aged children, Fabes, Eisenberg, & Eisenbud, 1993), as well as better emotion regulation rated by parents and teachers (Eisenberg et al., 1995). However, findings often were nonsignificant (Beauchaine, Hong, & Marsh, 2008) and sometimes negative for girls (Fabes et al., 1993). In addition, cognitive functioning was also related to RSA such that higher baseline RSA was predictive of better performance on Woodcock-Johnson III scales assessing fluid intelligence (Staton, El-Sheikh, & Buckhalt, 2009). Compared to their counterparts with lower RSA, 3.5-year-old children with higher baseline RSA performed significantly better in laboratory tasks examining executive function (Marcovitch et al., 2010).

In fact, baseline RSA is important because researchers believe that it reflects the dynamic range that allows RSA reactivity to occur (Beauchaine, 2001; Porges, 2007). It is possible that higher baseline RSA enables individuals to be aware of environmental stressors, and thus be more prepared to generate coping strategies to handle those stressors. The positive relation between children's higher baseline RSA and consistent RSA suppression during a number of tasks has been reported by Calkins (1997).

RSA reactivity. As aforementioned, RSA reactivity reflects the extent to which children are engaging with the environment and coping with the external challenges; it also reflects cooperation, organization and regulation of different systems (e.g., physiological, behavioral and cognitive systems). Porges (2007) argues in his polyvagal

theory that RSA suppression/withdrawal is the sign of successful RSA reactivity due to its effects in stimulating coping behaviors and facilitating adaptive flexibility. According to Porges, it is of evolutionary importance for humans to engage in appropriate social behaviors by distinguishing safe from threatening contexts, and failure in this distinction may contribute to the core characteristics of psychopathology. For instance, inability to inhibit defensive systems (i.e., lower RSA suppression) in a safe environment might be associated with the emergence of anxiety and depression (Porges, 2007).

Consistent with this argument, investigators tend to detect positive relations between higher RSA suppression and children's competent regulatory and social functioning, whereas lower RSA suppression or higher RSA augmentation has been associated with behavior problems (Boyce et al., 2001; Eisenberg, Valiente, & Sulik, 2009; Field & Diego, 2008). It was reported that children with higher RSA suppression demonstrated more effective emotion regulation strategies (such as attention orientation) during a task evoking negative emotions (Calkins, 1997) and better regulation of aggression (Millers et al., 2013). Children with high RSA suppression also tend to have high ratings of sociability and low ratings of shyness (Doussard-Roosevelt, Montgomery, & Porges, 2003).

In addition, low externalizing (El-Sheikh, Harger, & Whitson, 2001) and internalizing behaviors (Hinnant & El-Sheikh, 2009) have been displayed among children with high RSA suppression. Gentzler and colleagues (2009) found that greater RSA suppression was predictive of lower levels of clinician-rated depressive symptoms among children at risk for mood disorders. This positive linkage was also documented in an infant sample using the Still Face procedure such that higher RSA withdrawal from

neutral task to Still Face followed by quick recovery was related to more regulation activities (Bazhenova, Plonskaia, & Porges, 2001). Infants' higher RSA withdrawal also related to more experimenter-rated social approach behaviors (Stifter & Corey, 2001).

Conversely, compared to higher RSA suppression, less RSA suppression (sometimes combined with lower baseline RSA; Calkins, Graziano, & Keane, 2007) has been associated with easily displaying frustration during the stressor/challenging laboratory tasks (Calkins, Dedmon, Gill, Lomax, & Johnson, 2002), children's sleep problems assessed by both self-reported and actigraphy measures (El-Sheikh & Buckhalt, 2005), more aggressive/destructive behavior problems (Calkins & Dedmon, 2000), and more internalizing symptoms assessed by Child Behavioral Checklist (Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). Also, a clinical sample with ADHD demonstrated higher RSA augmentation during all novel emotion tasks as compared to normally developed children displaying distinct physiological responses to tasks only with different emotion valence (Musser et al., 2011). Overall, it is thought that RSA (both baseline RSA and RSA reactivity) is a physiological marker underlying child regulatory and attentional ability (Porges, 2007), and studies discussed above provide empirical evidence supporting the argument of examining RSA when investigating childhood regulation-related outcomes.

Of interest, children's externalizing and internalizing behaviors have been conceptually linked to the function of RSA because the presence of these problems is probably partially attributable to children's deficits in expressing and/or regulating emotions (Cole, Michel, & Teti, 1994; Denham et al., 2000; Shipman et al., 2007). Furthermore, given the potential role of RSA in children's responsiveness to external

requirements, it makes sense to take into consideration the interactive effects of RSA and environmental factors in predicting behavioral adaptation. In fact, researchers agree that the PNS cannot function in an isolated manner, and the identified physiological marker is not completely biologically determined (e.g., Beauchaine, 2001). Whether predisposed individuals have certain type of psychopathology is also largely dependent on their socialization exposure. Therefore, review of the associations among family influences, RSA, and childhood externalizing and internalizing problems is presented below.

Interactions of Environment with Physiological RSA

Conceptual models. Several alternative models of plasticity to environmental influences have been proposed to explain the environment and physiological reactivity interactions. The argument of the diathesis-stress model (or dual risk model) is that some individuals, due to a biologically based vulnerability, are disproportionately or even exclusively likely to be affected adversely by environmental stressors such as insensitive parenting or negative life events (see Figure 1a for graphical illustration). The diathesis/vulnerability is believed to occur only in the presence of life stress, and the vulnerability and external stress are both necessary components in the emergence and development of psychopathology (Monroe & Simons, 1991). In addition, the effects of diathesis-stress also depend on the degree/severity of diathesis and stress, which means, both factors should be considered as continuous variables varying in magnitude and loadings, rather than present versus absent categorical classifications. To the extent that diathesis factors combine with the requisite forms of life stress, highly predisposed people (i.e., vulnerable people), even in the general normal population, are more likely to develop psychopathology. Resilient people, on the other hand, display resistance to the

negative influences. Hence, this model highlights the necessary combination of diathesis and stress together in complex interactive ways to initiate and perpetuate psychopathology. Neither alone would function sufficiently.

Contrary to the realization of vulnerability only in the presence of adverse environment (i.e., stress), the argument of the vantage sensitivity model (Pluess & Belsky, 2012) is that some individuals are disproportionately sensitive or positively responsive to the advantageous environmental influences (see Figure 1b for graphic illustration). The terminology "vantage" is short for *advantage*; it implies gain and benefit, and refers to the predisposition, condition, or opportunity that provides individuals with the advantage. "Vantage sensitivity" reflects individuals' tendency to benefit from the positive/supportive or competence-promoting rearing environment (just as "vulnerability" conceptualizing individuals' tendency to have compromised outcomes under the negative environment in the diathesis-stress model). However, the failure of benefiting from the positive influences was labelled as "vantage resistance" (just as "resilience" in the diathesis-stress model). From the perspective of vantage sensitivity, therefore, individual variability emerges only in the supportive environmental exposure.

According to Pluess and Belsky (2012), this individual variability can be implied in the findings of intervention studies such that some generally effective intervention programs benefited some people (with vantage sensitivity) more than others (Kennard et al., 2006). Recent intervention research on genetic markers of child behaviors has also yielded results consistent with the vantage sensitivity model. For example, an intervention study reported the effect of the parenting intervention program in decreasing externalizing behaviors only among children with the DRD4 7-repeat allele (Bakermans-

Kranenburg, van IJzendoorn, Pijlman, Mesman, & Juffer, 2008). It may be the case that these children had high vantage sensitivity that enabled them to benefit from the intervention program (i.e., environmental advantages).

Furthermore, in contrast to the two models above that focus on one end of a continuum of environment quality, differential susceptibility posits that individuals are susceptible to environmental influences in a 'for better and for worse' pattern (see Figure 1c for graphic illustration; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007). According to this perspective, children's greater physiological reactivity may predispose them to demonstrate more behavior problems when exposed to negative environment, and less behavior problems under the condition of positive environment. Boyce and Ellis (2005) indicated that future is full of uncertainty; thus, it might be evolutionarily advantageous for flexible children to behave differently in environments of different quality. For example, it may be an adaptive strategy for children exposed to stressful environment to enact more aggression in order to obtain resources, whereas under supportive environment, those children may behave cooperatively and friendly to receive social support. The differential susceptibility model regards individuals as not just "vulnerable" to adversity or "sensitive" to advantages, but more generally as "susceptible/plastic" to both negative and positive environments. For example, as opposed to children with higher RSA, those with lower RSA were found to be less anxious when experiencing little marital conflict, but more anxious when growing up in high-conflict families (El-Sheikh et al., 2001).

Empirical evidence regarding family × RSA interactions. Starting from the theoretical models, the following review summarizes empirical evidence linking the

interactions of family environment and physiological RSA to children's behavior problems. Research findings in terms of whether higher or lower baseline RSA or RSA suppression confers vulnerability/vantage/plasticity are mixed.

Low baseline RSA/RSA suppression as vulnerability/vantage/plasticity? Some researchers have identified lower baseline RSA and lower RSA suppression as risk factors or vulnerabilities that exacerbate children's behavior problems in the context of high family conflicts (i.e., higher baseline RSA and RSA suppression are considered as protective). For example, as in the aforementioned El-Sheikh et al. (2001) study, when exposed to high levels of marital verbal conflicts, school-aged children with low baseline RSA exhibited the most mother-reported externalizing and child-reported internalizing problems. Also, positive relations between marital physical conflicts and children's general health problems (e.g., digestive, skin, fatigue and illness) were evident for those children with lower baseline RSA. Compared to their counterparts with higher baseline RSA, these children were found to be more reactive to the different contexts such that increased exposure to marital conflict was associated with significant increased levels of the problem behaviors. The same positive associations between martial conflicts and externalizing behaviors in children with lower baseline RSA were documented by Katz and Gottman (1995).

Using parental drinking problems as a negative environmental factor, El-Sheikh (2005a) found that maternal problem drinking was predictive of relatively high levels of externalizing and internalizing behaviors among children with low baseline RSA. In addition, the change scores of mother-reported externalizing behaviors across the two-year gap of the data collection were also higher in children with lower baseline RSA who

were exposed to high levels of parental problem drinking, compared to children with high RSA. Again, the simple slope of parental problem drinking with change scores of externalizing was significant only for children with low baseline RSA.

Also focusing on the paternal and maternal psychopathology, another group of researchers examined the moderating role of baseline RSA in the association between paternal anti-social personality disorder and children's conduct problems, and in the association between maternal melancholic depression and children's depression symptoms (Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). Low baseline RSA predicted high levels of children's conduct problems and depression with increased exposure to paternal and maternal psychopathology, respectively. In contract to the previous studies (e.g., El-Sheikh et al., 2001, El-Sheikh, 2005a), changes in behavior problems as a function of parental psychopathology were significant among children with high baseline RSA, but not low baseline RSA.

The aforementioned studies obtained findings consistent with the diathesis-stress model, however, Eisenberg et al. (2012), using growth curve analysis of aggression across early childhood, found that children with relatively high baseline RSA benefited most from the high family environment quality. That is, when exposed to higher family quality, these children not only demonstrated the lowest aggression level 3 years later, but also the sharpest decrease in their aggression behaviors across the 3 years, which was consistent with the vantage sensitivity model. Regardless, in this study, high baseline RSA was reported to be more reactive to the varying environment (similar to the Shannon et al., 2007 study).

Similar to baseline RSA, researchers have found that lower RSA suppression appears to confer risk for problem behaviors in childhood. In one study, El-Sheikh and Whitson (2006) found a positive association between martial conflict and children's internalizing behaviors 2 years later, but only for the children with lower RSA suppression. In another study, relative to those with higher RSA suppression, children with higher RSA augmentation (i.e., lower RSA suppression) during peer provocation procedure displayed more conduct problems when they were from families with higher family domestic violence (Katz, 2007). However, when exposed to a relatively low level of family domestic violence, the difference in their externalizing behaviors between these two groups was not significant. Also focusing on the peer interaction setting, Leary and Katz (2004) found that for preschoolers with relatively low RSA suppression, hostile-withdrawn co-parenting was positively associated with their high levels of conflict with peers. These studies consistently suggest that children with low RSA suppression are more reactive, such that the simple slopes of the interactions between a familial factor and RSA suppression were significant for the lower RSA suppression group.

Examining children's emotion regulation behaviors, Perry and colleagues (2012) reported a moderating effect of RSA suppression in the association between maternal nonsupportive reactions to children's negative expressivity and children's emotion regulation (e.g., the ability to modulate emotional arousal). Their findings indicated that maternal nonsupportive reactions predicted children's lower regulation ability only among children with lower RSA suppression.

Moreover, when combining the baseline RSA and RSA suppression together, El-Sheikh and colleagues (2011) predicted children's delinquent problems across 8, 9 and 10

years of age. The results replicated the above studies such that lower baseline RSA combined with higher RSA augmentation (i.e., lower RSA suppression), when in combination with higher family martial conflict, predicted the greatest increase in children's delinquency. In contrast, children with relatively high baseline RSA and high RSA augmentation did not show significant changes in delinquency as a function of levels of marital conflict.

High baseline RSA/RSA suppression as vulnerability/plasticity/vantage? On the other hand, however, some researchers have found evidence that high levels of baseline RSA and/or RSA suppression represent vulnerable factors. Specifically, 2 to 4-year old children with relatively high RSA suppression were reported to have the high social wariness when they were at preschool if their mothers had high protective overcontrolling parenting (Hastings et al., 2008). Another study conducted by this research team found that when mothers demonstrated more neglectful reactions to children's negative expressions (e.g., mothers ignored children's expression of sadness), lower and higher baseline RSA groups did not differ significantly in terms of their internalizing problems. However, children with lower baseline RSA exhibited significantly less internalizing if mothers' neglect of negative expressivity was low (Hastings & De, 2008). This pattern was somewhat consistent with the vantage sensitivity model such that the difference was significant only at the side of less negative parenting. However, we cannot say that children with lower RSA benefited most as we would do for the vantage sensitivity model because this side of the parenting behaviors might only indicate less detrimental, but not necessarily more positive/supportive, parenting.

Interpretations. From the studies discussed above, discrepancies exist in this area of exploring RSA interacting with familial environment to predict children's problem behaviors. Actually, in order to interpret the inconsistent findings, investigators have presented mainly two kinds of viewpoints to explain each of the issues of plasticity and reactivity.

First, the interpretations and explanations for the buffering role of high baseline RSA and RSA suppression rely on the arguments proposed by Porges (1996, 2007). As aforementioned briefly, Porges highlighted that baseline RSA may underlie individuals' readiness to respond to stress and challenges, and RSA suppression indicates the ability to slow physical arousal when the environmental demands are absent. Hence, the ability to suppress vagal is considered an index of children's self-regulation ability, and high baseline RSA represents the trait-like characteristic that may allow this ability to be exhibited. Theoretically, individuals with greater RSA suppression are more likely to demonstrate adaptive responses to the external stress by disengaging PNS with a short latency, followed by a quick recovering from the stress response once it is done. Also, RSA suppression is nicknamed as the "vagal brake" by Porges (1995), when facing external challenges, this brake can be removed to prepare individuals' coping process for dealing with the stress, especially among individuals with high RSA suppression.

Considering the argument that children with high baseline RSA and high RSA suppression are better self-regulated (Porges, 2007), these children are probably more capable of adjusting their behaviors and responses to the requirements of the changing environment than children with low baseline RSA or low RSA suppression. Consequently, higher baseline RSA and greater RSA suppression are proposed to be

associated with more flexibility/reactivity of children to the changing types of environments such that RSA functioning could buffer the detrimental effects of maternal depression in the development of child internalizing (Shannon et al., 2007) or enable children to benefit more from relatively high family quality in terms of decreased externalizing (Eisenberg et al., 2012).

In contrast, defenders of findings about the vulnerability of higher levels of baseline RSA and RSA suppression criticized that they may not be adaptive in all the situations (Hastings et al., 2008). These researchers argued that whether RSA reactivity is adaptive or maladaptive depends on the nature of the context. Specifically, it may be the case that children's high baseline RSA or RSA suppression is associated with better adjustment only in the presence of potential threats and harm. Under relatively safe environment, better self-regulation is probably not reflected in high RSA suppression, but contrarily, in the maintenance of low RSA suppression. In other words, if the stimulus were perceived to be safe or nonthreatening, vagal argumentation (i.e., low RSA suppression) would be adaptive in terms of supporting individuals' calm interactions with the environment (Hastings & Miller, 2014). From this viewpoint, Hastings et al. (2008) findings might be interpreted as that compared to the families with negative features (such as martial conflict, domestic violence or drinking parents), parental overprotectiveness may act as less harmful environment with respect to children's behavioral development. Hence, children's chronic RSA suppression as a reaction to overprotective parenting (which is relatively safe in nature) might also be a sign of regulation dysfunction because it implies an unnecessary mobilization of defensive systems toward a relatively safe environment.

When it comes to the flexibility/ reactivity, again Hastings and colleagues (2008) argue that higher RSA or high RSA suppression is related to children's better self-regulation within the undesirable environment; however, it also means that these children are expected to be relatively independent from the external regulatory resources provided by their socializers because they probably already have their own internal resources to which they may turn. In contrast, children who are supposed to lack this regulatory ability (i.e., those with lower baseline RSA and/or RSA suppression) have to rely more on their parents' socialization such that under adverse familial influences, these children demonstrate more problem behaviors, whereas they may also benefit more from positive influences (maybe consistent with differential susceptibility model). Therefore, lower RSA (baseline and suppression) should represent children's flexibility/ reactivity.

Despite the inconsistency, these intriguing arguments further call for the replications of studies in this area. More importantly, a large majority of past research considered only *negative* family environments and parenting practices, such that in their analyses, different levels of the environmental factors are in nature just less negative and highly negative. It is possible that a key piece of information about the influence of positive environment is missing. Appropriate maternal warmth, support, and lower intrusiveness may be particularly critical for the developmental period of early childhood (Early et al., 2002) because these parental behaviors are believed to contribute to children's security feelings, which could, in turn, promote children's competence and decrease the problem behaviors. This consideration, therefore, is also an important reason that current study involved a primarily positive parenting index (see the Measures session) as the external environmental factor.

Rationale for the current hypotheses regarding parenting × RSA interactions. As already mentioned, Porges (1996, 2007) associates higher baseline RSA with children's better regulation and lower baseline RSA with compromised regulatory ability. Baseline RSA has also been considered as a trait-like index that, to some extent, may reflect children's temperamental characteristics (Blandon, Calkins, Keane, & O'Brien, 2010). Based on the findings of most research discussed previously, it might be the case that higher baseline RSA functions as a buffer (or lower baseline RSA represent vulnerability) to children's problem behaviors in the context of environmental adversity (i.e., diathesis-stress model; although vantage sensitivity model has also been reported; Eisenberg et al., 2012). Specifically, children with lower baseline RSA may demonstrate more problem behaviors when exposed to relatively adverse environment, probably because their physiological functioning lacks the ability to promote the independent regulation of behaviors. However, the difference between these two groups might not be so pronounced when environmental quality is at average or highly positive level, both of which may be beneficial to all children with regard to reducing their problem behaviors (Van Zeijl et al., 2006).

A similar rationale may apply to the prediction of children's externalizing behaviors from the interactions between parenting and RSA suppression. Porges (1996, 2007), as well as the reviewed literature, all suggest a relation between sufficient RSA suppression and lower levels of externalizing symptoms. Again, when the environmental quality is disadvantageous, it makes sense that relative to the lower RSA suppression, children's higher RSA suppression (i.e., better self-regulation as proposed) may be especially important in diminishing their externalizing behaviors. That is, the significant

difference between children with higher (i.e., exhibiting fewer externalizing behaviors) versus lower (i.e., exhibiting more externalizing) RSA suppression may exist in the lower end of parenting quality.

On the other hand, the development of internalizing symptoms may be consistent with the idea of compromised outcomes for high RSA suppression group, but only when the parenting quality is high (i.e., vantage sensitivity for children with lower RSA suppression; Hastings et al., 2008). In fact, the two arguments about whether higher or lower RSA suppression represents plasticity may not be mutually exclusive. Rather, they may explain the differential mechanisms underlying different problem behaviors. Specifically, as aforementioned, chronic RSA suppression under nonthreatening environment has been identified as maladjusted because it may be an index of children's over-vigilant responses (e.g., Hastings & Miller, 2014). In other words, these children mobilize their coping resources to deal with the relatively safe stimuli when there is no need to do so.

This unselected vigilant engagement with one's environment has been reported as contributing to internalizing symptoms. For example, using cognitive challenging tasks (e.g., mental arithmetic), investigators found that for adult participants with anxiety, a reduction in cognitive stress was associated with increases in RSA suppression. However, this relation was not significant for participants with lower anxiety (Crowley et al., 2011). Therefore, individuals with internalizing symptoms (e.g., anxiety) may be more likely to respond with coping behaviors (indexed by the physiological changes of RSA suppression) to even less stressful external challenges. It also indicates that when internalizing symptoms are considered, the differences between children with higher (i.e.,

displaying more internalizing behaviors) or lower (i.e., displaying fewer internalizing behaviors) RSA suppression might occur in the context of high positive parenting (i.e., vantage sensitivity model).

Sex as a potential moderator. Besides the potential influence of environment × physiological/biological interactions in the psychopathology development, it is also plausible that child sex functions as a moderator in these interactions (Beauchaine et al., 2001, 2009). That is, how the effects of interactions between parenting quality and baseline RSA or RSA reactivity vary between boys and girls. Actually, previous research with older children (e.g., 8 to 12 years old) has shown a negative relation between baseline RSA and conduct problems only for boys, but not girls (Beauchaine, Hong, & Marsh, 2008; Beauchaine et al., 2008). Similarly, RSA suppression was negatively associated with kindergarten boys' externalizing behaviors as reported by teacher, whereas this prediction was not significant for girls (Graziano, Keane, & Calkins, 2007). However, evidence supporting the moderating role of sex in the influence of environment × physiological RSA interaction on children's psychopathology is still very scant (El-Sheikh, 2005b). The sex-moderated effect advocated by Beauchaine (2001) is apparently understudied, especially with children at early childhood. One exception, however, is that Eisenberg, et al., (2012) found that environmental quality interacted with children's baseline RSA to predict only father-reported aggression behaviors among girls, but not boys, at 18 months. Another study with school-aged children reported a significant three-way interaction among maternal depression, baseline RSA, and sex in predicting children's internalizing symptoms 2 years later. They found that when exposed to high level of maternal depression, girls with low baseline RSA and, in contrast, boys with high

baseline RSA, displayed the greatest levels of internalizing symptoms (Wetter & El-Sheikh, 2012).

In addition, the other component of the human autonomic nervous system (ANS), the sympathetic nervous system (SNS; measured by skin conductance level reactivity; SCLR), has been reported to interact with the environmental influence on children's problem behaviors, and this process is moderated by sex (Erath, El-Sheikh, & Cummings, 2009). For instance, using marital conflict as a predictor, El-Sheikh and colleagues (2007) found a stronger negative association between martial conflict and increased externalizing and internalizing symptoms among girls with higher SCLR; for boys, however, martial conflict positively predicted externalizing behaviors only among boys with lower SCLR. It is suggested that human ANS (both PNS and SNS) in general may demonstrate a sex-moderated effect that confers differential vulnerability or plasticity to boys versus girls (Obradović, Bush, & Boyce, 2011) and the extant research somewhat supports this argument. Nevertheless, it should be noted that most of the aforementioned sex-moderated findings (either on PNS or SNS) were found with school-aged children, evidence about early childhood is far from clear due to the paucity of studies with children at this age period, especially exploring their internalizing symptoms. Therefore, child sex was included as a moderator (and a covariate; discussed below) in the current study.

Differences between Externalizing and Internalizing

Although not all researchers have simultaneously explored child externalizing and internalizing behaviors, some did and found different patterns for externalizing versus internalizing. For instance, in a previously discussed study, Hastings and De (2008)

detected a different pattern in terms of children's externalizing relative to internalizing: for children with low baseline RSA (who were more reactive as a function of parenting), maternal neglect to children's negative emotion expression (such as sadness and fear) was negatively related to externalizing problems, but positively related to internalizing problems.

Indeed, internalizing behaviors, compared to externalizing problems, may have a distinct mechanism in regard to the interactions of environmental factors with RSA. Specifically, by classifying children into three groups (i.e., low in both behavior problems, pure externalizing problems and mixed externalizing/internalizing problems), Calkins et al. (2007) reported that children with mixed externalizing and internalizing demonstrated the greatest RSA suppression (which was contrary to the findings aforementioned), whereas children with pure externalizing demonstrated lowest RSA suppression, with the group low in both symptoms in the middle. One of the plausible explanations is that externalizing may be attributable to behavioral undercontrol, which is a reflection of lower RSA suppression, while internalizing may be due to extreme RSA suppression to the extent of over-vigilance and over-reactivity (Calkins et al., 2007; Thayer & Lane, 2000). Other researchers have obtained preliminary evidence that externalizing versus internalizing might be associated with distinctive patterns of autonomic activity (Boyce et al., 2001). For instance, Hinnant and El-Sheikh (2009) found that low baseline RSA combined with high RSA suppression was predictive of children's internalizing symptoms, while low baseline RSA in conjunction with high RSA augmentation was linked to externalizing.

Nevertheless, the overall pattern of linkages between RSA and different childhood problem behaviors is still unclear. This argument is true especially considering that some of the investigations discussed above detected positive associations between high baseline RSA and/or RSA suppression with low levels of both externalizing and internalizing problems. However, the differential developmental trajectories of, and the factors related to, externalizing versus internalizing that are discussed in the following literature review might imply the importance of exploring externalizing and internalizing in one study to compare their potential difference in relations with children's RSA.

Differences in the developmental trajectory. As discussed briefly at the beginning of the introduction, externalizing and internalizing behaviors demonstrate different trajectories through the period of childhood. Initial efforts of investigators were dedicated to tracing the average (i.e., normative) trajectories of these two domains across early childhood using longitudinal data (Achenbach, Howell, Quay, & Conners, 1991; Tremblay. 2000). These studies indicate changes in frequency and magnitude in both externalizing and internalizing problem through this period; however, the directions are somewhat opposite. Externalizing has been found to stably decline after a peak at approximately 30 months (Eisenberg et al., 2012), whereas internalizing was reported to generally increase across early childhood (Gilliom & Shaw, 2004; stable decreasing pattern was also found; Sterba et al., 2007). In fact, researchers pointed out that the development of these two behavior problems become differentiated at approximately 18 to 30 months (Mathiesen & Sanson, 2000).

Differences in the associated factors. Moving beyond the descriptive levels of the differences in developmental trajectories, most early work from developmentalists has

focused on the factors that are uniquely associated with these two types of symptoms. Just as for normal development, psychopathological development is considered as a complex, dynamic, and interactive function between organisms' characteristics and features of their environment.

Of course, several common environmental influences have been identified as affecting both behaviors, including lower family SES (Keiley et al., 2000), prenatal exposure to maternal smoking and drinking (Ashford, Van Lier, Timmermans, Cuijpers, & Koot, 2008), family stress (Kim, Conger, Elder, & Lorenz, 2003), and disorganized neighborhood environments (Hinshaw, 2002). Unique combinations among different familial and social factors, however, may differentially contribute to externalizing versus internalizing development.

In fact, the interpretations of the aforementioned differential developmental patterns are related to the maturation of children's cognitive functioning and the increasing involvement of parental socialization process. For example, the development of children's cognitive/verbal competence after 2 years old promotes their dyadic problem-solving skills with parents, which in turn, may predict the decrease in externalizing problems. By the same token, parental involvement, such as limit setting and authoritative control, is also considered as a potential predictor of the declining externalizing trajectory (Tremblay, 2000). Similarly, a series of studies from Barber and colleagues on parental behavioral control (e.g., monitoring and limit setting) also highlighted that high level of parental behavioral control was consistently predictive of lower rate of increase in externalizing (Barber, Stolz, Olsen, Collins, & Burchinal, 2005), but not internalizing.

