THE MODERATING ROLE OF AMYGDALAR AND HIPPOCAMPAL ACTIVATIONS IN THE EFFECTS OF FAMILY REARING ENVIRONMENTS ON YOUTH ADJUSTMENT

by

SIHONG LIU

(Under the Direction of Assaf Oshri)

ABSTRACT

Family rearing experiences are critical to youths' socioemotional and behavioral development. Supportive family environments contribute to positive youth adjustment. In contrast, youth exposed to adverse rearing experiences are at increased risk for developing psychopathology. Despite the documented effects of rearing environments on developmental outcomes, youth often show different responses even to similar family experiences. These differential responses are attributed to interactions between the environments (positive or negative) and youths' neurobiological architecture. Specific neurobiological traits potentiate the effect of rearing environments by rendering youth to be more biologically sensitive and behaviorally responsive to caregiving experiences. Thus, they are either more vulnerable to the harmful impacts of adverse rearing experiences or benefiting more from positive environments. Despite empirical evidence on youths' differential responses to family influences, the underpinning neural architecture remains unclear. The present dissertation investigated the neurobiological mechanisms underlying youths' differential behavioral reactivity to family rearing environments, with the amygdala and hippocampus as the key neurobiological foci. The activations of these two subcortical brain regions in response to emotional stimuli were hypothesized to moderate the linkage between rearing environments and youth adjustment.

This dissertation included two studies that methodologically complemented each other. The first study employed a large, nationally representative, and longitudinal dataset of the Adolescent Brain Cognitive Development study (N = 11,875). This dataset granted substantial statistical power to test the study hypotheses. The second study used a modest sample with multi-method and multi-reporter assessments from at-risk families in rural Georgia (N = 123). This study employed a rigorous measurement tool to assess the full continuum (from negative to positive) and dimensionality of rearing environments. Results showed that left amygdalar and hippocampal activations during emotional processing significantly exacerbated the effects of rearing environments on youth adjustment. Youth who evinced higher levels of left amygdalar and hippocampal activations and reported adverse family environments showed elevated problem behaviors. However, with heightened left amygdalar and hippocampal activations, youth also exhibited more adaptive responses to supportive rearing experiences. The findings of this dissertation may assist in tailoring prevention and intervention programs to target the most vulnerable and responsive youth for interventive practices.

INDEX WORDS: family rearing environments; internalizing and externalizing problems; prosocial behaviors; amygdala; hippocampus; neurobiological sensitivity to family environments

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INTRODUCTION

Extant literature in developmental science suggests that the quality of family caregiving experiences plays a critical role in shaping socioemotional and behavioral adjustment during childhood and adolescence (Luthar, 2006; A. S. Masten, 2001; Sturge‐Apple, Davies, & Cummings, 2010). Warm and supportive rearing environments are associated with positive youth development and reduced risk for psychopathology and risk-taking behaviors (G. H. Brody, Yu, Chen, Kogan, et al., 2013; Rohner, Khaleque, & Cournoyer, 2005; Wickrama & Kaspar, 2007). In contrast, adverse family experiences expose youth to elevated risks for a wide range of problem behaviors (Cummings, Davies, & Campbell, 2002; Kogan, Getahune, & Walsh, 2019; Oshri, Rogosch, & Cicchetti, 2013; Wickrama, Conger, & Abraham, 2008). This large body of literature provides support to the importance of rearing environments in youth development.

Despite consistent and robust relations between family rearing environments and youth adjustment, youth who experience comparable upbringing milieus vary significantly in their behavioral adjustment (Luthar, 2006; A. S. Masten & Obradović, 2006). Some youth exhibit elevated responses to rearing experiences (Ellis, Essex, & Boyce, 2005). These youth are more vulnerable to the harmful impacts of adverse rearing environments. However, they also show more developmental competence in response to supportive caretaking experiences (Boyce & Ellis, 2005; Ellis et al., 2005). Youth who exhibit heightened responses to positive and negative rearing environments are considered to be highly *sensitive* to the influences of caregiving experiences (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). In contrast, other youth are less responsive to the influences of family influences, so their developmental adjustment is not significantly affected by positive and negative rearing experiences (A. S. Masten, 2001; A. S. Masten & Obradović, 2006).

Several theoretical frameworks seek to explain why youth respond differently to similar caregiving experiences. The variability in youths' responses to family influences has been growingly attributed to individual differences in neurobiological factors, which moderate the linkage between rearing environments and youth adjustment (Ellis et al., 2011). Two theoretical perspectives received significant attention from empirical research, including the *diathesis-stress* model (Monroe & Simons, 1991; Zuckerman, 1999) and the *biological sensitivity to context* theory (Boyce & Ellis, 2005; Ellis et al., 2005). The diathesis-stress model seeks to explain the behavioral heterogeneity among youth raised in similarly adverse rearing environments. This model focuses on examining biological vulnerabilities that are hypothesized to predispose youth to the development of psychopathology and risk behaviors. According to the diathesis-stress model, some youth are disproportionally more vulnerable than others, thus are more likely to develop maladaptive outcomes in response to adverse environments (Monroe & Simons, 1991; Zuckerman, 1999). Research provides some support for this theory (e.g., Belsky, Hsieh, & Crnic, 1998; Caspi et al., 2003; Cummings, El-Sheikh, Kouros, & Keller, 2007). However, other studies point to its limitations. The main limitation of the diathesis-stress model lies in its lack of examination on youths' behavioral responses to the interactions between neurobiological factors and positive environmental influences (Ellis & Boyce, 2008; Ellis et al., 2011).

The diathesis-stress theory is expanded upon by the biological sensitivity to context theory, which proposes that neurobiological factors can also intensify youths' positive behavioral responses to supportive family environments (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, this theory purports that the same neurobiological factors that predispose youth for risk under adverse environments can also potentiate the effect of positive rearing environments (Boyce & Ellis, 2005). On the one hand, when exposed to adverse caregiving environments, youth with specific neurobiological factors may be predisposed to the development of maladaptive outcomes (Boyce & Ellis, 2005; Ellis et al., 2005). On the other hand, the same biological architecture can also serve as a potentiating factor that amplifies the benefits of supportive and nurturing caregiving experiences, which leads to positive youth development (Boyce & Ellis, 2005; Ellis et al., 2005). The biological sensitivity to context theory has been gaining growing empirical support (e.g., Bakermans-Kranenburg & Van Ijzendoorn, 2011; Cruz, Abreu-Lima, Canário, & Burchinal, 2018; Obradović, Bush, Stamperdahl, Adler, & Boyce, 2010; Oshri, Duprey, Liu, & Ehrlich, in press; Slagt, Dubas, Ellis, Van Aken, & Deković, 2019; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012). This theory provides a theoretical framework that guides the major hypotheses of the current dissertation.

The biological sensitivity to context theory suggests that individual heterogeneity in youths' responses to caregiving experiences is grounded by youths' differential "sensitivity" to family influences, which can be tested by modeling interactions between *neurobiological* processes and rearing environments (Boyce & Ellis, 2005). Therefore, youths' differential responses to family effects are referred to as *neurobiological sensitivity* (Ellis et al., 2011). Heightened neurobiological sensitivity indicates youths' increased vulnerability to adverse environments, as well as elevated adaptive responses to positive caregiving experiences. Research identifies a variety of behavioral and biological markers that reflect neurobiological sensitivity to family influences among youth. These sensitivity indicators include behavioral phenotypes (e.g., difficult temperament; Cruz et al., 2018; Slagt et al., 2019), genetic make-up (e.g., dopamine- and serotonin-related genes; Bakermans-Kranenburg & Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013; Van IJzendoorn et al., 2012), and psychophysiological processes (e.g., stress response systems; Obradović et al., 2010; Oshri et al., in press). However, studies that investigate the neural architecture (i.e., the brain structures and functions) underlying youths' differential sensitivity to family influences are scarce (Schriber & Guyer, 2016). The brain plays a central role in processing complex environmental information and regulating complicated human behaviors. Therefore, the lack of research on youth development in the context of underlying neurobiological sensitivity mechanisms is a major gap in the literature (Boyce & Ellis, 2005; Schriber & Guyer, 2016).

The present dissertation aimed to investigate the neural mechanisms underlying youths' differential neurobiological sensitivity to family rearing environments. In particular, two subcortical brain regions, the amygdala and hippocampus, were examined as the key foci of the neurobiological sensitivity mechanisms. The amygdala and hippocampus play essential roles in modulating youths' emotional and stress reactivity (Baxter & Murray, 2002; Phelps, 2004; Windle et al., 2018). Thus, research suggests that amygdalar and hippocampal functions can serve as candidate biomarkers of youths' neurobiological sensitivity to family influences (McLaughlin et al., 2014; Schriber & Guyer, 2016). In particular, this dissertation tested how amygdalar and hippocampal responses to emotional stimuli moderated the influences of positive and negative family rearing environments on youth adjustment. Figure 1.1 presented the conceptual framework for this dissertation.

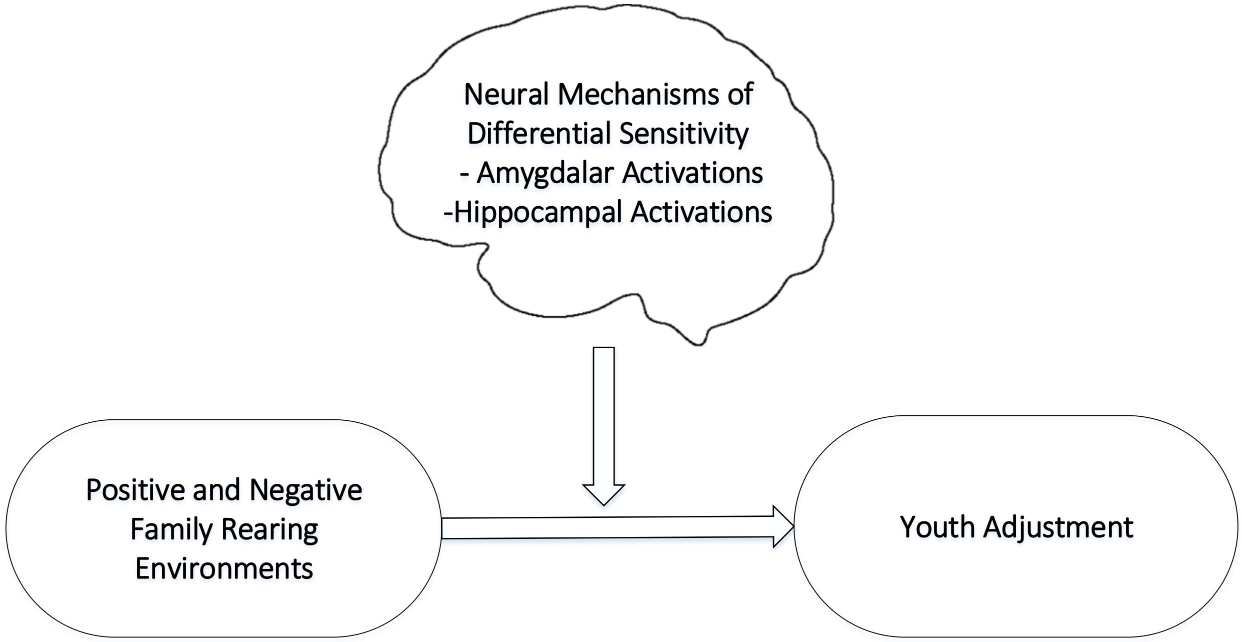

Figure 1.1. Conceptual Framework Guiding Study Hypotheses.

CHAPTER 2

THEORETICAL PERSPECTIVES AND LITERATURE REVIEW

In this chapter, two theoretical perspectives that informed this dissertation project were first reviewed, including the developmental psychopathology perspective (Cicchetti, 1993, 2010, 2016; Cummings et al., 2002; Davies & Cicchetti, 2004) and the evolutionary developmental perspective (Witherington & Lickliter, 2016). Then, adolescent neurodevelopment models that elaborated on the neural mechanisms underlying youths' responses to environmental influences were discussed (Casey, Getz, & Galvan, 2008; M. Ernst, 2014; Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg et al., 2008). Next, the literature on youths' differential sensitivity to the influences of family rearing environments was reviewed (Boyce & Ellis, 2005; Ellis et al., 2005; Monroe & Simons, 1991; Pluess & Belsky, 2013). The literature review discussed the empirical evidence that supported the neurobiological sensitivity to context models. This body of research included empirical evidence in behavioral phenotype (Chen, McElwain, Berry, & Emery, 2019), genetic (Bakermans-Kranenburg & Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013; Van IJzendoorn et al., 2012), psychophysiological (Obradović et al., 2010; Oshri et al., in press), and neuroimaging studies (Schriber & Guyer, 2016). Finally, an overview of the two studies conducted in this dissertation was stated.

Developmental Psychopathology and Family Science

Developmental Psychopathology is a paradigm that offers a comprehensive and integrative theoretical view for conceptualizing the emergence of psychopathology as a developmental phenomenon (Rutter, 2013; Sroufe, 2013). As a “macro-paradigm” (Cummings & Valentino, 2015), developmental psychopathology does not espouse a single theory that would account for all developmental phenomena (Cicchetti, 1993). Instead, it integrates knowledge across multiple levels of analyses and domains that examines the origins and courses of individual patterns of behavioral maladaptation (Rutter & Sroufe, 2000). A core tenet of the developmental psychopathology perspective is that psychopathology emerges from complex interactions across multiple systems over time. These systems include individuals and the social context they are embedded in (Cicchetti, 2016; Sroufe, 2009, 2013). Therefore, each case of psychopathology is unique in its etiology and developmental pathways. The emphasis on etiology across systems and time leads developmental psychopathology researchers to investigate how early life experiences influence the development of psychopathology over the life course (Cicchetti, 2010).

Multilevel Systems Perspective

The developmental psychopathology perspective suggests that psychopathology results from a developmental process that includes dynamic interactions among individuals’ biological, psychological, and social characteristics, and the multiple hierarchal social contexts (Cicchetti, 2010). Based on this framework, social contexts are critical components in youth development. Further, according to the developmental psychopathology framework, the interplay between individuals and the environment shapes the developmental processes (Cicchetti & Rogosch, 2002). Individuals’ experiences are not merely created by the environment; they are rather active participants in this process. Individuals actively create their experiences in their interactions with their environment (Cummings et al., 2002). For example, during childhood and adolescence, the family rearing environments are one of the closest social environment surrounding youth

(Bronfenbrenner & Morris, 2006). The interactions between youth and family members determine the quality of interpersonal relationships in the family rearing environments and play an essential role in shaping youth socioemotional and behavioral development (A. S. Masten, 2001; Steinberg et al., 2008; Steinberg & Morris, 2001).

The developmental psychopathology perspective is informed by *dynamic systems approaches* (Overton, 2013), which suggests that human development is embedded in multiple hierarchical systems. According to Bronfenbrenner's *bio-ecological perspectives on human development*, these systems include individuals' biological, psychological, and social characteristics. These bio-ecological systems are depicted as layers surrounding an individual, which are distinguished based on their physical and psychological proximity to the individual (Bronfenbrenner, 2005; Bronfenbrenner & Morris, 2006). The closest systems are referred to as *microsystems*; they encompass interpersonal relationships and direct interactions with immediate surroundings such as family, school, and neighborhood (Bronfenbrenner, 2005). The second layer is the *mesosystem*, which is defined as the interactions between two microsystems. For example, a relationship between a child's parents and school teachers is a part of the mesosystem affecting the child (Bronfenbrenner, 2005). The third layer is the *exosystem,* the settings that indirectly influence individuals through their direct effect on microsystems. For instance, parental job loss does not involve the child directly but might indirectly affect a child through influencing the family's financial situations (Bronfenbrenner, 2005). The *macrosystem* is the outer-most layer of Bronfenbrenner's bio-ecological systems model. It refers to the cultural and social contexts that influence child development, including laws, policies, and cultural values in society (Bronfenbrenner, 2005). Additionally, the *chronosystem* is defined as the occurrence and transition of environmental events throughout an individual's lifespan. These events include normative transitions (e.g., school entry, marriage), non-normative events (e.g., divorce, relocation, job loss), and the cumulative effects of these events over the life course (Bronfenbrenner, 2005). This dissertation focused on the influence of the family rearing environments on youth, which is one of the most critical microsystems in affecting youth development (Bronfenbrenner & Morris, 2006).

The dynamic systems approach also emphasizes the *holism* of various bio-ecological systems. These hierarchical and nested systems are inter-dependent as they influence each other and give meaning to the whole (Cummings & Valentino, 2015). Therefore, components of the systems should be examined in the social contexts, and the interdependency among different systems needs to be considered. In line with the bio-ecological perspective on human development, this dissertation examined how the interactions of the family rearing environments (i.e., a microsystem) and neural processes (i.e., biological characteristics) predict youths' socioemotional and behavioral adjustment.

Developmental Pathways: Multifinality and Equifinality

Developmental Psychopathology perspective suggests that developmental pathways to psychopathology are not linear (Cicchetti & Rogosch, 1996). Instead, diversity and heterogeneity exist in the etiology, processes, and outcomes associated with youth development. Specifically, maladaptation and psychopathology are conceptualized as developmental pathways that reflect the dynamic interplay between individuals' previous functioning, current individual status, and the hierarchal environmental contexts. This dynamic interplay forms a complex co-acting system (Cummings & Valentino, 2015). Researchers argue that developmental pathways are best characterized by probabilities rather than linear causality (Sroufe, 2013). Two principles—*multifinality* and *equifinality* —are informed by the probabilistic view of developmental pathways. Multifinality refers to the idea that an earlier origin point can result in multiple possible outcomes through different developmental pathways (Cicchetti & Rogosch, 1996). For example, children exposed to similar levels and patterns of family conflict may develop qualitatively different outcomes. Some youth may develop psychopathology (e.g., internalizing & externalizing problems) and health risk behaviors (e.g., substance use), while others may show resilience and competence (Afifi & MacMillan, 2011). The current research was designed to elucidate the neural underpinnings of *multifinality* in youth development, namely, why some youth were more likely to be affected, while others were less responsive to similar environmental influences of family interactions.

Equifinality is a concept that describes how the same developmental outcome can originate from different rearing environments (Cicchetti & Rogosch, 1996). According to the equifinality concept, in a bio-ecological system, the same outcome might be reached from different initial situations via different processes (Cicchetti & Rogosch, 1996). For example, adolescent substance use behaviors can be induced by risk factors from different social contexts in different microsystems, including family, peers, and the neighborhood. Stressors in the family rearing environments increase youths' risks for using the substance as a coping strategy to self-medicate their negative affect (Khantzian, 2013; S. Liu, Oshri, & Duprey, 2018). Therefore, family stress is a major etiology for youth development of substance use behaviors (Nash, McQueen, & Bray, 2005). Affiliation with deviant peers is another risk factor for youth substance use problems because youth affiliated with deviant peers are more likely to view substance use as normative (Hampson, Andrews, & Barckley, 2008; Kogan, Luo, Murry, & Brody, 2005). Deviant peer affiliation also increases youths' experiences of social pressures to use substances to conform to the deviant peer norms (Marshal & Chassin, 2000). Additionally, a disorganized neighborhood also poses risks for youths’ substance use problems (Lambert, Brown, Phillips, & Ialongo, 2004; Reyes et al., 2008). Therefore, youths’ substance use behaviors can be induced by different etiological factors, including family stress (S. Liu et al., 2018), deviant peer affiliation (Kogan et al., 2005; Marshal & Chassin, 2000), and disorganized neighborhood (Lambert et al., 2004). Equifinality is reflected in the current dissertation by investigating how different dimensions of caregiving experiences (i.e., family conflict, parental warmth, and family functioning) affect youth adjustment outcomes.

Developmental Timing

Another central tenet of the developmental psychopathology perspective is that developmental processes extend across the lifespan, and developmental timing has significant influences on the emergence of psychopathology (Cicchetti, 2010). Normality and psychopathology processes emerge throughout the entire life course (Cicchetti, 2011; Cicchetti & Rogosch, 2002). The same experience at different developmental stages might result in different developmental outcomes. Therefore, chronological age and developmental stages should be accounted for while examining the emergence of risky behaviors and psychopathology (Cicchetti, 2016).

Early experiences are particularly important for individuals to structure and organize their psychological and biological systems to adapt to the environment. Because of the importance of early life experiences, childhood is usually considered as a sensitive period of development (Cicchetti, 2011). This heightened sensitivity to childhood experiences can be partially attributed to multiple and rapid brain changes during infancy and early childhood (Guyer, Pérez-Edgar, & Crone, 2018). These brain changes foster development in fundamental domains of functioning, such as language acquisition, attention, working memory, and self-regulation (M. H. Johnson, 2000). Despite the importance of childhood experiences, adolescence is a second sensitive period marked by brain growth and change in higher-order functioning such as cognitive control (Casey et al., 2008). These changes support the unique tasks emerging from adolescence, including gradual independence from family rearing environments, increased academic requirements, and emerging working and romantic relationships (Guyer et al., 2018). Therefore, experiences during adolescence can also alter the psychological and biological development course (Cicchetti & Rogosch, 2002). Overall, the brain plays a vital role in regulating higher-order functions and modulating developmental outcomes during adolescence (Casey et al., 2008; Guyer et al., 2018; Romer, 2010). Therefore, the current investigation examined the neural mechanisms underlying youths' differential sensitivity during pre- and early-adolescence.

Developmental Psychopathology and Family Science

The developmental psychopathology perspective is integrated into research in family science to advance the understanding of the patterns of youth adjustment and maladjustment embedded in complex family dynamics (Davies & Cicchetti, 2004). Traditionally, developmental psychopathology and family systems theory are two distinct theoretical frameworks with differences between their primary unit of analysis. While the developmental psychopathology perspective examines individual-level normal and abnormal development (Cicchetti, 2010), the family system theory views the whole family as an investigation unit and focuses on the interplay, structure, and quality of individual relationships (M. J. Cox & Paley, 1997). However, these two frameworks share common philosophical and theoretical bases, such as the multilevel systems perspective (Davies & Cicchetti, 2004). The shared common philosophical and theoretical bases made it promising to integrate these two approaches in mutually-enriching ways.

Research has started closing the gap between the developmental psychopathology and family science frameworks by investigating how family dynamics and psychopathology interact and reciprocally influence each other (Cummings et al., 2002). Specifically, this field of research examines topics including the biopsychological antecedents and sequelae of family relationship quality across the life span, the roles of parental and child psychopathology in the context of family systems, and the multifinality of developmental pathways following family adversity (Davies & Cicchetti, 2004). This integration of the developmental psychopathology perspective into family science research provided essential conceptual guidance for the current dissertation. Indeed, this dissertation examined how the interpersonal relationship quality and family dynamics affect youths' normal and abnormal development, as well as the neurobiological mechanisms underlying the multifinality in these developmental processes (Cicchetti & Curtis, 2007; Davies et al., 2002).