In contrast, for internalizing behaviors, a meta-analysis on parental psychopathology and children's problem behaviors suggested that there were stronger relations between maternal than paternal psychopathology with children's internalizing, but this association was not present for externalizing (Connell & Goodman, 2002). Also, compared to externalizing, children are more likely to show a relatively high level of internalizing symptoms if they are exposed to parental psychological control (i.e., similar to authoritarian control whereby parents use coercive and intrusive controlling to control child's behaviors that undermines the development of psychological competence and self-direction; Barber, Olsen, & Shagle, 1994). The prediction of high levels of children's internalizing problems by parental psychological control/ intrusiveness has been further identified as a transactional process, such that children's higher internalizing behaviors were, in turn, associated with an increased level of child-perceived parental psychological control 2 years later (Albrecht, Galambos & Jansson, 2007). In fact, parenting practices characterized as detrimental to the development of self-process (e.g., self-reliance and self-identify) are believed to be more associated with children's internalizing than externalizing problems (Barber & Harmon, 2002), perhaps especially in Western cultures. Therefore, it appears that parental behavioral control is more effective in decreasing externalizing behaviors, whereas the relations of parental psychopathology and psychological control/ intrusiveness to internalizing behaviors are stronger.

In addition to examining the environmental/parenting factors associated with the general trend in the changes of externalizing and internalizing behaviors among normative children, researchers are also interested in the exploration of potential differential patterns of these trajectories of children from different backgrounds. Contrary

to the normative declining trajectory, children from families with extreme financial problems and conflicts demonstrated an increase or slower rate of decrease in their externalizing over time. For instance, Dearing et al. (2006) reported a positive association between chronic family financial deficits and the increase in externalizing among children across 4.5 years. Examining families with parental alcoholism, Loukas and colleagues (2003) found high disruptive behaviors among children at the school-entry age, and the detrimental effect of parental alcoholism in boys' externalizing was gradually stronger over time. Internalizing problems have been found to display an early onset and greater rate of increase among children from families with high marital conflict relative to children whose parents had less conflict (Kovacs & Devlin, 1998).

Sex differences in the behavior problems. During early childhood, sex differences have been found in a considerable amount of studies on externalizing, whereas there may not be as much evidence of the sex differences in internalizing problems. Specifically for externalizing, boys are reported to be more impulsive and have a greater tendency for anti-social behaviors (Bonger, Koot, Van Der Ende, & Verhulst, 2004). Else-Quest and colleagues' (2006) meta-analysis indicated that boys demonstrated significantly lower effortful control and higher surgency than girls did across toddlerhood to early adolescence. They argued that this pattern may imply the greater incidence of externalizing behaviors among boys. In fact, externalizing behaviors, such as aggression, have been documented to show sex differences as early as approximately 2 years of age (Alink et al., 2006). Another meta-analysis by Archer (2004) found consistent sex differences in aggression (with boys being higher), which were generally moderate in magnitude (e.g., $d = 0.55$) during childhood.

In addition, sex differences appear in the stability and changes of externalizing behaviors through childhood. Compared to the symptoms of girls, boys' externalizing was found to be more enduring and stable (Cai, 2004). In terms of the direction of changes of externalizing, Hammarberg and Hagekull (2006) reported that preschool girls were more likely to have decreased externalizing behaviors than preschool boys were approximately 8 months later. Results from one longitudinal latent profile analysis on externalizing behaviors (Hill, Degnan, Calkins, & Keane, 2006) suggested that relative to their counterparts, more girls were classified into the group labelled as modest-decreasing or normative profile (i.e., displayed modest levels of externalizing at age 2, and then lower levels of externalizing at age 4). However, more boys were classified into the group labelled as high-decreasing or subthreshold profile (i.e., demonstrated more initial externalizing behaviors at age 2, but had less externalizing at age 4). Similar findings were reported in German children from 11 to 15 years old (Castelao & Kröner-herwig, 2014). In addition, boys and girls have been found to be different in regard to the slopes of the developmental trajectories of externalizing such that the rate of decrease in externalizing for boys was significantly greater than for girls (Bonger et al., 2003). This difference might be partially due to the differential initial values for boys versus girls such that boys scored significantly higher than girls did in terms of their initial externalizing behaviors.

Finally, individual variability (i.e., variance) of the externalizing problems may be different for boys and girls as well. This point has been acknowledged in a recent review on sex differences in child development (Hyde, 2013). Statistically, the evaluation of the individual variability in the previous developmental works was a variance ratio (e.g., the

ratio of variance for boys divided by the variance of girls; Hyde, Lindberg, Linn, Ellis, & Williams, 2008; Lindberg, Hyde, Peterson, & Linn, 2010). If the result is larger than 1, it means that greater variability exists in boys; otherwise, if the ratio is less than 1, girls exhibit greater variability. From this approach, the aforementioned study by Else-Quest et al. (2006) found that the variability of inhibitory control of boys was greater than girls, whereas the variability of temperamental fear was greater among girls than boys. Although not much work has been done to compare the different magnitude of individual variability in externalizing behaviors among boys versus girls, it is interesting to integrate this investigation especially into growth curve modeling (although better statistical contrast indices are needed). For instance, by comparing the variance of the random intercepts and slopes of the growth curves of boys and girls, researchers can further get a better understanding of the individual variability in terms of the initial levels as well as rate of changes of the externalizing problems among boys versus girls. Nevertheless, because this approach is not the focus of the current study, it is not discussed in more detail.

Although there is substantial evidence of sex differences in externalizing behaviors, it is also important to note that the associations between sex and externalizing depends on some other factors, such as children's age, specific types of externalizing, and context. Specifically, Chaplin and Aldao's (2013) meta-analysis showed that sex differences in externalizing emotions (e.g., anger) became more pronounced as children age such that the gap in externalizing emotions between boys and girls was larger with the increasing of age. As for the different types of externalizing, researchers found no significant difference between boys and girls in oppositional and status violations at the

age of 9 years old (Lahey et al., 2000). Differences in relational aggression among preschoolers was also not significant among boys and girls (McEachern & Snyder, 2012), although female teenagers have been demonstrated significantly higher relational aggression than male teenagers (Dane & Marini, 2014). Moreover, sex differences in externalizing behaviors are believed to be stronger in the context of peer interactions in comparison to child-adult interactions (Chaplin & Aldao, 2013).

Contrary to the early-onset sex differences in externalizing problems, boys' and girls' internalizing problems were not found to be different until age 15 to 18 (Hankin, Abramson, Moffitt, Silva, & McGee, 1998). Puberty/early adolescence has been identified as a period during which girls start to experience more internalizing symptoms than boys (Hankin et al., 1998; Muris, Merckelbach, Gadet, & Moulaert, 2000). Twenge and Nolen-Hoeksema (2002) meta-analyzed studies on children's depression and their results indicated that at age 13-16 years old, sex differences in children's depression became significant with girls higher than boys ($d = 0.16$). From age 8 to 11, depression scores of girls were constant, followed by an increase between ages 12 to 16. However, boys' depression stayed stable through 8 to 16 years of age with higher scores at approximately age 12. It appears that sex differences in children's internalizing symptoms emerge around adolescence, and affective, biological, and cognitive factors are proposed to converge to create children's vulnerability to internalizing symptoms (Hyde, Mezulis, & Abramson, 2008).

In summary, externalizing, but not internalizing, behaviors appear to demonstrate consistent and significant sex differences during childhood. Along with the empirical evidence of the differential developmental trajectories of these two types of

psychopathology and the associated factors (discussed above), it implies that the development of externalizing and internalizing does not share the same mechanism, and as Rottenberg (2007) advocated, it is of importance for researchers to explore the role of RSA in the trajectory of externalizing and internalizing development simultaneously in one study in order to shed light on these relations. Due to the fact that children in the current study are in early childhood, it makes sense to control the effect of sex in the analyses (i.e., substantive models) on externalizing behaviors. Moreover, in order to compare externalizing and internalizing behaviors in the same model structure, sex was included as a covariate in the models with internalizing behaviors. As discussed previously, sex was also tested as a moderator in the prediction of parenting × RSA interaction to children's problem behaviors.

THE PRESENT STUDY

Based on the previous discussion, the current study focused on the associations between parenting quality and children's RSA as predictors of the developmental trajectories of childhood externalizing and internalizing symptoms. Sex was included in the current study as an important covariate or moderator.

Using an structural equation modeling (SEM) framework for latent growth curve analyses, this study explored the prediction of the development of child externalizing and internalizing across 24, 30, 42, 48, and 54 months by observed 18-month parenting and children's RSA, as well as their interaction (with sex as a covariate and moderator). The specific hypotheses of this study were:

I. There would be a linear or quadratic trend in the developmental trajectory of early childhood externalizing and internalizing behaviors. Specifically, it was

hypothesized that there would be a negative linear slope (i.e., decreasing linear trend) and/or a negative quadratic slope (i.e., increasing to a peak at approximately 36 months as was shown in our previous work; Eisenberg et al., 2012, and then decreasing after 36 months) across the ages of 24, 30, 42, 48, and 54 months in the developmental trajectory of externalizing problems. For internalizing behaviors, only a negative linear slope (e.g., Sterba et al., 2007) was hypothesized.

II. There would be negative associations between 18-month supportive parenting quality (i.e., higher in warmth, sensitivity, and authoritative control, and lower in intrusiveness) and the 24-month and/or 54-month intercepts of children's problematic behaviors (i.e., externalizing and internalizing). There would be negative associations between 18-month supportive parenting quality and the (linear) slopes of the trajectories of children's problematic behaviors. In other words, a high level of 18-month supportive parenting would be associated with a decreased level of behavior problems and with a more rapid rate of decline in children's behavior problems. For the potential quadratic trend (i.e., negative value) of externalizing symptoms, it was hypothesized to be negatively predicted from parenting quality. That is, higher parenting quality would be related to greater deceleration of the rate of change (i.e., numerically larger negative value) in externalizing.

III. Besides the main effect of parenting quality, there would be a significant interaction between 18-month supportive parenting quality and children's baseline RSA. Based on the aforementioned rationales (i.e., children with low

baseline RSA may lack the ability to independently regulate their behaviors; Hastings et al., 2008), supportive parenting quality was hypothesized to be negatively associated with problems at 24 and/or 54 months (i.e., intercept), rate of changes (i.e., linear slope) and deceleration of rate of changes (i.e., potential quadratic trend of externalizing) in problem behaviors (i.e., both externalizing and internalizing) only among children with low (i.e., -1 *SD*) baseline RSA. This pattern was hypothesized to be consistent with the diathesis-stress model (see Figure 1a for graphical illustration) where low baseline RSA was considered as the "vulnerability" (Shannon et al., 2007). Specifically, when exposed to lower parenting quality, in comparison to children with high (i.e., +1 *SD*) baseline RSA, children with low (i.e., -1 *SD*) baseline RSA would demonstrate more problem behaviors at 24 and/or 54 months. The rate of changes (i.e., hypothesized to be negative slopes) in problem behaviors among children with low (i.e., -1 *SD*) baseline RSA would be smaller (i.e., numerically smaller negative values representing flatter decline) relative to children with high (i.e., +1 *SD*) baseline RSA in the context of lower parenting quality. Also, for the potential quadratic slope of externalizing, children with low (i.e., -1 *SD*) baseline RSA and exposed to lower parenting quality would demonstrate smaller deceleration in the rate of change (i.e., numerically smaller negative values) as compared to their counterparts with high (i.e., +1 *SD*) baseline RSA.

IV. There would also be a significant interaction between 18-month supportive parenting quality and children's RSA reactivity. However, the nature of the

interaction might be different for externalizing and internalizing behaviors (as discussed above). Similar to low baseline RSA, negative relations of parenting quality with children's 24- and/or 54-month, and linear rate of changes in externalizing and internalizing, and potential quadratic slope of externalizing were hypothesized to be significant only among children with high (i.e., +1 *SD*) RSA reactivity, indicating greater RSA augmentation (Hastings et al., 2008). For externalizing behaviors, the pattern might be consistent with the diathesis-stress model, such that when exposed to lower parenting quality, in comparison to children with low RSA reactivity scores (i.e., -1 *SD* from the average of the residualized change scores), children with high RSA reactivity scores (i.e., +1 *SD* from the average of the residualized change scores) would demonstrate more externalizing behaviors at 24 and/or 54 months. Also, the expected declining linear slope and quadratic slope of externalizing for children with high RSA reactivity scores (i.e., +1 *SD* from the average of the residualized change scores) would be smaller (i.e., numerically smaller negative values) than for children with low RSA reactivity scores (i.e., -1 *SD* from the average of the residualized change scores) in the context of lower parenting quality. Therefore, it was hypothesized that the high RSA reactivity scores (i.e., interpreted as high RSA augmentation) represented the "vulnerability" conceptualized in the diathesis-stress model.

V. However, considering the proposed relations between internalizing and over-vigilant responses in safe environment (Beauchaine, 2001; Hastings & Miller, 2014), it was hypothesized that the differences between children with high RSA

reactivity and low RSA reactivity scores in terms of their internalizing behaviors would be detected at the side of higher parenting quality. Specifically, children who were high in RSA reactivity scores (i.e., +1 *SD* from the average of the residualized change scores) and were exposed to higher parenting quality would exhibit fewer internalizing problems at 24 and/or 54 months. In addition, the slope of internalizing among children with high RSA reactivity scores (i.e., +1 *SD* from the average of the residualized change scores) was hypothesized to be larger (i.e., numerically larger negative values representing steeper decline) relative to children with low RSA reactivity scores (i.e., -1 *SD* from the average of the residualized change scores) as response to higher parenting quality. This pattern was hypothesized to be consistent with the vantage sensitivity model (see Figure 1b for graphical illustration) where high RSA reactivity scores (i.e., +1 *SD* from the average of the residualized change scores) reflected the "vantage sensitivity" to benefit from the higher positive parenting.

VI. Sex was hypothesized to be a significant covariate in the models. Specifically, controlling for other covariates, boys would be higher in the average level of externalizing at 24 and/or 54 months (i.e., mean of the intercepts). There would be no significant differences between boys and girls in their average levels of internalizing behaviors at 24 and/or 54 months. In addition, it was hypothesized that the rate of changes on average (i.e., mean of slope) in externalizing would be higher for boys, whereas the rate of change in internalizing would be higher for girls.

VII. Furthermore, based on the argument of sex-moderated manifestations (i.e., potential moderating role of sex; Beauchaine et al., 2008; Beauchaine et al., 2009) in environment × physiology/biology interactions, sex was also examined as a moderator. Hypothesized based on the limited previous findings, the intercept and slope of externalizing behaviors were predicted to be negatively associated with baseline RSA among boys, but not girls (Beauchaine et al., 2008; El-Sheikh, 2005). It was also hypothesized that three-way interactions among parenting quality × baseline RSA × sex in predicting the externalizing intercept and slope might be significant. A significant three-way interaction in predicting externalizing might demonstrate that parenting quality × baseline RSA interaction would only be significant for girls, but not boys (Eisenberg, et al., 2012). Due to the scarcity of sex-moderated studies with RSA reactivity (the hypotheses were more exploratory), it was hypothesized merely that there might be a significant three-way interaction (among parenting quality × RSA reactivity × sex) when predicting the intercept and slope of externalizing symptoms. Similarly for internalizing, three-way interactions among parenting quality × baseline RSA × sex might be significant such that parenting quality interacted with baseline RSA to predict the intercept and slope of internalizing symptoms of girls but not boys (e.g., Wetter & El-Sheikh, 2012). Again, (the hypotheses were more exploratory), there was hypothesized to be significant prediction from the three-way interaction among the parenting quality × RSA reactivity × sex to the intercept and slope of the internalizing symptoms.

Innovations of the current study include the following. First, in contrast to the typical prior research focusing mainly on the broad family environment (i.e., SES, marital status, and neighborhood environment) and negative parenting, this study explored primarily positive parenting quality interacting with child RSA as predictors of the development of child problem behaviors. Second, sex was included as a covariate and moderator. Thus, the study provides more insight into the potential effect of sex in predicting psychopathological symptoms from the parenting × biology/physiology interactions. It might be the case that interactions between environmental influence and physiological factors are sex-moderated processes or manifestations (e.g., Lyons-Ruth, 2008) and an increasing amount of sex-moderated psychophysiological research has emerged in terms of childhood externalizing (e.g., Beauchaine et al., 2009) and internalizing symptoms (e.g., Wetter & El-Sheikh, 2012). Third, using mother- and father-reported child behavior problems are useful in reducing reporter bias. Otherwise, reliance on the same person (in the current study, mothers) for both parenting quality measure and children's problem behaviors measures may create spurious associations between them. Potential different findings about mother- versus father-reported problem behaviors could also be examined by modeling them separately. Finally, specifying separate models enabled me to investigate the potential different patterns associated with the intercepts and slopes of the changes of externalizing versus internalizing.

METHODS

Participants

Participants were part of a longitudinal study of young children's socio-emotional development. Families were recruited at birth through three hospitals in a large city. All

infants were healthy, full-term and from adult parents in English-speaking families. At 18 (N = 243), 30 (N = 223), 42 (N = 205), and 54 (N = 189) months of age, children and their mothers visited laboratory for an assessment lasing about 1.5 – 2 hours in addition to the questionnaire measures. At 24 (N = 225), 36 (N = 220) and 48 months (N = 194) of age, participants received a series of questionnaires mailed to their families. The questionnaires were answered by mothers, fathers, and caregivers separately.

In this study, parenting practices were coded from observations of mother-child free-play and puzzle tasks at 18 months. Child physiological measures (i.e., baseline RSA and reactivity) were collected at 18 months. In addition, at 24, 30, 42, 48, and 54 months, mother- and father-reported externalizing and internalizing symptoms were obtained. The current subsample included families that had complete physiological data at 18 months (N = 217; 120 boys and 97 girls; mean age at 18 months = 17.78, SD = .49). There were no significant differences between boys and girls in the variables of interest, except that mother-rated externalizing symptoms at 42 months were slightly higher among girls than boys; $t_{(167)} = 1.84$, $p = .07$.

Demographic information about this subsample was reported as following. Racial composition was 84.3% Caucasian, 5.5% African-American, 1.8% Asian, 5.1% Native American, 1.4% mix of two minority races, and 1.9% unknown races. In terms of the ethnicity, 22.6% of the sample were Hispanic/Latino ethnicity. Annual family income was measured on a seven-point scale, and the median annual family income at 18 months was between $45,000 and $60,000 ($M$ = 4.06, SD = 1.76). Specifically, 5.6% had less than $15,000 annual income, 14.7% had $15,000 to $30,000 annual income, 17.1% had $30,000 to $45,000 annual income, 18.9% had $45,000 to $60,000 annual income, 12.4%

had $60,000 to $75,000 annual income, 10.6% had $75,000 to $100,000 annual income and 11.1% had over $100,000 annual income, 10.1% had missing income data. Thus, this subsample contained families varying from fairly poor to middle-class level to upper-class level, yet the middle-class families were the primary participants. In terms of the highest parental education, there were 5.5% and 5.1% missing data on mother's education and father's education at 18 months, respectively. Rated on a seven-point scale (M = 4.30 and 4.20, SD = 1.09 and 1.22 for mother and father, respectively), 5.5% of mothers and 8.7% of fathers did not finish high school, 13.8% of mothers and 15.7% of fathers graduated high school, 31.8% of mothers and 33.2% of fathers had some college education, 34.1% of mothers and 24.4% of fathers graduated college, 10.2% of mothers and 12.9% of fathers had a master or Ph.D/JD/MD. Overall, mothers and fathers in this subsample were moderately well educated (i.e., at least had some college education).

Attrition

Attrition analyses were first conducted to explore whether the parental nonresponses to questionnaires (i.e., wave nonresponse) were associated with 18-month parenting quality, children's physiological variables, and parent-reported behavioral problems. Specifically, the correlations between the numbers of times each parents filled out the questionnaires (out of the five time waves) with parenting quality and children's RSA scores were examined. A positive correlation between number of times fathers completed the questionnaires and 18-month parenting quality was significant; $r = .22$, $p < .01$. None of the correlations among parental attrition and children's RSA scores was significant. In addition, externalizing and internalizing scores were separately averaged within each reporter across the five time points, and the correlations among these

averaged scores with mother attrition and father attrition were tested. Out of the eight correlations, only number of times fathers completed the questionnaires was significantly negatively correlated with mother-reported externalizing; $r = -.19$, $p < .01$. Summarizing the attrition analyses, they indicate that fathers completed more waves of questionnaires if the observed maternal parenting quality was high and if their children scored low on mother-reported externalizing problems.

Furthermore, I conducted attrition analyses to compare the families (i.e., participating families) who had reported data at both 24 (i.e., beginning of the current study) and 54 months (i.e., end of the current study) to the families who only had reported data at 24 months (i.e., attrited families). In terms of the demographic variables (i.e., child sex, race, mother and father education, and family income), *t*-tests showed no significant difference between the participating families and the attrited families. In terms of the key variables of interest, the only significant finding was that the attrited families had marginally lower observed parenting quality scores; $t_{(152)} = 1.71$, $p = .09$ (Levene's test suggested equal variance between the two groups of families in all the variables).

Measures

Parenting quality. During the laboratory visit of the participants, at 18 months (pertaining to the measures of the current study), a free-play and a challenging teaching task (i.e., puzzle task) were videotaped by the undergraduate assistants (i.e., experimenters conducted the experiments in the laboratory visit), who were extensively trained before the data collection. When conducting the laboratory experiment, the undergraduate assistants were supervised by a graduate student and the project director or a postdoctoral fellow. Later, two trained independent research assistant coders watched

the videos (of the free-play and challenging teaching task) and coded the maternal parenting behaviors (a main coder rated 100% of parent behaviors, and a reliability coder checked at least 25% of the videos for parent behaviors). For the free-play task, the mother and child were instructed to play with toys as they would at home for 3 minutes. During the teaching task, the mother was told to teach her child to finish a clown puzzle in 3 minutes using the strategies she would use at home.

Maternal sensitivity and intrusiveness/over-controlling behavior were rated at 15s intervals during free-play and 30s intervals during the teaching task (from 1 = no evidence of sensitivity displayed to 4 = high, very aware of the toddler and contingently responsive to his or her interests and affect; Fish, Stifer, & Belsky, 1991; e.g., providing age-appropriate stimulation, acknowledging and responding to child's affect, and pacing behavior/verbalizations according to the child's arousal level; *ICC*s = .86 and .71, respectively). Ratings of intrusiveness or over-controlling behavior ranged from 1 = no over-controlling behavior observed to 4 = mother demonstrates extreme intrusive or over-controlling behaviors (e.g., offering too many toys and over-stimulating the child; *ICC*s = .81 and .71 for free-play and the teaching task).

Maternal warmth and authoritative control were rated at 30-s intervals during the teaching task. For warmth, ratings were from 1 = ignoring the child most of the time or displayed primarily negative affect to 5 = engaging physically affectionate with child and exhibiting smiles and laughter with high frequency; e.g., displaying closeness, friendliness, encouragement, positive quality of conversation, *ICC* = .66). Ratings for authoritative control, which concerned maternal clear monitoring of child's progress (i.e., keeping children's behaviors focusing on task) with gentle and playful control, ranged

from 1 = none to 4 = strong authoritative control (e.g., using effective teaching strategies; *ICC* = .86).

All the observed parenting task scores were distributed normally except that maternal intrusiveness during the teaching task had moderate positive skewness (i.e., skewness = 2.21). This score was first transformed by taking the square root of the original values (i.e., best transformation of variable with distribution not including values ≤ zero and with moderate positive skewness), and after transformation, the skewness = 1.72 and kurtosis = 3.44. The transformed score was used for later analyses. Moreover, correlations among all the observed parenting measures (including the transformed intrusiveness score) were in the expected direction and significant (see Table 1; except that the correlation between free-play intrusiveness and teaching task warmth was marginally significant; $r = -.11$, $p < .10$). Therefore, I created a composite score of parenting quality at 18 months by averaging the standardized scores of maternal sensitivity, warmth, authoritative control, and reversed intrusiveness (*ICC* = .80 for the composite score).

Children's RSA. During the 18-month laboratory visit, children's RSA data were collected using a video containing two parts. The first portion (181 seconds) depicted a neutral state with pleasant music as the background of showing neutral to positive baby faces, and was used to measure the baseline RSA, whereas the second portion (42 seconds) featured crying and distressed babies and was used to obtain the data for calculation of RSA suppression (which was considered as a physiological index for children's physiological self-regulation; Porges, 1992). Two heart rate electrodes were attached near the bottom of children's ribs at children's lateral sides, while a third ground

electrode was attached to the children's backs. In addition, a respiration cord was wrapped around children's abdominal areas. In the presence of their parents, children's responses to the video (i.e., electrocardiograph; ECG and respiration) were recorded at 1 *ms* intervals through the electrodes and respiration cord connected to the James Long equipment. Using the peak-to-valley method (James Long Company, 1999), ECG data were analyzed with interbeat interval analysis software.

In order to eliminate the overlap between baseline RSA and RSA reactivity, a residualized change score was computed to index RSA reactivity (Calkins & Keane, 2004). Specifically, we regressed the RSA scores of the distressing portion of the video on the baseline RSA scores, and took the residuals of this regression as the RSA reactivity score that has partialled out the effects of baseline RSA scores. A positive value of this residualized change score indicates RSA augmentation (i.e., an increase from baseline level), whereas negative value indicates RSA suppression (i.e., a decrease from baseline level). In addition, examining box plots of the physiological data, there were three outliers (i.e., ± 3 *SD* away from the mean). The scores of the three outliers were recoded to slightly higher (i.e., .001) than the greatest value of the non-outliers.

Externalizing and internalizing symptoms. Mothers and fathers completed the Infant-Toddler Social and Emotional Assessment (ITSEA; item scoring ranged from 1 = not true to 3 = very true or often true; Carter, Little, Briggs-Gowan, & Kogan, 1999) at 24, 30, 42, 48, and 54 months to assess children's externalizing and internalizing problems. For the 217 children who had the parenting quality and physiological data at 18 months, mother-reported *N*s = 154, 185, 169, 147, and 155 at 24, 30, 42, 48, and 54 months, respectively for both externalizing and internalizing; father-reported *N*s = 118

and 118, 134 and 133, 108 and 109, 95 and 95, 96 and 96 at 24, 30, 42, 48, and 54 months for externalizing and internalizing, respectively.

Externalizing symptoms included subscales (subscale scores were calculated by averaging the item scores) of activity/impulsivity (6 items; αs at 24, 30, 42, 48, and 54 months were .70 and .74, .73 and .66, .71 and .65, .67 and .61, and .73 and .69, for mothers' and fathers' reports, respectively), defiance/aggression (6 items; αs at 24, 30, 42, 48, and 54 months were .78 and .75, .75 and .72, .81 and .75, .78 and .73, and .82 and .78, for mothers' and fathers' reports, respectively), and peer aggression (6 items; αs at 24, 30, 42, 48, and 54 months were .70 and .72, .72 and .68, .78 and .76, .74 and .65, and .78 and .69, for mothers' and fathers' reports, respectively). The αs for assessment of externalizing symptoms including the three aforementioned subscales (total as 18 items) were .82 and .79, .82 and .75, .83 and .81, .80 and .75, and .83 and .78 at 24, 30, 42, 48, and 54 months for mothers' and fathers' reports, respectively. In addition, there were moderate correlations among the subscales within each reporter (rs at different ages are from .31 to .57 for mothers' reports, and from .21 to .49 for fathers' reports; all are significant at $\alpha = .05$). Therefore, externalizing composite scores were computed by averaging scores from the corresponding subscales within each reporter at 24, 30, 42, 48, and 54 months individually.

Internalizing symptoms included subscales (subscale scores were calculated by averaging the item scores) of separation distress (6 items; αs at 24, 30, 42, 48, and 54 months were .57 and .60, .62 and .58, .65 and .52, .67 and .47, and .55 and .36, for mothers' and fathers' reports, respectively), depression/withdrawal (9 items; αs at 24, 30, 42, 48, and 54 months were .51 and .44, .38 and .53, .54 and .61, .54 and .61, and .61

and .55 for mothers' and fathers' reports, respectively), and general anxiety (7 items; αs at 24, 30, 42, 48, and 54 months were .70 and .71, .64 and .65, .70 and .70, .64 and .59, and .59 and .62, for mothers' and fathers' reports, respectively). The αs for internalizing symptoms containing the three measures (total as 22 items) were .63 and .76, .63 and .65, .73 and .76, .75 and .60, and .72 and .67 at 24, 30, 42, 48, and 54 months for mothers' and fathers' reports, respectively. Similar to externalizing, internalizing composite scores were calculated by averaging the corresponding subscales within each reporter at 24, 30, 42, 48, and 54 months.

Overall, the composite scores of mother- and father-reported externalizing and internalizing at the five ages were used in the current study.

RESULTS

Analytic Plan

From the approach of structural equation modeling (SEM), latent growth curve analyses were conducted in M*plus* 7.0 to predict the latent intercept and slope factors of children's externalizing and internalizing symptoms separately within each reporter from parenting quality, physiological RSA, and their interactions. Physiological variables in the current study were not normally distributed (in skewness and kurtosis); however, as the predicators, their nonnormal distributions may not be very problematic (the normality assumption requires normal distribution of the residuals; Poole & O'Farrell, 1971). Instead of transforming the physiological variables, I used the maximum likelihood robust estimation (MLR; i.e., maximum likelihood with robust standard errors for nonnormally distributed data) in both unconditional and conditional models to account for the nonnormality (of the physiological variables). MLR is a full information

maximum likelihood estimator (FIML) which also provides efficient handling of the presence of missing data under the Missing at Random assumption. In all analyses, variables of baseline RSA, residualized change scores (i.e., indicator of RSA reactivity), and observed parenting quality were all mean-centered to reduce non-essential multicollinearity among the interaction terms (Aiken & West, 1991). To explore the levels of internalizing and externalizing problems (i.e., intercepts) at the starting age of the study (i.e., 24 months) and the ending age of the study (i.e., 54 months), intercept factors of the models were centered at 24 months in one set of models and were centered at 54 months in a second set of models.

In addition, the SEM overall model fit indices used to assess the global model fit of the unconditional (Figure 2; discussed below) and conditional models (Figure 3 and 4; discussed below) in the current study were the root mean square error of approximation (*RMSEA*; smaller than .06 to be considered as an acceptable fit to the data), and the standardized root mean square residual (*SRMR*; small than .08 to indicate a good fit). It should be noticed that the comparative fit index (*CFI*), reported by M*plus* 7.0 was not used to determine the global model fit. This is because the incremental fit index estimates the improvement in fit of the specified model over the baseline model (i.e., the worst fitting model), and the baseline model for single-sample measured at a single time-point (i.e., the generic baseline model utilized by M*plus* 7.0 and other SEM software) is not the appropriate baseline model for the latent growth curve analysis (Widaman & Thompson, 2003). Specifically, the standard generic baseline model freely estimates the variances and means of the observed variables, but constrains the covariances to be zero (with the exception of among exogenous observed variables which M*plus* 7.0 allows to freely

covary); however, this baseline model is not nested within the most restricted substantive latent growth curve model. Pertaining to the current study, take the unconditional model of father-reported externalizing symptoms as an example, the most restricted substantive model contained 10 parameter estimates (i.e., mean of the intercept and linear slope, variance of the intercept and linear slope, covariance between the intercept and linear slope, 5 residual variances of the observed variables); however, the generic baseline model also had 10 parameter estimates (i.e., 5 variances and 5 means of the observed variables). Hence, this generic baseline model cannot be the appropriate baseline model because it is not nested within the most restricted substantive model. Rather, in this case, the intercept-only model may serve as the appropriate baseline model in which only mean and variance of the intercept factor were specified and five residual variances (of the observed variables) were freely estimated. Therefore, the baseline model (i.e., intercept-only model) would have 7 parameter estimates, which enables it to be nested within the most restrictive substantive model above (see Widaman & Thompson, 2003 for more details). This intercept-only model allows for the individual differences in the mean level, but does not allow for the growth over time. To summarize, based on the explanation above, using the inappropriate baseline model, the *CFI*s automatically calculated by M*plus* 7.0 are not the correct ones. However, I still reported the inaccurate *CFI*s (in parentheses) in the following models to provide an approximate reference of the values of *CFI*s.

In the series of conditional models predicting the growth factors of externalizing and internalizing, *main effect models* including sex (a dummy variable; 0 = boys, 1 = girls), 18-month parenting quality, and children's baseline RSA or RSA reactivity (but not

interactions) were first tested. Then, I examined the *two-way interaction models* (Figure 3) only specifying interactions between parenting quality and children's baseline RSA or RSA reactivity with sex included as a covariate. However, there was no significant effect of sex in any of the two-way interaction models. One possible reason is that sex interacted with other predictors to exert its influence. Therefore, *three-way interaction models* (Figure 4) with interactions among sex, parenting quality, and baseline RSA or RSA reactivity were further conducted to investigate the potential moderating role of sex. By doing this, the effect of sex could be better explored in the three-way interaction models; hence, all the two-way models (with only the interactions between parenting quality and baseline RSA or RSA reactivity) were rerun and reported with sex eliminated to simplify the two-way model specification.

For any significant three-way interactions, a multiple-group model was estimated in which sex was specified as the grouping variable to investigate whether the parenting quality × baseline RSA/RSA reactivity exhibited a different (significant) relation for boys versus girls. If the interactions (between parenting quality and physiological RSA variables) were significant in boys' and/or girls' models, simple effects (testing both directions) and the corresponding *RoS* were further tested and reported. No follow-up analyses were computed if the parenting × baseline RSA/RSA reactivity interaction was not significant.

In addition, because sex was coded as a dummy variable (0 = boys, 1=girls; i.e., it was not mean-centered), lower-level effects (i.e., effects of parenting quality, baseline RSA/RSA reactivity, and the parenting × baseline RSA/RSA reactivity interaction) in the three-way interaction models represented the relation for boys with average levels of the

other predictors. For example, the effect of parenting quality in the three-way interaction models represented the effect for boys with average baseline RSA/RSA reactivity.

To determine the pattern of interaction between environmental influences and child characteristics, Belsky et al. (2007, 2009) suggested the following: to test the differential susceptibility model (see Figure 1c for graphical illustration), the simple slope for putatively susceptible individuals should be significantly different from zero (at varying levels of environment). What is more, the putatively susceptible subgroups should be significantly different from the not-so-susceptible subgroups when exposed to both higher and lower levels of environmental quality, thus demonstrating the *for-better-and-for-worse* pattern. Statistically, the first criterion pertains to the simple effect of environmental influences (i.e., parenting quality in the current study), while the second criterion pertains to the simple effect of child characteristics (i.e., baseline RSA/RSA reactivity in the current study). Moreover, according to Roisman et al. (2012), the differential susceptibility and diathesis-stress accounts share the "*for-worse*" part, that is, (in addition to the significant simple slope), when experiencing negative environment, individuals who are with "risk" factors would demonstrate poorer adjustment than individuals without those "risk" factors (i.e., simple effect of baseline RSA/ RSA reactivity at lower end of positive parenting). Although not normally done in the previous research, recent environment × personal characteristics studies tested the simple effects in both ways (e.g., Belsky & Pluess, 2013; Eisenberg et al., 2012).

Therefore, for the significant interactions in the present investigation, simple effects of the predictors (i.e., parenting quality) were probed at three levels of moderator (i.e., -1 *SD*, average, and +1 *SD* of RSA) to determine whether the first criterion was met.

The regions of significance (*RoS;* discussed below) were also calculated to pinpoint the interactions using Johnson-Neyman technique (John & Neyman, 1936; Preacher, Curran, & Bauer, 2006). In addition, the simple effect and *RoS* of baseline RSA/RSA reactivity (i.e., parenting quality was switched as the moderator) were explored in order to decide whether the second criterion (of determining interaction pattern) was met. I reported up to 2 *SD* below and above the average (i.e., within the observed range) of baseline RSA/RSA reactivity or parenting quality as the boundaries for *RoS* testing (Kochanska, Kim, Barry, & Philibert, 2011; Roisman et al., 2012). That is, baseline RSA/RSA reactivity (or parenting quality) were considered to be unrelated to the outcomes if their relations were not significant at the values of up to 2 *SD* from the average of the parenting quality (or baseline RSA/RSA reactivity).

Testing the Regions of Significance (*RoS*)

The *RoS* testing provides the range of values of the moderator for which the simple effects of a predictor on an outcome variable are significantly different from zero. Generally, there are upper and lower boundaries of the *RoS*, and significant associations between the predictor and outcome exist when the values of moderator are smaller than the lower boundary and/or larger than the upper boundary (but not within the region of low and upper bounds; Preacher et al., 2006).

The reason of testing *RoS* is that the widely used ±1 *SD* from the average as specified levels of the moderator (i.e., continuous variable) in analyzing the simple effect are arbitrarily selected. Rather than examining the simple effect at ±1 *SD* levels, developmental methodologists recommend testing the *RoS* to determine the threshold where significant effects become nonsignificant (applying an $\alpha = .05$ cutoff) and vice

versa (e.g., Dearing & Hamilton, 2006). For example, statistically, assuming that the significant simple effect of parenting quality (on the outcomes) was detected among children with high (i.e., +1 *SD*), but not average or low (i.e., -1 *SD*) levels of baseline RSA, it means that the significant effect become nonsignificant at some value between average and +1 *SD* of the values of baseline RSA. To pinpoint the exact value, researchers need to test different values (usually the value is changed in increments of .01 *SD*) between average and +1 *SD* until the *p*-value of the simple effect of parenting quality equals .05. For instance, if at +.5 *SD* (of the value of baseline RSA), the effect (of parenting quality) is again significant (at alpha level = .05), then researchers should try +.49 *SD* (i.e., closer to average, that is 0 *SD*). If at +.49 *SD*, the effect is still significant, then +.48 *SD*, +.47 *SD* and so on should be tested until the *p*-value = .05, indicating the threshold differentiating the significant and nonsignificant effects. In this case, if this value equals +.25 *SD*, researchers could say that the significant effect of parenting on the outcomes is at the values of baseline RSA ≥ .25 *SD* above the average (i.e., identifying the full range of the moderator in which significant effect of predictor and outcome would be detected). The same calculation method can also be utilized to pinpoint the threshold where a nonsignificant effect becomes significant.

Descriptive Statistics

Descriptive statistics and zero-order correlations of key variables for the whole sample were presented in Table 2. Descriptive statistics and zero-order correlations of boys and girls separately were presented in Table 3 (for externalizing) and Table 4 (for internalizing). For both boys and girls, behavioral problems demonstrated rank-order stability as reflected in the significant positive correlations among different time points

reported by both mothers and fathers. Maternal parenting quality was significantly negatively (with values of *r*s from -.26 to -.50) correlated with girls' externalizing symptoms across most of the time points, except for 24- and 54-month externalizing rated by fathers; *r*s = .05 and -.09, *ns* respectively. For boys, relations between maternal parenting quality and externalizing behaviors were more likely to be significant for mothers' report than for fathers' report. Specifically, maternal parenting quality was significantly negatively correlated with mother-reported boys' externalizing at 30, 42, and 48 months; *r*s = -.23, -.23, and -.27, *p*s < .05, whereas the only negative relation of parenting quality with father-reported boys' 42-month externalizing was marginally significant; $r = .25$, $p < .10$. Moreover, baseline RSA was marginally significantly (negatively) correlated with father-reported 42-month boys' externalizing; $r = -.29$, $p < .10$. RSA reactivity was positively related to maternal parenting quality and negatively related to father-reported girls' externalizing at 48 months (both with marginal significance); *r*s = .16 and -.28, *p*s < .10, respectively.

For internalizing symptoms, in addition to the high correlations among different time points (of internalizing), negative correlations between maternal parenting quality and mother-reported boys' internalizing were marginally significant at 30 and 42 months; *r*s = -.21 and -.20, *p*s < .10, and significant at 48 and 54 months; *r*s = -.23 and -.28, *p*s < .05 and .01, respectively. Mother-reported 30-month and father-reported 30-month boys' internalizing was marginally and significantly positively associated with baseline RSA; *r*s = .18 and .23, *p*s < .10 and .05, for mothers' and fathers' reports, respectively. Also, the positive relation between baseline RSA and father-reported girls' internalizing at 48 months was marginally significant; $r = .26$, $p < .10$.

Therefore, it seems no particular correlation pattern for externalizing and internalizing symptoms, except that (1) both behaviors are relatively stable across 24 to 54 months, and (2) maternal parenting quality was negatively related to the mother-rated problem behaviors (especially for boys).

Unconditional Models of Externalizing and Internalizing Symptoms

Unconditional models were first tested by including fixed and random effects of quadratic growth factors in addition to fixed and random effects of intercept and linear slope growth factors. However, for both mother- and father-reported externalizing and internalizing, all the unconditional models with an estimated quadratic random effect (i.e., variance of the quadratic term) failed to converge. This suggests that individual variability in the quadratic trend could not be modeled. Thus, the models were rerun by fixing the variance of the quadratic factor to zero (i.e., only modelled the mean of the quadratic factor), and all the model estimations terminated normally after doing so.

In order to determine the need for estimating quadratic growth, chi-square difference tests were conducted to compare the models with intercept and linear growth factors as well as the quadratic mean estimated (i.e., freely-estimated model M_0) to the models specifying only intercept and linear growth factors (i.e., constrained model M_1). Due to the use of the MLR estimator, Satorra-Bentler scaled chi-square difference tests (Satorra & Bentler, 2010; correcting standard errors under nonnormally distributed data) were examined. The calculation takes the scaling correction factors into consideration. The formula is:

$\chi^2_{diff} = (\chi^2_{m1} \times c_{m1} - \chi^2_{m0} \times c_{m0}) \times (df_{m1} - df_{m0}) / (c_{m1} \times df_{m1} - c_{m0} \times df_{m0})$, where c_{m1} and c_{m0} are the corresponding scaling correction factors for constrained and freely

estimated model. Specifically, if the Satorra-Bentler scaled chi-square difference test is significant, it indicates that freely estimating the quadratic mean factor significantly improves the model fit. Hence, the M_0 models should be retained. However, if the Satorra-Bentler scaled chi-square difference test is not significant, the more parsimonious M_1 models need to be retained because constraining the quadratic mean factor does not significantly worsen the model fit (as compared to the M_0 models).

In addition to the Satorra-Bentler scaled chi-square difference tests, SEM overall model fit indices the *RMSEA* (smaller than .06 to indicate a good fit) and the *SRMR* (small than .08 to indicate a good fit) were also used to decide whether to specify the quadratic mean factor.

Third, the significance test for the quadratic mean factor should be significantly different from zero if to be specified in the unconditional models.

Externalizing symptoms. The overall model fit indices and the results from the Satorra-Bentler scaled chi-square difference tests are shown in Table 5. For mother-reported externalizing, the most appropriate unconditional models are the M_0 model with freely-estimated intercept, linear slope, and quadratic mean factor; $\chi^2_{(9)} = 36.82$, $p < .01$; (*CFI* = .93); *RMSEA* = .13; *SRMR* = .09. This decision was made because of the following factors, (1) the Satorra-Bentler scaled chi-square difference test was significant; $\chi^2_{diff(1)} = 11.38$, $p < .01$; (2) the overall model fit indices were better (although not good) for M_0 model; (3) the quadratic mean factor in M_0 was significantly different from zero; $b_{quadratic} = -.011$, $t = -3.27$, $p < .01$. Therefore, information from the three tests/sources consistently indicated that the fit of the M_0 model to the data with the quadratic mean factor was better.

However, it should be noticed that the overall model fit indices of M_0 were still not good, although they were better than M_1 models. To diagnose the misfit of M_0, I tested separate models with only a mean structure (i.e., saturated the variance/covariance structure so that it could be perfectly reproduced) and only a variance/covariance structure (i.e., saturated the mean structure so that it could be perfectly reproduced). By saturating the variance/covariance structure, any remaining model misfit can be attributable to the mean structure, and vice versa. Fit indices were as follows, $\chi^2_{(2)} = 32.58$, $p < .01$; (*CFI* = .93), *RMSEA* = .28, *SRMR* = .07 for the model with variance/covariance structure saturated, and $\chi^2_{(7)} = 10.40$, *ns*; (*CFI* = .99), *RMSEA* = .05, *SRMR* = .08 for the model with mean structure saturated. Therefore, it appears that the model misfit was due to the failure in reproducing the mean structure.

Furthermore, examining the residuals of externalizing variables at each time point suggested that the source of the mean structure misfit might be at 48 months. The plotted deviation of the model-estimated mean from the sample mean at 48 months can be easily seen in Figure 5 as well. Nevertheless, despite the relative lack of fit of M_0 (with the intercept set at 24 and 54 months), they still served as the unconditional models because they were the best among the possible growth curve models. Therefore, fixed and random effects of intercepts, linear slope, and fixed effect of quadratic factor were specified as the unconditional models of mother-reported externalizing (Figure 2 including the quadratic mean factor). Examining the fixed effect estimates (Table 6), it can be seen that there was an instantaneous increase of mother-reported externalizing on average at 24 months (i.e., $b_{linear24} = .027$, $t = 2.36$, $p < .05$), but the rate of change decelerated over time (i.e., $b_{quadratic} = -.011$, $t = -3.27$, $p < .01$; decelerated by .022 points in linear slope for

a 1-unit increase in time). Between 30 and 42 months, the trend started to decline, and finally at 54 months (i.e., end point of the current study), there was an instantaneous decrease in mother-reported externalizing (i.e., $b_{linear54}$ = -.038, t = -3.55, p < .01; See Figure 6a for the average growth trend).

Father-reported externalizing symptoms demonstrated a linear trend (Table 6), and M_1 models specifying an intercept and linear slope factors were the best unconditional models, $\chi^2_{(10)}$ = 6.74, p = .75; (*CFI* = 1.00); *RMSEA* = .00; *SRMR* = .06. This decision was made based on the following, (1) nonsignificant result of the Satorra-Bentler scaled chi-square difference tests; $\chi^2_{diff(1)}$ = 1.85, p = .93; (2) better overall SEM fit indices of M_1 models; (3) and nonsignificant quadratic mean factor in M_0 models; $b_{quadratic}$ = -.003, t = -1.34, p = .18. Therefore, in the unconditional models of father-reported externalizing, fixed and random effects of intercept and linear slope factors were included. On average, there was a declining trend in father-reported externalizing symptoms (Figure 4a).

Internalizing symptoms. Similar to externalizing symptoms, unconditional models of internalizing symptoms with quadratic mean factor (i.e., M_0 model) were also compared to the M_1 models with only intercepts and linear slope by the Satorra-Bentler scaled chi-square difference test. As is shown in Table 5, the Satorra-Bentler scaled chi-square difference tests were nonsignificant, $\chi^2_{diff(1)}$ = 2.72 and 3.10, ps = .10 and .08, for mothers' and fathers' report respectively, suggesting the preference of the more parsimonious M_1 models for internalizing symptoms rated by both reporters. In addition, overall fit indices of the M_1 models were better than the M_0 models and quadratic mean factors in M_0 models were all nonsignificant; $b_{quadratic}$s = -.003 and -.003, ts = -1.59 and -

1.58, *p*s = .11 for mothers' and fathers' reports, respectively (and means and variances of the intercepts and linear slope were significant; Table 5). Therefore, the best unconditional models of internalizing included the fixed and random effects of intercept and linear slope factors (see Figure 2 for the SEM model and Figure 6b for the model-implied mean growth trend). As is evident in Figure 6b, there was a declining trend in mother- and father-reported internalizing symptoms. The overall fit indices of unconditional models were $\chi^2_{(10)} = 25.43$, $p < .01$; (*CFI* = .96), *RMSEA* = .08, *SRMR* = .08, for mothers' reports, and $\chi^2_{(10)} = 10.68$, $p = .38$; (*CFI* = 1.00), *RMSEA* = .02, *SRMR* = .09, for fathers' reports.

Judging from the above global fit indices, the unconditional model of the mother-reported internalizing did not provide a good fit to the current data. To explore the potential reason, I followed the procedures discussed above by saturating either the variance/covariance structure (i.e., any remaining misfit was due to the mean structure) or saturating the mean structure (i.e., any remaining misfit was due to the variance/covariance structure). Model fit indices were $\chi^2_{(3)} = 22.39$, $p < .01$; (*CFI* = .95); *RMSEA* = .18, *SRMR* = .06 for the model with variance/covariance structure saturated, whereas fit indices for the model with the mean structure saturated were $\chi^2_{(7)} = 7.80$, *ns*; (*CFI* = 1.00); *RMSEA* = .03, *SRMR* = .08. Again, the lack of good fit was due to poor reproduction of the mean structure. In addition, residuals suggested that internalizing at 48 months may be the source of the mean structure's lack of good fit.

Conditional Models

Externalizing symptoms. The growth factors of externalizing included mother-reported 24-month intercept, 24-month linear slope, and quadratic slope (model 1),

mother-reported 54-month intercept, 54-month linear slope, and quadratic slope (model 2), father-reported 24-month intercept and linear slope (model 3), as well as father-reported 54-month intercept and linear slope (model 4). Therefore, four models (i.e., intercept centered at 24 and 54 months for mothers' and fathers' report; 2 × 2 = 4) with either baseline RSA or RSA reactivity as the moderator were tested to predict the above growth factors. Each of these models was conducted in three steps, with models for main effects, the 2-way interactions, and the 3-way interactions. Consequently, the numbers of main effect, two-way interaction as well as three-way interaction models each equals to eight (i.e., 4 baseline RSA + 4 RSA reactivity), which resulted in a total of 24 models for the growth curve of externalizing (see Table 7a). Note, however, that the two-way interaction models incorporated the main effects model for given predictors and a given outcome, and that the three-way interaction models were extensions of the models for 2-way interactions.

Baseline RSA. Findings from main effect models with baseline RSA (total number = 4; see Table 7a for the overall model fits) indicated that parenting quality negatively predicted 24-month and 54-month intercepts in all models, *b*s = -.097 and -.098, *t*s = -3.19 and -3.22, *p*s < .01, for the mother-reported 24- and 54-month intercepts, respectively; and *b*s = -.088 and -.058, *t*s = -2.86 and -1.96, *p*s < .01 and .05, for father-reported 24- and 54-month intercepts, respectively. Moreover, in mother-reported models, parenting quality was significantly associated with 24- and 54-month linear slopes and the quadratic slope of externalizing, *b*s= -.039, .038, and .008, *t*s= -2.27, 2.17, and 2.39, *p*s < .05, for 24- and 54-month linear slopes, and the quadratic slope, respectively. Therefore, children exposed to higher parenting quality at 18 months tended to exhibit

fewer externalizing symptoms at 24 and 54 months as rated by both parents. These children also exhibited a smaller instantaneous increase at 24-month in mother-reported externalizing behaviors. Their 54-month slope and quadratic slope became less negative as parenting quality increased. This indicated that there were smaller instantaneous decreases at 54 month as well as a relatively smaller deceleration in the rate of change in mother-reported externalizing behaviors. Main effects of sex and baseline RSA were nonsignificant (see Table 8a).

Next, as aforementioned, two-way interaction models (only specifying the parenting quality × children's baseline RSA with sex eliminated; 2 father-reported + 2 mother-reported = 4 models) and three-way interaction models (specifying interactions among parenting quality, children's baseline RSA, and sex; total = 4 models) were explored. Overall model fit statistics were the same for models with intercept set at 24 months and 54 months. The fit indices for the mother-reported two-way and three-way interaction models were $\chi^2_{(15)} = 41.55$, $p < .01$; (*CFI* = .95); *RMSEA* = .09; and *SRMR* = .06, and $\chi^2_{(23)} = 56.67$, $p < .01$; (*CFI* = .94); *RMSEA* = .09; and *SRMR* = .04, respectively. For father report, model fit statistics were $\chi^2_{(19)} = 14.02$, *ns*; (*CFI* = 1.00); *RMSEA* = .00; and *SRMR* = .04, for the two-way interaction model, and $\chi^2_{(31)} = 30.91$, *ns*; (*CFI* = 1.00); *RMSEA* = .00; and *SRMR* = .04, for the three-way interaction model.

Predicting the 24-month intercept, there was no significant interaction between parenting quality and baseline RSA in either the mother-reported model (Table 9a and 9b for two-way and three-way interaction models, respectively) or father-reported model (Tables 10a and 10b for two-way and three-way interaction models, respectively). For the 54-month intercept, the effects of the interaction in both two-way and three-way mother-

reported models and two-way father-reported models were nonsignificant. However, sex was found to interact with baseline RSA to predict the father-reported 54-month intercept of externalizing (Table 10b), $b = .047$, $t = 2.05$, $p < .05$. Specifically, a negative association between baseline RSA and 54-month (father-reported) intercept was significant among boys, $b = -.037$, $t = -3.59$, $p < .01$, but not girls, $b = .019$, $t = .99$, *ns*. Boys with low (i.e., -1 *SD*) baseline RSA displayed the highest level of externalizing symptoms at 54 months (rated by fathers), whereas boys with high (i.e., +1 *SD*) baseline RSA displayed the lowest (Figure 7).

In terms of the slopes (i.e., linear and quadratic slopes in mother-reported models, and only linear slope in father-reported models), no significant interactions between parenting quality and baseline RSA were detected for either mother- or father-reported models (see Tables 9a, 9b, 10a, and 10b for details).

RSA reactivity. Similar to the baseline RSA models, main effect models with RSA reactivity (i.e., residualized change score, with low scores indicating suppression or less augmentation) yielded negative associations between parenting quality and the growth factors (partialling out the other predictors) in mother- and father-reported models. All the coefficients were similar in values as the baseline RSA models (see Table 7a for the overall model fit statistics and Table 8a for the coefficients).

Moving onto the two-way and three-way interaction models, overall fit indices of mother-reported two-way and three-way interaction models were $\chi^2_{(15)} = 54.10$, $p < .01$; (*CFI* = .92); *RMSEA* = .12; and *SRMR* = .06 for the former, and $\chi^2_{(23)} = 73.17$, $p < .01$; (*CFI* = .91); *RMSEA* = .11; and *SRMR* = .05, for the latter. Model fits of father-reported two-way and three-way interaction models were $\chi^2_{(19)} = 16.84$, *ns*; (*CFI* = 1.00); *RMSEA*

= .02; *SRMR* = .04, and $\chi^2_{(31)}$ = 36.09, *ns*; (*CFI* = .98); *RMSEA* = .03; *SRMR* = .04 (Table 7a). Recall that the fits indices are the same for the 24-month intercept and the 54-month intercept models.

For the 24-month intercept, the interaction effects were all nonsignificant in the mother-reported models (Table 9a and 9b). In the father-reported models, there was a significant parenting quality × RSA reactivity interaction in predicting the 24-month intercept (Table 10a), b = -.049, t = -2.84, p < .01. To probe this interaction, the simple effect of parenting quality at a given level of RSA reactivity was first tested, and RSA reactivity were plotted across different values of parenting quality (Figure 8a). Parenting quality negatively predicted the 24-month father-reported externalizing intercept for children with average or high (i.e., +1 *SD*) RSA reactivity scores, *b*s= -.09 and -.16, *t*s= -3.20 and -4.28, *p*s < .01, whereas this relation was not significant for children with low (i.e., -1 *SD*) RSA reactivity scores, b = -.02, t = -.59, *ns*. Testing the *RoS* of the effect of parenting showed that negative association between parenting and the father-reported 24-month intercept was significant at the values of RSA reactivity ≥ .45 *SD* below the average (i.e., -.45 *SD*). Therefore, average and high RSA subgroups were more reactive to the varying levels of parenting quality.