Evolutionary Developmental Perspective

The multifinality concept in the developmental psychopathology perspective suggests that different outcomes can be induced by similar developmental etiology (Cicchetti & Rogosch, 1996). Specifically, under similar adverse rearing environments, some youth may develop psychopathology and risk behaviors, while others might be resilient to the negative effects of family adversity (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Similarly, when exposed to a supportive and nurturing family environments, some youth might benefit more from the positive environments (G. H. Brody, Yu, Chen, Kogan, et al., 2013; Pluess & Belsky, 2013). This multifinality can be explained by youths' differential sensitivity to the influences of family rearing environments (Ellis et al., 2011; Luthar, 2006; A. S. Masten & Obradović, 2006). Several models are proposed to use the evolutionary developmental perspective to explain why youth respond differently to similar caregiving experiences (Boyce & Ellis, 2005; Ellis et al., 2011; Ellis et al., 2005; Monroe & Simons, 1991; Pluess, 2017).

The evolutionary developmental perspective integrates developmental processes into evolutionary psychology theories, including Darwin's idea of natural selection as a shaping force behind phenotypic forms (Witherington & Lickliter, 2016). A core concept in the evolutionary developmental perspective is *adaptation*. Adaptation refers to the fitness outcomes (i.e., survival and reproduction) in the evolutionary sense rather than public health (e.g., physical health, mental well-being, and safety) outcomes (Ellis, Bianchi, Griskevicius, & Frankenhuis, 2017). Accordingly, natural selection favors *phenotypic plasticity* (i.e., the ability of a genotype to support multiple phenotypes in response to different ecological contexts). Through phenotypic plasticity, natural selection shapes individuals' *conditional adaptation* (i.e., development of specialized abilities as responses to different types of environmental contexts; Ellis, Bianchi, et al., 2017). This conditional adaptation renders individuals to detect and encode information about early rearing experiences internally, calibrate the development of stress response systems to match these early environments, and thus forms different levels of neurobiological sensitivity (Ellis et al., 2005). Overall, the evolutionary developmental perspective is widely used to explain individuals' development of adaptive and maladaptive outcomes under the influences of environments (Witherington & Lickliter, 2016). The evolutionary developmental perspective has been receiving growing empirical support (e.g., Khaleque, 2013; Obradović et al., 2010; Shonkoff, Boyce, & McEwen, 2009).

The current section on evolutionary developmental perspectives first provided a detailed review of three neurobiological sensitivity to context models, including the *diathesis-stress*, the *biological sensitivity to context*, and the *vantage sensitivity* models. Then, this section discussed how the evolutionary developmental perspective could be employed to explain the emergence of different neurobiological sensitivity patterns and levels. Reviews of the specialization and sensitization hypotheses, the biological sensitivity to context theory, and the adaptive calibration model were also provided.

Neurobiological Sensitivity to Context Models

Research establishes robust and consistent associations between family rearing experiences and youth developmental outcomes (G. H. Brody, Yu, Chen, Kogan, et al., 2013; Kogan et al., 2019; Oshri et al., 2018; Wickrama & Kaspar, 2007). However, significant variation in the psychological and physical adjustment of children exposed to similar early rearing environments is also documented in the literature (Luthar, 2006; A. S. Masten & Obradović, 2006). Youths' differential responses to caregiving environments stem partially from individuals' neurobiological processes that may moderate the effects of family experiences on youth behavioral adjustment. Therefore, the heterogeneity of youths' responsivity to family influences is hypothesized to be grounded in neurobiological sensitivity (Ellis et al., 2011). Three critical neurobiological sensitivity models have been proposed by evolutionary developmental scientists to reflect different sensitivity patterns. These models include the *diathesis-stress* (Monroe & Simons, 1991; Zuckerman, 1999), the *biological sensitivity to context* (Boyce & Ellis, 2005; Ellis et al., 2005), and the *vantage sensitivity* (Pluess & Belsky, 2013) models.

The diathesis-stress model. Historically, research efforts to delineate youths' differential responses to environmental influences start with the diathesis-stress model (Monroe & Simons, 1991; Zuckerman, 1999). The prevailing psychopathology analysis of developmental maladaptation and dysfunction induced by early adversity enables researchers to focus on the individual vulnerability and environmental risk of human development (Ellis et al., 2011). To explain the individual differences in response to environmental risk factors, the diathesis-stress model purports that specific neurobiological factors interact with rearing environments and predispose some youth to be more vulnerable to the negative effects of adverse life experiences (Zuckerman, 1999). In contrast, others who lack such vulnerabilities are resilient to the harmful impacts of adversity (Luthar, 2006; A. S. Masten & Obradović, 2006). The diathesis-stress model receives some empirical support (e.g., Belsky et al., 1998; Caspi et al., 2003; Cummings et al., 2007). However, the main limitation of this model is that it lacks an examination of youths' behavioral adjustment in response to the interactions between neurobiological factors and supportive family experiences (Ellis et al., 2011). Indeed, the diathesis-stress model implies no difference between vulnerable and resilient individuals in the absence of adverse experiences (Pluess & Belsky, 2013). To overcome this limitation, researchers propose the biological sensitivity to context (Boyce & Ellis, 2005; Ellis et al., 2005) and the vantage sensitivity (Pluess & Belsky, 2013) models.

The biological sensitivity to context model. The biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005) contends that some individuals are disproportionately more responsive to both negative (stress-promoting/developmental-vulnerability) and positive (development-enhancing) environments (Ellis et al., 2011). This differential responsivity to the full continuum of environmental influences is attributed to the interactions between youths' heterogenous neurobiological processes and rearing environments (i.e., neurobiological sensitivity; Boyce & Ellis, 2005; Ellis et al., 2005). According to the biological sensitivity to context hypothesis, youth with heightened neurobiological sensitivity exhibit elevated responsivity to the influences of qualitatively different rearing environments (Ellis et al., 2011). On the one hand, specific neurobiological factors interact with adverse caregiving experiences and eventuate youth with elevated risk for maladaptive outcomes. On the other hand, the same neurobiological architecture can also serve as a potentiating factor that amplifies the positive influences of supportive rearing environments on youth (Ellis et al., 2011). Empirical research documents convincing evidence that supports this biological sensitivity to context model. Research has identified multiple behavioral and biological sensitivity indicators, including behavioral phenotypes (e.g., Raver, Blair, & Willoughby, 2013), genotypes (e.g., Bakermans-Kranenburg & Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013), physiological processes (e.g., Obradović et al., 2010), and neurological mechanisms (e.g., Schriber et al., 2017).

The vantage sensitivity model. The vantage sensitivity hypothesis purports that individuals' positive developmental functioning is intensified by the interactions between specific biological sensitivity indicators and supportive rearing environments (Manuck, 2011; Pluess, 2017; Pluess & Belsky, 2013). This model suggests that some promotive factors that are grounded in neurobiological processes can intensify the beneficial effects of supportive environments among youth. The neurobiological vantage-sensitivity indicators render some youth to develop more adaptive responses in response to positive rearing environments (Pluess & Belsky, 2013). In contrast, other youth who lack these vantage-sensitivity factors exhibit vantage-resistance to the positive influences of supportive rearing environments (Pluess & Belsky, 2013). The vantage sensitivity hypothesis has received some preliminary empirical preliminary support (e.g., Pluess & Boniwell, 2015).

Overall, the diathesis-stress, biological sensitivity to context, and vantage sensitivity models all propose that individuals' elevated sensitivity significantly amplifies positive and/or negative environmental influences on youth adjustment (Boyce & Ellis, 2005; Pluess & Belsky, 2013; Zuckerman, 1999). Youths' sensitivity to environmental influences can be reflected by behavioral and neurobiological factors, including behavioral phenotypes, genotypes, physiological processes, and neural mechanisms (Ellis et al., 2011; Schriber & Guyer, 2016). These factors shape adolescents' perception and responsivity to environmental influences, change their behaviors, and ultimately moderate the impact of rearing environments on youths' development of psychopathology and competencies (Boyce & Ellis, 2005; Schriber & Guyer, 2016). In this dissertation, the neurobiological sensitivity to context models (Ellis et al., 2011; Schriber & Guyer, 2016) provide theoretical guidance for understanding the roles that the amygdala and hippocampus play in youths' differential responses to family rearing environments. Specifically, the diathesis-stress, biological sensitivity to context, and vantage sensitivity models represent different patterns in which neural factors interact with family environments and affect their socioemotional and behavioral adaptation (Del Giudice, 2017; Roisman et al., 2012).

The Conditional Adaptation of Neurobiological Sensitivity to Context Development

The neurobiological sensitivity models imply significant individual differences in youths' sensitivity under the influences of similar family rearing environments (Pluess & Belsky, 2013). From an evolutionary developmental perspective, theses individual differences originate from youths' active adaptation to early life experiences (Ellis et al., 2011). This adaptive pattern of neurobiological sensitivity is grounded in youths' *conditional adaptation*. Conditional adaptation indicates that a single genotype supports multiple phenotypic expressions that are contingent on environmental inputs, which allows youth to adapt to early caregiving environments (Boyce & Ellis, 2005). Accordingly, the developmental plasticity and evolved psychobiological mechanisms allow youth to calibrate the development of neurobiological sensitivity processes to adaptively match their early life experiences (Boyce & Ellis, 2005; Ellis et al., 2005). Below, several theories that elaborate on this conditional adaptation concept in neurobiological sensitivity to context models were reviewed. These theories include the specialization and sensitization hypotheses (Ellis, Bianchi, et al., 2017), the biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005), and the adaptive calibration model (Del Giudice, Ellis, & Shirtcliff, 2011). These theories provide conceptual support for the emergence of different levels and patterns of youth neurobiological sensitivity to context models.

The specialization and sensitization hypotheses. The specialization and sensitization hypotheses are drawn from the evolutionary developmental perspective to articulate how individuals adapt to rearing environments by forming specialized cognitive behaviors (Ellis, Bianchi, et al., 2017). The *specialization hypothesis* purports that under harsh, unpredictable, and uncontrollable environments, individuals develop adaptive cognitive abilities that are ecologically appropriate to solve problems that are unique to such environments (Ellis, Bianchi, et al., 2017; Frankenhuis & de Weerth, 2013). These cognitive abilities might be commonly viewed as maladaptive under a normative environment but can facilitate adaptation under adverse and stressful situations (Frankenhuis & de Weerth, 2013; Frankenhuis, Panchanathan, & Nettle, 2016; Lickliter & Honeycutt, 2013). For example, hyperreactive attention shifting among children is commonly regarded as a deficit that interferes with sustained attention and challenges academic performance in school (Daley & Birchwood, 2010). However, this attention shifting can enable youth to take advantage of opportunities that are quickly fleeting in a fast-changing environment, thus can be considered as adaptive in this context (Mittal, Griskevicius, Simpson, Sung, & Young, 2015). Further, the *sensitization hypothesis* suggests that the advantages of these specialized cognitive abilities (e.g., attention shifting) are primarily exhibited under currently stressful environments but not in benign and nonthreatening conditions (Mittal et al., 2015). In other words, individuals' early life experiences sensitize their stress response in later life (Ellis, Bianchi, et al., 2017). For example, youth exposed to adverse rearing environments may develop hyperreactive attention shifting as an evolutionary adaptive cognitive strategy. In their later years of life, this cognitive strategy of attention shifting mainly exhibits when youth experience stressful social and familiar environments (Ellis, Bianchi, et al., 2017)

The biological sensitivity to context theory. The biological sensitivity to context theory proposes an evolutionary model of the emergence of individuals' neurobiological susceptibility (Boyce & Ellis, 2005; Ellis et al., 2005). Accordingly, this theory identified a physiological mechanism through which the biological stress responses regulate individuals' sensitivity to social environments. Aligned with the specification and sensitization hypotheses, the biological sensitivity to context theory suggests that neurobiological sensitivity is shaped by individuals' early life experiences, following a U-shaped curvilinear pattern (Boyce & Ellis, 2005). In this U-shaped curvilinear pattern, both extremely adverse and supportive environments promote elevated neurobiological susceptibility. When exposed to an adverse childhood environment, neurobiological sensitivity is upregulated to enable youth with better abilities to detect danger and threats (Boyce & Ellis, 2005). When experiencing a particularly supportive and nurturing environment, neurobiological sensitivity is also upregulated so that youth can benefit the most from opportunities and resources in their immediate social context (Boyce & Ellis, 2005). In contrast, moderate levels of stress are associated with blunted sensitivity to rearing environments (Boyce & Ellis, 2005; Ellis & Boyce, 2008; Ellis et al., 2005). The downregulated sensitivity to environmental influences can buffer the negative influences of chronic stressors (Boyce & Ellis, 2005; Ellis et al., 2011). According to the biological sensitivity to context theory, individual differences in neurobiological sensitivity are induced by different childhood experiences to facilitate adaptation in fitness outcomes (Ellis et al., 2011).

The adaptive calibration model. The adaptive calibration model extends the biological sensitivity to context theory by positing four stress-response patterns in which conditional adaptation leads to individual differences in the neurobiological susceptibility (Del Giudice et al., 2011). According to the adaptive calibration model, youths' adaptive calibration to early experiences is achieved through adjusting their stress response systems comprising three circuits. These circuits include the sympathetic nervous system (SNS), the parasympathetic system (PNS), and the hypothalamic-pituitary-adrenal (HPA) axis. Four stress-response patterns are proposed in the adaptive calibration model, including sensitive (I), buffered (II), vigilant (III), and unemotional (IV) patterns (Del Giudice et al., 2011; Del Giudice, Hinnant, Ellis, & El-Sheikh, 2012; Ellis, Oldehinkel, & Nederhof, 2017). Under a low-stress environment, children develop a sensitive pattern characterized by high SNS responsivity to make them better detect beneficial opportunities (Del Giudice et al., 2011). In a moderately stressful context, children are more likely to have low SRS responsivity due to a cost/benefit balance (Del Giudice et al., 2011). Under a dangerous and unpredictable environment, children also develop high SRS responsivity so they can be more vigilant to potential harms (i.e., the vigilant pattern). Finally, when experiencing extremely stressful and traumatic situations, youth may develop a dampened SRS responsivity (i.e., the unemotional pattern) characterized by mating-oriented behaviors such as extreme risk-taking and antagonistic competition (Del Giudice et al., 2012). These four patterns proposed by ACM provide an integrative framework for future research in the field of neurobiological sensitivity to social context (Del Giudice et al., 2011).

Adolescent Neurodevelopment Models

Adolescence is a developmental stage characterized by heightened sensitivity towards the influences of social environments (Steinberg, 2008). Recent research has been making significant progress in understanding the associations between brain development and behaviors among adolescents. This body of research is guided by the perspective of *developmental cognitive neuroscience*. Developmental cognitive neuroscience is an evolving interdisciplinary field that investigates the links of developmental changes in neural mechanisms and youths' behaviors and cognitive performance (Baron-Cohen, Tager-Flusberg, & Cohen, 2000; M. H. Johnson, 2013; M. H. Johnson & De Haan, 2015; Munakata, Casey, & Diamond, 2004; Tager-Flusberg, 2013). The developmental cognitive neuroscience perspective integrates research in the field of psychology, neuroscience, developmental science, cognitive science, genetics, and social science (M. H. Johnson & De Haan, 2015; Munakata et al., 2004). The goal of the developmental cognitive neuroscience perspective is to provide a rich understanding of the interconnections of the developmental changes in youths' behaviors, cognitive abilities, emotions, and neural structures and functions (Munakata et al., 2004).

Research in developmental cognitive neuroscience yields prevailing theoretical models to elucidate the neural underpinnings of adolescents' heightened sensitivity and risk-taking behaviors (Casey et al., 2008; M. Ernst, 2014; M. Ernst & Fudge, 2009; Luna & Wright, 2016; Nelson et al., 2005; Steinberg, 2010; Steinberg et al., 2008). These neuroscience conceptual models attribute adolescents' heightened sensitivity to the differential weighing of the inputs from two key brain circuits, namely, the *social-affective* and the *cognitive control* systems (Schriber & Guyer, 2016). Therefore, these models are referred to as the "imbalance models" (Romer, Reyna, & Satterthwaite, 2017). These "imbalance models" provide theoretical support for investigating the amygdala and hippocampus, two major subcortical regions of the social-affective system, as candidate neurobiological sensitivity indicators in this dissertation.

Dual System Model

The advancement in developmental cognitive neuroscience, especially in the understanding of adolescent decision-making and risk-taking behaviors, fostered the emergence of the Dual System Model (Casey et al., 2008; Luna & Wright, 2016; Steinberg, 2010; Steinberg et al., 2008). This model argues that adolescents' risk-taking and impulsive decision-making are the products of the temporal disjoint between the interactions of two neural systems, including the social-affective and the cognitive control circuitries (Casey et al., 2008; Steinberg, 2008). The social-affective system is located at the limbic and paralimbic brain areas, including regions such as the amygdala, ventral striatum, orbitofrontal cortex, superior temporal sulcus, and the medial prefrontal cortex (Casey et al., 2008; Steinberg, 2008). The cognitive control circuit involves the lateral prefrontal and parietal cortices and the anterior cingulate cortex (Casey et al., 2008; Steinberg, 2008). Accordingly, during puberty, the increased dopaminergic activity in the social-affective brain circuitry predisposes youth toward higher levels of reward-seeking. However, the cognitive control system is undergoing slow and gradual maturation across adolescence, which leads to a temporal gap between the functions of the social-affective and the cognitive control systems (Luna & Wright, 2016; Steinberg, 2008). This temporal gap between these two systems accounts for adolescents' emotional and motivational reactions to social cues. Further, these emotional and motivational reactions orient youth towards over-reactivity, risk-taking, and impulsivity instead of self-control (Casey et al., 2008; Luna & Wright, 2016; Steinberg, 2010).

Triadic Model

The *Triadic Model* (M. Ernst, 2014; M. Ernst & Fudge, 2009; M. Ernst, Pine, & Hardin, 2006) is proposed to provide a framework for understanding motivated behaviors in cognitive neuroscience research. This model attributes individuals' motivated behaviors to the coordination and functions of three neural systems, including the *motivation/approach*, the *emotion/avoidance*, and the *regulation* systems (M. Ernst, 2014). The motivation/approach system involves dopaminergic networks mediated by the ventral striatum, which regulates behaviors that youth exert to achieve their goals, such as rewards and positive emotions (M. Ernst et al., 2006). The emotion/avoidance system is represented by the amygdala, which coordinates behaviors relevant to avoidance and negative emotions (M. Ernst et al., 2006). The regulation system exercises cognitive control functions on motivational and emotional neural responses through the functions of the prefrontal cortex (M. Ernst et al., 2006). Both the approach and the avoidance systems can respond to negatively- and positively-valenced stimuli (Gunther Moor, van Leijenhorst, Rombouts, Crone, & Van der Molen, 2010; Guyer et al., 2012; Guyer, Choate, Pine, & Nelson, 2011; Hamann, Ely, Hoffman, & Kilts, 2002; Vasa et al., 2011). Overall, the dynamic equilibrium states of the motivation/approach, emotion/avoidance, and regulation systems provide a framework to explain adolescent-specific behavioral features, such as cognitive impulsivity, risk-seeking, emotional reactivity, and social reorientation (M. Ernst, 2014). During adolescence, the regulation system exhibits a slow and gradual growth. However, the approach and avoidance systems show hypersensitivity to positive and negative stimuli, respectively (M. Ernst & Fudge, 2009; M. Ernst & Spear, 2009). This developmental gap among regulation, approach, and avoidance systems contributes to adolescents' heightened sensitivity to environmental influences (M. Ernst & Fudge, 2009; M. Ernst & Spear, 2009).

Social Re-Orientation Framework

The *Social Re-Orientation Framework* focuses on changes in adolescent social behaviors grounded in the neural social information processing network (SIPN; Nelson et al., 2005). According to this theory, SIPN can be broken down into three nodes, including the *detection*, *affective*, and *cognitive-regulatory* nodes (Nelson et al., 2005). The detection node includes brain regions such as the inferior occipital and temporal cortex, intraparietal sulcus, fusiform face area, superior temporal sulcus, and anterior temporal cortex. It is the first step of social information processing that regulates the perception and categorization of basic social stimuli (Nelson et al., 2005). The affective node involves regions primarily engaged in reward and punishment processing, including the amygdala, ventral striatum, hypothalamus, bed nucleus of the terminal stria, septum, and orbitofrontal cortex. This affective node is responsible for processing social and emotional information passed by the detection node (Nelson et al., 2005). Lastly, the cognitive-regulatory node includes multiple regions of the prefrontal cortex, which cognitively regulates youths' responses to the processed social and emotional information (Nelson et al., 2005). Similar to the Dual-System and the Triadic Model, the Social Re-Orientation framework proposes that the affective node exhibits an upsurge in reactivity during adolescence due to pubertal hormone changes (McEwen, 2001). However, the cognitive-regulatory node maintains a prolonged maturation into early adulthood (Nelson et al., 2005). This imbalanced maturation and reactivity of the affective and cognitive-regulatory nodes, coupled with flexibility in social behaviors, enable some youth to exhibit differential responses under various environmental influences (Nelson & Guyer, 2011).

Overall, these imbalance models of adolescent neurodevelopment provide a conceptual basis for identifying promising candidate neuro-biomarkers of adolescents' neurobiological sensitivity. Indeed, these models converge to suggest that the heightened sensitivity to social and environmental inputs among adolescents is grounded in the temporal disjoint of the social-affective and cognitive control neural systems (Schriber & Guyer, 2016). Through the co-regulation with different neural networks, the social-affective brain circuitry may harbor adolescents' neurobiological sensitivity to rearing environments (Schriber & Guyer, 2016). As the amygdala and hippocampus are two major subcortical regions of the social-affective brain circuitry, these two brain regions can serve as candidate neurobiological sensitivity indicators.

Criticism of the Imbalance Models

It is important to note that these imbalance models, including the Dual System Model, the Triadic Model, and the Social Re-Orientation Framework, bear the criticism of stereotyping adolescents with risk-taking behaviors (Do, Sharp, & Telzer, 2019; Romer et al., 2017). Specifically, Do et al. (2019) argue that adolescent risk-taking may be resultant from, rather than indicating a failure of, effective cognitive functioning. According to the Expected-Value-of-Control (EVC) computational model, decision-making comprises three critical factors. These factors include the expected payoff from the cognitive control, the amount of control required to achieve the payoff, and the cost of the cognitive control (Shenhav, Botvinick, & Cohen, 2013). During a decision-making process, adolescents employ their cognitive processes to evaluate the payoff, amount, and cost of cognitive control. Therefore, the EVC computation model suggests that heightened risk-taking behaviors during adolescence might be explained by the differences in valuation between adolescents and adults, rather than the immaturity of adolescents' cognitive control system (Do et al., 2019).

Similarly, Romer et al. (2017) propose an alternative Lifespan Wisdom Model. This model argues that while sensation-seeking (i.e., attraction to novel and exciting but risky experiences) peaks during adolescence (Romer, 2010; Zuckerman, 2007), the overall risk-taking behaviors and impulsive decision-making decline from childhood to adulthood (Romer, 2010; Van Den Bos, Rodriguez, Schweitzer, & McClure, 2015). This criticism of the imbalance models suggests that the observed high susceptibility for unhealthy risk-taking, reflected by the lack of control and excessive impulsivity among adolescents, can be attributed to individual differences (Romer et al., 2017). Indeed, although some adolescents exhibit excessive impulsivity and risk-taking behaviors, the vast majority of youth grow throughout adolescence without substance use problems, risky sexual behaviors, injuries, and mental health issues (National Research Council, 2011; Willoughby, Good, Adachi, Hamza, & Tavernier, 2014).

Literature Review

Family environments and Youth Development

The caregiving experiences in the family environments are robust predictors of youth socioemotional and behavioral adjustment (Kogan et al., 2010; Kogan et al., 2019; A. S. Masten, 2001; Steinberg et al., 2008; Steinberg & Morris, 2001; Wickrama & Kaspar, 2007). During the early years of life, from infancy through adolescence, youth spend most of their time and form tightest social interactions in the family rearing environments. Thus, the family serves as the primary source for shaping youths’ developmental outcomes (Bronfenbrenner, 2005; Bronfenbrenner & Morris, 2006). The family system comprises multidimensional characteristics and dynamic interpersonal relationships among family members, including parenting behaviors and style, quality of parent-child interactions, and family climate (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Schriber & Guyer, 2016; Steinberg, 2008). The present dissertation focused on three components of the family relationship quality, including family conflict, parenting warmth, and family functioning.

Despite the well-established connections between rearing environments and youth adjustment, not every adolescent is equally responsive to a given environmental stimulus or condition. Indeed, there is a striking variation in youths' sensitivity to the impact of caregiving experiences (Luthar, 2006; A. S. Masten & Obradović, 2006). According to the multifinality concept in the developmental psychopathology perspective (Cicchetti & Rogosch, 1996), a similar early rearing environment can result in multiple possible outcomes through different developmental pathways. For example, even though youth reared in disadvantaged families are at high risk for psychopathology, most youth who experienced early life stress survived and thrived from the adverse environment and demonstrated competence and resilience (A. S. Masten, 2001; A. S. Masten & Obradović, 2006). Understanding the mechanisms underlying this heterogeneity of sensitivity to family rearing environments among youth is crucial to advance knowledge in developmental psychopathology and positive youth development.

Youth Sensitivity to Rearing Environments: The Behavioral and Biological Indicators

The evolutionary developmental perspectives and neurobiological methodologies provide an opportunity for researchers to more precisely examine the neurobiological origins that underlie youths' differential responses to environmental inputs (Bjorklund & Pellegrini, 2002; Ellis et al., 2011; Witherington & Lickliter, 2016). The evolutionary developmental theories propose three models that could describe the patterns of environmental influences on youth adjustment. These theories include the diathesis-stress (Monroe & Simons, 1991; Zuckerman, 1999), biological sensitivity to context (Boyce & Ellis, 2005; Ellis et al., 2005), and vantage sensitivity (Pluess & Belsky, 2013) models.

Historically, the diathesis-stress model is proposed in concordance with the prevailing psychopathology analysis focusing on developmental maladaptation and early adversity (Ellis et al., 2011). This model suggests that some youth with vulnerability factors are disproportionally more susceptible to adverse rearing environments. However, others lack such vulnerability and are resilient to early adversity (Luthar, 2006; A. S. Masten & Obradović, 2006; Zuckerman, 1999). Despite some empirical support, this model bears significant limitations of ignoring youth development under positive rearing environments. To resolve this issue, modern theories, such as the biological sensitivity to context theory, are developed to encompass both negative and positive aspects of youth development (Boyce & Ellis, 2005; Ellis et al., 2005). The biological sensitivity to context theory purports that some sensitivity indicators enable youth to be more responsive to both negative (stress-promoting/developmental-vulnerability) and positive (development-enhancing) environments (Boyce & Ellis, 2005; Ellis et al., 2005). Further, the vantage sensitivity model is developed to reflect the “bright side” of youth development. The vantage sensitivity theory implies that some vantage-sensitivity can intensify youths’ the beneficial effects of a supportive environment on youth adjustment (Pluess & Belsky, 2013).

Despite the different focus on negative and positive components of youth development, these three models converge in suggesting that individuals’ sensitivity to environmental influences is grounded in the neurobiological interactions with caregiving experiences. Indeed, these neurobiological sensitivity models receive empirical support from behavioral phenotypes (Rothbart & Bates, 2007), genetic (Bakermans-Kranenburg & Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013; Caspi, Hariri, Holmes, Uher, & Moffitt, 2010), and psychophysiological research (Molly Davis, Suveg, Whitehead, Jones, & Shaffer, 2016; Obradović et al., 2010; Oshri et al., in press; Skowron, Cipriano‐Essel, Gatzke‐Kopp, Teti, & Ammerman, 2014). These factors shape adolescents’ perception and responsivity to environmental influences, change their behaviors, and ultimately moderate the influences of rearing environments on their development of psychopathology and competencies (Boyce & Ellis, 2005; Schriber & Guyer, 2016).

Behavioral phenotypes. Children's temperament is defined as constitutionally-based heterogeneity in emotional reactivity and self-regulation (Rothbart & Bates, 2007). Temperament is the most commonly-used behavioral indicators to youths' sensitivity to social context (Chen et al., 2019; Cruz et al., 2018; Gao, Ding, Feng, & Xing, 2018; Slagt et al., 2019; Xing, Gao, Liu, Ma, & Wang, 2018). Emotional reactivity reflects youths' threshold, intensity, and duration of emotional arousal to environmental changes, and is considered as the core behavioral facet of this sensitivity (Aron, Aron, & Jagiellowicz, 2012; Slagt, Dubas, van Aken, Ellis, & Deković, 2017). Children with difficult temperament (usually characterized as high emotional reactivity and negative affectivity) tend to show extended, intenser, and more frequent emotions in response to environmental input (Slagt et al., 2019). These youth with difficult temperament are usually more responsive to both positive and negative environmental influences. Aligned with studies examining the temperament by parenting interactions (Rothbart & Bates, 2007), research shows that high emotional reactivity and difficult temperament can exacerbate the effects of family relationship quality on a variety of developmental outcomes. These outcomes include social competence (Leerkes, Blankson, & O'Brien, 2009), behavioral problems (Gao et al., 2018; Xing et al., 2018), academic achievement (Curby, Rudasill, Edwards, & Pérez-Edgar, 2011), cognitive functioning (Raver et al., 2013), and self-regulation abilities (Cruz et al., 2018).

Genotypes. The genetic differential sensitivity to social environment (GDSE) model suggests that individuals with specific genetic makeup are more sensitive to both favorable and adverse environments (Mitchell et al., 2013). Most empirical studies that employ the GDSE model focus on the dopaminergic and serotonergic neural systems (Bakermans-Kranenburg &

Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013; Caspi et al., 2010). As neurotransmitters, dopamine and serotonin convey chemical signals among neurons and regulate individuals' emotions, motivations, reward-processing, attention, and learning (Brischoux, Chakraborty, Brierley, & Ungless, 2009; Caspi et al., 2010; Fan & Sklar, 2005; Propper, Willoughby, Halpern, Carbone, & Cox, 2007; Ungless, Magill, & Bolam, 2004; Williams et al., 2003; Young et al., 2002; Zald et al., 2008). Relevant dopamine-related genes include dopamine receptor D2 (DRD2), dopamine receptor D4 (DRD4), and dopamine-transporter (DAT). In a meta-analysis, Bakermans-Kranenburg and Van Ijzendoorn (2011) report supportive evidence to GDSE by synthesizing 15 effects (*N* = 1,232). This meta-analysis finds significant effect sizes between adverse rearing environment and behavioral problems (*r* = .37), and between parental support and positive developmental outcomes (*r* = .31), among youth who carry the high-sensitivity alleles. Another meta-analysis investigates the serotonin transporter genotype 5HTTLPR as a sensitivity marker among children and adolescents (Van IJzendoorn et al., 2012) based on 41 effect sizes (*N* = 5,863). This meta-analysis suggests that 5HTTLPR intensifies youths' susceptibility to environmental influences among Caucasian samples, thus also supports the GDSE model (Van IJzendoorn et al., 2012.

Psychophysiology and the stress response system. According to the biological sensitivity to context theory, youths' stress reactivity is a critical mechanism underlying their neurobiological sensitivity (Boyce & Ellis, 2005; Ellis et al., 2005). Stress reactivity is physiologically regulated by two stress response systems, including the autonomic nervous system (ANS) and the hypothalamus-pituitary-adrenal (HPA) axis. These two systems form counter-regulatory circuits, which control physical arousal as responses to acute stressors (Boyce & Ellis, 2005). Specifically, the ANS regulates the immediate "fight or flight" response, which initiates physical changes, including heart and respiratory rate accelerations and sweat production (Berntson, Cacioppo, & Quigley, 1993a, 1993b). ANS reactivity is usually reflected by several biomarkers such as heart rate variability (HRV), galvanic skin response (GSR), and pre-ejection periods (PEP). HPA axis activation is a lagged response that occurs within minutes after the acute stress, which regulates blood pressure, glucose metabolism, and immunity via glucocorticoid (e.g., cortisol) secretion (Sapolsky, Romero, & Munck, 2000). Thus, HPA axis activation is usually indicated by cortisol reactivity in response to acute stressors. The literature yields compelling evidence that supports ANS and HPA axis reactivity as neurobiological sensitivity biomarkers. These physiological stress response indicators modify youth responses' when exposed to differential environments, and further affect their socioemotional adjustment. Specifically, research shows that youths' heightened stress reactivity intensifies the influences of rearing environments on youths' developmental outcomes (Molly Davis et al., 2016; Obradović et al., 2010; Oshri et al., in press; Skowron et al., 2014).

Neural Functioning as Sensitivity Indicators: The Roles of the Amygdala and Hippocampus

Extant literature that supports the neurobiological sensitivity to context models mostly focuses on behavioral phenotypes (Rothbart & Bates, 2007), genetic variants (Bakermans-Kranenburg & Van Ijzendoorn, 2011), and psychophysiological processes (Molly Davis et al., 2016; Obradović et al., 2010; Oshri et al., in press; Skowron et al., 2014). However, direct measures of brain structure and functions as mechanisms underlying this differential sensitivity are left largely unexamined (Schriber & Guyer, 2016). The focus on the neural mechanisms is particularly necessary because individuals' neurobiological sensitivity is regulated by the brain. Indeed, the brain directly and interactively (with genetic and environmental factors) determines physiological processes and human behaviors (Pluess, 2017). Emerging adolescent neurodevelopment models (Casey et al., 2008; M. Ernst, 2014; M. Ernst & Fudge, 2009; Luna & Wright, 2016; Nelson et al., 2005; Steinberg, 2010; Steinberg et al., 2008) suggest the social-affective brain circuitry as a promising candidate brain circuitry that might mediate youths' differential responses under the influences of family interactions. However, there are sparse empirical studies that directly test the roles of the social-affective brain circuitry as neurobiological sensitivity indicators (Schriber & Guyer, 2016). This lack of investigation on the neural underpinnings of youth sensitivity to environmental influences is a major gap in the literature.

To fill this gap, this dissertation aimed to examine how youths' social-affective neural responses to emotional stimuli affect their positive and negative developmental outcomes under family environments influences. The social-affective circuitry includes two key subcortical brain regions—the amygdala and hippocampus—which could mediate youths' neurobiological sensitivity (Schriber & Guyer, 2016) via modulating emotional and stress reactivity (Baxter & Murray, 2002; Phelps, 2004). Therefore, the current dissertation focused on testing amygdalar and hippocampal responses to emotional processing as neurobiological sensitivity indicators that moderated the associations between family environments and youth adjustment.

Overview of the amygdala.

Amygdalar structure. The amygdala is bilateral almond-shaped subcortical nuclei (i.e., a discrete mass of gray matter formed by a cluster of neurons in the central nervous system) that is located in the frontal portion of the temporal lobe (Carlson, 2012; LeDoux, 2000). Structurally, the amygdala is divided into three subregions, including the basolateral amygdala groups, the cortical-like groups, and the centromedial groups (Sah, Faber, Lopez de Armentia, & Power, 2003; Yang & Wang, 2017). The basolateral amygdala groups can be further divided into the lateral nucleus, the basal nucleus (including both anterior and posterior part), and the basomedial nucleus. The cortical-like groups include the nucleus of the lateral olfactory tract and the cortical nuclei. The centromedial groups comprise the medial and central nuclei (Sah et al., 2003). The amygdala receives highly processed sensory input from the hippocampus, thalamus, and cerebral cortex (Carlson, 2012). Specifically, the basolateral nuclei receive higher-order cortical input, and the corticomeidal nuclei receive information from basic systems such as the thalamus and the autonomic nervous system (Pitkänen et al., 1995; Yang & Wang, 2017). The information processed by the amygdala is then relayed to a broad range of brain regions such as orbital frontal cortex, hippocampus, dorsal and ventral striatum (Michael Davis & Whalen, 2001). This information can be further used for emotional perception, integration into cognitive functions such as decision-making, providing valence and salience that motivates learning, and influencing the arousal of the autonomic nervous system as basic emotional responses (Brodal, 2016).

Amygdalar functions. The amygdala is a critical subcortical region in which emotional processing occurs (Dolan, 2002; LeDoux, 1998; Phillips, Drevets, Rauch, & Lane, 2003a, 2003b), especially the perception of emotional salience of social stimuli (Michael Davis & Whalen, 2001). Traditionally, researchers indicate that the amygdala is a hub of emotional responses to *aversive* stimuli, especially fear (Carlson, 2012; Michael Davis & Whalen, 2001). Animal studies suggest the neural transmissions in the amygdala is a major mechanism through which an aversive stimulus triggers fear responses. These fear responses are reflected in somatic (e.g., sweating, trembling), autonomic (e.g., increased heartbeat), and endocrine (e.g., increased cortisol reactivity; a stress hormone) signs (Michael Davis & Whalen, 2001). In other words, the activations of the amygdala project to other brain regions and initiate specific behavioral and physiological symptoms of fear (Michael Davis & Whalen, 2001). Neuroimaging studies in human samples further confirm the presence of elevated amygdalar activations when individuals are experiencing aversive conditions (e.g., Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000; Hamann & Mao, 2002).

Recent research, however, reports that the amygdala is responsive to not only aversive social situations but also *positively valenced* stimuli, such as happy facial expressions (Garavan, Pendergrass, Ross, Stein, & Risinger, 2001; Hennenlotter et al., 2005; Somerville, Kim, Johnstone, Alexander, & Whalen, 2004; Weymar & Schwabe, 2016). Taken together, the amygdala shows elevated activations when individuals are experiencing both aversive and positive emotional processing. Further, research suggests that the amygdala regulates individuals' positive and negative emotional processing through modulating attention and vigilance (Michael Davis & Whalen, 2001; P. C. Holland & Gallagher, 1999). Under aversive conditions, amygdalar responses to fear enable individuals with higher vigilance to dangerous social situations so they can avoid potential harm (Tottenham, Hare, & Casey, 2009). When exposed to positive stimuli, heightened amygdalar responses reflect individuals' elevated attention so that they can benefit the most from the supportive social context (Weymar & Schwabe, 2016). Overall, the amygdala is related to individuals' learning about the emotional significance of the general social environment (Tottenham & Sheridan, 2010). Therefore, the amygdala harbors individuals' information processing of social interactions and relationships, such as social perception, affiliation, and aversion (Bickart, Dickerson, & Barrett, 2014). Conversely, the amygdala also plays an important role in the psychological and psychiatric disorders associated with emotion (Clark, Sweet, Morgello, Philip, & Cohen, 2017).

The amygdala is also involved in regulating stress responses by directly modifying the activities of the ANS and the HPA axis (Herman, Ostrander, Mueller, & Figueiredo, 2005). Research shows the amygdala can activate the HPA axis stress responses via its projections to the basal forebrain, hypothalamus, and brainstem (Swanson & Petrovich, 1998). Additionally, the amygdala can also activate the sympathetic branch of the ANS via its connections with the hypothalamus (Pessoa, 2010). Overall, the amygdala is critical for individuals' emotional and stress reactivity and regulation (Ueyama, 2012).

Lateralization of amygdalar functions. Research suggests that significant lateralization (i.e., the tendency for some brain functions to be specialized to one side/hemisphere of the brain) exists in the amygdala. Functionally, the right amygdala is more engaged in immediate, autonomic, and global (i.e., holistic) emotional responses (Cahill, 2003; Gläscher & Adolphs, 2003). In contrast, the left amygdala is more involved in local (i.e., fine-grained details) and elaborate information processing (Gläscher & Adolphs, 2003; Markowitsch, 1999; Wright et al., 2003). Researchers have conducted several meta-analyses to analyze lateralized amygdalar functions (Baas, Aleman, & Kahn, 2004; Fusar-Poli et al., 2009; Murphy, Nimmo-Smith, & Lawrence, 2003; Sergerie, Chochol, & Armony, 2008; Wager, Phan, Liberzon, & Taylor, 2003). The majority of these meta-analyses report a larger proportion of activation foci in the left amygdala compared to the right amygdala. These findings suggest that activations are reported more often in the left amygdala when participants are presented with emotional stimuli (Baas et al., 2004; Murphy et al., 2003; Sergerie et al., 2008; Wager et al., 2003). However, the comparison of activation effect sizes indicates no statistically significant difference between the right and left amygdala (Fusar-Poli et al., 2009; Sergerie et al., 2008). This finding suggests that, when activations are reported, the magnitude of responses is similar for the left and right amygdala (Sergerie et al., 2008).

Furthermore, the lateralization of amygdalar activations is influenced by other factors such as experimental design, valence, and gender (Sergerie et al., 2008; Wager et al., 2003). For example, Sergerie et al. (2008) synthesize 148 studies and report that lateralized amygdalar activation only appears in block-design experiments rather than event-design paradigms. Wager et al. (2003) aggregate 65 studies and report that the elevated left amygdalar activations are particularly significant for negatively valenced stimuli. This study also finds significant gender X brain-hemisphere effects by showing that males exhibit greater lateralization of amygdalar activations (i.e., more significant hemisphere differences between the left and right amygdalar activations) compared to females (Wager et al., 2003). Overall, these studies on lateralized amygdalar activations converge to suggest that the left amygdala is more likely to activate in responses to emotional stimuli (Baas et al., 2004; Murphy et al., 2003; Sergerie et al., 2008; Wager et al., 2003). However, this lateralization of amygdalar responses may be influenced by other study methods and sample characteristics such as gender, stimuli valence, and experiment design (Oshri, Gray, et al., 2019; Sergerie et al., 2008; Wager et al., 2003).

Overview of the hippocampus.

Hippocampal structure. The hippocampus is bilateral subcortical nuclei located in the temporal lobe, close to the amygdala. The hippocampus and is the central region of the hippocampal formation along with other areas such as the dentate gyrus, subiculum, and entorhinal area, and comprises subfields, including the CA1, CA2, CA3, and CA4 (Brodal, 2016). Hippocampal formation receives input from multiple cortical (e.g., sensory and motor associations cortexes) and subcortical (e.g., ventral tegmental area, amygdala, and medial septal nucleus) regions. These inputs first arrive in the entorhinal cortex and then are forwarded to the dentate gyrus and the hippocampus. Information processed by the hippocampus is sent reciprocally to the input cortical and subcortical regions via the subiculum and the entorhinal cortex (Carlson, 2012).

Hippocampal functions. The hippocampus is a critical region for learning, memory, stress response, and emotional regulation. First, the hippocampus is responsible for memory consolidation, through which explicit memories (i.e., the memory of facts and events that individuals can consciously recall) are formed (Manns, Hopkins, & Squire, 2003). In other words, the hippocampus is responsible for converting short-term memories to long-term explicit memories. However, the hippocampus is not involved in the formation of implicit memories (i.e., memory that is not consciously recalled, such as skills; Carlson, 2012). For example, patients with bilateral lesions of the hippocampus cannot learn new explicit information but have intact short-term and implicit memories (Eichenbaum, 2004). Even though the mechanisms underlying the formation of explicit memories remain unclear, researchers believe that the hippocampus modifies and links essential elements of memories so that individuals can easily remember them (Carlson, 2012). These mechanisms may be achieved through hippocampal efferent connections (i.e., outwards or away from the hippocampus) with subcortical areas such as the amygdala and the basal ganglia (Brodal, 2016). Research shows that the hippocampus is responsible for consolidating the emotional elements of explicit memories (Manns et al., 2003)

Another function of the hippocampus is regulating stress responses through a negative feedback mechanism that modifies the HPA axis response to acute stressors (Mizoguchi, Ishige, Aburada, & Tabira, 2003; Wingenfeld & Wolf, 2014). When an individual is experiencing acute stressors, the HPA axis gets activated and induces a neuroendocrine cascade that culminates in the synthesis and secretion of glucocorticoids (Herman, McKlveen, Solomon, Carvalho-Netto, & Myers, 2012). Among human beings, the glucocorticoids are primarily cortisol, which is a steroid hormone that facilitates energy redistribution to promote the individual's survival capacity when facing stressors (Herman et al., 2005). After secretion, the glucocorticoids subsequently bind with glucocorticoid receptors in the brain and forms a negative feedback inhibition of its own production (Evanson, Tasker, Hill, Hillard, & Herman, 2010; Gómez, De Kloet, & Armario, 1998). The hippocampus is engaged in this negative feedback by expressing glucocorticoid receptors and inhibiting stress responses (Herman, 1993).

The hippocampus can also interact with the amygdala and regulate individuals' emotional responses and the encoding of emotional memories (Phelps, 2004; Richardson, Strange, & Dolan, 2004; Richter-Levin & Akirav, 2000). In particular, the amygdala moderates the hippocampal formation of declarative memories through elevating the attention and vigilance towards the emotional component of these memories (Fox, Russo, Bowles, & Dutton, 2001; Öhman, Flykt, & Esteves, 2001). The amygdala also regulates the secretion of stress hormones on hippocampal memory consolidation (McGaugh & Roozendaal, 2002). Reciprocally, the explicit memories formed through the hippocampus alters amygdalar responses to emotional stimuli (Ochsner, Bunge, Gross, & Gabrieli, 2002; S. M. Schaefer et al., 2002). Indeed, the associations between the amygdala and hippocampus during an individual's encoding of memories with emotional significance might be bi-directional and reciprocal (Richardson et al., 2004).