Furthermore, I tested the simple effect and *RoS* of the RSA reactivity at a given level of parenting quality (Belsky & Pluess, 2009; Ellis et al., 2011; Roisman et al., 2012). RSA reactivity positively predicted the 24-month father-reported intercept when parenting quality was relatively low (i.e., -1 *SD*), b = .043, t = 2.22, p < .05, whereas this relation was negative at a marginal level of significance at the side of high parenting quality (i.e., +1 *SD*), b = -.02, t = -1.93, p < .10. RSA reactivity was unrelated to 24-

month intercept when parenting quality was at average level, $b = .012$, $t = 1.07$, *ns* (Figure 8b). *R*o*S* testing suggested that a positive association between RSA reactivity and 24-month intercept was significant at the values of parenting quality ≤ .60 *SD* below the average (i.e., -.60 *SD*), and a significant negative association at the values of parenting quality ≥ 1.10 *SD* above the average (i.e., lower and upper boundaries were within the observed range of parenting quality). Therefore, findings from the follow-up analyses met the statistical criteria for evaluating differential susceptibility (i.e., Belsky & Pluess, 2009; significant differences were obtained for both comparisons), revealing *a for-better-and-for-worse* pattern of the effects of parenting quality with relatively high RSA reactivity score (i.e., low RSA suppression) representing the "susceptibility/plasticity."

For 54-month intercepts and slopes, no significant interactions between parenting quality and RSA reactivity were detected for either mother- or father-reported models (see Tables 8a, 8b, 9a, and 9b for details).

Internalizing symptoms. The growth factors of internalizing included mother-reported 24-month intercept and linear slope (model 1), mother-reported 54-month intercept and linear slope (model 2), father-reported 24-month intercept and linear slope (model 3), and father-reported 54-month intercept and linear slope (model 4). Hence, similar to externalizing, four models (i.e., intercept centered at 24 and 54 months for mother and father report; 2 × 2 = 4) with either baseline RSA or RSA reactivity as the moderator were tested to predict the above models. The number of main effect, two-way interactions, and three-way interaction models each equals eight (i.e., 4 baseline RSA + 4 RSA reactivity), which results in a total of 24 models for the growth models of internalizing (see Table 7b for the overall model fit indices).

Baseline RSA. Similar to externalizing models, I first investigated the main effects of sex, parenting quality, and children's baseline RSA on the intercepts and slope of internalizing symptoms within each reporter (Table 8b). Partialling out the effects of other predictors, the 24-month intercept was marginally significantly positively predicted from children's baseline RSA in father-reported models, $b = .015$, $t = 1.73$, $p < .10$. The 54-month intercept and the linear slope were only significantly negatively predicted from parenting quality in mother-reported models, bs= -.070 and -.011, ts= -3.16 and -2.72, ps < .01 (for 54-month intercept and the linear slope, respectively). All the other main effects of baseline RSA, parenting, or sex were nonsignificant (see Table 8b).

Proceeding to the two-way interaction (only specifying the parenting quality × children's baseline RSA with sex eliminated; 2 father-reported + 2 mother-reported = 4 models) and the three-way interaction (specifying interactions among parenting quality, children's baseline RSA, and sex; total = 4) conditional models, the overall model fit indices suggested an acceptable fit to our current data (Table 7b). Specifically, for mother-reported two-way and three-way interaction models, the fit statistics were $\chi^2_{(19)} = 35.71$, $p < .05$; (*CFI* = .96); *RMSEA* = .06; and *SRMR* = .05, and $\chi^2_{(31)} = 42.95$, *ns*; (*CFI* = .97); *RMSEA* = .04; and *SRMR* = .04, respectively. For father report, the model fits were $\chi^2_{(19)} = 30.81$, *ns*; (*CFI* = .93); *RMSEA* = .06; and *SRMR* = .07, and $\chi^2_{(31)} = 49.04$, *ns*; (*CFI* = .95); *RMSEA* = .06; and *SRMR* = .07, for the two-way and three-way interaction models, respectively.

In regard to the significant interactions, mother- and father-reported 24-month intercepts were significantly associated with baseline RSA × sex interaction, bs = -.035 and -.040, ts = -2.04 and -2.55, ps < .05 (for mothers' and fathers' report, respectively;

Tables 11b and 12b) and the patterns were similar. For boys, baseline RSA was found to be positively significantly related to mother- (Figure 9) and father-reported (Figure 10) 24-month intercepts, *b*s = .018 and .027, *t*s = 2.29 and 3.23, *p*s < .05 and .01, whereas this relation was not significant for girls, *b*s = -.012 and -.010, *t*s = -.85 and -.79, *ns*, for mother- and father-reported 24-month internalizing, respectively. Therefore, it appears (see Figure 9 and Figure 10) that relative to boys with high (i.e., +1 *SD*) baseline RSA, boys with low (i.e., -1 *SD*) baseline RSA tended to demonstrate fewer parent-reported internalizing behaviors at 24 months, whereas girls with high or low baseline RSA were not significantly different from each other. All the other interactions in models predicting the 24-month intercepts reported by mother and father were nonsignificant (see Tables 11a, 11b, 12a, and 12b for the coefficients).

In addition, the 54-month intercept was significantly predicted from the parenting quality × baseline RSA × sex (three-way) interaction in the mother-reported model (Table 11b), $b = .072$, $t = 2.18$, $p < .05$. The results from the multiple-group model indicated that the interaction between parenting quality and baseline RSA was significant for girls, $b = .045$, $t = 2.12$, $p < .05$, but not for boys, $b = -.027$, $t = -1.07$, *ns*. Therefore, follow-up analyses were conducted only with the girls' model.

Specifically for girls, the negative association between parenting quality and 54-month intercept of girls' internalizing was significant when baseline RSA was at average or low (i.e., -1 *SD*) levels, *b*s = -.07 and -.14, *t*s = -2.64 and -2.83, *p*s < .01, but not at a high level (i.e., +1 *SD*), $b = -.01$, $t = -.31$, ns. The *RoS* test of the effect of parenting detected a significant association between parenting quality and girls' 54-month intercept at the values of baseline RSA ≤ .37 *SD* above the average (Figure 11a). Moreover,

examining the tests of simple effects for baseline RSA indicated that the association between baseline RSA and the mother-reported 54-month intercept of girls' internalizing was marginally significant at lower (i.e., -1 *SD*) parenting quality, b = -.040, t = -1.87, p < .10, but not significant at either average or higher (i.e., +1 *SD*) parenting quality, bs= -.010 and .019, ts= -.57 and .76, *ns*. Moreover, according to *RoS* testing, when the value of parenting quality ≤ 1.13 *SD* below the average (i.e., -1.13 *SD*), the negative relation between baseline RSA and mother-reported girls' 54-month intercept was significant (Figure 11b).

All the other interactions in models predicting 54-month intercepts reported by mothers and fathers were nonsignificant (see Tables 11a, 11b, 12a, and 12b for the coefficients).

Besides the 24- and 54-month intercepts, the mother-reported internalizing slope was significantly predicted from the interaction between parenting quality and baseline RSA (Table 11a), b = .006, t = 2.08, p < .05. According to tests of simple effects, parenting was negatively predictive of the mother-reported internalizing slopes for children with average or low (i.e., -1 *SD*) baseline RSA, bs = -.01 and -.02, ts = -2.80 and -2.99, ps < .01. For children with high (i.e., +1 *SD*) baseline RSA, this relation was nonsignificant, b = -.003, t = -.60, *ns* (Figure 12a) and the level of internalizing was low regardless of level of parenting. From the *RoS* analysis, negative prediction from parenting quality to the mother-reported internalizing slope was significant at the values of baseline RSA ≤ .32 *SD* above the average. When simple effects were computed with parenting as the moderator, baseline RSA was predictive of the mother-reported internalizing slope only when parenting quality was low, b = -.006, t = -2.21, p < .05.

When parenting quality was at average or high level, the prediction was not significant, *b*s= -.002 and .002, *t*s= -1.22 and .76, *ns* (Figure 12b). In fact, baseline RSA negatively significantly predicted the mother-reported internalizing slope at the values of parenting quality ≤ .51 *SD* below the average (i.e., -.51 *SD*).

The father-reported internalizing slope was positively significantly related to the interaction between baseline RSA and sex (Table 12b), $b = .012$, $t = 2.23$, $p < .05$. Specifically, the girls' internalizing slope was significantly positively associated with baseline RSA, $b = .011$, $t = 3.34$, $p < .01$, but the boys' internalizing slope was unrelated to baseline RSA, $b = -.003$, $t = -1.08$, *ns* (Figure 13). Therefore, relative to girls with high (i.e., +1 *SD* baseline RSA, girls with low (i.e., -1 *SD*) baseline RSA demonstrated a greater rate of decline in father-reported internalizing symptoms.

All other interactions in models predicting the internalizing slopes reported by mothers and fathers were nonsignificant (see Tables 11a, 11b, 12a, and 12b for the coefficients).

RSA reactivity. Main effect models including RSA reactivity yielded similar values of coefficients to the baseline RSA main effect models (Table 8b). Specifically, partialling out the effects of other predictors, the mother-reported 54-month intercept and slope were significantly negatively predicted from parenting quality, *b*s= -.069 and -.012, *t*s= -3.08 and -2.83, *p*s < .01 (for 54-month intercept and slope, respectively). All the other main effects of RSA reactivity, parenting, or sex were nonsignificant (see Table 8b).

For two-way and three-way interaction models of mothers' reports, the overall model fit statistics were $\chi^2_{(19)} = 40.24$, $p < .01$; (*CFI* = .95); *RMSEA* = .07; and *SRMR* = .06, and $\chi^2_{(31)} = 52.49$, $p = .01$; (*CFI* = .95); *RMSEA* = .06; and *SRMR* = .04,

respectively. For fathers' reports, the model fit statistics were $\chi^2_{(19)}$ = 17.88, *ns*; (*CFI* = 1.00); *RMSEA* = .00; and *SRMR* = .07, and $\chi^2_{(31)}$ = 30.77, *ns*; (*CFI* = 1.00); *RMSEA* = .00; and *SRMR* = .06, for the two-way and three-way interaction models, respectively (also see Table 7b).

An interaction between RSA reactivity and parenting quality predicted the father-reported intercept at 24 months (Table 12a), b = -.027, t = -2.02, p < .05. Probing the simple effect of parenting, negative prediction by parenting quality on the (father-reported) 24-month intercept was found only for children with high (i.e., +1 *SD*) RSA reactivity, b = -.047, t = -1.98, p < .05. Parenting was unrelated to the 24-month intercept among children with average or low (i.e., -1 *SD*) RSA reactivity, bs = -.007 and .031, ts= .34 and .94, *ns* (Figure 14a). The *RoS* analysis of the parenting effect suggested that the significantly negative association between parenting quality and 24-month intercept was at the values of RSA reactivity ≥ .98 *SD* above the average. When simple effects were computed with parenting as the moderator, RSA reactivity was negatively related to the father-reported 24-month internalizing intercept under the high positive parenting environment, b = -.023, t = -2.09, p < .05. RSA reactivity was unrelated to the 24-month intercept under low or average parenting quality, bs = .012 and -.005, ts= .87 and -.61, *ns* (Figure 14b). In the test of the *RoS* of RSA reactivity, RSA reactivity was significantly negatively related to the father-reported 24-month intercept at the values of parenting quality ≥ .82 *SD* above the average.

Therefore, this pattern appears to be consistent with the vantage sensitivity model, such that compared to children with average or low RSA reactivity scores, children with high RSA reactivity scores (i.e., low RSA suppression) were more reactive to the

different levels of parenting quality, and also they appeared to benefit more from the positive parenting environment in terms of their lower level of father-reported internalizing symptoms six months later.

All the other interactions in models with RSA reactivity predicting 24-month and 54-month internalizing intercepts reported by mothers and fathers were nonsignificant (see Tables 11a, 11b, 12a, and 12b for the coefficients).

Finally, the three-way interaction among parenting quality, RSA reactivity, and sex was significant in predicting the mother-reported internalizing slope (Table 11b), $b = .008$, $t = 1.99$, $p < .05$. The multiple-group model had a marginally significant interaction between parenting quality and RSA reactivity only among boys, $b = -.004$, $t = -1.72$, $p < .10$, but not among girls, $b = .003$, $t = .93$, *ns*. Therefore, follow-up analyses testing the simple effects and corresponding *RoS* were only conducted for boys.

Specifically for boys, parenting quality was significantly negatively associated with the mother-reported internalizing slope when RSA reactivity was at an average or high level (i.e., +1 *SD*; Figure 15a), bs = -.012 and -.019, ts = -2.69 and -2.89, ps < .01, but not at a low level (i.e., -1 *SD*), $b = -.006$, $t = -1.05$, *ns*. *RoS* testing of the parenting effect suggested that for boys, the significant negative link between parenting and the internalizing slope was at the values of RSA reactivity ≥ .47 *SD* below the average (i.e., -.47 *SD*). In addition, in simple effect analyses with parenting as the moderator, RSA reactivity was significantly associated with the boys' internalizing slope when parenting quality was at low (-1 *SD*) and average levels, bs = .007 and .004, ts = 2.43 and 2.35, ps < .05, but not at the high (+1 *SD*) level, $b = .002$, $t = .78$, *ns* (Figure 15b). The *RoS* test of RSA reactivity effect only yielded a lower boundary for boys such that RSA reactivity

significantly positively predicted boys' internalizing slope at the values of parenting quality ≤ .60 *SD* above the average.

Therefore, for the mother-reported internalizing slope, boys with average or high RSA reactivity (less suppression and/or more augmentation) were more reactive to the changing levels of environment. Boys' pattern may be consistent with the diathesis-stress model in which the rate of decline in internalizing symptoms is relatively slow for boys with high RSA reactivity scores (i.e., low RSA suppression) when they are exposed to average or less positive parenting environment.

DISCUSSION

Aims of the current study included describing the developmental trajectories of children's externalizing and internalizing symptoms across 24, 30, 42, 48, and 54 months of age, and exploring the role of the 18-month (observed) parenting quality × children's baseline RSA/RSA reactivity interaction in predicting the trajectories. In addition, I investigated whether the prediction of externalizing and internalizing trajectories from the parenting × baseline RSA/RSA reactivity interaction was moderated by child sex. To determine the patterns of the interaction (Roisman et al., 2012), both simple effects of parenting and baseline RSA/RSA reactivity and the corresponding *RoS* were tested.
It was hypothesized that children with low baseline RSA would be more reactive than children with higher baseline RSA in response to levels of parenting quality. Children with low baseline RSA were expected to have poorer adjustment (i.e., exhibiting more externalizing and internalizing symptoms at 24 and/or 54 months, and slower decline in externalizing and internalizing over time) when exposed to less positive parenting, in a pattern consistent with the diathesis-stress model. In addition, children with high RSA

reactivity (more augmentation and/or less suppression) were expected to be more reactive to the varying parenting quality. Moreover, it was hypothesized that when parenting support was low, children with high RSA reactivity would demonstrate more 24-, and/or 54-month externalizing and a slower decline in externalizing as they aged (i.e., results consistent with the diathesis-stress model). However, in terms of internalizing symptoms, children with high RSA reactivity were hypothesized to benefit most from high quality parenting, displaying fewer 24- and/or 54-month internalizing and faster decline in internalizing over time (i.e., the vantage-sensitivity model). Partially supporting these hypotheses, the most consistent finding in the current study was that children with low baseline RSA or high RSA reactivity (i.e., high augmentation, which reflects less RSA suppression), in comparison to those with high baseline RSA or low RSA reactivity, were more reactive as a function of early parenting quality in regard to the development of early childhood problem symptoms.

Parenting Quality × RSA in Predicting the Internalizing Trajectory

In predicting the developmental trajectory of internalizing symptoms, both baseline RSA (supporting hypothesis III) and RSA reactivity (supporting hypothesis IV) moderated the effects of early parenting quality. The findings regarding the moderating role of baseline RSA were consistent with the diathesis-stress model, with low baseline RSA representing the "vulnerability." For the mother-reported intercept at 54 months, but not 24 months, the interaction between parenting quality and baseline RSA was further moderated by sex (i.e., significant for girls but not boys). The negative association between parenting quality and 54-month internalizing was significant for girls with low and average baseline RSA, but not girls with high baseline RSA. It appears that compared

to girls with high baseline RSA who were relatively low in mother-reported 54-month internalizing regardless of the parenting quality, girls with low baseline RSA were more reactive to the varying levels of parenting quality (Figure 11a), and exhibited more internalizing symptoms in the context of less positive parenting (Figure 11b). The pattern of mother-reported internalizing slope was similar to the pattern for the 54-month intercept (except that the slope finding was not moderated by sex): children with low baseline RSA were more reactive as a function of parenting quality (Figure 12a) and the difference between different levels of RSA was significant only for low quality parenting. Moreover, in analyses with parenting as the moderator, for children experiencing low quality parenting, those with low baseline RSA had a slowest rate of decline (the slope was negative) with age (Figure 12b). To summarize, under the condition of less preferable early parenting quality, low baseline RSA appeared to function as a "vulnerability" (in the diathesis-stress model), conferring risk for more internalizing symptoms 3 years later (at 54 months) among girls, and, for the whole sample, being associated with a slower decline in their internalizing over time.

The findings regarding the moderating role of RSA reactivity consistently showed that children with high RSA reactivity (i.e., more inclined to RSA augmentation) were more reactive as a function of parenting quality relative to those with low RSA reactivity, especially for boys. However, the patterns were slightly different for fathers' and mothers' reports. For father-reported internalizing at 24 months, relative to those with low or average RSA reactivity, children with high RSA reactivity appeared to be more reactive to parenting quality (Figure 14a), and the difference among the three levels of RSA reactivity was significant at high levels of positive parenting (Figure 14b). When

parenting was positive, children with high RSA reactivity exhibited the lowest level of 24-month internalizing symptoms; however, individual differences were not significant when parenting was average or less positive. Hence, this pattern seems to be consistent with the vantage sensitivity model. For mother-reported internalizing slope, the interaction between parenting quality and RSA reactivity was significant for boys, but not for girls. For boys, quality of parenting was related to the slope of internalizing for boys with high and average RSA reactivity (Figure 15a). Within the context of low supportive parenting (Figure 15b), boys with high RSA reactivity demonstrated the slowest decline in internalizing symptoms. Significant individual differences (in mother-reported boys' slope) were not observed for high parenting quality. Thus, this pattern was found to be consistent with the diathesis-stress model. In the following sections, interpretation of these findings is discussed.

Low baseline RSA and high RSA reactivity were more reactive (in predicting the internalizing trajectory). As elaborated in the literature review, low baseline RSA has been associated with relatively poor physiological functioning because it may reflect a trait-like characteristic that restricts the exhibition of individuals' regulation ability. By the same token, high RSA reactivity in the form of RSA augmentation sometimes is regarded as an index of maladjusted self-regulation ability (Porges, 1996, 2007). Due to the lack of better self-regulation, children with low baseline RSA or high RSA reactivity may have to depend on the external regulatory resources or assistance provided by their socializers, demonstrating high responsivity to changing parenting quality (Hastings et al., 2008). In contrast, children with high baseline RSA or low RSA reactivity may already have their own internal resources to use, and thus may act relatively

independently from the outside environment in regulating their behaviors. The empirical studies discussed in the introduction also reported the more reactive role of low baseline RSA (e.g., Shannon et al., 2007; Wetter & El-Sheikh, 2012) and high RSA reactivity (e.g., El-Sheikh & Whitson, 2006) in the development of children's internalizing symptoms.

Patterns of the interaction (in predicting the internalizing trajectory). As mentioned previously, a pattern consistent with the diathesis-stress hypothesis emerged for the significant mother-report models with either baseline RSA or RSA reactivity as the moderator. Low baseline RSA (especially in girls; more internalizing symptoms at 54 months for girls and slower decline for the whole sample) or high RSA reactivity (especially in boys; slower decline over time), along with low parenting quality, was identified as the "dual risk" factors that exacerbate children's internalizing symptoms rated by mothers. These findings are consistent with the previous research where school-aged children with low baseline RSA were more vulnerable to marital conflict (e.g., El-Sheikh et al., 2001), parental drinking (e.g., El-Sheikh, 2005) or maternal depression (e.g., Shannon et al., 2007), exhibiting high levels of internalizing behaviors (El-Sheikh et al., 2001). Although few studies have reported a significant moderating role of RSA reactivity in the association between parenting/environment and young children's internalizing (El-Sheikh & Whitson, 2006, was an exception), Perry et al. (2012) found that the emotion regulation ability was compromised by maternal nonsupportive reactions to children's negative expressivity, but only among preschoolers with high RSA reactivity. Given the positive association between poor emotion regulation and high levels of internalizing behaviors among preschoolers (Blair, Denham, Kochanoff, &

Whipple, 2004), high RSA reactivity might also function as the "vulnerability" for internalizing symptoms in the context of less advantageous parenting quality.

The detected diathesis-stress pattern indicates that associations between baseline RSA or RSA reactivity and mother-reported internalizing tend to be stronger in the context of low parenting quality (relative to high parenting quality). This pattern might be explained by considering RSA as a physiological index of response to the external challenges or stress (Porges, 2007). Low quality parenting (e.g., low maternal sensitivity such that children's needs cannot be addressed in a timely manner, or low maternal warmth where mothers seldom display affection toward their children) could be regarded as a stressful context for the adaptive development of young children. As a response to this stressful environment, it would make sense that children with different physiological regulation abilities (indexed by RSA) exhibited different levels of problem behaviors in the context of less positive parenting. In addition, low baseline RSA or high RSA reactivity (i.e., more augmentation or less suppression) is believed to indicate physiological maladjustment and impaired ability of self-regulation (Porges, 2007). Low baseline RSA or high RSA reactivity may be more vulnerable to a relatively adverse environment (compared to high baseline RSA or low RSA reactivity), thus conferring physiological risk for the development of internalizing symptoms (reported by mothers). Nevertheless, while experiencing high quality caregiving, perhaps children with low baseline RSA or high RSA reactivity could perform as well as their counterparts (with high baseline RSA or low RSA reactivity).

However, despite the consistent diathesis-stress pattern for (mother-reported) 54-month girls' intercept and slope in the models with baseline RSA, and (mother-reported)

boys' slope in the model with RSA reactivity, the prediction of father-reported 24-month intercept from parenting quality × RSA reactivity interaction showed the vantage sensitivity pattern. Although the high RSA reactivity was still more reactive (as in mother-reported models), individual differences were significant only at the higher end of the parenting quality in the father-reported model. High RSA reactivity was associated with fewer internalizing problems when parenting was supportive.

A plausible explanation of this vantage-sensitivity pattern (which is different from the diathesis-stress pattern of mother report) is that father engagement in families with high quality maternal parenting provides a better caregiving environment from which children with high RSA reactivity could benefit, resulting in lower levels of their (father-reported) internalizing symptoms. Indeed, it has been documented that father involvement in childrearing is contingent upon maternal parenting, especially during early developmental periods (Bradford & Hawkins, 2006). More effective fathering practices (e.g., high paternal sensitivity, encouragement of exploration and low paternal negative intrusiveness) have been observed in families where maternal parenting quality is high (e.g., Barnett, Deng, Roger, Willoughby, & Cox, 2008, for infants; McBride & Rane, 1998, for childhood, and Pleck & Hofferth, 2008, for adolescents). This relation may also be partially revealed in the current attrition analyses such that fathers participated in more waves of data collection when higher maternal parenting quality was observed. This effective fathering (in addition to the advantageous mothering) might be especially beneficial for children with high RSA reactivity due to their potential lack of self-regulation ability. Moreover, the advantage of father engagement might be more

evident during father-children interactions (Paquette, 2004), which in turn was associated with fathers' perceiving of lower levels of internalizing symptoms.

In contrast, as mentioned in the introduction, low RSA reactivity under the condition of high positive parenting might reflect children's chronic RSA suppression as a reaction to the environment which is safe in nature. This (RSA suppression) might also be a sign of maladaptive regulation because it implies an unnecessary mobilization of the defensive systems to the unthreatening environment (Hastings & Miller, 2014). As a consequence, children with low RSA reactivity might demonstrate high levels of (father-reported) internalizing when exposed to high parenting quality.

Moderation by sex (in predicting the internalizing trajectory). In mother-reported models, it is not clear why a significant parenting quality × baseline RSA interaction was only obtained for the 54-month intercept of girls (but not boys) and a parenting quality × RSA reactivity interaction was only significant for boys' slope (but not girls'). In fact, one of the few studies that examined the three-way environment × baseline RSA × sex interaction also reported similar findings for girls. Wetter and El-Sheikh (2012) found that school-aged girls (but not boys) with low baseline RSA and were exposed to high levels of maternal depression (i.e., negative environment) displayed the highest levels of internalizing symptoms 2 years later.

However, the significant three-way interaction among parenting/environment, RSA reactivity, and sex in predicting the trajectory of internalizing problems has not been reported. Considering the age range of the current study, it might be the case that mothers started to be attuned to girls' internalizing symptoms during preschool years (e.g., 54 months) rather than during late toddlerhood (e.g., 24 months), whereas boys' internalizing

symptoms might have been viewed as more problematic by mothers from toddlerhood (e.g., 24 months) to the preschool years (e.g., 54 months; Keenan & Shaw, 1997; more discussion below). For girls at 2 years old, within most societies, the demonstration of high internalizing symptoms in the form of separation distress, anxiety, or social withdrawal might be more likely to be accepted as normative or even encouraged by caregivers (Bayer et al., 2006; particularly over girls' externalizing symptoms). Therefore, young girls' internalizing problems may develop either unnoticed or unaddressed by socializers until preschool years when these symptoms begin to elicit attention from parents, especially if girls express high levels of distress and anxiety in the school context (Keenan & Shaw, 1997). If this is the case, the involvement of parental socialization might be more important for girls during the preschool years (e.g., around 54 months in the current study) in terms of decreasing their internalizing behaviors. In other words, lack of positive socialization or effective parenting on internalizing may exert greater detrimental effects for preschooler girls than at earlier time points. Also, given the argument of the physiological maladjustment of low baseline RSA (and high RSA reactivity), the adversity of less positive parenting may be more pronounced for girls with low baseline RSA at this age.

For boys, in contrast, although they may experience internalizing problems at a similar level as girls, mothers may tend to regard boys' internalizing symptoms as more unacceptable due to the gender stereotype that boys should be assertive, less distressed or less withdrawn than girls (Rubin, Coplan, & Bowker, 2009). Therefore, relative to girls, from an early age, boys' high internalizing symptoms might elicit sustained regulatory efforts from mothers, which may account for the decline in boys' internalizing behaviors

over time, rather than boys' internalizing intercept at a specific time point. Specifically, based on the simple effects within high parenting, positive parenting was associated with a relatively fast decline in boys' mother-reported internalizing regardless of the level of RSA reactivity. When exposed to low parenting quality or lack of advantageous caregiver socialization, nevertheless, boys with high RSA reactivity (i.e., physiologically maladaptive) displayed a significantly slower decrease in their internalizing over 24 to 54 months reported by mothers. In light of this interpretation, the significant parenting quality × RSA reactivity interaction in predicting the mother-reported internalizing slope among boys would make sense.

Parenting Quality × RSA Reactivity in Predicting the Externalizing Trajectory

The most important finding about externalizing symptoms was that parenting quality × RSA reactivity interaction was significantly predictive of father-reported (externalizing) intercept at 24 months. Consistent with the internalizing findings, high RSA reactivity was identified as more reactive to levels of parenting quality, relative to the more stable, low RSA reactivity. For children with high RSA reactivity, more 24-month externalizing symptoms were exhibited under low parenting quality, whereas fewer symptoms were found under high parenting quality, implying a differential susceptibility pattern. Thus, the high RSA reactivity reflects the "susceptibility/plasticity" for externalizing symptoms, with high RSA reactivity children being not only most vulnerable to the environmental adversity but also benefiting most from the desirable parenting. However, because a total as 16 (two-way and three-way interaction) models predicting the trajectory of externalizing were tested, and only one significant interaction was found (between parenting and RSA reactivity), this result might be due to chance.

Further replication efforts are needed to examine the prediction of early childhood externalizing from the interaction between parenting quality and RSA reactivity.

Baseline RSA × Sex in Predicting the Internalizing and Externalizing Trajectories

Baseline RSA also interacted with sex in predicting the trajectories of internalizing and externalizing symptoms. For internalizing symptoms, the effect of baseline RSA × sex interaction was significant for the 24-month intercept (similar patterns reported by both parents) and linear slope (rated by fathers). Boys, but not girls, with high baseline RSA displayed more internalizing at 24 months, whereas boys with low baseline RSA displayed less (Figure 9 for mother-reported findings, and Figure 10 for father-reported findings). Despite the nonsignificantly statistical difference among girls with varying levels of baseline RSA, girls with low baseline RSA had slightly higher levels of internalizing, as compared to their counterparts with high baseline RSA. On the other hand, the father-reported internalizing slope (Figure 13) appeared to be different among girls, but not boys, with different baseline RSA. The decline in father-reported internalizing symptoms over time was greater among girls with low baseline RSA, in comparison to girls with high baseline RSA. For boys, the decline in (father-reported) internalizing was slightly (but not statistically significantly) smaller among boys with low baseline RSA.

Examining the model predicting father-reported 24-month intercept and linear slope, the correlation between the latent intercept and slope factors r = -.11. Because low baseline RSA boys had fewer internalizing symptoms at the beginning (of the current study; i.e., 24 months), the decrease of their internalizing over time was not likely to be as large as the decrease for the high baseline RSA boys (who started with higher

internalizing levels). By the same token, girls with high baseline RSA started slightly lower in their internalizing levels (at 24 month) and, perhaps for that reason, they did not decline as fast as girls with low baseline RSA. In fact, judging from the Figure 13, there was a small increase of internalizing among girls with high baseline RSA.

For externalizing, the interaction between baseline RSA and child sex was significantly associated with the father-reported 54-month intercept. Boys, but not girls, with different levels of baseline RSA exhibited significantly different levels of 54-month externalizing (rated by fathers). However, opposite to the pattern for internalizing symptoms at 24 months reported by parents, boys with low baseline RSA exhibited more externalizing at 54 month, whereas boys with high baseline RSA displayed less. Yet, girls with low baseline RSA appeared to show slightly lower levels of externalizing at 54-month, relative to girls with high baseline RSA (although this difference was not statistically significant). The significant finding for boys was consistent with hypothesis VII (regarding the moderating role of sex) and with the previous research reported a negative relation between baseline RSA and boys' externalizing behaviors (Beauchaine et al., 2008 for school-aged children).

Integrating the aforementioned findings about externalizing and internalizing symptoms, one consistent aspect was that the associations (either negative or positive) between baseline RSA and the problem behaviors (i.e., parents-reported internalizing at 24 months, and father-reported externalizing at 54 months) were stronger among boys than girls during early childhood. In other words, physiological regulation might play a relatively more important role in young boys' psychopathology symptoms than girls'. One plausible explanation of this sex difference lies in the differential genetic effects on

externalizing and internalizing symptoms for boys versus girls, which may be reflected in the differential psychophysiological response patterns (Beauchaine et al., 2008). In fact, there is some evidence of the sex-specific genetic effect. The source of genetic influences on externalizing (Rose et al., 2004) and internalizing (Kendler et al., 2000) symptoms might be different for males and females, which may be accounted for by certain biological factors, such as hormonal levels during early development (e.g., Collaer & Hines, 1995). Also, the heritability of externalizing (e.g., Dick, Viken, Kaprio, Pulkkinen, & Rose 2005) and internalizing symptoms (e.g., Haberstick, Schmitz, Young, & Hewitt, 2005) was reported to be higher among boys versus girls. Thus, it might be true that boys' and girls' psychopathological symptoms do not share the same biological/physiological markers, and boys are more influenced by the biological factors.

In contrast to the consistent finding that the relation between baseline RSA and problem behaviors was stronger for boys, the inconsistent finding regarding externalizing and internalizing symptoms was that they tended to have different relations with baseline RSA, especially for boys in the early childhood. Specifically, boys with high baseline RSA, as compared to boys with low baseline RSA, displayed lower levels of father-reported externalizing at 54 months, yet higher levels of parent-reported internalizing symptoms at 24 months,.

First, it is not difficult to interpret this negative link between baseline RSA and boys' externalizing behaviors (if it was not significant due to chance), when one considers higher baseline RSA may reflect better physiological functioning (as argued by the polyvagal theory elaborated earlier; Porges, 2007). Specifically, better physiological functioning (as reflected in higher baseline RSA) may not only provide the prerequisite

for adaptive regulation to occur (Porges, 2007), but also help to free up metabolic resources for cognitive process, which in turn, promote the regulation of externalizing behaviors (Graziano et al., 2007). Conversely, poor physiological performance may disrupt motivational and regulatory processes; this disruption could give rise to high impulsivity and behavioral dysregulation that may contribute to emotional lability and aggression (Beauchaine, 2008).

The positive link between higher baseline RSA and boys' internalizing was unexpected given the putative facilitating role of higher baseline RSA in emotion and behavior regulation. This pattern, to some extent, was reliable across mothers' and fathers' reports of internalizing, and may suggest that externalizing and internalizing were driven by different etiological mechanisms. Alternatively, the different associations between baseline RSA and boys' externalizing versus internalizing are more likely to suggest that gender differences exist in the relation between baseline RSA and behavioral problems, but that these associations are complex. For example, baseline RSA may interact with other variables (such as parenting quality and other aspects of the home environment, or possibly other child characteristics) to predict the development of externalizing versus internalizing symptoms.

The Main Effect of 18-month Parenting Quality in Predicting the Internalizing and Externalizing Trajectories

Eighteen-month parenting quality was hypothesized to be predictive of lower levels of externalizing and internalizing behaviors (at 24 and 54 months), and more rapid rates of decline in externalizing and internalizing over time. Partially consistent with this hypothesis, parenting quality was found to be significantly (either negatively or

positively) associated with the development of externalizing in both mother- and father-reported models (except for father-reported linear slope), and mother-reported internalizing models (except for mother-reported 24-month intercept), whereas parenting quality was unrelated to models predicting father-reported internalizing symptoms.

For externalizing, high 18-month parenting quality was associated with low levels of externalizing symptoms at 24 and 54 months reported by both parents. These findings are in line with the previous research with school-aged children (e.g., Miner & Clarke-Stewart, 2008) and adolescents (e.g., Galambos et al., 2003; Simons et al., 2005). In addition, for mother report of externalizing in the current study, high parenting quality was predictive of a small instantaneous increase at 24 months, and a small instantaneous decline at 54 months in externalizing behaviors. The deceleration in the rate of change was also small for children with mothers high in parenting quality. This pattern suggested that, relative to their counterpart with less positive parenting quality, children whose mothers were high in early parenting quality demonstrated a smaller increase in externalizing 6 months later, and a less sharp deceleration of rate of change over time with a flatter decline of externalizing 30 months later. Thus, it appears that these children did not have a dramatic increase or decrease in their mother-reported externalizing symptom over time.

The significant main effect of parenting quality in predicting most of the growth factors of parent-reported externalizing (except for the father-reported linear slope) suggests that high parenting quality was related to low levels of children's externalizing symptoms and relatively less dramatic change (i.e., a less marked inverted U quadratic effect) over time. This tended to be true for all children regardless of their baseline

RSA/RSA reactivity levels or sex. The positive association between high parenting quality and lower levels of externalizing behaviors has been widely documented (e.g., Adam et al., 2004). For instance, as elaborated in the introduction, positive association between maternal warmth and decreased externalizing was found not only among children at childhood (Eiden et al., 2007), but also among young adolescents (McKee et al., 2008). The same relation was also reported for at-risk African Americans (Jone et al., 2008), children with conduct problems (Katz & Windecker-Nelson, 2004), and children from low-income families participating in the NICHD study (Bradley & Corwyn, 2007). The prominent main effect of parenting quality may also, to some extent, provide an explanation for the lack of significant interaction effects in externalizing trajectory (i.e., only 2 out of 16 models found significant interactions) in the current study (more in the general discussion section).

Nevertheless, the findings of the conditional models predicting the mother-reported externalizing need to be interpreted with caution because the unconditional model provided poor reproduction of the mean structure of the mother-reported externalizing trajectory (i.e., the model fits indices were $\chi^2_{(9)} = 36.82$, $p < .01$; (*CFI* = .93); *RMSEA* = .13; *SRMR* = .09; more discussion below). The conditional models were built on inaccurate unconditional growth model for mothers' reports. Therefore, when predictors were added to predict the mean-level latent growth factors, these predictions may not be reliable because the unconditional model was not reflective of the true growth trend to begin with.

For internalizing, parenting quality was negatively predictive of mother-reported 54-month intercept and linear slope, which suggested that high parenting quality

contributed to the low levels of internalizing symptoms 30 months later and greater decline in internalizing over time. This finding makes sense because (as discussed with details in the introduction) when children exhibit distress or anxiety, parents high in warmth and sensitivity/responsivity tend to provide timely emotional support and regulation assistance in attempts to reduce these internalizing symptoms (Bayer et al., 2006). These parents are also likely to establish a secure attachment with their children, and thus may promote the development of children's emotion regulation, which in turn is associated with decreasing levels of internalizing during childhood (see Brumariu & Kerns, 2010, for a review on the relation between attachment and childhood internalizing). However, the main effect of parenting quality became less important when taking into consideration its interactions with baseline RSA and sex (in predicting mother-reported 54-month intercept and slope). Prediction by parenting quality was moderated by the other predictors.

Regarding the father-reported models of internalizing, the failure to detect a significant main effect of parenting quality might be due to the lack of statistical power. Although the FIML estimates have greater statistical power in significance tests than ML (Yung & Zhang, 2011), the greater amount of missing data in father-reported internalizing behavior across time may still limit the ability to detect the significant effects. For example, in the current study, missing data for father-reported internalizing at 54 months were 121, as compared to 62 for mother-reported internalizing at 54 months. In fact, the lack of statistical power in research on father-reported psychopathology due to the small sample size is not uncommon (Phares, Lopez, Fields, Kamboukos, & Duhig, 2005). It has been advocated that researchers should make concrete efforts to increase the engagement

of fathers in the psychopathology study, and therefore, provide separate analyses for mothers and fathers in a parallel manner (Zimmerman et al., 2000). Another plausible reason for the low statistical power is the low reliability of father-reported internalizing symptoms at different time points. For instance, in comparison to (father-reported) externalizing reliability at 54 months (i.e., $\alpha = .78$), the Cronbach's alpha for internalizing at 54 months was .67. This may partly explain why a significant main effect of parenting was detected in father-report externalizing models (i.e., predicting father-reported 24 and 54 months), but not internalizing models.

Developmental Trajectories of Externalizing and Internalizing Symptoms

Before proceeding to discuss the specific trajectories, again, it should be acknowledged that the unconditional model of mother-reported externalizing failed to adequately reproduce the mean structure of the observed data at 48 months. In fact, procedures of 48 months in the current study were slightly different from the other time points such that questionnaires were mailed to the participating families rather than their visiting laboratory to finish the observation tasks and questionnaires (as in 30, 42 and 54 months). This different procedure might influence the collection of questionnaire data from the participants, resulting in reduced total sample *N* at 48 months (e.g., 147 at 48 months for mother report as compared to 169 at 42 months, and 155 at 54 months). In addition, examining Figure 5 suggested that the model-estimated mean (i.e., the mean that would have obtained had one's data been completed) was larger than the observed sample mean at 48 months; this indicated that some mothers whose children demonstrated relatively high levels of externalizing symptoms did not return their questionnaires at 48 months.

Knowing the possible reason for the misfit of the mean structure at 48 months, I moved on to interpret the mother-reported externalizing trajectory. Specifically, a negative quadratic slope with a positive 24-month linear slope and a negative 54-month linear slope was found. This implied that during the study period (i.e., 24 to 54 months), there was an overall deceleration in the rate of change with an instantaneous increase at the beginning and an instantaneous decrease at the end of the study in mother-reported externalizing symptoms. The plot of the mother-reported externalizing symptoms (Figure 6a) indicated a peak between 30 and 42 months, followed by a decline until the end of study (i.e., 54 months). This finding was consistent with our previous work reporting a peak of mother-reported aggression at approximately 36 months in the quadratic trend across 18 to 54 months (Eisenberg, et al., 2012). A similar deceasing trend for externalizing behaviors from the age of 36 months onward was also found by Alink et al. (2006) studying the developmental trajectory of externalizing over the period of 12 months to 48 months.

However, in contrast to mothers' reports in the current study, father-reported externalizing symptoms (across 24 to 54 months) demonstrated a linear decline (Figure 6a), which is consistent with previous research documenting a steep decrease in father-reported externalizing for ages 2 through ages 7 (Hussong et al., 2007). The different patterns in children's externalizing problems reported by mothers and fathers have also been documented in the previous research, such that mothers reported more externalizing behaviors than fathers did, and the discrepancy between mothers' and fathers' reports became larger at 24 months relative to the earlier ages (Stanger & Lewis, 1993).

This disagreement between parents may be attributed to the different socialization roles played by mothers versus fathers. Compared to fathers, mothers are more likely to serve as the primary caregivers during toddlerhood and to spend more time taking care of their toddlers. Children may have more opportunities to demonstrate externalizing behaviors while interacting with mothers (e.g., they can act out at home when they are tired or during grocery shopping when they really want a toy). Hence, it is possible that mothers are more aware of their children's acting out and the nuance in the change of children's externalizing behaviors than fathers do (Miner & Clarke-Stewart, 2008). Mothers' reports may be more valid. However, this argument may not necessarily hold because in the present study, the difference between mother- and father-reported externalizing was only marginally significant at 42 months; $t_{(107)} = 1.67$, $p < .09$, but not other time points.

The normative increase in mother-reported externalizing symptoms from 24 months to a point between 30 and 42 month may be partially explained by the development of autonomy feelings in children at ages 2 to 3 (e.g., Campbell, 2002). Due to their growing self-awareness during this time, toddlers start to display new exploratory behaviors that might seem unacceptable to their parents, which may elicit parents' controlling practices, such as limit setting. The potential conflict between parental control and children's desire for independent exploration of the external world may contribute to the increase in externalizing behaviors (Alink et al., 2006). Again, this development may be more likely to be perceived by mothers than fathers because mothers' interactions with toddlers are higher in frequency and longer in duration compared to fathers' (Miner & Clarke-Stewart, 2008). Nevertheless, the decline in the parent-reported prevalence of

externalizing starting from approximately 3 years old may be accounted for by the development of children's self-regulation through the process of parental socialization (Rothbart & Bates, 2006). During early childhood, children begin to internalize their parents' rules and requirements, and learn to control and regulate their behaviors in a more socially appropriate way. Their rapidly growing language ability also enables them to verbally communicate their feelings and situations with people around, which may further promote children's constructive regulation of externalizing behaviors (Nelson, Benner, Neill, & Stage, 2006).

In contrast to externalizing problems, both mothers and fathers reported a linear decline in internalizing symptoms from 24 to 54 months of age. This finding is consistent with the Carter et al. (2010) study. Nevertheless, another study on early childhood reported an increase in children's internalizing behaviors over age 2 to 6 (Gilliom & Shaw, 2004). When comparing it to the current investigation, one important distinction should be pointed out. The participants in Gilliom and Shaw (2004) study were from financially disadvantaged families (i.e., annual income was $11,568 for a family of four), whereas the majority of participating families in the current study were middle-class families. The financial difficulties experienced by parents in the Gilliom and Shaw (2004) study might constrain their ability to address the children's psychological and emotional needs by either not perceiving children's' signals in a timely manner, or lacking the ability to provide positive and sufficient support to children's distress. Thus, the acquisition of autonomous coping skills of children from poverty families might be jeopardized, and these children may be more likely to display increasing, rather than decreasing, internalizing symptoms during early childhood.

In addition to the mean-level trajectories of externalizing and internalizing symptoms, the variances of the intercepts and linear slopes in mother- and father-reported (externalizing and internalizing) unconditional models were significant. Although there were overall quadratic or linear trajectories for externalizing or internalizing behaviors, individual variability in their developmental trends was also significant, indicating that this variability may be accounted for by adding predictors.

General Discussion

Baseline RSA versus RSA reactivity. The current study identified the moderating effect of baseline RSA in most of the significant two-way and three-way interaction models (i.e., 6 out of 9). Compared to the baseline RSA, interactions concerning RSA reactivity were significant in only 3 models (with the one predicting the father-reported externalizing at the 24 months possibly being due to chance). Given the proposed role of RSA reactivity as an index of individuals' physiological ability to respond to external environment/challenges (Porges, 2007), one might have expected to find a more pronounced interactive effect of parenting with RSA reactivity than with baseline RSA. The lack of significant findings of RSA reactivity was also reported in previous work with the same sample as in this study (i.e., Eisenberg et al., 2012). One plausible reason underlying the less prominent moderating role of RSA reactivity concerns the stimulus used in the current study to assess it—film of crying babies). Some other RSA studies assessed RSA reactivity with cognitive challenging tasks (e.g., Hinnant & El-Sheikh, 2009) or situations designed to elicit physiological stress (e.g., Calkins et al, 2007); in contrast to those measures, the film of crying babies might be more socially orientated and less cognitively stressful, and thus might involve less of an attentional load

and require fewer physiological coping resources. Perhaps a significant moderating effect would be detected by using more evocative tasks or tasks with greater cognitive stress.

Another possible explanation is that parenting × RSA reactivity interaction may be more evident for older children than children in early childhood. In other research that has reported this significant interaction, the participants generally were children or adolescents (e.g., El-Sheikh et al., 2001). El-Sheikh et al. (2001) reported that higher RSA suppression appeared to protect 8- to 12-year-old boys against externalizing problems associated with parental verbal conflict. Although El-Sheikh et al. (2001) assessed RSA reactivity through a more evocative and stressful task (i.e., hearing adult argument), Gentzler et al. (2009) study used sad film clip (which was similar to the crying babies film in eliciting sadness) as a measure of RSA suppression and found that RSA suppression had significantly positive associations with adaptive emotion regulation and fewer clinical depression symptoms. Again, the participants of Gentzler et al. (2009) were 5- to 13-year-old children. Hence, it seems that the relation between RSA reactivity and children's psychopathology is stronger among older children or young adolescents (although the measures might be different). However, because of the scarcity of the studies predicting early childhood psychopathology from the interaction between parenting and RSA reactivity, the above argument is still speculative.

24-month intercept versus 54-month intercept. Among the nine significant interactions for externalizing and internalizing symptoms, four predicted the 24-month intercepts (i.e., one for externalizing and three for internalizing), two predicted the 54-month intercepts (i.e., one for externalizing and the other one for internalizing), and three predicted the slopes (i.e., three for internalizing). It seems that the effects of 18-month

parenting quality, physiological RSA variables, and sex were more pronounced for the 24-month intercept than for the 54-month intercept. In addition, correlations between parenting quality and the parent-reported problem behaviors were larger in magnitude for earlier time points relative to later. This pattern might be explained by considering the three-year gap between 18 months and 54 months, as compared to the six-month gap between 18 months and 24 months. Three years between 18 and 54 months might be too long for the effect of parenting quality or physiological RSA variables to last, especially considering the potential change in parenting behaviors and children's increasing exposure to various influences outside of the families. Therefore, it makes sense that the predicted effects were larger for the outcomes measured at adjacent time points relative to time points more apart.

Limitations

To my knowledge, the current investigation is the first one to examine prediction of both externalizing and internalizing problems by the interaction of parenting quality with physiological RSA across early childhood. Testing simple effects and *RoS* in both directions produced a stronger test regarding the type of interaction pattern than merely testing the simple effects of the predictor (i.e., parenting quality). Examining the moderation by sex of the parenting quality × RSA interactions shed further light on how these interactions might differentially predict the problem behaviors of boys and girls. The differences between mother- and father-reported models might imply different characteristics of mother-child versus father-child interactions. Despite these strengths, several limitations of the current study should be acknowledged.

First, although several races and different ethnicities were included in this sample, the majority of participants were still non-Hispanic Caucasian, which restricted our ability of generalizing the current findings to other cultures. The homogeneity of this sample with regard to SES might not only limit the statistical power to detect significant effects, but also limit the generalizability of obtained effects to low SES families. In addition, these children were not from clinical sample, which means that their problem symptoms were still generally within the normative ranges for children between 24 to 54 months. Therefore, different patterns might emerge for children with clinically diagnosed behavioral problems, although the current findings, to some extent, should provide insight into the development of psychopathology among young children.

Second, although the use of multiple reporters reduced the reporter bias of perceived children's problem behaviors, there were still limitations in the measures of the current study. Maternal parenting quality was assessed solely by observation in the laboratory context, which is possibly the most accurate measure of maternal behaviors during early childhood. Yet, the observed measures could have been supplemented by the self-reported questionnaire data, although the validity and reliability of the self-reported data may still be problematic. In addition, because paternal parenting was not evaluated in the current study, I could not explore the potential differences between paternal parenting and maternal parenting in their relations with young children's physiological RSA variables and psychopathology. It is possible that a different pattern of results would be obtained with paternal parenting as a predictor, especially regarding the moderating role of child sex.

As briefly discussed earlier, RSA reactivity was measured by the film of crying babies in the current study, which probably was not as emotionally evocative or cognitively challenging as measures used in other RSA studies. It would be interesting to see whether the obtained patterns of parenting × RSA reactivity interactions would still be found with other measures of RSA reactivity in independent samples.

Children's externalizing and internalizing symptoms were measured by the ITSEA in the current study. Within each reporter, externalizing were the averaged scores of subscales of activity/impulsivity, defiance/aggression, and peer aggression, whereas internalizing were the averaged scores of subscales of separation distress, depression/withdrawal, and general anxiety. It is questionable whether these subscales have the same age-appropriate weight. For instance, one may question whether activity/impulsivity is a better measure than peer aggression for 24-month externalizing, and whether separation distress, rather than depression/withdrawal, is a better measure of internalizing at 24 months. In fact, this concern may be the general issue for longitudinal data in developmental research. On the one hand, researchers need to establish measurement invariance or homogeneity in order to examine the children's change over time. On the other hand, the same measures might be more valid for earlier time point than later time point, or vice versa, especially for longitudinal studies with 30 months apart like the current one. However, given that the ITSEA has been shown as a valid and reliable measure of children's problem behaviors across early childhood (Briggs-Gowan & Carter, 2007; Carter, Briggs-Gowan, Jones, & Little, 2003), the current measures could be considered as age appropriate.

The current investigation ran a large number of models, and some of the significant results (e.g., father-reported externalizing intercept at 24 months) might be due to chance. Replication using independent samples is desirable. Moreover, because I examined the externalizing and internalizing symptoms in different models, the research design did not take into account the possible co-occurrence of externalizing and internalizing behaviors during early childhood. Would the developmental trajectory of the co-occurring symptoms and the findings predicting these co-occurring symptoms be different from the pure externalizing or internalizing? In addition, considering the moderate prevalence of co-occurring symptoms in childhood (Oland & Shaw, 2005), it is possible that the obtained significant predictions of externalizing were through the pathways associated with internalizing, or vice versa. Perhaps a latent class growth analysis (or latent profile analysis for continuous variables) identifying distinct classes of individual trajectories for pure externalizing symptoms, pure internalizing symptoms, and co-occurring externalizing and internalizing symptoms would be a strategy to deal with the co-occurrence of the two behaviors.

Furthermore, the current study used physiological RSA variables at early age to predict the trajectories of children's problem behaviors across later time points. This did not consider the potential changes in children's RSA throughout the study period. Baseline RSA has been documented to be stable across 2 months to 5 years of age (e.g., Bornstein & Suess, 2000), whereas RSA reactivity (in the form of RSA suppression) decreased from 2 to 4 years of age (e.g., Calkins & Keane, 2004). Therefore, it is unclear if the relations between the physiological RSA variables, especially RSA reactivity, and children's problematic behaviors change as children age.

Future Directions

Future studies of children's physiological RSA variables should first strive to adopt standardized measures of RSA reactivity. As aforementioned, different measures might result in different findings in terms of the moderating function of RSA reactivity. Indeed, it would be difficult to interpret the physiological indices of vagal tone without accounting for the attention and emotion loading of the measures. Perhaps our understanding of RSA/vagal tone (i.e., PNS) would also be supplemented by assessing the SNS (i.e., sympathetic nervous system) in addition to PNS in the psychopathology research. Cardiac activity is believed to be influenced by the interactions between acceleratory SNS activation and deceleratory PNS activation (Beauchaine, 2001). When facing external challenges, competent coping behaviors require appropriate RSA suppression, which presumably promotes the readiness of the SNS to respond to the upcoming demands. Therefore, it might be more important to investigate the coordination in responses across the systems, rather than one system in isolation, in facilitating the adaptive engagement. Specific to the psychopathology study, it would be interesting to examine in the future whether different combinations of the PNS and SNS would have different associations with the problem behaviors.

Moreover, the current investigation detected a three-way interaction of sex × parenting quality × physiological RSA variables when predicting internalizing development in early childhood. However, the mechanisms underlying the moderation by sex were unclear. Are there sex differences in physiological functioning among children at this young age? If so, is the physiological difference an endophenotype of the possible genetic difference between males and females? Given that sex typically has been ignored

as a moderator of the relation of RSA to problem behaviors, prospective studies can benefit greatly from exploring the possible biological markers of the sex differences.

Finally, in order to obtain a fuller understanding of the childhood psychopathology, the explorations of typical and atypical development should be juxtaposed. The current study consistently identified low baseline RSA or high RSA reactivity as more responsive to the varying levels of parenting quality with respect to both externalizing and internalizing development; thus, it appears that low baseline RSA or high RSA reactivity is a nonspecific marker of maladaptive regulation. However, would similar results be detected in children with clinically diagnosed disorders? To answer this question, perhaps direct comparisons between typically developing children with symptoms and clinical samples with externalizing or internalizing disorders are desirable in future investigations.