The hippocampus is one of only a few brain regions where neurogenesis (e.g., producing new neurons) occurs throughout life-span development (A. Ernst et al., 2014). Neurogenesis is essential for hippocampal functions in memory formation and emotional regulation (Deng, Aimone, & Gage, 2010; Kirby et al., 2012; Winocur, Wojtowicz, Sekeres, Snyder, & Wang, 2006; Zhao, Deng, & Gage, 2008). Particularly, adolescents present up to four times higher neurogenesis rate compared to adulthood (Cowen, Takase, Fornal, & Jacobs, 2008; He & Crews, 2007; Knoth et al., 2010; Kuhn, Dickinson-Anson, & Gage, 1996; Spalding et al., 2013). Because of the fast neurogenesis rate, adolescence is a key developmental phase for researchers to investigate how the functions of the hippocampus affect youths' socioemotional development.

Lateralization of hippocampal functions. Research documents significant lateralization of hippocampal functions. Specifically, the lateralization of hippocampal functions in consolidating explicit memories is associated with memory types (Klur et al., 2009; Maguire, 2001). Explicit memories include both spatial and verbal memories. Spatial memory is the memory responsible for the recording of information about an individual's environment and spatial orientation (Bannerman et al., 2014; Broadbent, Squire, & Clark, 2004; Brodal, 2016), which is localized to the right hippocampus (Klur et al., 2009; Maguire, 2001). The left hippocampus, in contrast, specializes in the consolidation of verbal memories, which refers to the memory of verbally presented information (Bonner-Jackson, Mahmoud, Miller, & Banks, 2015; de Toledo-Morrell et al., 2000; Kelley et al., 1998). Furthermore, empirical studies also show that the links among hippocampal functions, early life experiences, and the development of stress-related psychopathology are particularly lateralized to the left brain hemisphere (MacMaster & Kusumakar, 2004; Schriber et al., 2017; Vythilingam et al., 2002; Whittle et al., 2013). It is possible that the greater expressions of corticosteroid receptors on the left hippocampus render the left brain hemisphere to be more engaged in regulating the HPA axis stress responses, compared to the right hippocampus (Hou, Yang, & Yuan, 2013; Madsen et al., 2012).

The links between the amygdala and hippocampus and other neurobiological sensitivity indicators. The functions of the amygdala and hippocampus are highly interconnected with the behavioral, genetic, and physiological processes that have been demonstrated as sensitivity indicators. Behaviorally, amygdalar (Schwartz, Wright, Shin, Kagan, & Rauch, 2003) and hippocampal (Sakai et al., 2005) functions are linked to youths' negative affectivity, a characterizing trait of difficult temperament among youth. Genetically, the amygdala and hippocampus are involved in the dopaminergic and serotonergic neural pathways that are regulated by dopamine-related genes (e.g., DRD2, DRD4, and DAT) and serotonin transporter gene 5HTTLPR, respectively (Carlson, 2012). These genetic make-ups are also reported as robust sensitivity indicators that can interact with the family environments and affect youths' developmental outcomes (Bakermans-Kranenburg & Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013; Van IJzendoorn et al., 2012). Physiologically, research also reports close relations and co-regulations between the amygdala and hippocampus and stress response systems, including the HPA axis (Herman et al., 2005) and the ANS (Buijs & Van Eden, 2000; DiCara, 2012). Overall, the hippocampus and amygdala may be the loci of youths' neurobiological sensitivity to environmental influences (Schriber & Guyer, 2016). The amygdala and hippocampus can interact with the genetic and ecological factors to regulate individuals' physiological processes and behaviors (Baxter & Murray, 2002; Phelps, 2004).

The amygdala and hippocampus as neurobiological sensitivity indicators: structural evidence. Brain structure has a strong genetic basis (Jansen, Mous, White, Posthuma, & Polderman, 2015), and may serve as a robust sensitivity factor that reflects youths' differential responses to the influences of family rearing environments. Emerging empirical studies identify amygdalar and hippocampal morphometry as among the most promising candidate neural susceptibility factors (Schriber & Guyer, 2016). As suggested by the social brain hypothesis, greater volumes of social-affective brain circuitry regions suggest better processing capacity, thus may indicate heightened sensitivity among adolescents (Dunbar, 2009). Specifically, the large amygdalar volume can reflect an elevated engagement of the amygdala in generating negative affect when youth were reacting to negative social cues (Whittle et al., 2008). Large hippocampal volume, similarly, can support more complex social interactions (Dunbar, 2009) and cognitive functions during emotionally salient situations (Østby, Tamnes, Fjell, & Walhovd, 2011; Redondo et al., 2014). Thus, the larger hippocampal volume may render a higher HPA axis response to acute social stressors (Pruessner et al., 2005). Therefore, the social brain hypothesis suggests that large volumes of the amygdala and hippocampus might confer greater neurobiological sensitivity to social context among adolescents (Dunbar, 2009).

To date, only a few studies exist that examine amygdalar and hippocampal morphometry as direct indicators to youths’ neurobiological sensitivity (Schriber et al., 2017; Whittle et al., 2011; Yap et al., 2008). These studies mostly focus on adolescents’ development of depressive symptoms under the influence of family relationship quality. For example, Whittle et al. (2011) examine hippocampal volume as a moderator of the associations between maternal aggression and adolescents’ depressive symptoms. This study supports the biological sensitivity to context model. Specifically, girls with larger bilateral hippocampal volumes are more responsive to both positive and negative effects of low and high maternal aggression levels, respectively. Similarly, Schriber et al. (2017) also support the biological sensitivity to context theory by reporting that hippocampal volume interacts with family connectedness and community crime and affects Mexican-origin adolescents’ depressive symptoms. Adolescents with large left hippocampal volume, particularly, exhibit higher sensitivity to the protective effect of family connectedness.

However, with a larger left hippocampus, youth also exhibit elevated vulnerability to the negative effects of community crime. In another study, Yap et al. (2008) find significant sex differences in the link between aggressive maternal behaviors and adolescents' depressive symptoms with amygdalar volume as a moderator. This study suggests that girls with smaller bilateral amygdala exhibit a biological sensitivity to context pattern. Specifically, girls who have smaller bilateral amygdala and experience low maternal aggression report fewer depressive symptoms. However, with a small amygdala, girls who report high levels of maternal aggression evince high levels of depressive symptoms. In contrast, boys with larger right amygdala present a vantage sensitivity pattern. In particular, boys with larger right amygdala and report low maternal aggression show reduced depressive symptoms under low maternal aggression. However, when reporting high levels of maternal aggression, the right amygdala volume is not significantly associated with boys' depressive symptoms.

These findings shed light on the roles of amygdalar and hippocampal morphometry as candidate indicators to youths' neurobiological sensitivity. However, more empirical research is needed to clarify some discrepancies in this body of research. First, significant sex differences and lateralization of amygdalar and hippocampal functions are found in these studies and require further investigation. Second, even though large hippocampal volume seems to confer high neurobiological sensitivity, it remains unclear whether small or large amygdalar volume serves as a sensitivity indicator. It also remains unclear whether amygdalar morphometry mediates youths' sensitivity in the biological sensitivity to context or vantage sensitivity pattern. Additionally, all three studies focus on adolescents' depressive symptoms, which only capture a small part of the whole picture of youth development under the influence of the family environments. More studies that test different aspects of developmental outcomes (both positive and negative) are needed for researchers to comprehensively understand how amygdalar and hippocampal structures account for adolescents' neurobiological sensitivity to differential inputs from family interactions.

The amygdala and hippocampus as neurobiological sensitivity indicators: functional evidence. Compared to brain structures, brain functions reflects more immediate and direct responses to perceived environmental influences (Schriber & Guyer, 2016). The activations of amygdala and hippocampus during youths' reactions to socioemotional stimuli can represent their neurobiological sensitivity to rearing environments. Specifically, increased amygdalar and hippocampal reactivity could indicate higher neurobiological sensitivity (McLaughlin et al., 2014; Swartz, Knodt, Radtke, & Hariri, 2015). Activations of brain regions of interest (ROIs) are commonly measured through functional magnetic resonance imaging (fMRI) using experimental tasks with conditions designed to elicit ROI responses to certain stimuli. fMRI measures the activations of brain regions indirectly through blood-oxygen level-dependent (BOLD) signals (i.e., levels of oxygen in the brain's blood vessels). Theoretically, the BOLD signal is based on the fact that elevated reactivity in particular brain regions stimulates blood flow and thus increases the local blood oxygen to the de-oxyhemoglobin ratio (Carlson, 2012). Using fMRI BOLD signals, amygdalar and hippocampal functions of regulating emotional reactivity and stress responsivity can be indirectly assessed through their activations in response to positively and negatively valenced emotional stimuli (Tottenham, Tanaka, et al., 2009). Further, these activations might indicate youths' neurobiological sensitivity to both supportive and adverse environmental influences (Schriber & Guyer, 2016).

Limited empirical research exists on directly examining amygdalar and hippocampal activations as neurobiological sensitivity indicators (Admon et al., 2009; McLaughlin et al., 2014; Swartz et al., 2015). Most of these studies take advantage of unique and stressful social events such as natural disasters or terrorist attacks. For example, Swartz et al. (2015) investigate how amygdalar reactivity to emotional stimuli interacts with natural disaster experiences and predicts children's internalizing and externalizing problems. The findings of this study support the biological sensitivity to context model. Specifically, heightened neural activations to unpleasant emotional stimuli exacerbate the impact of hurricane-related stress on youths' externalizing problems. Additionally, heightened neural reactivity to pleasant emotional stimuli protects youth from the negative influences of hurricane-related stress.

Additionally, McLaughlin et al. (2014) examine how amygdalar and hippocampal reactivity to negative stimuli predicts the onsets of posttraumatic stress disorder (PTSD) symptoms after the 2013 Boston Marathon terrorist attack. This study reports significant associations between increased PTSD symptoms and left amygdalar activations during negative emotional processing. Similarly, Admon et al. (2009) investigate brain responses and stress experiences among Israeli Defense Forces soldiers. This study finds that increased amygdalar and hippocampal reactivity to stress-related stimuli is associated with elevated PTSD symptoms over time. The findings of these two studies (Admon et al., 2009; McLaughlin et al., 2014) are aligned with the diathesis-stress model by suggesting amygdalar and hippocampal reactivity to negative emotional information as vulnerability biomarkers to PTSD symptoms. Overall, these studies indicate that amygdalar and hippocampal activations in response to emotionally-valenced social stimuli are promising candidate indicators to adolescents' neurobiological sensitivity (Admon et al., 2009; McLaughlin et al., 2014; Swartz et al., 2015).

Existing yet limited research provides preliminary support on the roles of amygdalar and hippocampal functions as neurobiological sensitivity factors. However, more studies are needed to clarify how the activations of the amygdala and hippocampus during emotional processing can account for this sensitivity. First, research that directly tests the neurobiological sensitivity models by capturing the full spectrum (from negative to positive) of family environmental context is lacking. Existing studies in this body of research (e.g., Admon et al., 2009; McLaughlin et al., 2014; Swartz et al., 2015) mostly utilize samples from unique and high-stress social settings (e.g., natural disaster, terrorist attacking) and fail to assess youths' sensitivity to supportive social environments. To accurately test and differentiate the mechanisms underlying adolescent neurobiological sensitivity (i.e., diathesis-stress, biological sensitivity to context, and vantage sensitivity patterns) under family influences, researchers need to consider utilizing adequate measurement instruments that can capture both positive and negative extremes of the family rearing environments (Boyce & Ellis, 2005; Ellis et al., 2011; Ellis et al., 2005). To reveal the comprehensive picture of adolescent development, the influences of environmental context on both positive and negative developmental outcomes should also be incorporated in empirical research. Additionally, given that the amygdala and hippocampus exhibit reactivity to both aversive and positive social stimuli (Hennenlotter et al., 2005; Somerville et al., 2004), it is necessary to incorporate both unpleasant and pleasant emotional situations in the fMRI experiment paradigm to elicit amygdalar and hippocampal activations (Schriber & Guyer, 2016). Further, more studies are needed to clarify the sex differences and lateralization of amygdalar and hippocampal functions in the neurobiological sensitivity models.

Sex Differences

Biological sex differences in youth adjustment. The biological sex differences in youth socioemotional adjustment are widely assessed and established in developmental research (Chaplin & Aldao, 2013). Research suggests that male youth present more externalizing problems while females exhibit increased internalizing problems (e.g., Gleason et al., 2011; Henrichs et al., 2013; LaGasse et al., 2012). A meta-analysis that investigates sex differences in youth's emotional expression also supported this robust sex differences (Chaplin & Aldao, 2013). This study analyzes 555 effect sizes from 166 studies (*N* = 21,709) and reports elevated internalizing emotions (e.g., sadness, anxiety; *g* = -.10) among girls and increased externalizing emotions (e.g., anger; *g* = .09) among boys with small but significant effect sizes. Additionally, female youth tend to exhibit higher levels of prosocial behaviors (i.e., behaviors and intentions to benefit other people or society as a whole) than male youth (Kuhnert, Begeer, Fink, & de Rosnay, 2017).

The significant and consistent sex differences in youths' socioemotional and behavioral development are attributed to the interactions of biological differences and gender-specific socialization experiences (L. R. Brody, 2000; L. R. Brody & Hall, 2008; Chaplin & Aldao, 2013). Specifically, the biologically-based temperamental predispositions stem from differences in genetic expressions and the influences of sex hormones. These biological differences further contribute to the brain and behavior distinctions between males and females (Zahn-Waxler et al., 2008). Meanwhile, youth develop behaviors that are consistent with gender roles through socialization experiences to meet gender-specific social expectations (Liben & Bigler, 2002). Under these social expectations, females are anticipated to be more emotionally expressive (L. R. Brody & Hall, 2008) and to display more empathetic behaviors (Zahn-Waxler, 2000). In contrast, male youth are allowed to express externalizing emotions such as anger (L. R. Brody & Hall, 2008). Overall, the combination of innate predispositions (based on genetic expressions and hormones) and socialization experiences from parents, teachers, and peers contribute to the development of sex differences in youths' socioemotional and behavioral adjustment (L. R. Brody, 2000).

Biological sex differences in neurobiological sensitivity. Biological sex differences are also consistently documented in neuroimaging studies. Structurally, male youth are found to have a larger amygdala and a smaller hippocampus compared to female youth (Durston et al., 2001). Functionally, amygdalar responses to emotional stimuli are more salient among males compared to females (Fine, Semrud-Clikeman, & Zhu, 2009; Wrase et al., 2003). These neural sex differences are induced by genetic factors and hormone manipulations (Kret & De Gelder, 2012), which evolutionarily promote youth’s adaptations to a presumed social structure with specific gender expectations (Vigil, 2008).

Furthermore, sex differences also emerge in studies that investigate the neurobiological sensitivity models using neuroimaging methods. For example, while examining amygdalar volume as a sensitivity indicator, Yap et al. (2008) find that smaller bilateral amygdala represents higher levels of sensitivity among girls, whereas larger right amygdala indexes boy’s elevated sensitivity. In another study, Whittle et al. (2011) report that girls with larger bilateral hippocampal volumes are more responsive to both positive and negative effects of low and high maternal aggression levels, respectively. However, the directionality of biological sex effects still remains unclear because of the limited neuroimaging studies in neurobiological sensitivity models, and thus requires further investigation.

Aims and Overview of Studies

In light of the above literature review and discussed theories, this dissertation addressed gaps in research on the neural underpinnings of youths’ neurobiological sensitivity to the influences of positive and negative family environments. Specifically, this dissertation examined how positive and negative family rearing environments interacted with amygdalar and hippocampal functions and affected youth adjustment. Amygdalar and hippocampal functions were assessed by their activations in responses to positive and negative emotional stimuli when youth completed an emotional-cognitive processing task (Emotional N-Back task, EN-Back; Barch et al., 2013; A. Cohen, Conley, Dellarco, & Casey, 2016a).

This dissertation comprised two studies. The first study tested the moderating effect of amygdalar and hippocampal activations on the linkage between family rearing environments and youth developmental outcomes. Family rearing environments were assessed via family conflict levels and parental warmth (i.e., one positive and one negative family environment indicator). Youths' developmental outcomes included internalizing and externalizing problems and prosocial behaviors. This study tested hypotheses with data from the Adolescent Brain Cognitive Development (ABCD) study, a large, nationally representative, and longitudinal dataset (Volkow et al., 2018). This sample contained data from 11,875 families, which provided ample statistical power to analyze different patterns of neurobiological sensitivity. The findings of this dataset can be generalized to U.S. youth (Volkow et al., 2018).

Although the ABCD study had distinct advantages, large-scale studies rarely included in-depth measurements of every construct. For example, the ABCD study failed to systematically capture the full spectrum (from negative to positive) and dimensionality of family relationship quality, which was necessary for testing the biological sensitivity to context model (Ellis et al., 2011). Another issue in the ABCD study was the trimming of measurement tools or the use of brief questionnaires in order to reduce participant burden and research costs. The use of trimmed or shortened measurement tools often resulted in reduced validity in the assessment of key constructs (Oshri et al., 2018). To address these issues in the ABCD study dataset, hypotheses were tested using a second sample that included targeted at-risk adolescents and their primary caregivers (*N* = 123) in the Development of Risk and Resilience among Rural Youth (DORRY) project. This project included multi-method (i.e., surveys and fMRI) and multi-reporter (i.e., primary caregivers and youth) assessments of families recruited from rural Georgia. In the second study, a comprehensive set of measurement tools was used to assess the full continuum (i.e., from negative to positive) of the multiple domains of family rearing environments (Olson, Gorall, & Tiesel, 2006). This assessment included cohesion, flexibility, and communication dimensions of the quality of interpersonal relationships in family environments (i.e., family functioning). In particular, this study investigated how amygdalar and hippocampal responses to emotional stimuli moderated the associations between family functioning and adolescents' developmental outcomes. Taken together, these two studies methodologically complemented each other and provided a comprehensive investigation of youths' neurobiological sensitivity to family rearing environments.

CHAPTER 3

FAMILY CONFLICT, PARENTAL WARMTH, AND YOUTH ADJUSTMENT: THE MODERATING ROLES OF THE AMYGDALAR AND HIPPOCAMPAL RESPONSES TO EMOTIONAL SOCIAL CUES[1]

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[1] Liu, Sihong. To be submitted to *Development and Psychopathology*

Abstract

Family rearing experiences, as reflected by conflicts among family members and parenting behaviors, are critical to youths' socioemotional adjustment. However, youth often do not respond equally even to similar family rearing environments. Some youth are more responsive to family influences. Thus, they are either more vulnerable to the harmful impacts of adverse environments or benefiting more from supportive family experiences. Other youth are not significantly influenced by caregiving influences. The current study aimed to examine the neural mechanisms underlying youths' differential responses to family environments, with the activations of two subcortical brain regions, the amygdala and hippocampus, as neurobiological foci. Specifically, this study tested the moderating effects of amygdalar and hippocampal activations during emotional stimuli on the links between family environments and youths' development of psychopathology and prosocial behaviors. Data were drawn from the Adolescent Brain Cognitive Development (ABCD) study, a large, nationally-representative, and longitudinal study (N = 11,875). Family rearing environments were assessed through family conflict levels and parental warmth. Youths' amygdalar and hippocampal activations during positive and negative emotional processing were obtained via an fMRI Emotional N-back paradigm. Results indicated that left amygdalar responses to negative emotional stimuli significantly exacerbated the impacts of family conflict on youths' externalizing problems. Left hippocampal activations significantly amplified the effects of family conflict and parental warmth on youths' internalizing problems. Results suggested that left amygdalar and hippocampal activations during emotional processing were associated with youths' sensitivity to the influences of family rearing environments.

Introduction

During pre-adolescence, the emotional climate and caregiving experiences in the family environments significantly shape youths' socioemotional and behavioral adjustment (Davies & Cummings, 1994; Morris et al., 2007). Adverse emotional climate induced by family conflict might disrupt youths' emotional security and foster the development of maladaptive behaviors (Davies et al., 2002; McCoy, Cummings, & Davies, 2009; Morris et al., 2007; Sheeber, Hops, Alpert, Davis, & Andrews, 1997). In contrast, positive rearing experiences such as warm parenting behaviors can fulfill youths' affective needs from family members and contribute to positive youth development (Rohner, 2008; Rohner, Khaleque, & Cournoyer, 2004; Rohner et al., 2005). However, youth vary in their responses to the influences of family relationships (Ellis et al., 2011). Some youth are highly responsive to both negative and positive influences of the family environments (A. S. Masten, 2001; A. S. Masten & Obradović, 2006). These youth develop more maladaptive outcomes when they are exposed to adverse rearing environments. However, they can also exhibit more developmental competence in response to supportive family rearing environments (Boyce & Ellis, 2005). In contrast, other youth are less responsive to family environments. For less responsive youth, there is no significant association between rearing experiences and their developmental outcomes (Boyce & Ellis, 2005).

The biological sensitivity to context theory suggests that youths' differential responses to family rearing environments (i.e., differential sensitivity) are partially stemmed from the interactions between neurobiological processes and caregiving experiences (Boyce & Ellis, 2005). Therefore, youths' differential responsivity to family influences is referred to as neurobiological sensitivity (Schriber & Guyer, 2016). In interaction with adverse rearing environments, elevated neurobiological sensitivity exacerbates youths' vulnerability to develop psychopathology and risk behaviors. However, high neurobiological sensitivity also interacts with positive caregiving experiences and potentiate youth to more positive developmental outcomes (Boyce & Ellis, 2005). Despite the empirical evidence on youths' differential responses to family influences, the neural mechanisms underlying this neurobiological sensitivity remains unclear (Schriber & Guyer, 2016) The current study aimed to reveal the neural underpinnings of youths' differential sensitivity to family influences, with the functions of the amygdala and hippocampus, two subcortical brain regions, as the key foci. Specifically, this study examined how amygdalar and hippocampal responses to emotional stimuli moderated the effects of family conflict and parental warmth on youths' socioemotional adjustment. Youths' developmental outcomes were assessed via internalizing and externalizing problems and prosocial behaviors.

Family rearing environments and Youth Adjustment

Family conflict. Conflict in family environments (i.e., active opposition between family members) has a significant and prolonged impact on youth development (Cummings et al., 2002). Family conflict may involve different combinations of family members, including interparental conflicts, parent-child conflicts, and sibling conflicts (Zahn-Waxler et al., 2008). Families who frequently engage in intra-familial conflict are characterized by stressful and aversive familial emotional climate (Morris et al., 2007). This aversive emotional climate ultimately disrupts youths' sense of emotional security (Davies et al., 2002; McCoy et al., 2009).

According to the emotional security theory (Davies et al., 2002), children have a higher-order goal of wanting the family environments to be secure. When this goal is compromised, they develop behavior patterns that are appropriate for threatening context to protect themselves from potential danger (Davies et al., 2002; McCoy et al., 2009). These adaptive behaviors may be perceived as maladjustment and harmful under normative environments (Frankenhuis et al., 2016). Therefore, an adverse and insecure family emotional climate induced by high levels of family conflict can lead to children’s and adolescents’ adjustment difficulties (Cummings et al., 2002). These adjustment difficulties include emotional dysregulation (Morris et al., 2007), anxiety and depression (Queen, Stewart, Ehrenreich-May, & Pincus, 2013), and aggressive behaviors (Tanaka, Raishevich, & Scarpa, 2010). For example, in a meta-analysis, Kitzmann, Gaylord, Holt, and Kenny (2003) report significant associations between witnessing domestic violence and children’s internalizing, externalizing, social problems, and academic failure. Another meta-analysis also suggests associations between inter-parental conflict and youths’ cognitive, affective, behavioral, and physiological maladaptation (Rhoades, 2008). Additionally, family conflict poses challenges for youth to achieve positive developmental outcomes, such as prosocial behaviors (McCoy et al., 2009).

Parental warmth. Parenting behaviors are another essential dimension of the family environments that has profound effects on youth development (Kwon & Wickrama, 2014; Masarik & Conger, 2017). Parental warmth reflects parenting behaviors that can be assessed on a continuum with one end as parental acceptance and another end as parental rejection (Rohner et al., 2005). Parental acceptance is usually exhibited by positive affect, responsiveness, acceptance, and support towards youth (Rohner, Khaleque, & culture, 2002). In contrast, parental rejection is commonly presented as the absence or withdrawal of positive affect and support, and the presence of physically and psychologically hurtful behaviors and emotions towards youth (Rohner et al., 2005). According to the parental acceptance and rejection theory (PARTheory; Rohner, 2008; Rohner et al., 2004, 2005), children have the evolutionally acquired needs for affective and supportive bonds with their parents to achieve physical and emotional security.

When youths’ needs for parental affection and support are not satisfied, children might perceive the early rearing environment as harsh, adverse, and unpredictable. These youth tend to develop negative personality dispositions as an adaption to achieve evolutionary goals of survival (Rohner, 2008). These negative personality dispositions are linked to a series of maladaptive behaviors such as aggressive behaviors, impulsivity, and high-vigilance (Khaleque, 2013; Rohner et al., 2005).

Research provides ample evidence supporting the PARTheory (Franck & Buehler, 2007; Khaleque, 2013; Kim & Rohner, 2002; Mogro-Wilson, 2008; Padilla-Walker, Nielson, & Day, 2016). For example, Mogro-Wilson (2008) reports that parental warmth is associated with significant decreases in alcohol use among Latino adolescents. Additionally, Franck and Buehler (2007) suggest that parental warmth can protect youth from developing internalizing and externalizing problems under family rearing environments with depressed parents. In parallel, parental warmth also contributes to elevated positive developmental outcomes such as academic achievement (Kim & Rohner, 2002) and prosocial behaviors (Khaleque, 2013; Padilla-Walker et al., 2016).

Individual Differences and Neurobiological Sensitivity

Despite the consistent links between the quality of family interpersonal interactions and youth adjustment, youth do not respond equally even to similar family rearing environments (Luthar, 2006; A. S. Masten & Obradović, 2006). Specifically, some youth are hypersensitive to their rearing environments. These youth are more likely to develop maladaptive behaviors under adverse family environments. However, they also exhibit elevated positive outcomes under the influence of supportive rearing experiences (Ellis et al., 2011). However, other youth are less responsive to family influences. For less responsive youth, caregiving experiences do not significantly affect their developmental outcomes (Boyce & Ellis, 2005). These heterogeneous responses to caregiving experiences are suggested to be attributed to youths' neurobiological factors, which moderate the linkage between rearing environments and youth adjustment. Therefore, youths' differential responsivity to family environments is referred to as neurobiological sensitivity (Ellis et al., 2011; Schriber & Guyer, 2016). Accordingly, high levels of neurobiological sensitivity interact with positive or negative rearing environments and amplify youths' adaptive or maladaptive adjustment (Boyce & Ellis, 2005).

Researchers propose three conceptual models to articulate why and how youth respond differently to similar caregiving experiences. These models include the *diathesis-stress* (Monroe & Simons, 1991), the *biological sensitivity to context* (Boyce & Ellis, 2005; Ellis et al., 2005), and the *vantage sensitivity* (Pluess & Belsky, 2013) hypotheses. The diathesis-stress hypothesis is first purported to elaborate youths' differential *vulnerability* under negative environments. This hypothesis suggests that certain neurobiological sensitivity indicators render some youth to be disproportionally more vulnerable to the impacts of early life stress (Monroe & Simons, 1991; Zuckerman, 2007). Despite some empirical support, the diathesis-stress model bears significant limitations. The major limitation lies in its lack of examination of the neurobiological mechanisms underlying youths' behavioral responses to supportive rearing environments (Ellis et al., 2011).

The biological sensitivity to context theory extends the diathesis-stress model by incorporating the interaction between neurobiological traits and youth response to *both positive and negative* environments. The biological sensitivity to context theory indicates that specific neurobiological sensitivity indicators interact with positive and negative rearing environments and induce qualitatively different behavioral adjustment among youth. In interaction with supportive environments, specific neurobiological factors amplify youths' development competence. In contrast, the same neurobiological factors can also interact with adverse caregiving experiences and lead to elevated risks for psychopathology and risk behaviors (Boyce & Ellis, 2005; Ellis et al., 2005). This biological sensitivity to context theory has been gaining increasing empirical support (e.g., Bakermans-Kranenburg & Van Ijzendoorn, 2011; Cruz et al., 2018; Obradović et al., 2010; Oshri et al., in press; Slagt et al., 2019; Van IJzendoorn et al., 2012).

Pluess and Belsky (2013) also advance the vantage sensitivity model, which focuses on the positive extreme of youth development under an *advantageous* caregiving environment. The vantage sensitivity model indicates that certain neurobiological indicators render youth to be more sensitive to the advantageous effects of positive environments. In interaction with supportive rearing experiences, these neurobiological factors can intensify youths' adaptive responses and developmental competence (Pluess, 2017; Pluess & Belsky, 2013). Overall, to comprehensively and accurately examine these neurobiological sensitivity models and distinguish different sensitivity patterns, it is critical for researchers to incorporate both negative and positive rearing environment assessments (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012).

The neurobiological sensitivity to context models have been receiving growing empirical support. This body of research has identified numerous sensitivity indicators, including the behavioral phenotypes (e.g., Chen et al., 2019), genetic make-up (Bakermans-Kranenburg & Van Ijzendoorn, 2011; G. H. Brody, Yu, Chen, Kogan, et al., 2013; Caspi et al., 2010), and psychophysiological stress responses (e.g., Obradović et al., 2010; Oshri et al., in press). The behavioral phenotypes, genetic make-up, and stress responses are all connected to youths' abilities to regulate their emotions and stress responses (Ellis et al., 2011). Nevertheless, what is lacking in this body of research is the neural architecture (i.e., brain structures and functions) that underlies youths’ differential sensitivity to the influences of the family environments (Schriber & Guyer, 2016). The brain plays a key role in regulating human behaviors in response to environmental inputs (Schriber & Guyer, 2016). Thus, the lack of knowledge on the neural sensitivity mechanisms may obscure the further understanding of the links between neurobiological sensitivity and youth adjustment. Therefore, the current study aimed to advance knowledge in this area by focusing on the functions of two subcortical brain regions, including the amygdala and hippocampus.

The Amygdala and Hippocampus as Neurobiological Sensitivity Indicators

The amygdala and the hippocampus are two major subcortical regions of the social-affective brain circuitry, which is responsible for modulating youths’ emotional and stress reactivity (Casey et al., 2008; M. Ernst, 2014; M. Ernst & Fudge, 2009; Luna & Wright, 2016; Nelson et al., 2005; Steinberg, 2010; Steinberg et al., 2008). Specifically, the amygdala is the hub of emotional processing in response to both positive and negative stimuli (Garavan et al., 2001; LeDoux, 2000; Windle et al., 2018). The hippocampus, on the other hand, is responsible for consolidating explicit memories (especially emotional elements of memories; Manns et al., 2003). The hippocampus also regulates the hypothalamic-pituitary-adrenal (HPA) axis acute stress responses (Mizoguchi et al., 2003). Overall, the functions of the amygdala and hippocampus in regulating youths’ emotional and stress responses correspond with the characteristics of previously-validated neurobiological sensitivity indicators (including the behavioral phenotype, dopamine- and serotonin-relevant genotype, and psychophysiological stress responses). Therefore, the amygdala and hippocampus might serve as candidate neurobiological sensitivity indicators (Schriber & Guyer, 2016). The current study, therefore, aimed to investigate how the functions of the amygdala and hippocampus interacted with family conflict (i.e., negative context) and parental warmth (i.e., positive context) and affected youth developmental outcomes.

In the investigation of the amygdala and hippocampus as neurobiological sensitivity indicators, it is also necessary to examine whether the neurobiological sensitivity functions are lateralized to a certain brain hemisphere (left or right). Research documents significant lateralization (i.e., the tendency for certain brain functions to be specialized to one brain hemisphere) on amygdalar and hippocampal activations in response to emotional stimuli. For example, when testing amygdalar responses to emotional stimuli, activations are reported more often in the left amygdala compared to the right amygdala (Baas et al., 2004; Murphy et al., 2003; Sergerie et al., 2008; Wager et al., 2003). However, when activations are reported, the magnitude of effect sizes is similar for the left and right amygdala (Fusar-Poli et al., 2009; Sergerie et al., 2008). Additionally, research documents lateralized temporal dynamics of amygdalar activities (Sergerie et al., 2008). For example, the left amygdala is more engaged in detailed and elaborated emotional processing (Gläscher & Adolphs, 2003). In contrast, the right amygdala responds more to the immediate emotional input (Gläscher & Adolphs, 2003). For the lateralization of hippocampal functions, research indicates that the left hippocampus exhibits stronger connections with the experiences of early life stress and the development of psychopathology, compared to the right hippocampus (MacMaster & Kusumakar, 2004; Schriber et al., 2017; Vythilingam et al., 2002; Whittle et al., 2013). Taken together, it is possible that the neurobiological sensitivity indicators are specifically lateralized to the left brain hemisphere (i.e., the left amygdala and hippocampus).

Further, significant sex differences are documented in studies that examine amygdalar and hippocampal roles as neurobiological sensitivity indicators. Functionally, amygdalar responses to emotional stimuli are more salient among males compared to females (Fine et al., 2009; Wrase et al., 2003). In empirical research that tests for the neural mechanisms underlying youths' differential responses to family influences, Yap et al. (2008) report that a smaller bilateral amygdala represents heightened sensitivity among girls. However, a larger right amygdala reflects boys' elevated neurobiological sensitivity to family rearing environments. Additionally, Whittle et al. (2011) show that the hippocampus only serves as a neurobiological sensitivity indicator among girls. Overall, due to the limited number of empirical studies that examine youths' neurobiological sensitivity, the directionality of sex differences still remains unclear and thus requires further investigation.

The Present Study

The aims and hypotheses of this study were five-fold (Figure 3.1). First, the direct effects of family conflict and parental warmth on youth adjustment were examined. It was hypothesized that (hypothesis 1) elevated family conflict was associated with negative developmental outcomes (i.e., increased behavioral problems and decreased prosocial behaviors). Similarly, high levels of parental warmth were hypothesized to be linked to youth's positive outcomes (i.e., decreased behavioral problems and increased prosocial behaviors).

Next, the moderating roles of amygdalar and hippocampal responses to emotional stimuli on the links between family environments and youth outcomes were tested. It was hypothesized that the moderating effects of amygdalar (hypothesis 2) and hippocampal (hypothesis 3) activations on the associations between family rearing environments and youth adjustment would abide by hypotheses generated by the biological sensitivity to context theory. This pattern is presented in Figure 3.2. In particular, higher amygdalar and hippocampal activations during negative social cues were expected to amplify the relations between family conflict and negative developmental outcomes (hypotheses 2a and 3a). Higher amygdalar and hippocampal responses to positive social cues were hypothesized to intensify the associations between parental warmth and positive developmental outcomes (hypotheses 2b and 3b). Youth with low levels of amygdalar activation during emotional processing were hypothesized to show lower behavioral responses to family interactions, as characterized by family conflict and parental warmth (hypotheses 2c and 3c).

Further, this study also investigated the lateralization of amygdalar and hippocampal activations in response to emotional stimuli, as well as the lateralization of amygdalar and hippocampal roles as neurobiological sensitivity indicators. The left amygdala and hippocampus were hypothesized to exhibit higher levels of activations in response to emotional stimuli (hypothesis 4a). Additionally, it was hypothesized that left amygdalar and hippocampal would exhibit stronger moderating effects on the associations between family functioning and youth adjustment, compared to the right amygdala and hippocampus (hypothesis 4b). Lastly, sex differences in the neurobiological sensitivity models were also tested. Because of the mixed findings in gender and biological sex effects on neurobiological sensitivity, no specific hypothesis was determined for how the tested models would differ by biological sex.

Study hypotheses were tested with data from the ABCD study, a large, nationally representative, and longitudinal dataset of adolescent brain development (Volkow et al., 2018). The ABCD dataset comprised sufficient statistical power to distinguish different patterns of neurobiological sensitivity models (i.e., the diathesis-stress, biological sensitivity to context, and vantage sensitivity models) and to specify biological sex differences. This large dataset also strengthened the external validity (i.e., generalizability) for study findings (Garavan et al., 2018).

Methods

Participants

The current study used a sample of 11,875 youth and their primary caregivers (PC) from the Adolescent Brain Cognitive Development (ABCD) study. The ABCD study was a large longitudinal study of adolescent brain development and health in the United States with the aim of recruiting children and following them for ten years, starting from September 2016 (Garavan et al., 2018; Volkow et al., 2018). The data used in the present study were based on the ABCD Data Release 2.0 (released in March 2019) and ABCD Fix Release 2.0.1 (released in July 2019), which included data from 11,875 participants. Upon data release 2.0.1, 11,875 participants have completed the baseline visits (T1), 8,623 have completed the 6-month follow-up (T2), 4,951 have completed the 1-year follow-up (T3), and 1,919 have completed the 18-month follow-up (T4). Participants were aged 9 to 10 years old at baseline (M_{age} = 9.48, SD_{age} = .51). The biological sex distribution of youth was approximately even, with 47.9% (*n* = 5,681) female, 52.1% (*n* = 6,188) male, and .1% (*n* = 6) intersex. For race/ethnicity distribution, there were 52.3% (*n* = 6,178) White, 15.1% (*n* = 1,780) Black, 20.4% (*n* = 2,409) Hispanic/Latino(a), 2.2% (*n* = 255) Asian, and 10.1% (*n* = 1,199) Other (including American Indian & Alaska Native, Native Hawaiian and Pacific Islander, and Mixed). Of all the participants in this study, 68.6% (*n* = 8,147) were the only child of the family involved in the study, 13.4% (*n* = 1,593) had a sibling who also participated, 17.7% (*n* = 2,105) were twins, and .3% (*n* = 30) were triplets.

Most of primary caregivers participated in this ABCD study were female (89.0%, *n* = 10,564, M_{age} = 39.97, SD_{age} = 6.84). There were 73.7% (*n* = 8,679) of primary caregivers who were married or living together with a partner during the baseline data collection. Large variance existed in participants' sociodemographic characteristics. For example, there were 5.0% (*n* = 592) primary caregivers whose highest education were below high school diploma, 9.5% (*n* = 1,131) who obtained high school diploma or GED, and 26.0% (*n* = 3,078) who had some college education. Additionally, there were 25.4% (*n* = 3,014) of primary caregivers who got a Bachelor degree, and 34.1% (*n* = 4,043) who obtained post graduate degrees. The average household annual income was approximately $97,340 (*SD* = $62,231). There were 29.70% of families who were under 200% federal poverty level during baseline assessments (i.e., annual income below $51,500 for a family of four).

Participants were recruited at 21 sites throughout the United States, using probability sampling of U.S. schools around the 21 sites as major sampling strategies. In selected schools, the recruitment materials and/or electronic copies were given to all 9-10-year-old children and their primary caregivers. Interested and eligible (screened through a brief phone call) families were enrolled in the study and scheduled for assessments. Other supplemental sampling and recruitment methods (such as snowballing referral and twin registry) were also employed in order to include children who might be excluded using the major strategies, such as and under-representative and home-schooled youth. The demographics of enrolled participants were closely monitored. Emerging deviations were adjusted quickly with the ultimate goal of recruiting a diverse sample that represents the national demographic and socio-economic characteristics of the population of U.S. 9-10-year-old children (Garavan et al., 2018). Additionally, case-specific analysis weights were obtained using the propensity-based methods (Heeringa, West, & Berglund, 2017; Rosenbaum & Rubin, 1983a, 1983b) to compensate for existing selection biases.

Procedures

The ABCD study was a ten-year longitudinal study, with full assessments (6-7 hours completed in one or two visits) taking place biennially. During the alternative year, a 2-3 hour assessment was completed. Additionally, every 3-6 months, a brief (15-minute) follow-up through online or phone interviews were administered on participants. For a full assessment, youth completed a series of surveys, neurocognitive assessments, interviews, bio-specimen collection, and neuroimaging, while primary caregivers completed surveys and interviews. For the alternative year, youth completed surveys, neurocognitive assessments, interviews, and bio-specimen collection, while primary caregivers completed surveys and interviews. The detailed study procedure varied by site and by time-points. The current study employed data collected during the baseline and 1-year follow-up assessments.

fMRI procedures. The neuroimaging scanning occurred in either one or two sessions, varying by sites. A one-session scanning consisted of a fixed order of a localizer, the 3D T1-weighted scanning, resting-state fMRI (2 runs), and resting-state fMRI (1-2 runs). Then, three task-based fMRI were administered in a randomized order across participants, including the Monetary Incentive Delay (MID) Task, Stop Signal Task (SST), and Emotional N-Back Task (EN-back). For the two-session scanning, the first session usually included a localizer, the 3D T1-weighted scanning, resting-state fMRI (2 runs), DTI, and resting-state fMRI (1-2 runs). The youth then took a short break and completed the second session, which included a localizer and three randomized (in order) task-based fMRI scan (Casey et al., 2018).

Before the neuroimaging, participants went through a pre-scan session (25-45 minutes). This pre-scan session included an MRI contraindication rescreening, MRI simulation and motion compliance training, practice on the three fMRI tasks, and a pre-scan questionnaire that assessed youths’ emotional arousal (e.g., relaxed, happy, scared). After each scanning session, the same questionnaire on emotional arousal was administered to youth again. At the end of neuroimaging, youth also completed an EN-Back recognition memory task and a monetary incentive delay (MID) task post-scan questionnaire.

Measures

Family environment.

Family conflict (T1). Family conflict was assessed through the Family Conflict Subscale of the Family environments Scale (FES; Moos & Moos, 1994), which includes nine items. This scale was widely used and well-validated in family studies (King, 1998; Moos & Moos, 1994). Youth reported “yes (1)” or “no (0)” on nine statements reflecting the overt family conflict. Example items included “We fight a lot in our family” and “Family members sometimes get so angry they throw things.” Four items were reversely coded so that “1” reflects higher family conflict (e.g., “Family members rarely become openly angry”). Then, a sum score of these nine items (minimum = 0, maximum = 9) was calculated and used in analyses ($\alpha = .68$), with higher score reflecting higher levels of interpersonal conflicts in the family rearing environments.

Parental warmth (T1). Parental warmth was assessed through the Acceptance subscale from the Child Report Parenting Behavior Inventory-Short version (CRPBI; E. S. Schaefer, 1965). Youth responded to five items that reflected their primary caregivers’ warmth, acceptance, and responsiveness (Zucker et al., 2018). The responses ranged from 1 (“not like him/her”) to 3 (“a lot like him/her”). Example items included “My parent is a person who makes me feel better after talking over my worries with him/her” and “My parent is a person who smiles at me very often.” A sum score of these five items (minimum = 5, maximum = 15) was calculated and used in the analyses (α = .71). Higher scores indicated higher levels of parental warmth and acceptance, while lower scores suggested parental rejection.

Adolescent adjustment.

Internalizing and externalizing problems (T1 & T3). Youths' internalizing and externalizing problems were assessed by Child Behavior Checklist (CBCL; Achenbach, Dumenci, & Rescorla, 2001) reported by primary caregivers at the baseline (T1) and one-year follow-up (T3). This scale has been widely used and well-validated to measure youths' behavior problems (Achenbach et al., 2001). Parents were instructed to answer how much a series of behavior problems fit their children using a 3-point scale with responses ranging from 0 (not true) to 2 (very often true). The internalizing problems were quantified using three syndrome scales: anxious/depressed, withdrawn, and somatic complaints. The externalizing problems were quantified with two syndrome scales: rule-breaking and aggressive behaviors. The CBCL manual suggests that raw scores for behavioral syndromes and problem scales are more precise and uniform than T-scores, especially at the end of the distribution. As there were no significant age differences among youth in the ABCD sample, raw scores of internalizing and externalizing problems were used in analyses. Internal consistency of internalizing (α = .60) and externalizing (α = .70) problems were adequate.

Adolescent prosocial behaviors (T1 & T3). Youths' prosocial behaviors were obtained through the parent-report Prosocial Behavior Scale, a subscale from the Strengths and Difficulties Questionnaire (SDQ; Goodman, Meltzer, & Bailey, 1998; Goodman & Scott, 1999). The original 5-item scale was shortened to include three items with the highest factor loadings, including "My child is considerate of other people's feelings," "My child is helpful if someone is hurt, upset, or feeling ill," and "My child often offers to help others (parents, teachers, children)”. Primary caregivers responded on a 3-point scale ranging from 0 (not true) to 2 (certainly true). Sum scores of T1 and T3 (range 0 – 6) were calculated ($\alpha = .71$) and used in further analyses.

Demographics. Primary caregivers reported on youths’ and their own demographics and socioeconomic characteristics. Youths’ biological sex was coded as 1 for females and 2 for males. The race/ethnicity variable was recoded into three binary variables to reflect participants’ racial/ethnic status of being White, Black, and Hispanic/Latino(a). Parental highest education was coded into an ordinal variable that included five values: “less than high school diploma,” “high school diploma/ GED,” “some college,” “Bachelor,” and “post-graduate degree.” Participants reported their household income based on the total combined family income in the past 12 months, including income (before tax), wages, rent from properties, social security, benefits, compensation, and help from relatives. The household income was originally an ordinal variable with ten values ranging from 1 (less than $5,000) to 10 ($200,000 and greater). Then, the household income variable was coded into a continuous variable that reflected the mid-point of each category (e.g., $2,500, $7500). Parental marital status was coded as 1 for married or living together, and 0 for not married or living together.