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Table 1

Zero-order Correlations among the 18-month Observed Parenting Tasks

		1	2	3	4	5	6
1	Free play-Sensitivity	–					
2	Free play-Intrusiveness	-.41***	–				
3	Teaching-Sensitivity	.20**	-.23**	–			
4	Teaching-Warmth	.26**	-.11†	.45***	–		
5	Teaching-authoritativeness	.16*	-.19**	.70***	.58***	–	
6	Teaching-Intrusiveness (transformed)	-.18**	.21**	-.75***	-.16*	-.36***	–

Note: Free play-Sensitivity indicates maternal sensitivity during free play task.
*** $p < .001$. ** $p < .01$, * $p < .05$, † $p < .10$

Table 2

Descriptive Statistics and Zero-order Correlations for Key Variables (Presented for the Whole Sample)

		1	2	3	4	5	6	7	8	9	10	11	12	13
	Mean	1.96	.00	.01	1.45	1.45	1.43	1.36	1.37	1.45	1.45	1.43	1.40	1.38
	SD	1.43	1.43	.65	.17	.17	.20	.19	.19	.16	.17	.20	.17	.18
	N (INT)	–	–	–	154	185	169	147	155	118	133	109	95	96
1	Baseline RSA	–	.00	.04	.07	.10	.03	.04	-.00	.09	.16†	.01	.12	.16
2	RSA reactivity	.00	–	.11	-.15	-.05	-.10	-.06	-.05	-.09	-.60	-.02	.05	-.09
3	Parenting quality	.04	.11	–	-.02	-.02	-.17	-.21*	-.20*	-.01	-.08	-.09	-.04	-.03
4	Mother 24m	.07	.02	-.21**	–	.67**	.59**	.55**	.56**	.37**	.37**	.31**	.25*	.33**
5	Mother 30m	.02	-.16*	-.30**	.76**	–	.58**	.52**	.54**	.30**	.41**	.36**	.30**	.34**
6	Mother 42m	-.09	-.06	-.32**	.72**	.70**	–	.70**	.63**	.25*	.41**	.42**	.30**	.30**
7	Mother 48m	-.04	-.00	-.38**	.63**	.62**	.76**	–	.69**	.34**	.35**	.29**	.39**	.27*
8	Mother 54m	-.04	-.09	-.18*	.69**	.67**	.70**	.75**	–	.28**	.31**	.21*	.27*	.36**
9	Father 24m	.03	.06	-.17†	.48**	.41**	.24*	.26**	.35**	–	.57**	.44**	.37**	.40**
10	Father 30m	.04	-.07	-.26**	.51**	.44**	.44**	.42**	.46**	.65**	–	.47**	.61**	.58**
11	Father 42m	-.14	-.04	-.28**	.47**	.39**	.46**	.43**	.42**	.57**	.57**	–	.51**	.54**
12	Father 48m	-.10	.02	-.19†	.47**	.28**	.51**	.48**	.47**	.51**	.56**	.66**	–	.63**
13	Father 54m	-.09	-.05	-.16	.27*	.32**	39**	.42**	.51**	.48**	.57**	.65**	.70**	–
	N (EXT)	–	–	–	154	185	169	147	155	118	134	108	95	96
	Mean	1.96	.00	.01	1.52	1.57	1.58	1.47	1.52	1.51	1.52	1.51	1.45	1.46
	SD	1.43	1.43	.65	.27	.28	.28	.25	.27	.24	.24	.24	.21	.21

Note: Mother 24m = mother-reported behavioral symptoms (i.e., externalizing and internalizing) at 24 months. *N* (INT) and *N* (EXT) = *N*s for internalizing and externalizing. Correlations in the lower triangle below the main diagonal are for externalizing symptoms, and upper triangle above the main diagonal are for internalizing symptoms. ** $p < .01$ or less, * $p < .05$, † $p < .10$

Table 3

Descriptive Statistics and Zero-order Correlations for Key Variables (Separately for Boys' and Girls' Externalizing Symptoms)

		1	2	3	4	5	6	7	8	9	10	11	12	13
	Mean	2.06	-.07	-.09	1.54	1.59	1.61	1.47	1.53	1.54	1.55	1.50	1.44	1.49
	SD	1.67	1.65	.61	.30	.30	.28	.27	.30	.26	.25	.23	.21	.21
1	Baseline RSA	–	-.08	.09	.03	-.04	-.16	-.09	-.06	.08	.01	-.29*	-.22	-.17
2	RSA reactivity	.23*	–	.16†	.04	-.16	-.03	-.01	-.07	.06	-.08	-.04	.15	-.07
3	Parenting quality	-.04	.03	–	-.13	-.23*	-.23*	-.27*	-.13	-.19	-.14	-.25†	-.15	-.14
4	Mother 24-EXT	.13	-.05	-.32**	–	.80**	.73**	.70**	.75**	.51**	.51**	.57**	.50**	.28†
5	Mother 30-EXT	.12	-.16	-.39**	.70**	–	.73**	.66**	.70**	.35**	.36**	.40**	.28*	.23
6	Mother 42-EXT	.03	-.12	-.40**	.70**	.64**	–	.77**	.71**	.26†	.42**	.52**	.52**	.40**
7	Mother 48-EXT	.09	.01	-.50**	.51**	.57**	.76**	–	.79**	.22	.50**	.48**	.50**	.44**
8	Mother 54-EXT	-.01	-.11	-.26*	.51**	.63**	.68**	.69**	–	.38**	.50**	.51**	.47**	.44**
9	Father 24-EXT	-.01	.05	-.11	.45**	.48**	.23	.32*	.28†	–	.77**	.52**	.63**	.45**
10	Father 30-EXT	.07	-.04	-.39**	.53**	.54**	.46**	.35*	.40**	.52**	–	.67**	.60**	.57**
11	Father 42-EXT	.06	-.06	-.32*	.37*	.29*	.41**	.37*	.34*	.63**	.50**	–	.70**	.71**
12	Father 48-EXT	.10	-.28†	-.27†	.42**	.29	.52**	.46**	.47**	.39*	.50**	.62**	–	.71**
13	Father 54-EXT	.04	-.03	-.09	.28†	.41**	.38	.43**	.62**	.49**	.51**	.61**	.70**	–
	Mean	1.84	.09	.01	1.49	1.54	1.53	1.46	1.49	1.48	1.47	1.51	1.47	1.41
	SD	1.09	1.10	.70	.22	.26	.28	.24	.24	.24	.22	.25	.20	.21

Note: EXT = externalizing, Mother 24-EXT = mother-reported externalizing symptoms at 24 months. Correlations shown in the lower triangle under the main diagonal are for girls, and higher triangle above the main diagonal are for boys. Mean and *SD* are for the whole subsample. ** $p < .01$ or less, * $p < .05$, † $p < .10$

Table 4

Descriptive Statistics and Zero-order Correlations for Key Variables (Separately for Boys' and Girls' Internalizing Symptoms)

		1	2	3	4	5	6	7	8	9	10	11	12	13
	Mean	2.06	-.07	-.09	1.45	1.45	1.42	1.36	1.34	1.46	1.44	1.43	1.41	1.36
	SD	1.67	1.65	.61	.18	.18	.20	.18	.18	.18	.18	.17	.18	.24
1	Baseline RSA	–	-.08	.09	.17	.18†	.10	.12	.08	.19	.23*	.08	.04	.21
2	RSA reactivity	.23*	–	.16	-.18	-.12	-.13	-.06	-.06	-.14	-.09	-.07	.04	-.20
3	Parenting quality	-.04	.03	–	-.06	-.02†	-.20†	-.23*	-.28**	-.05	-.11	-.04	-.00	-.10
4	Mother 24-INT	-.11	-.11	.02	–	.66**	.62**	.58**	.67**	.45**	.24†	.21	.20	.41**
5	Mother 30-INT	-.06	.06	-.03	.68**	–	.58**	.52**	.60**	.26*	.37**	.33*	.30*	.39**
6	Mother 42-INT	-.08	-.07	-.15	.57**	.58**	–	.63**	.62**	.26†	.40**	.23†	.33*	.35*
7	Mother 48-INT	-.09	-.07	-.19	.52**	.54**	.77**	–	.68**	.40**	.28*	.30*	.46**	.30*
8	Mother 54-INT	-.14	-.05	-.13	.42**	.47**	.64**	.69**	–	.41**	.33*	.15	.24†	.38**
9	Father 24-INT	-.05	.00	.09	.32*	.36**	.25†	.27†	.10	–	.49**	.33*	.36*	.39**
10	Father 30-INT	.05	-.01	-.07	.51**	.46**	.42**	.43**	.29*	.68**	–	.43**	.58**	.59**
11	Father 42-INT	-.05	.03	-.16	.38*	.40**	.57**	.28†	.25†	.54**	.51**	–	.59**	.66**
12	Father 48-INT	.26†	.10	-.08	.34*	.30†	.27†	.32*	.30†	.38*	.63**	.47**	–	.62**
13	Father 54-INT	.09	.12	-.02	.24	.28†	.24	.23	.33*	.42**	.58**	.45**	.68**	–
	Mean	1.84	.09	.01	1.46	1.46	1.44	1.37	1.40	1.43	1.47	1.44	1.40	1.40
	SD	1.09	1.10	.70	.17	.17	.20	.20	.20	.14	.16	.23	.15	.19

Note: INT = internalizing, Mother 24-INT = mother-reported internalizing symptoms at 24 months. Correlations shown in the lower triangle under the main diagonal are for girls, and higher triangle above the main diagonal are for boys. Mean and *SD* are for the whole subsample. ** $p < .01$ or less, * $p < .05$, † $p < .10$

Table 5

Satorra-Bentler Scaled Chi-square Different Tests Comparing Unconditional Models

Model	Outcome	Overall fit for model with intercept and linear factors	Overall fit for model with quadratic mean factor	Satorra-Bentler scaled chi-square test
1	Mother-reported Externalizing	$\chi^2_{(10)} = 46.67$, $p < .01$; (CFI = .91)[3]; RMSEA = .14; SRMR = .08	$\chi^2_{(9)} = 36.82$, $p < .01$; (CFI = .93); RMSEA = .13; SRMR = .09[2]	$\chi^2_{diff(1)} = 11.38$, $p = .001$
2	Father-reported Externalizing	$\chi^2_{(10)} = 6.74$, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .06	$\chi^2_{(9)} = 5.07$, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .07	$\chi^2_{diff(1)} = 1.85$, *ns*
3	Mother-reported Internalizing	$\chi^2_{(10)} = 25.43$, $p < .01$; (CFI = .96); RMSEA = .08; SRMR = .08	$\chi^2_{(9)} = 22.75$, $p < .01$; (CFI = .96); RMSEA = .08; SRMR = .09	$\chi^2_{diff(1)} = 2.72$, *ns*
4	Father-reported Internalizing	$\chi^2_{(10)} = 10.68$, *ns*; (CFI = 1.00); RMSEA = .02; SRMR = .09	$\chi^2_{(9)} = 8.21$, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .12	$\chi^2_{diff(1)} = 3.10$, *ns*

Note: [1.]Means and variances of the intercept and linear slope factors are all significantly different from zero at both 24 and 54 months. [2.]Overall model fit indices were the same for unconditional models with intercept centered at 24 and 54 months. [3.]The values of the CFI were presented in parentheses, indicating the inaccurate CFI values automatically reported by M*plus* 7.0.

Table 6

Unconditional Models of Mother- and Father-reported Problem Symptoms

	Mother-reported EXT			Father-report EXT[1]			Mother-reported INT			Father-reported INT		
Fixed effects	*b*	*t*	*p*	*b*	*t*	*p*	*b*	*t*	*p*	*b*	*t*	*p*
Intercept (24m)	1.54			1.52			1.47			1.50		
Intercept (54m)	1.49			1.46			1.36			1.39		
Linear slope (24m)	.027	2.36	*	-.012	-2.87	**	-.021	-7.70	***	-.014	-4.33	***
Linear slope (54m)[2]	-.038	-3.55	***									
Quadratic slope	-.011	-3.27	**									
Random effects	μ	*t*	*p*	μ	*t*	*p*	μ	*t*	*p*	μ	*t*	*p*
Intercept (24m)	.059	7.51	***	.040	6.02	***	.020	5.79	***	.016	4.61	***
Intercept (54m)	.056	7.68	***	.035	6.33	***	.026	6.99	***	.022	5.45	***
Linear slope (24m)	.001	3.35	***	.001	2.32	*	.001	2.75	**	.001	2.13	*
Linear slope (54m)	.001	2.94	**									
Covariance[3]	-.002	-2.08	*	-.002	-1.96	*	-.001	-.94		.000	-.63	

Note: [1.]EXT = externalizing, whereas INT = internalizing; [2.]Linear slope (54m) = 54-month linear slope (only applicable to mother-reported externalizing) [3.]covariances between intercept and 24-month linear slope; *** $p < .001$, ** $p < .01$, * $p < .05$

Table 7a

Overall Model Fit Indices of Mother- and Father-reported Externalizing Symptoms (Baseline RSA and RSA reactivity)

Main effect models	Moderator	Outcome	Overall model fit indices
1	baseline RSA	Mother report	$\chi^2_{(15)}$ = 47.40, p < .01; (CFI = .94); RMSEA = .10; SRMR = .06
2	baseline RSA	Father report	$\chi^2_{(19)}$ = 19.68, *ns*; (CFI = 1.00); RMSEA = .02; SRMR = .05
3	RSA reactivity	Mother report	$\chi^2_{(15)}$ = 57.94, p < .01; (CFI = .92); RMSEA = .12; SRMR = .06
4	RSA reactivity	Father report	$\chi^2_{(19)}$ = 21.79, *ns*; (CFI = .99); RMSEA = .03; SRMR = .05
Two-way interaction models			
5	baseline RSA	Mother report	$\chi^2_{(15)}$ = 41.55, p < .01; (CFI = .95); RMSEA = .09; SRMR = .06
6	baseline RSA	Father report	$\chi^2_{(19)}$ = 14.02, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .04
7	RSA reactivity	Mother report	$\chi^2_{(15)}$ = 54.10, p < .01; (CFI = .92); RMSEA = .12; SRMR = .06
8	RSA reactivity	Father report	$\chi^2_{(19)}$ = 16.84, *ns*; (CFI = 1.00); RMSEA = .02; SRMR = .04
Three-way interaction models			
9	baseline RSA	Mother report	$\chi^2_{(23)}$ = 56.67, p < .01; (CFI = .94); RMSEA = .09; SRMR = .04
10	baseline RSA	Father report	$\chi^2_{(31)}$ = 30.91, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .04
11	RSA reactivity	Mother report	$\chi^2_{(23)}$ = 73.17, p < .01; (CFI = .91); RMSEA = .11; SRMR = .05
12	RSA reactivity	Father report	$\chi^2_{(31)}$ = 36.09, *ns*; (CFI = .98); RMSEA = .03; SRMR = .04

Note: Overall fit indices are the same for models with intercept centered at 24 and 54 months within each reporter; the values of the CFI were presented in parentheses, indicating the inaccurate CFI values automatically reported by M*plus* 7.0.

Table 7b

Overall Model Fit Indices of Mother- and Father-reported Internalizing Symptoms (Baseline RSA and RSA reactivity)

Main effect models	Moderator	Outcome	Overall model fit indices
1	baseline RSA	Mother report	$\chi^2_{(19)}$ = 33.09, $p < .05$; (CFI = .96); RMSEA = .06; SRMR = .06
2	baseline RSA	Father report	$\chi^2_{(19)}$ = 19.04, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .08
3	RSA reactivity	Mother report	$\chi^2_{(19)}$ = 32.09, $p < .05$; (CFI = .97); RMSEA = .06; SRMR = .06
4	RSA reactivity	Father report	$\chi^2_{(19)}$ = 19.17, *ns*; (CFI = 1.00); RMSEA = .02; SRMR = .09
Two-way interaction models			
5	baseline RSA	Mother report	$\chi^2_{(19)}$ = 35.71, $p < .05$; (CFI = .96); RMSEA = .06; SRMR = .05
6	baseline RSA	Father report	$\chi^2_{(19)}$ = 30.81, *ns*; (CFI = .93); RMSEA = .06; SRMR = .07
7	RSA reactivity	Mother report	$\chi^2_{(19)}$ = 40.24, $p < .01$; (CFI = .95); RMSEA = .07; SRMR = .06
8	RSA reactivity	Father report	$\chi^2_{(19)}$ = 17.88, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .07
Three-way interaction models			
9	baseline RSA	Mother report	$\chi^2_{(31)}$ = 42.95, *ns*; (CFI = .97); RMSEA = .04; SRMR = .04
10	baseline RSA	Father report	$\chi^2_{(31)}$ = 49.04, *ns*; (CFI = .95); RMSEA = .06; SRMR = .07
11	RSA reactivity	Mother report	$\chi^2_{(31)}$ = 52.49, $p = .01$; (CFI = .95); RMSEA = .06; SRMR = .04
12	RSA reactivity	Father report	$\chi^2_{(31)}$ = 30.77, *ns*; (CFI = 1.00); RMSEA = .00; SRMR = .06

Note: Overall fit indices are the same for models with intercept centered at 24 and 54 months within each reporter; the values of the CFI were presented in parentheses, indicating the inaccurate CFI values automatically reported by M*plus* 7.0.

Table 8a

Main Effect Models of Mother- and Father-reported Externalizing Symptoms (Baseline RSA and RSA reactivity)

Fixed effects	Mother 24month[1]		Mother 54month		Mother 24m linear slope[2]		Mother 54m linear slope		Mother quadratic slope	
	b	*t*	*b*	*t*	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.58		1.52		.04		-.07		-.01	
Sex	-.031	-.84	-.015	-.38	-.007	-.29	.014	.59	.002	.47
Parenting quality	***-.097***	-3.19	***-.098***	-3.22	**-.039**	-2.27	**.038**	2.17	**.008**	2.39
Baseline RSA	.010	.91	-.008	-.64	*-.010*	-1.78	.003	.47	.001	1.15
Residual variances	***.052***	7.60	***.049***	7.77	***.001***	3.14	***.001***	3.14		
Intercept (model)	1.58		1.51		.04		-.06		-.010	
Sex	-.034	-.91	-.013	-.35	-.004	-.19	.012	.54	.002	.38
Parenting quality	***-.096***	-3.15	***-.098***	-3.18	**-.038**	-2.35	**.037**	2.14	**.008**	2.39
RSA reactivity	.002	.13	.000	-.01	-.006	-.79	.005	.68	.001	.77
Residual variances	***.053***	7.63	***.050***	7.99	***.001***	3.40	***.001***	3.40		

Fixed effects	Father 24month		Father 54month		Father linear slope	
	b	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.59		1.52		-.015	
Sex	-.043	-1.12	-.033	-.88	.002	.23
Parenting quality	***-.088***	-2.86	**-.058**	-1.96	.006	.84
Baseline RSA	.004	.40	-.014	-1.26	-.004	-1.45
Residual variances	***.036***	5.67	***.033***	6.74	**.001**	2.21
Intercept (model)	1.59		1.52		-.015	
Sex	-.042	-1.11	-.034	-.89	.002	.21
Parenting quality	***-.088***	-2.85	**-.062**	-2.08	.005	.71

RSA reactivity	.003	.30	.006	.59	.001	.33
Residual variances	***.036***	5.67	***.033***	6.77	**.001**	2.21

Note: [1.]Mother 24month = mother-reported 24-month intercept; [2.]Mother 24m linear slope = mother-reported 24-month linear slope; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$.

Table 8b

Main Effect Models of Mother- and Father-reported Internalizing Symptoms (Baseline RSA and RSA reactivity)

Fixed effects	Mother 24month[1]		Mother 54month		Mother linear slope	
	b	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.43		1.30		-.03	
Sex	.025	1.06	.040	1.48	.003	.57
Parenting quality	-.013	-.64	***-.070***	-3.16	***-.011***	-2.72
Baseline RSA	.012	1.58	.004	.38	-.001	-.82
Residual variances	***.019***	5.94	***.024***	7.11	**.001**	2.57
Intercept (model)	1.43		1.31		-.025	
Sex	.023	1.00	.039	1.44	.003	.60
Parenting quality	-.008	-.41	***-.069***	-3.08	***-.012***	-2.83
RSA reactivity	-.013	-1.15	.000	.025	.003	1.53
Residual variances	***.019***	6.27	***.024***	7.11	**.001**	2.47
Fixed effects	Father 24month		Father 54month		Father linear slope	
	b	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.48		1.34		-.03	
Sex	-.011	-.44	.032	.99	.009	1.24
Parenting quality	-.010	-.46	-.038	-1.42	-.005	-.93
Baseline RSA	*.015*	1.73	.020	1.60	.001	.27
Residual variances	***.015***	4.50	***.021***	5.38	**.001**	2.18
Intercept (model)	1.48		1.35		-.03	
Sex	-.013	-.50	.031	.96	.009	1.26
Parenting quality	-.006	-.25	-.034	-1.23	-.006	-.95
RSA reactivity	-.010	-1.24	-.001	-.12	.002	.69
Residual variances	***.016***	4.58	***.022***	5.32	**.001**	2.08

Note: [1.]Mother 24month = mother-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$.

Table 9a

Two-way Interaction Models Predicting Mother-reported Externalizing Symptoms from Parenting Quality and Baseline RSA or RSA reactivity

Two-way models Mother-reported Externalizing	Mother-reported 24month[1]		Mother-reported 54month		Mother-reported 24m linear slope[2]	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.54		1.50		.29	
Baseline RSA	.013	1.16	-.008	-.59	**.011**	-2.26
Parenting quality	***-.100***	-3.34	-.101	-3.28	**-.039**	-2.30
Baseline RSA * Parenting quality	-.027	-1.04	.001	.041	.014	.88
Residual variances	***.052***	7.54	***.050***	7.60	***.001***	3.01
Intercept (model)	1.53		1.50		.03	
RSA reactivity	.002	.084	.000	.00	-.005	-.64
Parenting quality	***-.097***	-3.17	***-.10***	-3.18	**-.039**	-2.46
RSA reactivity*Parenting quality	.001	.036	-.004	-.12	-.008	-.63
Residual variances	***.054***	7.50	***.050***	7.87	***.001***	3.46

Two-way models Mother-reported Externalizing	Mother-reported 54m linear slope		Mother-reported quadratic slope	
Fixed effects	*b*	*t*	*b*	*t*
Intercept (model)	-.045		-.007	
Baseline RSA	.003	.45	.001	1.30
Parenting quality	**.038**	2.18	**.008**	2.37
Baseline RSA * Parenting quality	-.002	-.15	-.002	-.53
Residual variances	***.001***	3.01		
Intercept (model)	-.045		-.008	
RSA reactivity	.004	.54	.001	.61
Parenting quality	**.038**	2.20	**.008**	2.48
RSA reactivity*Parenting quality	.006	.52	.001	.61
Residual variances	***.001***	3.46		

Note: [1.]Mother-reported 24month = mother-reported 24-month intercept; [2.]Mother-reported 24m linear slope = mother-reported 24-month linear slope; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$.

Table 9b

Three-way Interaction Models Predicting Mother-reported Externalizing Symptoms from Parenting Quality, Baseline RSA or RSA reactivity, and Sex

Three-way models Mother-reported Externalizing	Mother-reported 24month[1]		Mother-reported 54month		Mother-reported 24m linear slope[2]	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.55		1.51		.03	
Baseline RSA	.009	.51	.000	-.02	**-.018**	-2.47
Parenting quality	-.072	-1.27	-.085	-1.61	-.039	-1.51
Baseline RSA * Parenting quality	-.013	-.21	-.069	-1.09	.017	.76
Sex	-.031	-.82	-.016	-.42	-.006	-.27
Sex * Parenting quality	-.046	-.72	-.031	-.48	-.002	-.05
Sex * Baseline RSA	.006	.25	-.003	-.13	*.026*	1.77
Sex * Baseline * Parenting quality	-.018	-.28	.113	1.67	-.003	-.10
Residual variances	***.052***	7.35	***.048***	7.74	***.001***	2.65
Intercept (model)	1.55		1.51		.03	

RSA reactivity	.006	.22	.004	.06	-.005	-.53
Parenting quality	-.069	-1.22	-.06	-.50	-.042	-1.52
RSA reactivity * Parenting quality	.020	.40	-.001	-.011	-.007	-.42
Sex	-.027	.72	-.014	-.35	-.005	-.22
Sex * Parenting quality	-.050	-.79	-.029	-.46	.004	.13
Sex * RSA reactivity	-.022	-.70	-.002	-.07	.003	.15
Sex * Reactivity * Parenting quality	-.049	-.09	-.003	-.04	-.002	-.07
Residual variances	***.052***	7.46	***.050***	7.96	***.001***	3.28

Three-way models Mother-reported Externalizing	Mother-reported 54m linear slope		Mother-reported quadratic slope	
Fixed effects	*b*	*t*	*b*	*t*
Intercept (model)	-.05		-.01	
Baseline RSA	*.014*	1.75	**.003**	2.13
Parenting quality	.034	1.16	.007	1.39
Baseline RSA * Parenting quality	*-.040*	-1.72	-.006	-1.28
Sex	.012	.52	.002	.41

Sex * Parenting quality	.007	.22	.001	.15
Sex * Baseline RSA	-*.030*	-1.67	-*.006*	-1.83
Sex * Baseline * Parenting quality	*.055*	1.81	.006	1.04
Residual variances	***.001***	2.65		
Intercept (model)	-.05		-.01	
RSA reactivity	-.002	-.06	.001	.43
Parenting quality	.032	.51	.008	1.43
RSA reactivity * Parenting quality	-.023	-.52	.001	.14
Sex	.010	.44	.001	.35
Sex * Parenting quality	.004	.12	.000	.00
Sex * RSA reactivity	.005	.30	.000	.08
Sex * Reactivity * Parenting quality	.021	.77	.002	.43
Residual variances	***.001***	3.28		

Note: [1.]Mother-reported 24month = mother-reported 24-month intercept; [2.]Mother-reported 24m linear slope = mother-reported 24-month linear slope; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$. Refer to the main effects models for the main effects of parenting quality, and baseline RSA or RSA reactivity; and refer to the two-way interaction models for the effects of parenting quality × baseline RSA or RSA reactivity interaction.

Table 10a

Two-way Interaction Models Predicting Father-reported Externalizing Symptoms from Parenting Quality and Baseline RSA or RSA reactivity

Two-way models Father-reported Externalizing	Father-reported 24month[1]		Father-reported 54month		Father-reported linear slope	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.53		1.47		-.013	
Baseline RSA	.007	.70	-.017	-1.43	-.005	-1.67
Parenting quality	***-.093***	-3.26	**-.062**	-2.16	.006	.97
Baseline RSA * Parenting quality	-.020	-.92	.014	.61	.007	1.11
Residual variances	***.036***	5.48	***.033***	6.61	**.001**	2.12
Intercept (model)	1.53		1.47		-.013	
RSA reactivity	.012	1.07	.006	.47	-.001	-.55
Parenting quality	***-.092***	-3.20	**-.066**	-2.29	**.008**	2.28
RSA reactivity * Parenting quality	***-.049***	-2.84	-.008	-.47	.005	.75
Residual variances	***.035***	5.40	***.033***	6.62	**.001**	2.00

Note: [1.]Father-reported 24month = father-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less, in **bold** are significant at $p < .05$, in *italic* are significant at $p < .10$; values in shade indicate significant interaction.

Table 10b

Three-way Interaction Models Predicting Father-reported Externalizing Symptoms from Parenting Quality, Baseline RSA or RSA reactivity, and Sex

Three-way models	Father-reported		Father-reported		Father-reported	
Father-reported Externalizing	24month[1]		54month		linear slope	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.54		1.48		-.01	
Baseline RSA	.001	.09	***-.034***	-3.15	**-.007**	-2.26
Parenting quality	-.077	-1.78	-.039	-.98	.007	.94
Baseline RSA * Parenting quality	.032	.71	.005	.15	-.005	-.56
Sex	-.042	-1.15	-.024	-.63	.004	.47
Sex * Parenting quality	.000	-.00	-.057	-.94	-.011	-.83
Sex * Baseline RSA	-.001	-.04	**.047**	2.05	.010	1.62
Sex * Baseline * Parenting quality	-.079	-1.53	.021	.49	*.020*	1.68
Residual variances	***.036***	5.71	***.031***	6.06	*.001*	1.93
Intercept (model)	1.56		1.51		-.01	
RSA reactivity	.015	1.14	.021	.45	-.001	-.56

Parenting quality	**-.094**	-2.30	-.014	-.13	.008	.99
RSA reactivity * Parenting quality	**-.048**	-2.05	.006	.09	.008	1.84
Sex	-.054	-1.41	-.038	-.80	.004	.49
Sex * Parenting quality	.018	.31	-.028	-.36	-.008	-.51
Sex * RSA reactivity	-.010	-.40	-.016	-.43	-.001	-.18
Sex* Reactivity* Parenting quality	-.016	-.44	.000	.00	.002	.19
Residual variances	***.034***	5.56	***.039***	5.29	**.001**	2.00

Note: [1.]Father-reported 24month = father-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$. Values in shade indicate significant interaction. Refer to the main effects models for the main effects of parenting quality, and baseline RSA or RSA reactivity; and refer to the two-way interaction models for the effects of parenting quality × baseline RSA or RSA reactivity interaction.