Amygdala and hippocampus activation assessment.

Paradigm. The amygdala and hippocampus responses to positive and negative emotional stimuli were assessed when youth were completing the Emotional N-back (EN-back) task in the functional MRI (fMRI) paradigm (Barch et al., 2013; A. Cohen et al., 2016a). The EN-back task engaged youths’ working memory and emotion regulation processes. The working memory component contained both high (i.e., 2-back) and low (i.e., 0-back) memory load conditions. The comparison of 2- vs. 0-back allowed for the measurement of brain activations specifically related to working memory as opposed to general cognitive functions. This working memory component of the EN-back task was similar to traditional n-back tasks (Barch et al., 2013). The traditional n-back task has been validated among youth (Barch et al., 2013; Casey et al., 2018) and shown to induce reliable brain activations across subjects (Drobyshevsky, Baumann, & Schneider, 2006) and time (Caceres, Hall, Zelaya, Williams, & Mehta, 2009). Therefore, the n-back task has been widely used in neuroimaging studies (Owen, McMillan, Laird, & Bullmore, 2005).

The emotional component of this EN-back task included a set of negative (e.g., fearful) and positive (e.g., happy) facial expressions as stimuli (Conley et al., 2018; Tottenham, Tanaka, et al., 2009). This emotional component reflected youths' emotional reactivity and regulation (Gee et al., 2013; Hare et al., 2008). The facial expressions were drawn from the NimStim emotional stimulus set (Tottenham, Tanaka, et al., 2009) and the Racially Diverse Affective Expressions (RADIATE) set of stimuli (Conley et al., 2018). These two emotional stimulus set addressed the racial/ethnicity diversity of ABCD study participants (Casey et al., 2018). Youths' ability to distinguish neutral faces from negative or positive faces in this task also reflected their sensitivity to special emotionally evocative stimuli (Casey et al., 2018).

This EN-back task included two types of working memory blocks, the 0-back block, and the 2-back block. For the 0-back, participants responded "match" when the current stimulus matched the target presented at the beginning of the block. For the 2-back, participants responded "match" when the current stimulus matched the one that was shown two trials before. Both 0-back and 2-back tasks included four types of blocks that corresponded to four emotional conditions: non-facial expressions (places), neutral, negative (fear), and positive (happy). Overall, this EN-back task included two runs in total. Each run of the EN-back task included eight task blocks (four 0-back and four 2-back blocks) and four fixation blocks (15 seconds each). Each block consisted of 10 trials of the same emotional condition (2.5 seconds each). At the beginning of each block, a 2.5-second cue was presented to indicate whether to perform the 2-back or 0-back condition. Then, the ten trials were presented, with two targets (i.e., matched stimulus), two to three non-target lures, and the rest as non-lures (i.e., stimuli only presented once). At the end of each block, a 500-millisecond colored cross was presented to alert children of the task switch. Each trial included a 2-second stimulus followed by a 500-millisecond fixation cross to alert children of the next trial. In total, there were 160 trials in this EN-back task, with 96 unique stimuli of four different stimulus types (none, neutral, negative, and positive). Each emotional stimulus type included 24 unique stimuli.

Paradigm behavioral assessment. Youths' behavioral performance measures on the EN-back task were calculated, including the number, rate, and the reaction time of correct trials. The rate of correct trails (i.e., performance accuracy) was obtained by calculating the percentage of correct trails. The reaction time was obtained by calculating the mean reaction time of correct trials. These behavioral performance metrics were calculated by condition (i.e., 2-back and 0-back conditions), stimuli type (i.e., none, neutral, positive, and negative), and combinations of the two (e.g., 2-back positive stimuli, 2-back negative stimuli, etc.). In the present study, participants' accuracy (i.e., the percentage of correct trials) was used as the major indicator of the EN-back behavioral performance (Casey et al., 2018). Specifically, the percentage of correct trials were obtained for the total 160 trails, for 0-back and 2-back conditions separately, for the four different emotional types (i.e., positive, negative, neutral, and places) separately, and for the working memory condition-specific and stimuli-specific blocks (e.g., 2-back negative emotions; 2-back positive emotions, etc.). Per suggestions from Frings et al. (2006), the fMRI data of participants who exhibited lower than 60% accuracy on the 2-back or 0-back tasks (*n* = 2) were excluded from the analyses.

MRI acquisition. Across the 21 sites, three 3T scanner platforms (i.e., Siemens Prisma, General Electric (GE) 750, and Philips) were employed, and the neuroimaging protocol was harmonized accordingly. Standard adult-size multi-channel coils capable of multiband echo-planar imaging (EPI) acquisitions were used across sites. The EN-back task was programmed in E-Prime Professional 2.0 version 2.0.10.356 or later (Psychology Software Tools, PA), which was reliable for Windows 8.1 or earlier. When lying supine on the scanner table, participants viewed the task through rear projection or goggles (varied by sites) and responded to the task through a Current Design 2-button response box using their dominant hand. The structural images were obtained for anatomical reference using a high-resolution 3D T1-weighted magnetization-prepared rapid acquisition gradient echo scan (for cortical and subcortical segmentation; Hagler Jr et al., 2019). Functional images were collected using high spatial and temporal resolution simultaneous multi-slice (SMS)/multiband EPI fMRI scans with fast integrated distortion correction (Hagler Jr et al., 2019). Table 3.1 presented the harmonized imaging scanning parameters for three platforms (Casey et al., 2018). In sites using the Siemens scanner platform, the Frame-wise Integrated Real-time Motion Monitoring (FIRMM) software (Dosenbach et al., 2017) was used to assess real-time head motions. It is important to note that, in sites using the Philips scanner, the data were post-processed incorrectly, which lead to a distortion in processed fMRI images. Therefore, the MRI data obtained using the Philips scanner (7.5%, *n* = 894) were excluded from the current analyses.

Imaging processing. The current study employed processed neuroimaging data and tabulated ROI-based analysis results that were publicly shared via the National Institute for

Mental Health Data Archive (Casey et al., 2018; Hagler Jr et al., 2019). The neuroimaging data processing was conducted using the Analysis of NeuroImages (AFNI) software (R. W. Cox, 1996). In the fMRI data processing, to correct for head motions, each frame was registered to the first using AFNI'S 3dvolreg (R. W. Cox, 1996; Hagler Jr et al., 2019). This process also provided head motion time courses, which were further incorporated into task-based fMRI analyses. Then, the reversing gradient method (D. Holland, Kuperman, & Dale, 2010) was used to correct B_0 distortions, and the displacement field was estimated from spin-echo field map scans, adjusted for between-scan head motions, and applied to the series of gradient-echo images. After correcting for distortions due to gradient nonlinearities (Jovicich et al., 2006) and resampling all fMRI scans for each participant's imaging visits with cubic interpolation, automated registration between T2-weighted, spin-echo B0 calibration scans, and T1-weighted structural images was performed to atlas brains using mutual information (Wells III, Viola, Atsumi, Nakajima, & Kikinis, 1996). To identify cortical and subcortical ROIs, brain segmentation was performed in FreeSurfer v5.3.0 (Fischl et al., 2002), which has been validated for a children sample (Ghosh et al., 2010). Specifically, subcortical structures were labeled with an automated, atlas-based, volumetric segmentation procedure (Fischl et al., 2002). Cortical regions labeled with the Desikan atlas-based classification (Desikan et al., 2006). Initial TRs (8 TRs for Seimens and Philips, 5TRs for GE DV25, and 16 TRs for GE DV26) that made up the pre-scan reference were removed before analyses.

A voxelwise General Linear Model (GLM) procedure was implemented to quantify the task-based brain regions activation at the individual level, which was conducted in AFNI's 3dDeconvolve (R. W. Cox, 1996). Motion estimates were included as independent variables (Power et al., 2014), and time points with framewise displacement (FD) greater than .9mm were censored (Hagler Jr et al., 2019; Siegel et al., 2014). The positive and negative emotional stimuli were introduced into the models as predictors, and the neutral emotional stimuli were modeled as the linear contrast baseline. Instruction screens, fixation, and non-facial stimuli (faces) were left as resting states. Then, linear contrasts were obtained for contrasts of positive vs. neutral faces and negative vs. neutral faces. These contrasts reflected youths' emotional reactivity towards positive and negative facial expressions and were aligned with the major study hypotheses. For each voxel of every participant, a GLM was conducted, with the temporal pattern of the positive or negative emotion block and covariates (e.g., head movement values) as independent variables. In the GLM, the blood-oxygen-level-dependent (BOLD) signals over time were modeled as the dependent variable.

To define ROIs, cortical surface-based regions were labeled with FreeSurfer's standard, anatomically-defined Desikan's parcellation (Desikan et al., 2006). Subcortical regions, including the amygdala and hippocampus, were labeled with atlas-based, automated, and volumetric segmentation procedure in FreeSurfer v5.3.0 (Fischl et al., 2002). Brain regions were resampled from atlas-space to individual space (Hagler Jr et al., 2019). For each subcortical ROI (including amygdala and hippocampus), the average GLM beta coefficients and standard errors of the mean across voxels were computed for each run. Then, the mean GLM beta coefficients and standard errors of the mean across two runs for each participant were calculated, weighted by nominal degrees of freedom (Hagler Jr et al., 2019). The nominal degrees of freedom were equal to the number of frames after motion censoring minus the number of model parameters. In the data processing, runs with fewer than 50 degrees of freedom were excluded (Hagler Jr et al., 2019).

Imaging quality control. The quality control process followed the recommended inclusion criteria for fMRI data generated by the ABCD study (Casey et al., 2018). To be included in the analysis, the participants needed to (1) pass the EN-back task fMRI data quality control, (2) pass the 3D T1 data quality control for successful registration of T1 images, (3) pass FreeSurfer quality control, (4) meet the motion compliance criteria (EN-back average beta weights degree of freedom > 200), and (5) have acceptable performance in the task (i.e., both 2-back and n-back total accuracy ≥ 60%, as suggested by the ABCD study). The exclusion included 15.6% (*n* = 1,858) participants who did not pass the quality control; 1.1% (*n* = 136) 3D T1 data not passing the quality control; 3.1% (*n* = 370) not passing the FreeSurfer quality control; 8.5% (*n* = 1,013) having excessive motions, and 10.9% (*n* = 1,297) who did not meet the behavioral performance criteria. Additionally, the incorrect data post-processing on the Philips scanner resulted in a distortion in processed fMRI images and led to an additional 7.5% (*n* = 894) of participants' fMRI data being excluded from analyses. Further, per the suggestions from the ABCD data user manual, the top and bottom .25% (*n* = 30) of fMRI ROI activation data were considered as outliers and thus also excluded from analyses. Therefore, there were 52.87% (*n* = 6,278) participants' fMRI data for the EN-back task included in the final analyses. Independent sample t-tests and chi-square tests were conducted to compare youth whose fMRI data were excluded due to quality control reasons with other youth whose fMRI data were included in analyses.

Analytical Plan

Preliminary analyses plan. Preliminary data analysis (including descriptive and correlation analyses of study variables) were conducted in IBM SPSS software version 25.0 (Armonk, NY). Independent-sample t-tests were also conducted in SPSS to compare the sex differences of the means of study variables. Paired-sample t-tests were used to compare the mean difference of brain hemispheres (left vs. right) and valences (positive vs. negative) in amygdalar and hippocampal activations. Then, study hypotheses were tested using structural equation models (SEM) with maximum likelihood estimation with robust standard errors (Yuan & Bentler, 2000) in Mplus Version 7.4 (L. Muthén & Muthén, 2012). This dataset was nested within two levels: the families and the sites. The intra-class correlation coefficients were calculated for all the study variables across the 21 sites and presented in Table 3.2. The between-group variance only accounted for a small fraction (ranged from 0.01% to 2.2%) of the total variance. Therefore, the analyses did not account for the interdependence between sites. In order to correct for stratification and observation non-independence induced by the nesting of participants in families, methods designed by Mplus for complex survey data (L. Muthén & Muthén, 2012) were used in the analyses. Specifically, family ID was set as a cluster indicator, and the propensity scores were used as weights.

Structural equation models analyses plans. First, direct effect models were tested to examine the associations between the family environments and youths' developmental outcomes, including internalizing and externalizing problems and prosocial behaviors. Then, the moderating effects of amygdalar and hippocampal response to positive and negative emotional social cues on the associations between family rearing environments and youths' developmental outcomes were tested. The three developmental outcomes were modeled together and covaried with each other. The left and right amygdalar and hippocampal responses to positive and negative social cues were tested separately as moderators in order to examine the region- and hemisphere-specific effects. Specifically, in the model with family conflict as the independent variable, brain responses to *negative* social cues were examined because family conflict mostly captured the adverse spectrum of the family environment. In contrast, in the parental warmth model, brain responses to positive social cues were tested due to the *positive* nature of parental warmth. Overall, there were eight models tested: four models for the amygdala and four models for the hippocampus. In all the SEM models, youths' biological sex, race/ethnicity, household income, and parental marital status were controlled for (i.e., added as independent variables that predicted youth adjustment), and covaried with family rearing environments. Given that the direct effect models were nested in the moderating models, chi-square tests were conducted to compare model fit improvements from the baseline (direct effect) models to the moderation models.

In each model, an interaction term of the brain responses and the family rearing environment variables (mean-centered) was created and added to the model. A significant linear association between the interaction term and youth adjustment at T3 indicated a moderating effect of brain responses on the link between the family rearing environments and youth adjustment. The Johnson-Neyman plot (P. O. Johnson & Neyman, 1936) and the simple slope method (Aiken, West, & Reno, 1991; Dawson, 2014) were used to probe and display significant moderating effects. The Johnson-Neyman technique presented the effect of family rearing environments on youth adjustment, its 95% confidence interval, and regions of significance on different values of moderators (P. O. Johnson & Neyman, 1936). The simple slope method presented the slope of the main effect of family rearing environments on youth adjustment contingent on low and high values of the moderator (Aiken et al., 1991; Dawson, 2014). This simple slope method has been used to complement the Johnson-Neyman plot to allow a clearer visualization of the different sensitivity patterns (Roisman et al., 2012).

Distinguishing different sensitivity patterns. To distinguish different sensitivity patterns (i.e., biological sensitivity to context, diathesis-stress, and vantage sensitivity), Roisman et al. (2012) proposed three critical tests, including the Regions of Significance (RoS), the proportion of interaction (PoI), and the proportion affected (PA) tests. In these tests, the independent variable, namely, family conflict or parental warmth, was denoted as X; the moderator (i.e., amygdalar and hippocampal responses during positive and negative emotional stimuli) was denoted as Z; and the outcome (i.e., behavioral problems and prosocial behaviors) was denoted as Y. First, in the RoS test, the regions of significance on the X were probed to determine whether the Y and Z are significantly associated at *both high and low ends* of X and whether the RoS is bounded by a $\pm$2SD range from the mean of X. Second, in the PoI test, the proportion of interaction was calculated as indices of sensitivity patterns. This test divided youths' differential responses to rearing environments into two regions, including the "for better" and the "for worse" regions. The "for better" metaphor referred to youth who were biologically vulnerable to the influences of adverse caregiving experiences (Boyce & Ellis, 2005). The "for worse" metaphor, in contrast, indicated youth who were neurobiologically predisposed to develop more adaptive responses to positive rearing environments (Boyce & Ellis, 2005). PoI stands for the proportion of "for better" region compared to "for worse" region, with a value close to .50 indicating biological sensitivity to context, a value close to zero indicating diathesis-stress, and a value close to one indicating vantage sensitivity patterns. Roisman et al. (2012) suggested that a PoI value between .40 and .60 indicated a possible biological sensitivity to context pattern. Third, the proportion affected (PA) was defined as the proportion of the population that was differentially affected by the moderator. Roisman et al. (2012) purported an acceptable PA index of over 16%.

Recent research tested the performance of these three critical tests and proposed revisions to these tests (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017). For example, Jolicoeur-Martineau et al. (2017) suggested that the critical tests based on crossover points of the interaction effects (e.g., the PoI and the PA tests) yielded more accurate results compared to the RoS test, especially under the circumstance of moderate effect size and large sample size. Additionally, Del Giudice (2017) argued that the criteria of PoI between .40 and .60, as proposed by Roisman et al. (2012), bore the limitation of producing false negatives (i.e., type-two error). Therefore, Del Giudice (2017) purported a broader window of PoI values (between 0.2 and 0.8) as suggesting a potential biological sensitivity to context pattern. The present study incorporated these revisions into distinguishing the sensitivity patterns. Specifically, this study utilized the PoI and the PA tests, with a PoI value between .20 and .80 coupled with a PA index between 16% and 84% confirming a significant biological sensitivity to context pattern.

Testing for biological sex differences. To test for biological sex differences, multiple group analysis (B. Muthén & Asparouhov, 2002) were conducted for each model with youths' biological sex as the grouping variable. When differences were found between male and female participants, the chi-square test was used to examine if the group difference was significant. Specifically, the parameter of the effect where the group difference was found was constrained to be the same across two groups. The significance of the chi-square difference between the constrained and non-constrained models was examined. Only a significant chi-square difference indicated a significant sex difference.

Missing data and model fit indices. In this study, the missing data rate ranged from 0.1% to 58.4%, with an average of 17.48% across all study variables. The major missingness occurred on the T3 youth adjustment variables (internalizing & externalizing problems and prosocial behaviors) due to the incomplete release of the ABCD study data. The full-informative maximum likelihood (FIML) algorithm was used to estimate missing data (R. J. Little & Rubin, 2019). The model fit was assessed using the chi-square test, the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR). Satisfactory model fit was determined based on criteria including a minimum value of .95 for CFI and TLI, a minimum value of .08 for SRMR, and a minimum value of .06 for RMSEA (Hu & Bentler, 1999).

Results

Descriptive and Correlation Analyses

The descriptive statistics of participants' EN-back behavioral performance were presented in Table 3.3. Overall, the performance indicated that most participants understood and could perform the task well across the two runs, with an average accuracy rate of 79.51% for 2-back and 86.56% for 0-back tasks. Accuracy was slightly higher for 0-back compared to the 2-back task. Independent sample t-tests and chi-square tests were conducted to compare youth whose fMRI data were excluded due to quality control reasons with other youth whose fMRI data were included in analyses. Results indicated that youth with excluded fMRI data were significantly younger ($t(11716.87) = -10.28$, $M_{\text{difference}} = -.10$, 95%$CI$ of $M_{\text{difference}}$ [-.11, -.08], $p < .001$), were raised in significantly lower household SES as exhibited by lower parental education ($t(11162.87) = -14.80$, $M_{\text{difference}} = -.32$, 95%$CI$ of $M_{\text{difference}}$ [-.36, -.28], $p < .001$) and lower household annual income ($t(10855) = -11.94$, $M_{\text{difference}} = -16.65$, 95%$CI$ of $M_{\text{difference}}$ [-19.39, -13.92], $p < .001$). Additionally, youth with excluded fMRI data reported significantly higher levels of family conflict ($t(11616.53) = 4.04$, $M_{\text{difference}} = .15$, 95%$CI$ of $M_{\text{difference}}$ [.08, .22], $p < .001$) and lower levels of parental warmth ($t(11428.90) = -2.37$, $M_{\text{difference}} = -.01$, 95%*CI* of $M_{\text{difference}}$ [.-.02, -.002], $p < .05$). These youth also exhibited significantly higher levels of externalizing problems during the follow-up assessments ($t(4549.41) = 4.01$, $M_{\text{difference}} = .62$, 95%*CI* of $M_{\text{difference}}$ [.31, .92], $p < .001$). Chi-square analyses further suggested that males ($\chi^2(1) = 4.81$ ($p < .05$)), and Black youth ($\chi^2(1) = 142.54$ ($p < .001$) had higher rate of excluded fMRI data.

Table 3.4 presented the descriptive statistics and correlation analyses of study variables. Youth reported relatively low levels of family conflict (*M* = 2.05, *Min*. = 0, *Max*. = 9) and high levels of parental warmth (*M* = 2.78, *Min*. = 0, *Max*. = 3) with small to moderate levels of variability (family conflict: *SD* = 1.95; parental warmth: *SD* = .30). Youth also exhibited significant internalizing and externalizing problems. At baseline assessment (T1), there were 9.9% (*n* = 1,175) of youth's internalizing problems and 6.1% (*n* = 724) of externalizing problems above the clinical cut-off score (T-score ≥ 64). At the 1-year follow-up assessment (T3), there were 9.7% (*n* = 479) of youth's internalizing problems and 5.0% (*n* = 245) of externalizing problems above this clinical cut-off score. Additionally, 7.2% (*n* = 854) of youth at T1 and 6.2% (*n* = 306) of youth at T3 had total problems T-scores above the clinical cutoff (≥ 64). Given that the clinical cutoff (T ≥ 64) corresponds with 90 percentile, youth in the ABCD dataset presented normal rates of internalizing problems and relatively low levels of externalizing problems. Parents also reported high levels of prosocial behaviors among youth (T1: *M* = 5.25, *SD* = 1.21; T3: *M* = 5.28, *SD* = 1.23).

Study variables were correlated in the hypothesized directions. Specifically, higher levels of family conflict were positively associated with elevated internalizing (T1: $r = .09$, 95% *CI*[.07, .11], $p < .001$; T3: $r = .07$, 95% *CI*[.04, .10], $p < .001$) and externalizing (T1: $r = .18$, 95% *CI*[.16, .20], $p < .001$; T3: $r = .16$, 95% *CI*[.13, .19], $p < .001$) problems, as well as reduced prosocial behaviors (T1: *r* = -.09, 95% *CI*[-.11, -.07], *p* < .001; T3: *r* = -.08, 95% *CI*[-.11, -.05], *p* < .001). Higher levels of parental warmth were linked to internalizing (T1: *r* = -.05, 95% *CI*[-.07, -.03], *p* < .001; T3: *r* = -.06, 95% *CI*[-.09, -.03], *p* < .001) and externalizing (T1: *r* = -.10, 95% *CI*[-.12, -.08], *p* < .001; T3: *r* = -.10, 95% *CI*[-.13, -.07], *p* < .001) problems, as well as increased prosocial behaviors (T1: *r* = .13, 95% *CI*[.11, .15], *p* < .001; T3: *r* = .13, 95% *CI*[.10, .16], *p* < .001). Youth internalizing and externalizing problems were positively correlated with each other across two assessments (*r* ranged from .42 to .74, *p* < .001), and were negatively related to prosocial behaviors (*r* ranged from -.37 to -.14, *p* < .001). Youths' amygdalar and hippocampal responses to both negative and positive emotional stimuli were all moderately and positively correlated (*r* ranged from .21 to .58, *p* < .001). Additionally, right amygdalar responses to negative stimuli was related to reduced externalizing problems at T1 (*r* = -.03, 95% *CI*[-.05, -.01], *p* < .01). Left amygdalar responses to positive stimuli was linked to increased internalizing problems at T3 (*r* = .04, 95% *CI*[.002, .08], *p* < .05).