Table 11a

Two-way Interaction Models Predicting Mother-reported Internalizing Symptoms from Parenting Quality and Baseline RSA or RSA reactivity

Two-way models Mother-reported Internalizing	Mother-reported 24month[1]		Mother-reported 54month		Mother-reported linear slope	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.47		1.36		-.021	
Baseline RSA	.012	1.53	.002	.15	-.002	-1.22
Parenting quality	-.012	-.63	***-.069***	-3.17	***-.011***	-2.89
Baseline RSA * Parenting quality	-.014	-.82	.017	.90	**.006**	2.08
Residual variances	***.019***	6.09	***.024***	6.98	**.000**	2.47
Intercept (model)	1.47		1.36		-.021	
RSA reactivity	-.012	-.94	.002	.17	.003	1.51
Parenting quality	-.008	-.39	***-.068***	-3.05	***-.012***	-2.80
RSA reactivity * Parenting quality	-.005	-.029	-.009	-.50	-.001	-.027
Residual variances	***.019***	6.18	***.025***	7.04	**.000**	2.47

Note: [1.]Mother-reported 24month = mother-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less, in **bold** are significant at $p < .05$, in *italic* are significant at $p < .10$; values in shade indicate significant interaction.

Table 11b

Three-way Interaction Models Predicting Mother-reported Internalizing Symptoms from Parenting Quality, Baseline RSA or RSA reactivity, and Sex

Three-way models Mother-reported Internalizing	Mother-reported 24month		Mother-reported 54month		Mother-reported linear slope	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.46		1.35		-.02	
Baseline RSA	**.023**	2.30	.012	.99	-.002	-1.00
Parenting quality	-.026	-.87	**-.077**	-2.49	**-.010**	-2.31
Baseline RSA * Parenting quality	-.039	-1.24	-.027	-1.06	.002	.55
Sex	.020	.85	.037	1.39	.003	.65
Sex * Parenting quality	.018	.46	.005	.13	-.003	-.33
Sex * Baseline RSA	**-.035**	-2.04	-.023	-1.05	.002	.60
Sex * Baseline * Parenting quality	.040	1.12	**.072**	2.18	.007	1.11
Residual variances	***.019***	6.61	***.023***	6.95	**.001**	2.47
Intercept (model)	1.46		1.31		-.02	
RSA reactivity	-.016	-.88	.017	.51	**.004**	2.37

Parenting quality	-.007	-.24	-.073	-1.09	***-.013***	-2.76
RSA reactivity * Parenting quality	-.005	-.18	-.072	-1.35	-.005	-1.54
Sex	.022	.94	.035	1.27	.003	.49
Sex * Parenting quality	-.003	-.07	.002	.05	.001	.12
Sex * RSA reactivity	.012	.52	-.011	-.49	-.005	-1.34
Sex* Reactivity* Parenting quality	.004	.11	.044	1.33	**.008**	1.99
Residual variances	***.019***	6.30	***.024***	6.88	**.001**	2.29

Note: [1.]Mother-reported 24month = mother-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$. Values in shade indicate significant interaction. Refer to the main effects models for the main effects of parenting quality, and baseline RSA or RSA reactivity; and refer to the two-way interaction models for the effects of parenting quality × baseline RSA or RSA reactivity interaction.

Table 12a

Two-way Interaction Models Predicting Father-reported Internalizing Symptoms from Parenting Quality and Baseline RSA or RSA reactivity

Two-way models Father-reported Internalizing	Father-reported 24month[1]		Father-reported 54month		Father-reported linear slope	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.46		1.39		-.014	
Baseline RSA	*.015*	1.65	*.023*	1.69	.001	.39
Parenting quality	-.013	-.60	-.031	-1.29	-.004	-.68
Baseline RSA * Parenting quality	-.004	-.30	-.019	-.76	-.003	-.50
Residual variances	***.015***	4.53	***.021***	5.46	**.000**	2.11
Intercept (model)	1.47		1.39		-.014	
RSA reactivity	-.005	-.61	.004	.40	.002	.60
Parenting quality	-.007	-.34	-.027	-1.06	***-.004***	-.70
RSA reactivity * Parenting quality	**-.027**	-2.02	-.025	-1.30	.000	.07
Residual variances	***.015***	4.52	***.021***	5.48	**.000**	2.08

Note: [1.]Father-reported 24month = father-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less, in **bold** are significant at $p < .05$, in *italic* are significant at $p < .10$; values in shade indicate significant interaction.

Table 12b

Three-way Interaction Models Predicting Father-reported Internalizing Symptoms from Parenting Quality, Baseline RSA or RSA reactivity, and Sex

Three-way models Father-reported Internalizing	Father-reported 24month[1]		Father-reported 54month		Father-reported linear slope	
Fixed effects	*b*	*t*	*b*	*t*	*b*	*t*
Intercept (model)	1.47		1.38		-.02	
Baseline RSA	***.030***	3.55	.018	1.21	-.002	-.68
Parenting quality	-.026	-.73	-.030	-.82	-.001	-.09
Baseline RSA * Parenting quality	-.004	-.15	-.004	-.97	-.007	-.72
Sex	-.014	-.55	.031	.94	.009	1.23
Sex * Parenting quality	.031	.68	-.032	-.57	-.013	-.93
Sex * Baseline RSA	**-.040**	-2.55	.018	.74	**.012**	2.23
Sex* Baseline * Parenting quality	-.010	-.30	.034	.66	.009	.72
Residual variances	***.015***	4.43	***.021***	5.24	.001	1.42
Intercept (model)	1.47		1.38		-.02	
RSA reactivity	-.009	-.81	.000	-.04	.002	.47

Parenting quality	-.014	-.42	-.018	.51	-.016	-.09
RSA reactivity * Parenting quality	-.016	-.79	-.030	-1.24	-.003	-.43
Sex	-.021	-.84	.036	1.04	.011	1.52
Sex * Parenting quality	.032	.74	-.049	-.82	-.016	-1.21
Sex * RSA reactivity	.014	.82	.015	.53	.000	.06
Sex* Reactivity* Parenting quality	-.033	-1.31	.030	.67	.013	1.32
Residual variances	***.015***	4.56	***.021***	5.62	**.001**	2.03

Note: [1.]Father-reported 24month = father-reported 24-month intercept; Values in ***italic bold*** are significant at $p < .01$ or less; values in **bold** are significant at $p < .05$; values in *italic* are significant at $p < .10$. Values in shade indicate significant interaction. Refer to the main effects models for the main effects of parenting quality, and baseline RSA or RSA reactivity; and refer to the two-way interaction models for the effects of parenting quality × baseline RSA or RSA reactivity interaction.

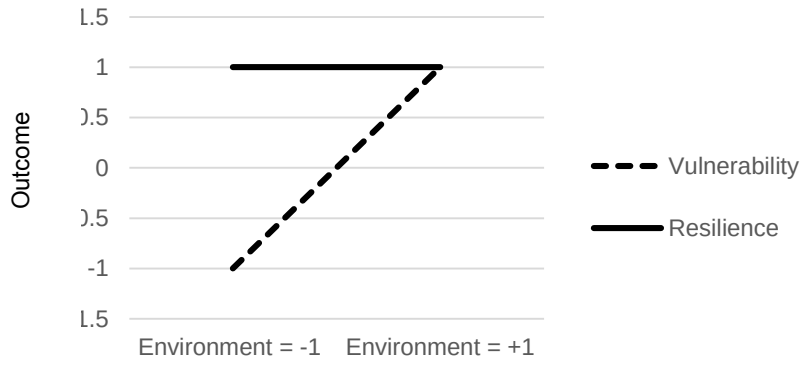

Figure 1a. Graphical Illustration of the Diathesis-Stress Model

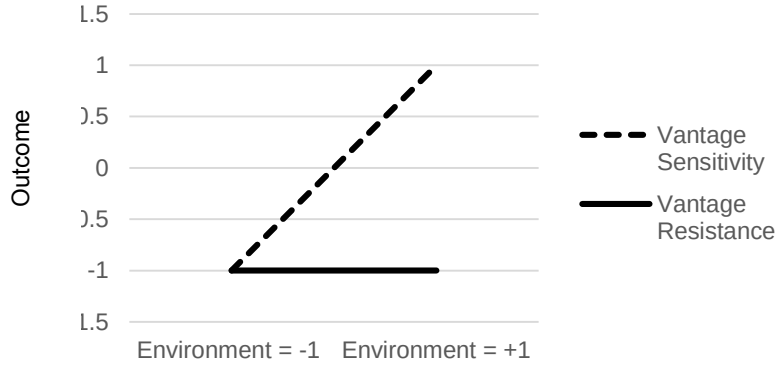

Figure 1b. Graphical Illustration of the Vantage Sensitivity Model

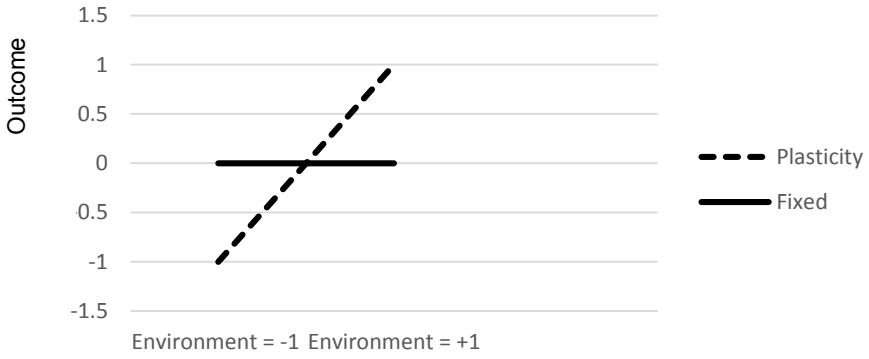

Figure 1c. Graphical Illustration of the Differential Susceptibility Model

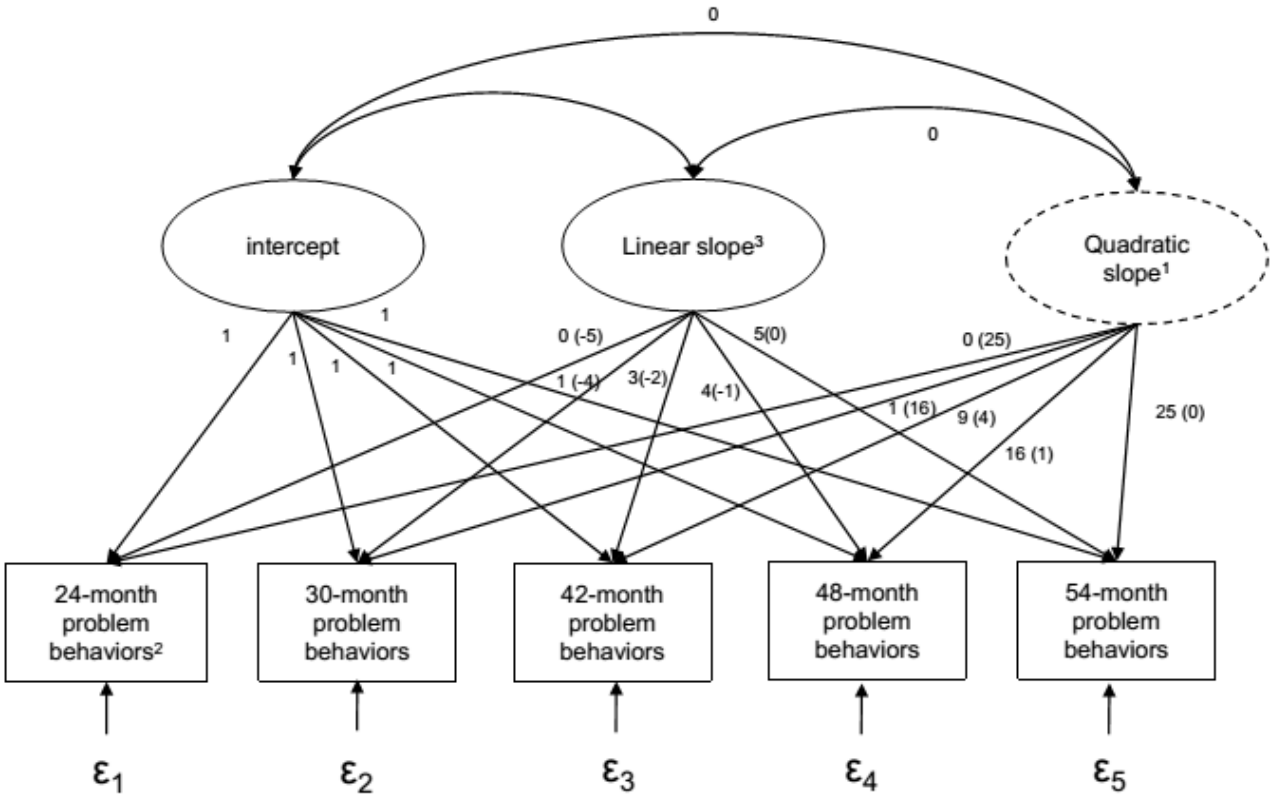

Figure 2. SEM Unconditional Models for the Latent Growth Curve of Problem Behaviors. *Note*: [1]Quadratic slope (i.e., average trend) was only included in the **mother-reported externalizing models**. Because this quadratic variance was fixed to zero, its covariances with intercept and linear slope factor were zero. However, covariances between intercept and linear slope were freely estimated. [2]Problem behaviors indicate mother- and father-reported externalizing and internalizing separately for different models. [3]Slope loadings indicate time centering at 24 months, and the loadings in parentheses indicate time centering at 54 months.

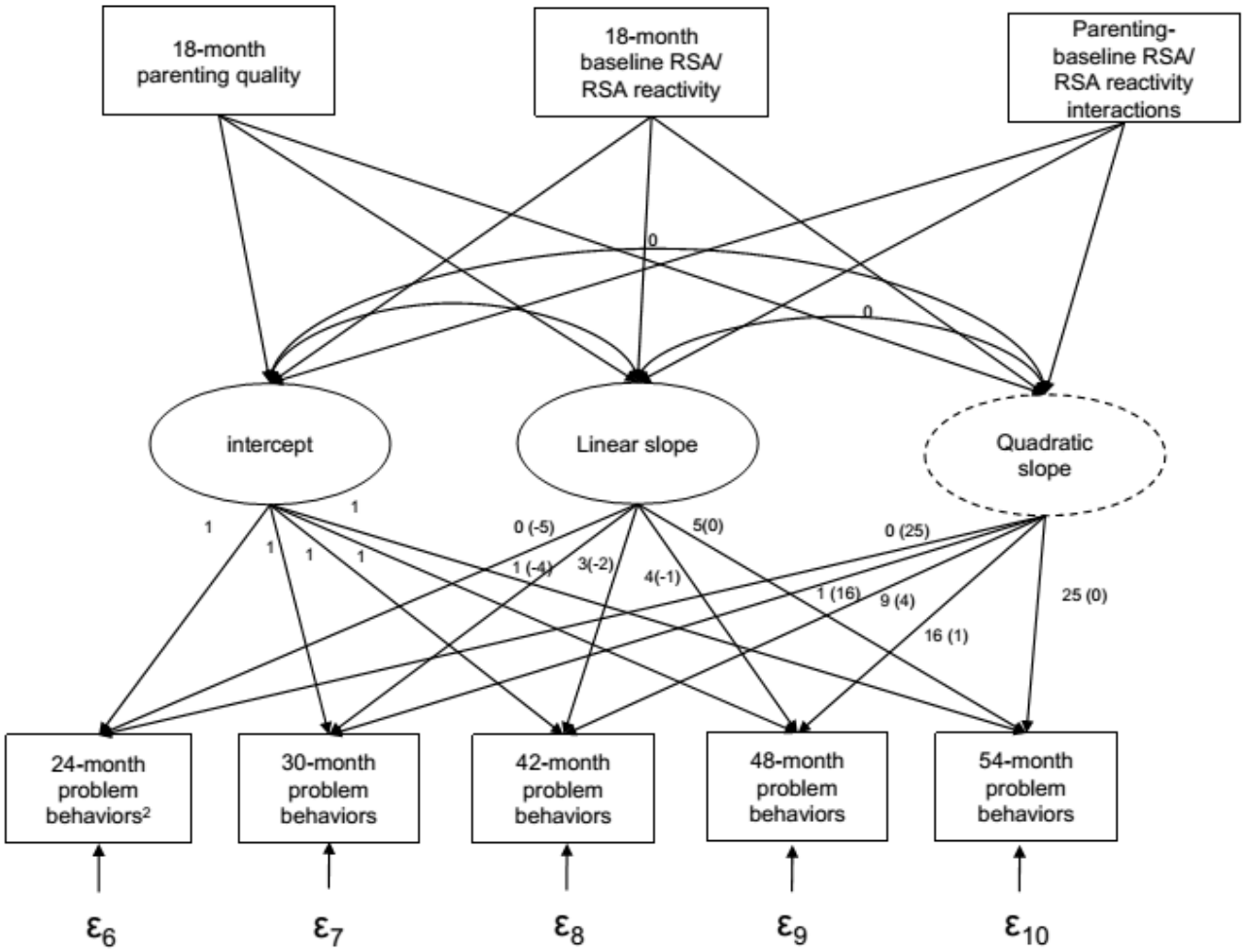

Figure 3. SEM Two-way Interaction (i.e. parenting quality × baseline RSA/RSA reactivity) Conditional Models Predicting Problem Behaviors. *Note:* Child sex was included as covariate; Unconditional model rules apply to the conditional model.

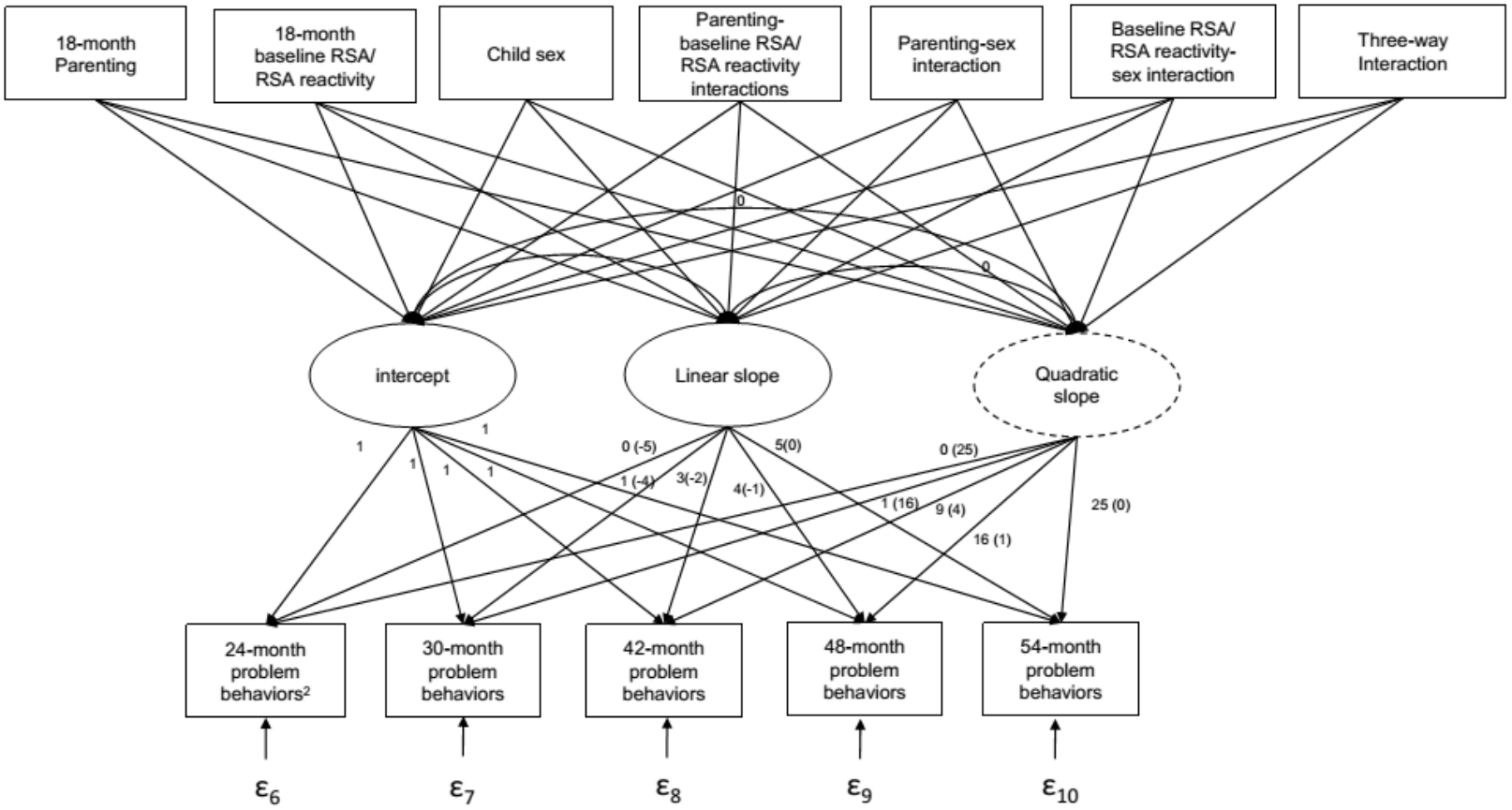

Figure 4. SEM Three-way Interaction (i.e. parenting quality × baseline RSA/RSA reactivity × sex) Conditional Models Predicting Problem Behaviors. *Note*: Unconditional model rules apply to the conditional model.

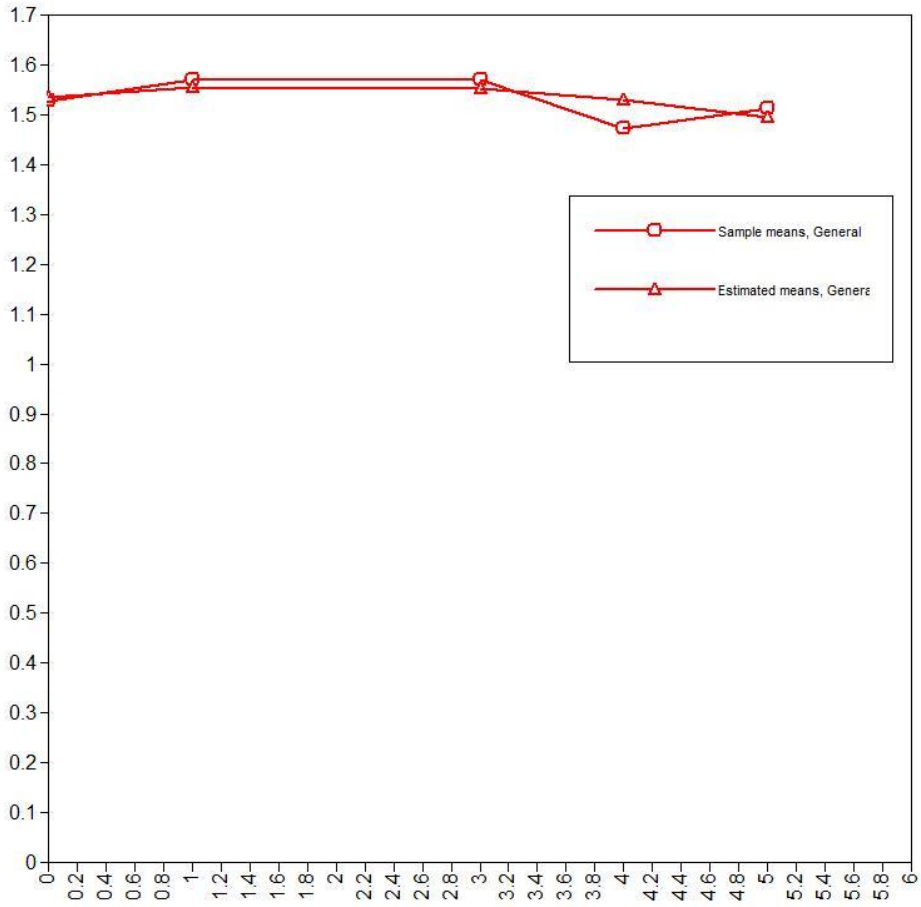

Figure 5. Plot of Sample Means and Model-Estimated Means of Mother-reported Externalizing

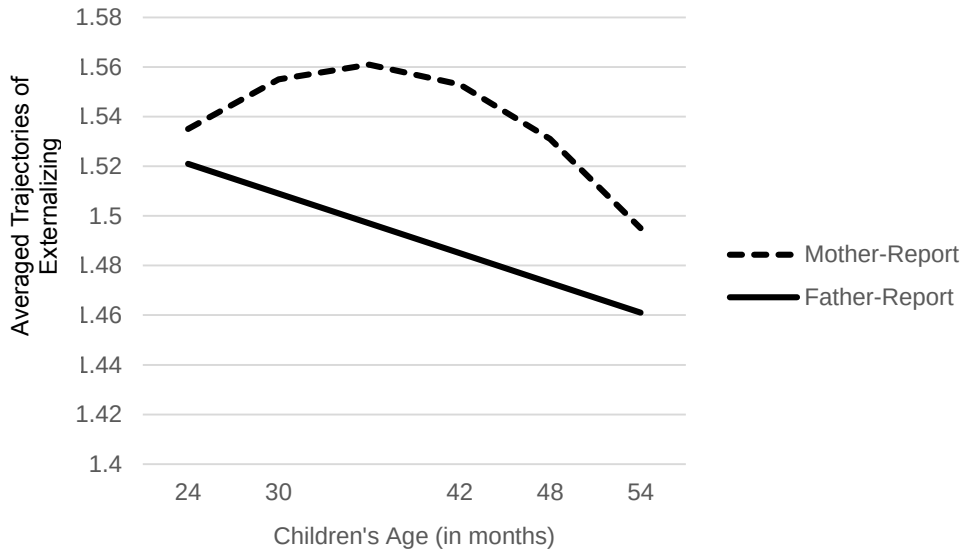

Figure 6a. Average Trajectories of Mother- and Father-reported Externalizing Symptoms

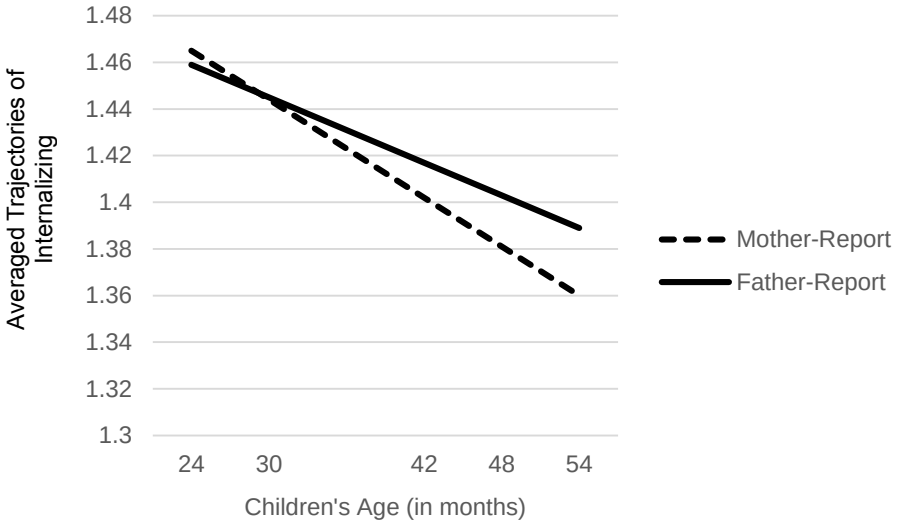

Figure 6b. Average Trajectories of Mother- and Father-reported Internalizing Symptoms