Compared to female, male reported higher levels of perceived family conflict (*r* = -.06, 95% *CI*[-.08, -.04], *p* < .01) and lower levels of perceived parental warmth (*r* = .06, 95% *CI*[.04, .08], *p* < .001). Males also exhibited more externalizing problems (T1: *r* = .12, 95% *CI*[.10, .14], *p* < .001; T3: *r* = .11, 95% *CI*[.08, .14], *p* < .001) and fewer prosocial behaviors (T1: *r* = -.14, 95% *CI*[-.16, -.12], *p* < .001; T3: *r* = -.14, 95% *CI*[-.17, -.11], *p* < .001). White families reported higher annual household income (*M* = 128,325, *SD* = 68,552) compared to Black (*M* = 47,578, *SD* = 50,235) and Hispanic (*M* = 69,171, *SD* = 59,745) families. White youth reported fewer family conflicts (*r* = -.05, 95% CI[-.07, -.03], *p* < .001), higher levels of parental warmth (*r* = .03, 95% *CI*[.01, .05], *p* < .01), and decreased externalizing problems compared to youth of other race/ethnicity (T1: *r* = -.05, 95% *CI*[-.07, -.03], *p* < .001; T3: *r* = -.04, 95% CI[-.07, -.01], *p* < .001).

Black youth experienced more family conflicts (*r* = .09, 95% *CI*[.07, .11], *p* < .001) and less parental warmth (*r* = -.02, 95% *CI*[-.04, -.002], *p* < .001). Additionally, Black youth also evinced increased externalizing (T1: *r* = .06, 95% *CI*[.04, .08], *p* < .001; T3: *r* = .05, 95% *CI*[.02, .08], *p* < .001) and reduced internalizing (T1: *r* = -.05, 95% *CI*[-.07, -.03], *p* < .001; T3: *r* = -.05, 95% *CI*[-.08, -.02], *p* < .001) problems. Black youth also had lower levels of left hippocampal (*r* = -.04, 95% *CI*[-.06, -.02], *p* < .01) and amygdalar (*r* = -.03, 95% CI[-.05, -.01], *p* < .01) responses to positive emotional stimuli compared to youth of other race/ethnicity. Hispanic/Latino(a) youth showed increased internalizing problems at T1 (*r* = .03, 95% *CI*[.01, .05], *p* < .01) and higher levels of prosocial behaviors at both assessments (T1: *r* = .04, 95% *CI*[.02, .06], *p* < .001; T3: *r* = .04, 95% CI[.01, .07], *p* < .01). Higher levels of household income were related to fewer family conflict (*r* = -.13, 95% *CI*[-.15, -.11], *p* < .001) and increased parental warmth (*r* = .06, 95% CI[.04, .08], *p* < .01). Additionally, families with married (or living-together) parents reported fewer family conflicts (*r* = -.06, 95% *CI*[-.08, -.04], *p* < .001) and increased parental warmth (*r* = .02, 95% *CI*[.002, .04], *p* < .01). Youth reared by married or living-together caregivers exhibited reduced internalizing (T1: *r* = -.13, 95% *CI*[-.15, -.11], *p* < .001; T3: *r* = -.13, 95% *CI*[-.16, -.10], *p* < .001) and externalizing problems (T1: *r* = -.08, 95% *CI*[-.10, -06], *p* < .01; T3: *r* = -.08, 95% *CI*[-.11, -.05], *p* < .001).

Neural Activity During the EN-back Task

Paired-sample t-tests were conducted to examine the valence (positive vs. negative emotional stimuli) differences in amygdalar and hippocampal activations during emotional processing (Table 3.5). Results suggested that both left (t(6259) = 10.23, $M_{\text{difference}}$ = .06, 95%*CI* of $M_{\text{difference}}$ [.05, .07], $p < .001$) and right ($t(6257) = 10.53$, $M_{\text{difference}} = .06$, 95%*CI* of $M_{\text{difference}}$ [.05, .07], $p < .001$) amygdala exhibited higher levels of activations during negative emotional processing compared to positive emotional processing. Similarly, left ($t(6256) = 8.47$, $M_{\text{difference}} = .03$, 95%*CI* of $M_{\text{difference}}$ [.03, .04], $p < .001$) and right ($t(6260) = 8.83$, $M_{\text{difference}} = .04$, 95%*CI* of $M_{\text{difference}}$ [.03, .05], $p < .001$) hippocampus also showed higher levels of responses to negative emotional stimuli compared to positive emotional stimuli.

Hypothesis 1: Direct Effects of Family rearing environments on Youth Adjustment

Structural equation models were tested to examine the direct effects of family conflict and parental warmth on youth adjustment (Table 3.6 and Figure 3.3). Family conflict and parental warmth were tested separately. Both models exhibited acceptable fit indices (for family conflict model: $\chi^2(15) = 507.683$ ($p < .001$); CFI = .909; TLI = .800; RMSEA = .053; SRMR = .030; for parental warmth model: $\chi^2(15) = 494.735$ ($p < .001$); CFI = .907; TLI = .795; RMSEA = .052; SRMR = .030). Results showed that higher levels of family conflict was significantly related to youth's increased externalizing problems ($\beta = .04$, 95%*CI* [.02, .09], $p < .01$) and decreased prosocial behaviors at T3 ($\beta = -.11$, 95%*CI* [-.05, -.03], $p < .001$), after controlling for corresponding constructs at T1, youth sex, race/ethnicity, household income, and parental marital status. However, no significant association was found between internalizing problems and family conflict ($\beta = .02$, 95%*CI* [-.03, .42], $p = .09$). High levels of parental warmth was significantly linked to youth's decreased externalizing ($\beta = -.03$, 95%*CI* [-.60, -.04], $p < .05$) and internalizing ($\beta = -.04$, 95%*CI* [-4.88, -.28], $p < .05$) problems, as well as higher levels of prosocial behaviors ($\beta = .13$, 95%*CI* [.23, .39], $p < .01$). Youth's externalizing, internalizing problems and prosocial behaviors exhibited significant stability over time ($p < .001$).

Hypothesis 2: The Amygdalar Activations as Moderators on the Associations Between Family rearing environments and Youth Adjustment

Negative emotional stimuli & family conflict. Table 3.7, Figure 3.4, and Figure 3.5 presented the results of models with amygdalar responses to negative emotional stimuli as moderators on the associations between family conflict and youth adjustment. Compared to the baseline direct effect model, these two models exhibited significant improvement in chi-square model fit indices ($p < .001$).

Left amygdalar activations as moderator. The left column of Table 3.7 and Panel (A) of Figure 3.4 presented the model with left amygdalar activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 61.46$ ($p < .001$); CFI = .99; TLI = .94; RMSEA = .03; SRMR = .01. Higher levels of family conflict was related to increased externalizing problems ($\beta = .05$, 95%*CI* [.01, .03], $p < .001$) and reduced prosocial behaviors ($\beta = -.12$, 95%*CI* [-.02, -.01], $p < .001$), but not significantly associated with changes of internalizing problems from baseline to the one-year follow-up assessment ($\beta = .02$, 95%*CI* [-.01, .02], $p = .15$). Left amygdalar activations during negative emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: $\beta = -.02$, 95%*CI* [-.13, .04], $p = .30$; Internalizing: $\beta = .04$, 95%*CI* [-.01, .18], $p = .06$; Prosocial behaviors: $\beta = -.01$, 95%*CI* [-.03, .03], $p =.64$). The interaction term of family conflict and left amygdalar activations was significantly and positively associated with externalizing problems ($\beta = .04$, 95%*CI* [.004, .09], $p < .05$), but not internalizing problems ($\beta = .04$, 95%*CI* [-.02, .08], $p = .26$) or prosocial behaviors ($\beta = .00$, 95%*CI* [-.01, .02], $p = .90$). Overall, this model showed that left amygdalar responses to negative emotional stimuli significantly intensified the associations between elevated family conflict and youths' increased externalizing problems.

The upper panel of Figure 3.8 presented the Johnson-Neyman plot (P. O. Johnson & Neyman, 1936) as an interpretation of the moderating effects of left amygdalar activations during negative emotional stimuli on the associations between family conflict and youth's externalizing problems. In this figure, the X-axis indicated the moderator (i.e., left amygdalar activations), and the Y-axis represented the effects of family conflict on youth's externalizing problems. For youth with relatively high left amygdalar activations (76.1% of participants), high levels of family conflict were significantly associated with youths' increased externalizing problems. For youth with low amygdalar activations (23.9% of participants), the effect of family conflict on youth externalizing problems was not statistically significant.

The lower panel of Figure 3.8 displayed the simple slope figure and the differential susceptibility pattern critical tests of the moderating effects of left amygdalar activations during negative emotional stimuli on the associations between family conflict and youth's externalizing problems. Among participants with higher left amygdalar activations (presented by the solid line), low levels of family conflict were associated with reduced externalizing problems among youth. Among participants with lower left amygdalar activations (presented by the dotted line), family conflict was not significantly linked to youth's externalizing problems. A PoI of .72 and a PA of 66% were also obtained from this interpretation. Overall, this effect supported the biological sensitivity to context hypothesis (Boyce & Ellis, 2005; Ellis et al., 2005). Youth with high levels of activations in the left amygdala during negative emotional processing exhibited fewer externalizing problems in response to low-conflict family rearing environments. However, with the high levels of left amygdalar activations, youth who reported high levels of family conflict also showed elevated externalizing problems (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012).

Right amygdalar activations as moderator. The right column of Table 3.7 and Figure 3.5 presented the model with right amygdalar activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 32.64$ ($p < .001$); CFI = 1.00; TLI = .97; RMSEA = .02; SRMR = .01. High levels of family conflict was related to increased externalizing problems (β = .03, 95%*CI* [.01, .03], $p < .01$) and reduced prosocial behaviors (β = -.12, 95%*CI* [-.02, -.01], $p < .001$), but not significantly associated with changes of internalizing problems (β = .02, 95%*CI* [-.01, .02], $p = .17$). Right amygdalar activations during negative emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: β = .00, 95%*CI* [-.08, .07], $p = .90$; Internalizing: β = .02, 95%*CI* [-.04, .11], $p = .35$; Prosocial behaviors: β = -.03, 95%*CI* [-.04, .01], $p =.26$). The interaction term of family conflict and left amygdalar activations was not significantly associated with externalizing problems (β = .02, 95%*CI* [-.01, .06], $p = .22$), internalizing problems (β = .01, 95%*CI* [-.02, .05], $p = .45$), or prosocial behaviors (β = .02, 95%*CI* [-.01, .02], $p = .31$).

Positive emotional stimuli & parental warmth. Table 3.8, Figure 3.6, and Figure 3.7 presented the results of models with amygdalar responses to positive emotional stimuli as moderators on the associations between parental warmth and youth adjustment. Compared to the baseline direct effect model, these two models exhibited significant improvement in chi-square model fit indices ($p < .001$).

Left amygdalar activations as moderator. The left column of Table 3.8 and Figure 3.6 presented the model with left amygdalar activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 63.48$ ($p < .001$); CFI = .99; TLI = .94; RMSEA = .03; SRMR = .01. High levels of parental warmth was related to decreased externalizing problems (β = -.03, 95%*CI* [-.12, -.01], $p < .05$) and increased prosocial behaviors (β = .13, 95%*CI* [.07, .12], $p < .001$), but not significantly associated with changes of internalizing problems (β = -.02, 95%*CI* [-.18, .02], *p* = .11). Left amygdalar activations during positive emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: β = -.01, 95%*CI* [-.12, .07], *p* = .601; Internalizing: β = .02, 95%*CI* [-.04, .15], *p* = .25; Prosocial behaviors: β = -.03, 95%*CI* [-.03, .01], *p* =.20). The interaction term of parental warmth and left amygdalar activations was not significantly associated with externalizing problems (β = -.02, 95%*CI* [-.36, .13], *p* = .35), internalizing problems (β = .01, 95%*CI* [-.17, .33], *p* = .52), or prosocial behaviors (β = -.02, 95%*CI* [-.07, .07], *p* = .92).

Right amygdalar activations as moderator. The right column of Table 3.8 and Figure 3.7 presented the model with right amygdalar activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 36.61$ ($p < .001$); CFI = 1.00; TLI = .97; RMSEA = .02; SRMR = .01. High levels of parental warmth was related to increased prosocial behaviors (β = .02, 95%*CI* [.17, .31], *p* < .001), but not significantly associated with changes of externalizing (β = -.02, 95%*CI* [-.49, .02], *p* = .07) or internalizing (β = .01, 95%*CI* [-.27, .21], *p* = .81) problems. Right amygdalar activations during positive emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: β = .02, 95%*CI* [-.09, .05], *p* = .51; Internalizing: β = .02, 95%*CI* [-.06, .09], *p* = .68; Prosocial behaviors: β = .02, 95%*CI* [-.03, .01], *p* =.29). The interaction term of parental warmth and right amygdalar activations was not significantly associated with externalizing problems (β = .01, 95%*CI* [-.44, 97], *p* = .46), internalizing problems (-.02, 95%*CI* [-1.28, .61], *p* = .49), or prosocial behaviors (β = .02, 95%*CI* [-.14, .31], *p* =.46).

Hypothesis 3: The Hippocampal Activations as Moderators on the Associations Between Family rearing environments and Youth Adjustment

Negative emotional stimuli & family conflict. Table 3.9, Figure 3.9, and Figure 3.10 presented the results of models with hippocampal responses to negative emotional stimuli as moderators on the associations between family conflict and youth adjustment. Compared to the baseline direct effect model, these two models exhibited significant improvement in chi-square model fit indices ($p < .001$).

Left hippocampal activations as moderator. Specifically, the left column of Table 3.9 and Figure 3.9 presented the model with left hippocampal activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 31.75$ ($p < .001$); CFI = 1.00; TLI = .98; RMSEA = .02; SRMR = .01. High levels of family conflict was related to increased externalizing problems (β = .03, 95%*CI* [.01, 03], $p < .01$) and reduced prosocial behaviors (β = -.12, 95%*CI* [-.02, -.01], $p < .001$), but not significantly associated with changes of internalizing problems (β = .02, 95%*CI* [-.01, .02], $p = .21$). Left hippocampal activations during negative emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: β = -.03, 95%*CI* [-.21, .01], $p = .08$; Internalizing: β = .00, 95%*CI* [-.11, .11], $p = .95$; Prosocial behaviors: β = -.02, 95%*CI* [-.05, .02], $p =.46$). The interaction term of family conflict and left hippocampal activations was significantly and positively associated with internalizing problems (β = .04, 95%*CI* [.01, .13], $p < .05$), but not externalizing problems (β = .02, 95%*CI* [-.03, .09], $p = .27$) or prosocial behaviors (β = .01, 95%*CI* [-.02, .02], $p = .78$). Overall, this model showed that left hippocampal responses to negative emotional stimuli significantly exacerbated the associations between increased levels of family conflict and youths' elevated internalizing problems.

The upper panel of Figure 3.13 presented the Johnson-Neyman plot (P. O. Johnson & Neyman, 1936) as an interpretation of the moderating effects of left hippocampal activations during negative emotional stimuli on the associations between family conflict and youth's internalizing problems. In this figure, the X-axis indicated the moderator (i.e., left hippocampal activations), and the Y-axis represented the effects of family conflict on youth's internalizing problems. This figure showed that for youth with relatively high left hippocampal activations (32.50% of participants), high levels of family conflict had a significant impact on youth's increased internalizing problems. For youth with low hippocampal activations (67.50% of participants), the effect of family conflict on youth internalizing problems was not statistically significant.

The lower panel of Figure 3.13 displayed the simple slope figure and the differential susceptibility pattern critical tests of the moderating effects of left hippocampal activations during negative emotional stimuli on the associations between family conflict and youth's internalizing problems (Del Giudice, 2017; Roisman et al., 2012). Among participants with higher left hippocampal activations (presented by the solid line), low levels of family conflict were associated with reduced internalizing problems among youth. Among participants with lower left hippocampal activations (presented by the dotted line), family conflict was not significantly linked to youth's internalizing problems. A PoI of .49 and a PA of 66% were also obtained from this interpretation. Overall, this effect supported the biological sensitivity to context hypothesis (Boyce & Ellis, 2005; Ellis et al., 2005). Youth with high left hippocampal activations during negative emotional processing exhibited fewer externalizing problems in response to low-conflict family rearing environments. However, with the high left hippocampal activations, youth who reported high levels of interpersonal conflict in the family context also evinced increased externalizing problems (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012).

Right hippocampal activations as moderator. The right column of Table 3.9 and Figure 3.10 presented the model with right hippocampal activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 31.77$ ($p < .001$); CFI = 1.00; TLI = .97; RMSEA = .02; SRMR = .01. High levels of family conflict was related to increased externalizing problems (β = .03, 95%*CI* [.01, .03], $p < .01$) and reduced prosocial behaviors (β = -.12, 95%*CI* [-.02, -.01], $p < .001$), but not significantly associated with changes of internalizing problems (β = .02, 95%*CI* [-.01, .02], $p = .19$). Right hippocampal activations during negative emotional stimuli were associated with lower levels of externalizing problems (β = -.04, 95%*CI* [-.27, -.03], $p < .05$) but not significantly linked to internalizing problems (β = -.01, 95%*CI* [-.17, .08], $p = .50$) or prosocial behaviors (β = -.03, 95%*CI* [-.05, .01], $p =.18$). The interaction term of family conflict and left hippocampal activations was not significantly associated with externalizing problems (β = .01, 95%*CI* [-.04, .06], $p = .71$), internalizing problems (β = .00, 95%*CI* [-.06, .06], $p = .91$), or prosocial behaviors (β = .01, 95%*CI* [-.01, .02], $p =.68$).

Positive emotional stimuli & parental warmth. Table 3.10, Figure 3.11, and Figure 3.12 presented the results of models with hippocampal responses to positive emotional stimuli as moderators on the associations between parental warmth and youth adjustment. Compared to the baseline direct effect model, these two models exhibited significant improvement in chi-square model fit indices ($p < .001$).

Left hippocampal activations as moderator. The left column of Table 3.10 and Figure 3.11 presented the model with left hippocampal activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 35.93$ ($p < .001$); CFI = 1.00; TLI = .97; RMSEA = .02;

SRMR = .01. High levels of parental warmth was related to increased prosocial behaviors (β = .12, 95%*CI* [.13, .23], *p* < .001), but not significantly associated with changes of externalizing (β = -.02, 95%*CI* [-.38, .02], *p* = .08) or internalizing (β = -.01, 95%*CI* [-.23, .15], *p* = .67) problems. Left hippocampal activations during positive emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: β = -.01, 95%*CI* [-.13, 08], *p* = .61; Internalizing: β = .01, 95%*CI* [-.06, .14], *p* = .44; Prosocial behaviors: β = -.01, 95%*CI* [-.03, .02], *p* =.52). The interaction term of parental warmth and left hippocampal activations was negatively linked to internalizing problems (β = -.04, 95%*CI* [-1.73, -.10], *p* < .05) but not significantly associated with externalizing problems (β = -.02, 95%*CI* [-1.38, .34], *p* = .24) or prosocial behaviors (β = -.01, 95%*CI* [-.26, .18], *p* = .73). Overall, this model showed that left hippocampal responses to positive emotional stimuli significantly exacerbated the associations between increased levels of parental warmth and youths' reduced internalizing problems.

The upper panel of Figure 3.14 presented the Johnson-Neyman plot (P. O. Johnson & Neyman, 1936) as an interpretation of the moderating effects of left hippocampal activations during positive emotional stimuli on the associations between parental warmth and youth's internalizing problems. In this figure, the X-axis indicated the moderator (i.e., left hippocampal activations), and the Y-axis represented the effects of parental warmth on youth's internalizing problems. This figure showed that for youth with relatively high left hippocampal activations (10.8% of participants), parental warmth was linked to youth's internalizing problems significantly and negatively. For youth with moderate to low hippocampal activations (86.5% of participants), the effect of parental warmth on youth internalizing problems was not statistically significant. For a small portion of participants (2.7%) with extremely low hippocampal activations during the positive emotional stimuli, lower levels of parental warmth were significantly associated with reduced internalizing problems.

The lower panel of Figure 3.14 displayed the simple slope figure and the differential susceptibility pattern critical tests of the moderating effects of left hippocampal activations during positive emotional stimuli on the associations between parental warmth and youth's internalizing problems (Del Giudice, 2017; Roisman et al., 2012). Among participants with higher left hippocampal activations (presented by the solid line), low levels of parental warmth were associated with increased internalizing problems among youth. Among participants with lower left hippocampal activations (presented by the dotted line), parental warmth was not significantly linked to youth's internalizing problems. A PoI of .34 and a PA of 50% were also obtained from this interpretation. Overall, this effect supported the biological sensitivity to context hypothesis (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, youth with high left hippocampal activations during positive emotional processing exhibited increased internalizing problems in response to family rearing environments with low parental warmth. However, these youth also showed reduced internalizing problems in response to high levels of parental warmth (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012). Additionally, a small percentage of participants with extremely low hippocampal activations during positive emotional processing exhibited a "steeling" effect (i.e., moderate levels of stress strengthen youths' resilience and thus promote their positive developmental outcomes; Rutter, 2006). These youth showed reduced internalizing problems under family rearing environments with low parental warmth.

Right hippocampal activations as moderator. The right column of Table 3.10 and Figure 3.12 presented the model with right hippocampal activations as the moderator. This model exhibited excellent model fit: $\chi^2(6) = 36.54$ ($p < .001$); CFI = 1.00; TLI = .97; RMSEA = .02; SRMR = .01. High levels of parental warmth was related to increased prosocial behaviors (β = .12, 95%*CI* [.17, .31], $p < .001$), but not significantly associated with changes of externalizing (β = -.02, 95%*CI* [-.49, .03], $p = .08$) or internalizing (β = -.01, 95%*CI* [-.27, .21], $p = .77$) problems. Right hippocampal activations during positive emotional stimuli were not significantly linked to any developmental adjustment outcomes (Externalizing: β = -.02, 95%*CI* [-.17, .04], $p = .21$; Internalizing: β = .01, 95%*CI* [-.07, .12], $p = .65$; Prosocial behaviors: β = -.03, 95%*CI* [-.03, .25], $p =.18$). The interaction term of parental warmth and right hippocampal activations was not significantly associated with externalizing problems (β = .00, 95%*CI* [-1.32, 1.52], $p = .88$), internalizing problems (β = -.02, 95%*CI* [-1.69, .94], $p = .34$), or prosocial behaviors (β = -.02, 95%*CI* [-.52, .35], $p =.45$).