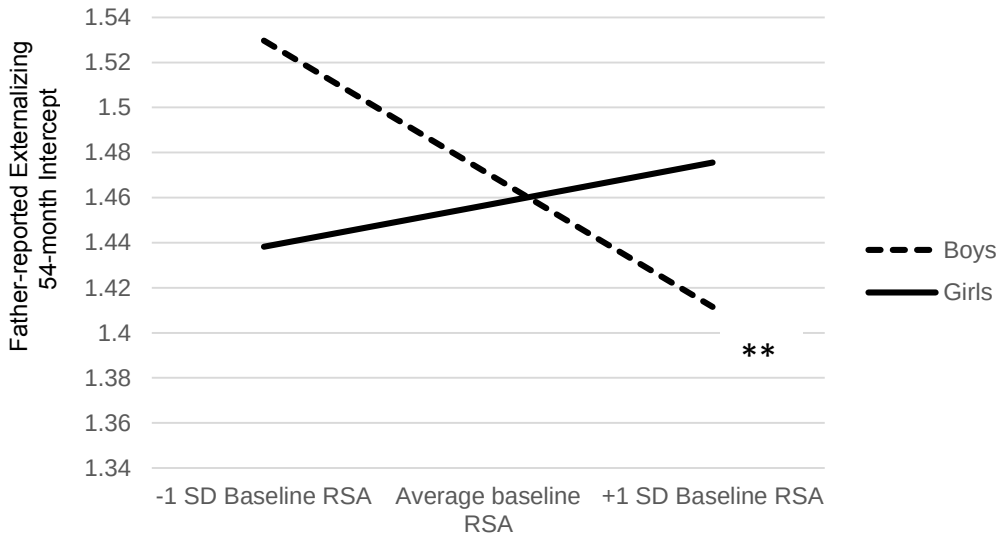

Figure 7. Predicting the Father-reported Externalizing Intercept at 54 months from the Interaction between Children's Sex and Baseline RSA

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

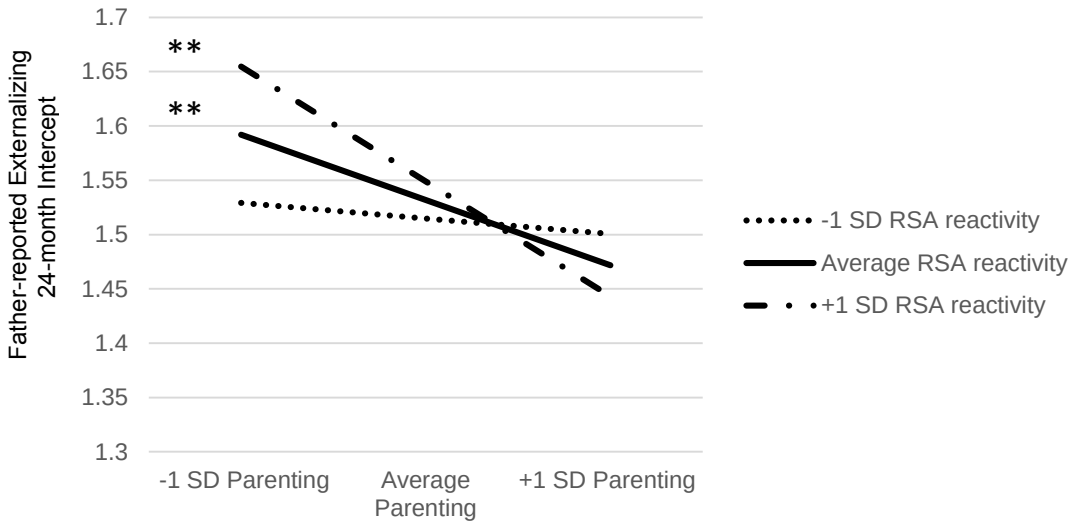

Figure 8a. Predicting the Father-reported Externalizing Intercept at 24 months from the Interaction between 18-months Parenting Quality and Children's RSA reactivity (RSA reactivity as the moderator)

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

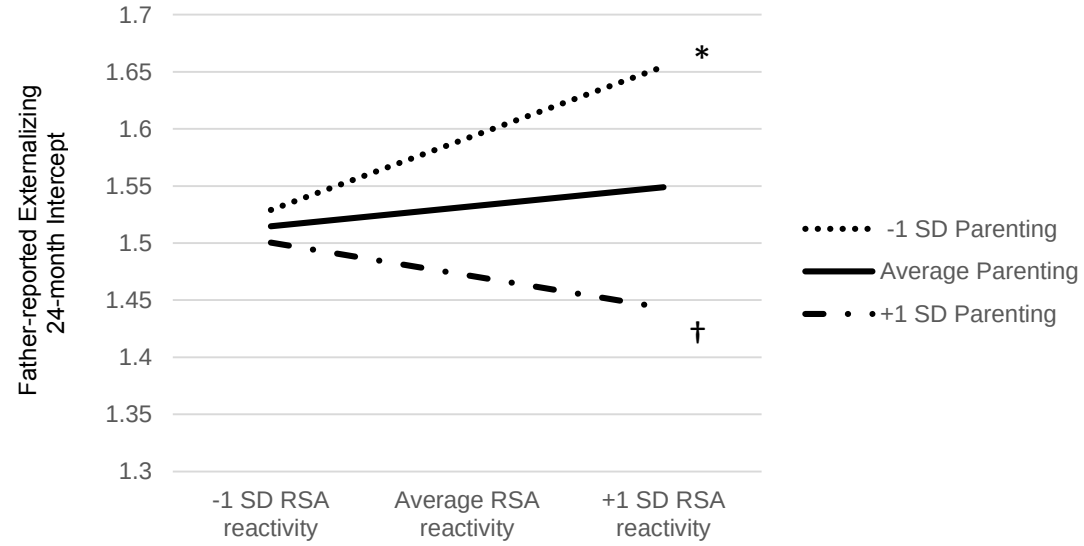

Figure 8b. Predicting the Father-reported Externalizing Intercept at 24 months from the Interaction between 18-months Parenting Quality and Children's RSA reactivity (parenting quality as the moderator)

* $p < .05$, † $p < .10$

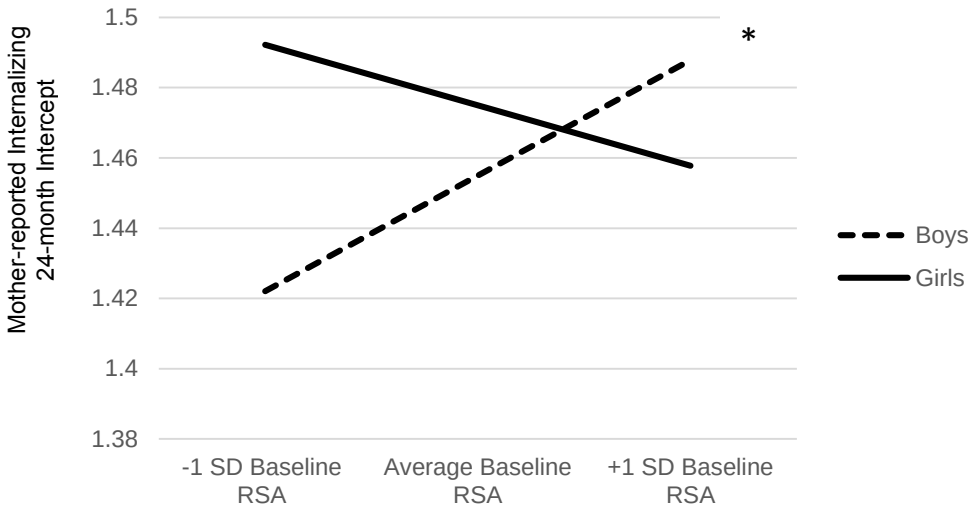

Figure 9. Predicting the Mother-reported Internalizing Intercept at 24 months from the Interaction between Children's Sex and Baseline RSA

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

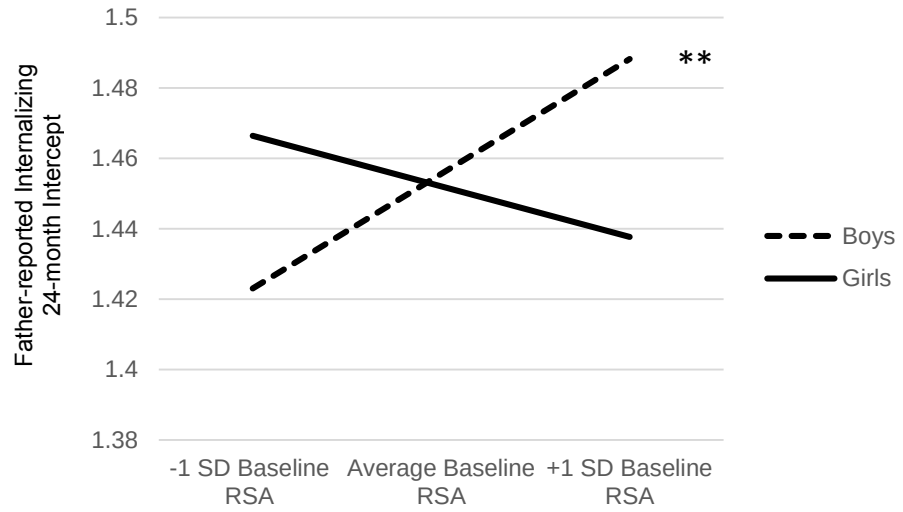

Figure 10. Predicting the Father-reported Internalizing Intercept at 24 months from the Interaction between Children's Sex and Baseline RSA

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

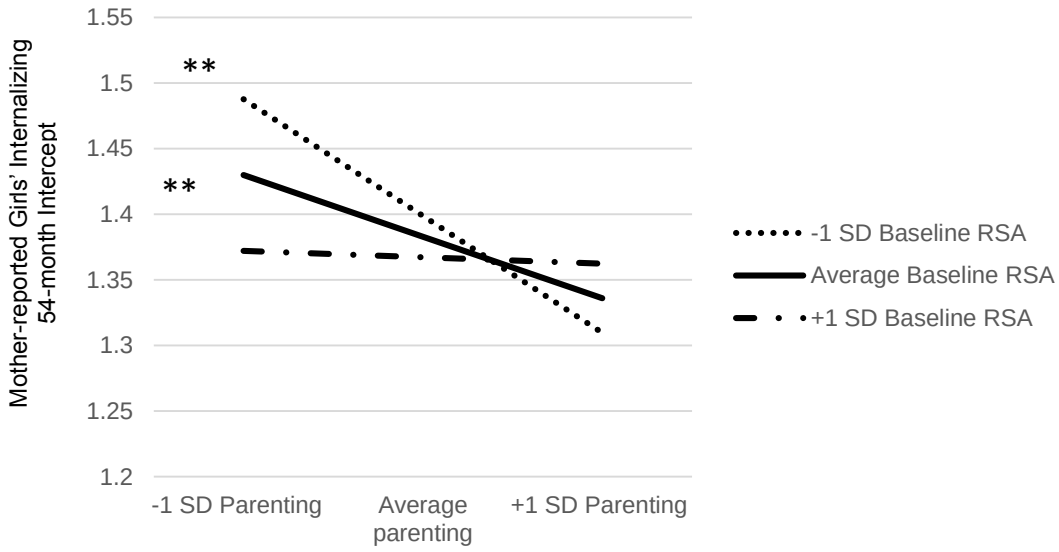

Figure 11a. Predicting the Mother-reported Girls' Internalizing Intercept at 54 months from the Interaction between 18-month Parenting Quality and Girls' Baseline RSA (baseline RSA as the moderator)

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

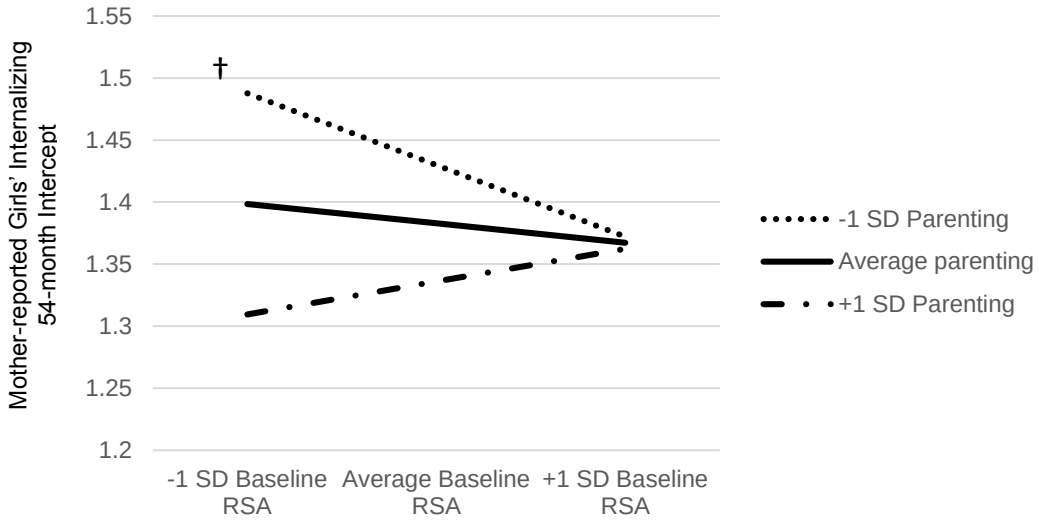

Figure 11b. Predicting the Mother-reported Girls' Internalizing Intercept at 54 months from the Interaction between 18-month Parenting Quality and Girls' Baseline RSA (parenting quality as the moderator)

† $p < .10$

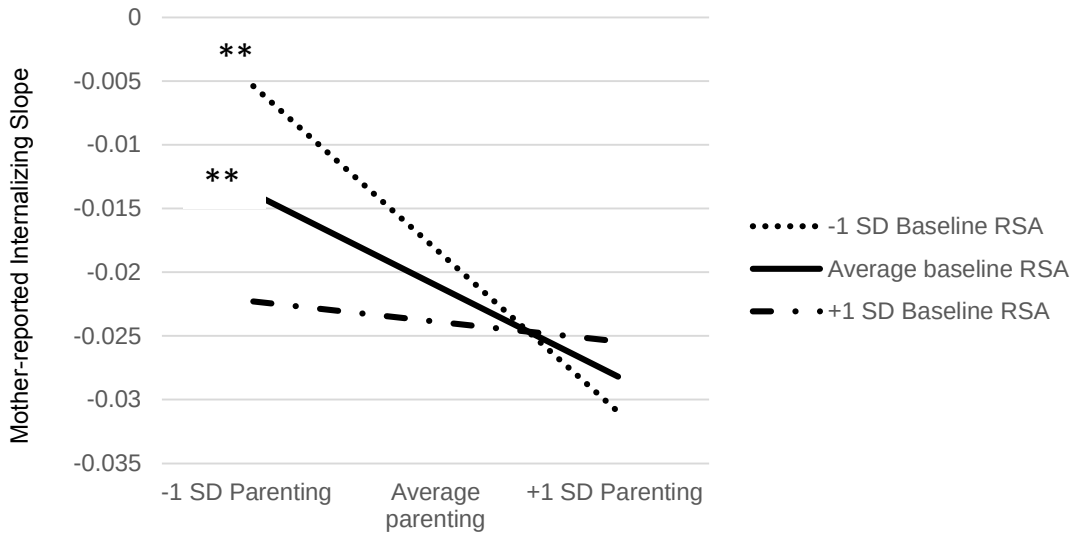

Figure 12a. Predicting the Mother-reported Internalizing Slope from the Interaction between 18-month Parenting Quality and Children's Baseline RSA (baseline RSA as the moderator)

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

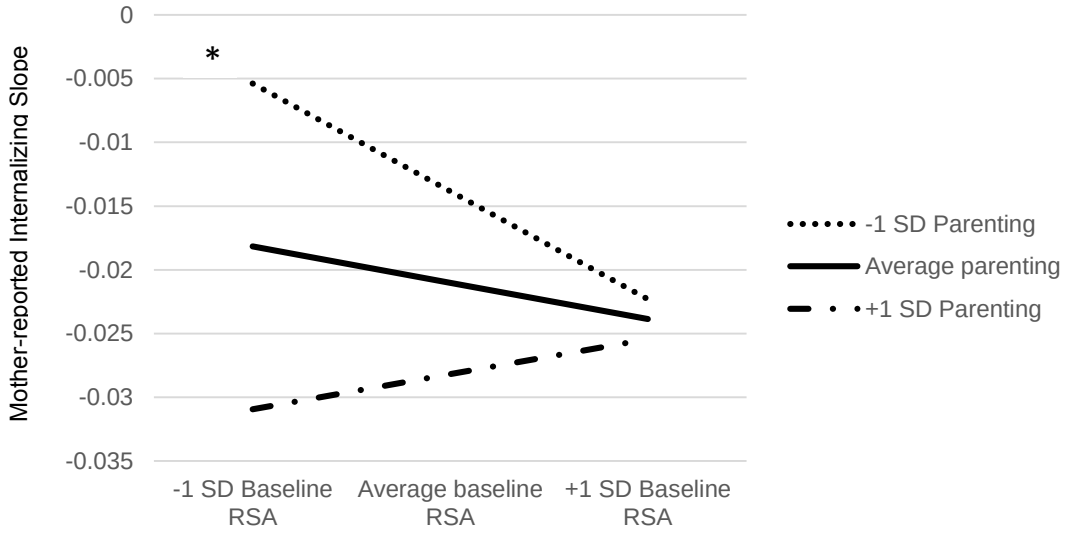

Figure 12b. Predicting the Mother-reported Internalizing Slope from the Interaction between 18-month Parenting Quality and Children's Baseline RSA (parenting quality as the moderator)

* $p < .05$.

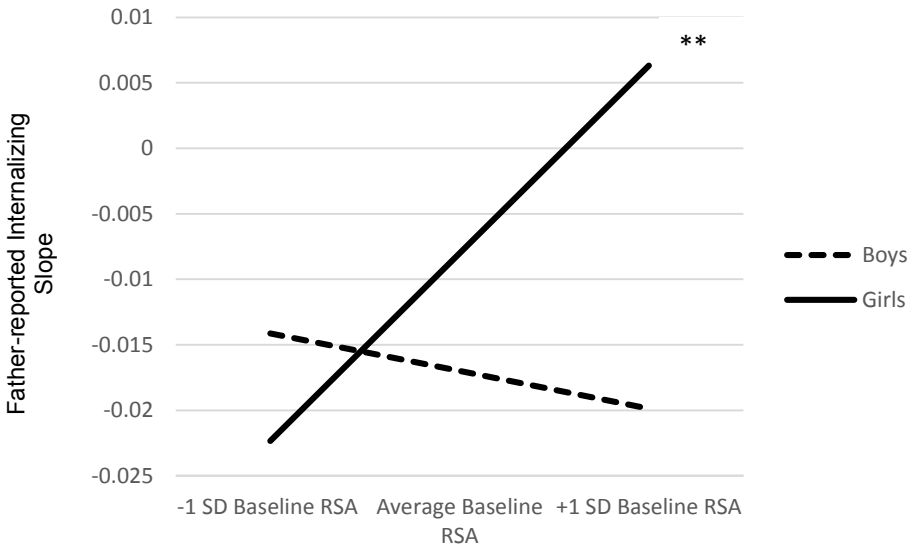

Figure 13. Predicting the Father-reported Internalizing Slope from the Interaction between Children's Sex and Baseline RSA

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

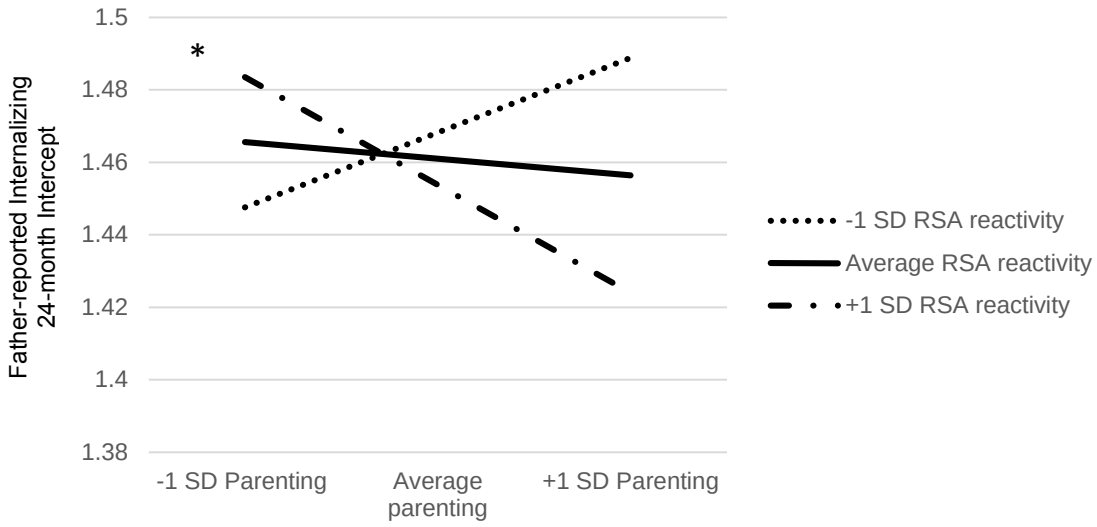

Figure 14a. Predicting the Father-reported Internalizing Intercept at 24 months from the Interaction between 18-month Parenting Quality and Children's RSA reactivity (RSA reactivity as the moderator)

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

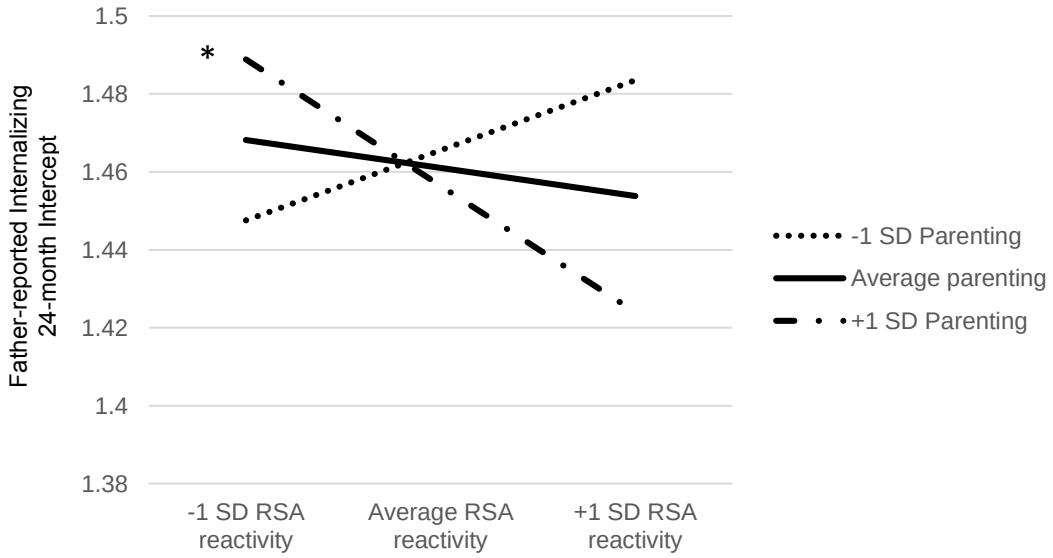

Figure 14b. Predicting the Father-reported Internalizing Intercept at 24 months from the Interaction between 18-month Parenting Quality and Children's RSA reactivity (parenting quality as the moderator)

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

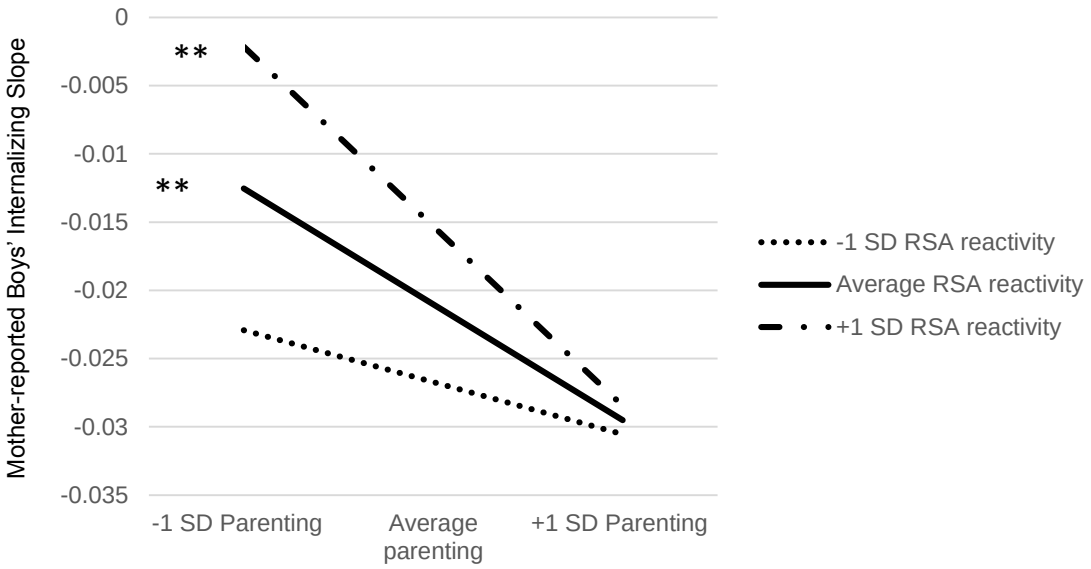

Figure 15a. Predicting the Mother-reported Boys' Internalizing Slope from the Interaction between 18-month Parenting Quality and Boys' RSA reactivity (RSA reactivity as the moderator)

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

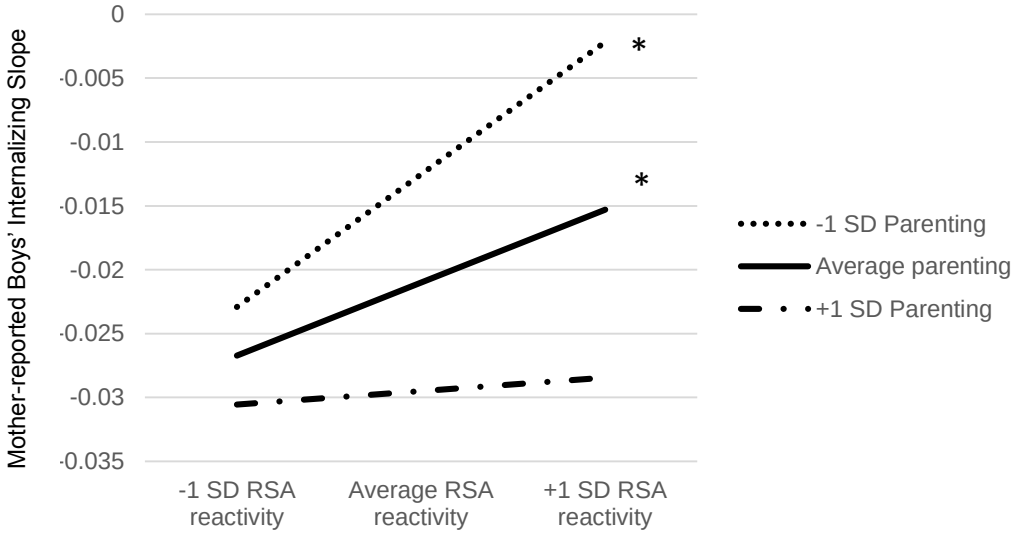

Figure 15b. Predicting the Mother-reported Boys' Internalizing Slope from the Interaction between 18-month Parenting Quality and Boys' RSA reactivity (parenting quality as the moderator)

* $p < .05$.