Hypothesis 4: Lateralization of Amygdalar and Hippocampal Activations

Table 3.11 presented the paired-sample t-tests on the brain hemisphere (left vs. right) differences of amygdalar and hippocampal activations during positive and negative emotional processing. Results showed that in response to both negative ($t(6261) = -3.16$, $M_{\text{difference}} = -.02$, 95%*CI* of $M_{\text{difference}}$ [-.03, -.01], $p < .01$) and positive ($t(6259) = -2.22$, $M_{\text{difference}} = -.01$, 95%*CI* of $M_{\text{difference}}$ [-.02, -.001], $p < .05$) emotional stimuli, the right amygdala exhibited higher levels of activations than the left amygdala. However, no significant difference was found for left vs. right hippocampal activations during either negative ($t(6261) = -.59$, $M_{\text{difference}} = -.002$, 95%*CI* of $M_{\text{difference}}$ [-.01, .004], $p = .520$) or positive ($t(6260) = .69$, $M_{\text{difference}} = .002$, 95%*CI* of $M_{\text{difference}}$ [-.004, .01], $p = .490$) emotional processing. Additionally, only the left amygdala and hippocampus were found to significantly exacerbate the influences of family rearing environments (parental warmth and family conflict) on youth adjustment. Specifically, left amygdalar responses to negative stimuli intensified the links between family conflict and youth's externalizing problems. Left hippocampal responses to positive and negative stimuli interacted with parental warmth and family conflict, respectively, and significantly predicted internalizing problems among youth. No significant moderating effect for right amygdalar or hippocampal functions was found. Therefore, left amygdalar and hippocampal activations during emotional processing were suggested to be more engaged in regulating youths' neurobiological sensitivity under the influences of family rearing environments.

Hypothesis 5: Biological Sex Differences

Table 3.12 presented the independent-sample t-tests for sex differences among study variables. Results showed that compared to male, female youth perceived lower levels of family conflict ($t(11792) = -6.50$, $M_{\text{difference}} = -.23$, $95\%CI$ of $M_{\text{difference}}$ [-.30, -.16], $p < .001$) and higher levels of parental warmth ($t(11830) = 6.27$, $M_{\text{difference}} = .04$, $95\%CI$ of $M_{\text{difference}}$ [.02, .05], $p < .001$). Females also exhibited reduced externalizing problems (T1: $t(11690) = -12.72$, $M_{\text{difference}} = 1.35$, $95\%CI$ of $M_{\text{difference}}$ [-1.56, -1.14], $p < .001$; T3: $t(4891) = -8.08$, $M_{\text{difference}} = -1.21$, $95\%CI$ of $M_{\text{difference}}$ [-.14, -.92], $p < .001$) and elevated prosocial behaviors during both baseline and 1-year follow-up assessments (T1: $t(11745) = 15.43$, $M_{\text{difference}} = .34$, $95\%CI$ of $M_{\text{difference}}$ [.30, .38], $p < .001$; T3: $t(4910) = 10.30$, $M_{\text{difference}} = .35$, $95\%CI$ of $M_{\text{difference}}$ [.29, .42], $p < .001$) compared to males. Further, female showed lower levels of left amygdalar ($t(6262) = -2.54$, $M_{\text{difference}} = -.02$, $95\%CI$ of $M_{\text{difference}}$ [-.03, -.004], $p < .05$) and hippocampal ($t(6265) = -2.12$, $M_{\text{difference}} = -.02$, $95\%CI$ of $M_{\text{difference}}$ [-.04, -.002], $p < .05$) activations during positive emotional processing.

To test for biological sex differences, multiple group analysis (B. Muthén & Asparouhov, 2002) was conducted for each model with youth's biological sex as a grouping variable. Biological sex differences were found on the roles of hippocampal activations as neurobiological sensitivity indicators. Specifically, hippocampal responses to positive stimuli only significantly exacerbated the effect of parental warmth on youth internalizing problems among male (β = -.04, 95%*CI* [-2.50, -.17], $p < .05$), not female (β = -.03, 95%*CI* [-2.63, .70], $p = .26$). Hippocampal responses to negative stimuli only significantly intensified the effect of family conflict on youth internalizing problems among female (β = .08, 95%*CI* [.03, .25], $p < .05$), not male (β = .01, 95%*CI* [-.06, .09], $p = .72$). However, the chi-square tests showed that these sex differences were not statistically significant (for negative stimuli: $\chi^2(1) = 3.18$, $p = .08$; for positive stimuli: $\chi^2(1) = .48$, $p = .49$). Therefore, no significant sex difference was found in these neurobiological sensitivity to family rearing environments models.

Discussion

Family caregiving experiences are critical for shaping youths' socioemotional and behavioral adjustment (Bronfenbrenner, 2005; Bronfenbrenner & Morris, 2006; Kogan et al., 2019; Oshri, Gray, et al., 2019; Oshri et al., 2013; Wickrama et al., 2008). Despite the robust and consistent associations between family rearing environments and youth developmental outcomes, considerable individual differences exist in youths' neurobiological sensitivity to the influences of family rearing environments (Ellis et al., 2011). Certain neurobiological factors, such as the hypersensitivity of the amygdala and hippocampus, render some youth to be more sensitive to the impacts of positive and negative family environments (Boyce & Ellis, 2005; Ellis et al., 2005). These factors interact with positive and negative rearing environments and significantly affected youths' socioemotional development. Youth with neurobiological sensitivity indicators are more likely to develop maladaptive outcomes when they experience adverse rearing environments. However, they also evince elevated adaptive responses to supportive caregiving experiences. In contrast, youth who lack such neurobiological sensitivity are not responsive to the influences of family rearing environments (Boyce & Ellis, 2005; Ellis et al., 2005). The current study examined the functions of the amygdala and hippocampus as neurobiological indicators to youths' sensitivity to family environments. Specifically, this study employed a sample of large, longitudinal, and nationally representative youth and their primary caregivers from the ABCD study (Volkow et al., 2018). The moderating effects of amygdalar and hippocampal responses to positive and negative emotional stimuli on the associations among family conflict, parental warmth, and youth adjustment were investigated.

Results overall supported the study hypotheses. First, family conflict and parental warmth were found to have significant effects on youths' development of psychopathology and prosocial behaviors. Second, left amygdalar responses to *negative* emotional stimuli significantly exacerbated the effects of family conflict on youths' externalizing problems. Similarly, left hippocampal responses to *negative* stimuli were found to be a significant moderator that intensified the associations between family conflict and internalizing problems. Left hippocampal activations during *positive* emotional stimuli significantly amplified the links between parental warmth and youths' reduced internalizing problems. All these moderating effects followed a biological sensitivity to context pattern (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, with high levels of amygdalar or hippocampal activations, youth who experienced adverse family rearing environments developed more problem behaviors. However, these youth who had elevated amygdalar or hippocampal activations also exhibited reduced problem behaviors as adaptive responses to positive rearing environments. Further, the right amygdala exhibited higher levels of activations during negative and positive emotional processing compared to the left amygdala. Lastly, males evinced higher levels of left amygdalar and hippocampal responses to positive emotional stimuli. However, there weren't any significant moderating effects of biological sex on the neurobiological sensitivity to family rearing environments.

Neural Activity During the EN-back Task

The current study found that youth exhibited higher levels of amygdalar and hippocampal activations in response to negative emotional stimuli, compared to positive emotional stimuli. Therefore, amygdalar and hippocampus were more engaged in processing negative emotional information, as presented by fearful facial expressions. These findings are expected for youth during the transition from childhood to adolescence. As youth transit to adolescence, there is an acceleration in the development of memory and reward sensitivity, as evident in dopaminrgic activity, which is linked to heightened neuorbiological sensitivity to social feedback (Silva, Shulman, Chein, & Steinberg, 2016; Somerville, 2013; Tottenham & Galván, 2016). Therefore, findings on the heightened amygdalar and hippocampal activations in response to negative emotional stimuli may suggest youths' increased salience and awareness of negative social evaluations and social feedback (Monk et al., 2008). Additionally, given the hippocampal roles in regulating stress reactivity (Wingenfeld & Wolf, 2014), it is expected to see increased activations and engagement of the hippocampus in response to stressful stimuli, such as fearful facial expressions. This elevated engagement in negative emotional processing can explain why the hippocampus exhibited higher levels of activations in response to negative stimuli, compared to positive stimuli.

It is important to note that the neural activity presented in the EN-back task reflected youths' emotional reactivity under sustained cognitive challenges (A. Cohen, Conley, Dellarco, & Casey, 2016b; Schweizer et al., 2019). The EN-back paradigm included two components, including working memory and emotional reactivity. Youth were instructed to complete 0-back and 2-back conditions in response to pictures of different emotional stimuli, including negative (fearful) faces, positive (happy) faces, neutral faces, and non-facial (places) stimuli. The emotional EN-back paradigm was first developed to examine the influence of affective information on working memory performance (Schweizer et al., 2019). Preliminary imaging analyses of an ABCD sub-sample (*n* = 517) have validated the working memory condition of the EN-back paradigm (Casey et al., 2018) among youth. Specifically, neuroimaging analyses show that youth exhibited significant fronto-parietal and fronto-thalamic activities in the 2-back vs. 0-back contrast (Casey et al., 2018). These activation patterns are consistent with previous working memory literature (Borst & Anderson, 2013; Engström, Vigren, Karlsson, & Landtblom, 2009; Oshri, Hallowell, et al., 2019; Philip et al., 2016).

Furthermore, the emotional EN-back paradigm can also be adopted to investigate the neural mechanisms underlying emotional processing during challenging cognitive tasks, especially working memory tasks (Casey et al., 2018). The emotional reactivity condition of the EN-back paradigm has been supported by some preliminary research (Schweizer et al., 2019). For example, in a meta-analysis, Schweizer et al. (2019) suggest that processing emotional information during working memory tasks is associated with activations of the amygdalo-hippocampal complex and the temporo-occipital cortex (including the fusiform gyrus). The amygdalo-hippocampal complex and temporo-occipital cortex are all involved in the processing of emotional information or facial recognition (Brose, Lövdén, & Schmiedek, 2014; LeDoux, 2000). Therefore, this meta-analysis provides a preliminary validation of the emotional component of the EN-back task (Schweizer et al., 2019). However, researchers also need to account for the possible suppressions of some social-affective neural activity during the on-going cognitive tasks (Stretton et al., 2012) when using the EN-back fMRI paradigm.

Hypothesis 1: Direct Effects of Family rearing environments on Youth Adjustment

In support of the first hypothesis, findings of the current study suggested that higher levels of family conflict were associated with youth's elevated externalizing problems and reduced prosocial behaviors. These findings corroborate empirical evidence on the robust and prolonged impact of family conflict on youth maladaptation (Kitzmann et al., 2003; Rhoades, 2008). According to the emotional security theory (Davies & Cummings, 1994), high levels of interpersonal conflict in the rearing environment form a hostile emotional climate and disrupt youth's emotional security. Emotional security is defined as youths' appraisal of positive and stable bondings among family members, even under stressful situations (Davies et al., 2002). As a regulatory system, emotional security organizes youths' responses to interpersonal discords in the family rearing environments through emotional reactivity and behavioral reactions (Cummings, Goeke-Morey, & Papp, 2004). When emotional security is disrupted, youth are more likely to respond to family conflict via negative emotional reactivity and maladaptive behaviors, such as increased externalizing problems and reduced prosocial behaviors (Kouros, Cummings, & Davies, 2010). Further, under a rearing environment with high levels of interpersonal conflicts, youth who are unable to cope with family stress might blame themselves for the inter-parental conflicts and feel responsible for helping solve the disagreements (Grych, Harold, & Miles, 2003). Youth who experience increased family conflict tend to internalize the feelings of guilt, sadness, shame, and self-blame, and are more likely to develop problem behaviors (Buehler, Lange, & Franck, 2007).

The current study also indicated that higher levels of parental warmth were linked to reduced internalizing problems and increased prosocial behaviors among youth. This finding corroborates the parental acceptance and rejection theory (PARTheory; Rohner, 2008; Rohner et al., 2004, 2005; Rohner et al., 2002). According to the PARTheory, youth are universally (across culture, gender, age, race/ethnicity) in need of emotional and affective support and warmth from their primary caregivers (Rohner et al., 2004). Youth form their perceptions of parental acceptance and rejection based on four classes of behaviors, including warmth/affection, hostility/aggression, indifference/neglect, and undifferentiated rejection (Rohner & Khaleque, 2010). When youths' emotional needs from their primary caregivers are not satisfied, they are more likely to self-blame for the harsh parenting behaviors and thus develop internalizing problems (Rohner et al., 2002). In contrast, when youth experience parental warmth and have affective needs fulfilled by primary caregivers, youth are more likely to learn from warm parenting behaviors, which promotes their development of prosocial behaviors (Rohner et al., 2005).

The associations between family environments, including family conflict and parental warmth, and youth adjustment are aligned with the specialization hypothesis in the evolutionary developmental perspective (Ellis, Bianchi, et al., 2017). Accordingly, youth who perceive the family environments as harsh, dangerous, and unpredictable are more likely to develop evolutionally-adaptive strategies (e.g., high vigilance, attention shifting, impulsivity) to cope with threatening and rapidly changing social environments (Ellis, Bianchi, et al., 2017; Frankenhuis & de Weerth, 2013; Frankenhuis et al., 2016; Lickliter & Honeycutt, 2013). The cognitive strategies specialized for harsh rearing experiences may be effective when youth were reared in an adverse environment characterized by high levels of interpersonal conflicts and low parental warmth. However, when the social context is not dangerous anymore during their later years of life, these strategies are not appropriate to the social context, and thus might be perceived as maladjustment (Frankenhuis & de Weerth, 2013). This mismatch of behaviors and environment might pose potential harm to youths' mental health and socio-developmental success (Frankenhuis et al., 2016; Lickliter & Honeycutt, 2013).

Hypothesis 2: The Amygdalar Activations as Moderators on the Associations Between Family rearing environments and Youth Adjustment

To test the second hypothesis, this study examined the moderating effects of the left and right amygdalar responses to positive and negative emotional stimuli on the associations between family rearing environments (including parental warmth and family conflict) and youth adjustment. Findings suggested that left amygdalar activations during negative emotional processing significantly exacerbated the effects of family conflict on youths' externalizing problems. Specifically, under a rearing environment with high interpersonal conflict, youth with higher levels of amygdalar activations exhibited elevated externalizing problems compared to youth with low amygdalar activation. Under family rearing environments with lower levels of conflict, in contrast, youth with elevated amygdalar responses reported reduced externalizing problems. Overall, this sensitivity pattern was aligned with the biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, heightened amygdalar activations intensified youths' problem behaviors when they reported high levels of family conflict. These heightened amygdalar activations also promoted reduced externalizing problems in response to family rearing environments with low levels of conflict. This finding suggests that left amygdalar activations during negative emotional stimuli are a neurobiological sensitivity indicator that may exacerbate the influences of family rearing environments on youth adjustment.

This role of the left amygdala in youths' neurobiological sensitivity can be attributed to its functions in modulating emotional reactivity. Decades of research suggests that the amygdala plays a critical role in negative emotional processing, especially fear (LeDoux, 1998, 2000).

Interpersonal conflict in the family rearing environments is a major source of negative emotional stimuli. Thus, amygdalar activations to negative emotional stimuli render youth to detect the potential threats rendered by family conflict and generate adaptive responses (LeDoux, 2000). In contrast, youth who lack amygdalar activations in response to negative emotional stimuli might be less sensitive in detecting these potential threats, and thus are less impacted by family stress. Overall, amygdalar responses during negative emotional processing can indicate youths' neurobiological sensitivity, which interacts with family rearing environments, as characterized by family conflict levels, and affect youths' externalizing problems.

Hypothesis 3: The Hippocampal Activations as Moderators on the Associations Between Family rearing environments and Youth Adjustment

To test the third hypothesis, this study examined the moderating effects of the left and right hippocampal responses to positive and negative emotional stimuli on the associations between family rearing environments and youth adjustment. Results showed that left hippocampal responses to negative emotional stimuli significantly exacerbated the impact of family conflict on youth internalizing problems. Similarly, left hippocampal activations to positive emotional stimuli significantly intensified the associations between parental warmth and youth internalizing problems. Both moderating effects followed a biological sensitivity to context pattern (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, when reporting stressful family rearing environments (i.e., high levels of conflict or low levels of parental warmth), youth with high activations in the left hippocampus exhibited elevated levels of internalizing problems. In contrast, youth who reported supportive rearing environments (i.e., high levels of parental warmth and low family conflict) and evinced high left hippocampal responses exhibited reduced internalizing problems. These findings indicate that left hippocampal responses to emotional stimuli can serve as a neurobiological sensitivity indicator that exacerbates the influences of family rearing environments on youth adjustment.

The role of the hippocampus as a neurobiological sensitivity indicator is relevant to its functions in regulating emotional reactivity and stress responses (Schriber et al., 2017). The hippocampus plays an essential role in binding the emotional and environmental elements of experiences and consolidating explicit memories (Burgess, Maguire, & O'Keefe, 2002) and discriminating between threat and safety (Ji & Maren, 2007). Further, the hippocampus is a part of a negative feedback mechanism that modifies the HPA axis responses to acute stressors (Mizoguchi et al., 2003; Wingenfeld & Wolf, 2014). Therefore, high levels of hippocampal activations may indicate elevated emotional and stress reactivity during emotional processing. The elevated emotional and stress reactivity renders youth to be more vulnerable under adverse rearing environments, but meanwhile, also be able to benefit more from a nurturing and supportive family rearing environments (Ellis et al., 2011).

Lastly, for a small percentage of youth with high hippocampal deactivations (i.e., negative activation beta coefficients) during positive emotional processing, lower levels of parental warmth were related to reduced internalizing problems. This finding is aligned with the “steeling” effect (R. T. Liu, 2015; Repetti & Robles, 2016; Rutter, 2006). The “steeling” effect refers to that the exposure to moderate levels of certain stressful experiences can strengthen youths’ resilience to future stressors, and thus promotes positive developmental outcomes (R. T. Liu, 2015; Repetti & Robles, 2016; Rutter, 2006). In the current study, hippocampal deactivations may indicate the suppression of the social-affective brain circuitry by the activations of other neural systems during emotional processing during the EN-back task, such as the cognitive control system (Stretton et al., 2012). Further, the activities of the cognitive control neural circuitry are associated with youths' executive functioning, which has been shown to promote the "steeling" effect of youth development under early life stress (Finch & Obradović, 2017).

Hypothesis 4: Lateralization of Amygdalar and Hippocampal Activations

Lateralization of amygdalar and hippocampal activations in response to emotional stimuli. The results of the current study showed that the right amygdala exhibited higher magnitudes (i.e., effect sizes) of activations in response to both positive and negative emotional stimuli compared to the left amygdala. In contrast, no significant brain hemisphere difference was found in hippocampal activations during either positive or negative emotional stimuli. These findings did not support the original hypothesis (i.e., hypothesis 4a). The heightened activations in the right amygdala may be attributed to the lateralized amygdalar activities in response to different types of stimuli (Hamid, 2014; Markowitsch, 1999). Studies suggest that the left amygdala is more reactive to semantic information (e.g., language and narratives). The right amygdala, in contrast, is more closely related to non-semantic and visual (e.g., pictorial or image-related) stimuli (Hamid, 2014; Markowitsch, 1999; Sergerie et al., 2008). Given that the EN-back task employs visual stimulation, it is possible that the right amygdala is more engaged in this task, and thus exhibits higher levels of emotional reactivity. Additionally, despite the statistically significant mean differences between left and right amygdalar activations, the effect sizes of these mean differences were small (Cohen's d ranged from -.025 to -.024). These small effect sizes corroborate the meta-analysis findings that suggest similar magnitudes of amygdalar activations across the left and right brain hemispheres (Baas et al., 2004; Sergerie et al., 2008).

Neurobiological sensitivity indicators lateralized to the left brain hemisphere. The current study also found that only left amygdalar and hippocampal activations to emotional stimuli significantly exacerbated the effects of family rearing environments on youth adjustment. These findings supported hypothesis 4b by suggesting that the left amygdala and hippocampus were more salient brain regions that indicate youths' neurobiological sensitivity to family rearing environments indicators. The localization of neurobiological sensitivity indicators on the left brain hemisphere was aligned with both structural (e.g., Schriber et al., 2017) and functional (e.g., McLaughlin et al., 2014) evidence in previous empirical studies that examined youths' neural sensitivity to environmental influences.

Left amygdalar activations were found to intensify the impact of family conflict on youths' externalizing problems. The differences between the right and left amygdalar functions in neurobiological sensitivity can be attributed to the temporal dynamics of the amygdala in response to emotional stimuli (Sergerie et al., 2008). Research suggests that the right amygdala is involved in the rapid and automatic detection of an emotional stimulus. However, the left amygdala mediates the more detailed and elaborate analyses of positive and negative emotions (Gläscher & Adolphs, 2003; Markowitsch, 1999; Wright et al., 2003). The detailed and specific evaluation of emotional information localized on the left amygdala may play a more salient role for youth to determine the emotional characteristics of the family rearing environments and generate behavioral reactions accordingly (Wright et al., 2003). Therefore, compared to the right amygdala, the left amygdala may be more critical for shaping youths' sensitivity to family rearing environments.

Furthermore, left hippocampal activations were found to exacerbate youths' development of internalizing problems under the influences of high family conflict and low parental warmth. This finding contributes to the body of research that links stress-related psychopathology to the left hippocampus in particular (MacMaster & Kusumakar, 2004; Schriber et al., 2017;

Vythilingam et al., 2002; Whittle et al., 2013). Empirical evidence suggests that the impact of early life experiences on hippocampal structures and functions is particularly localized to the left rather than the right (Schriber et al., 2017; Vythilingam et al., 2002; Whittle et al., 2013). Further, the left hippocampus is more closely linked to youths' development of psychopathology, especially internalizing problems, compared to the right hippocampus (MacMaster & Kusumakar, 2004). This localization of neurobiological sensitivity on the left hippocampus may be relevant to the greater expression of corticosteroid receptors and elevated engagement in HPA axis regulation in the left hippocampus (Hou et al., 2013; Madsen et al., 2012). Because of the localization of corticosteroid receptors and HPA axis regulation engagement, the left hippocampus is more engaged in regulating youths' acute stress responses and thus plays a more significant role in neurobiological sensitivity compared to the right hippocampus.

Hypothesis 5: Sex Differences

Biological sex differences in youth adjustment. In the current study, primary caregivers reported higher levels of externalizing problems and reduced prosocial behaviors for male youth, compared to female youth, with small but statistically significant effect sizes. These findings are aligned with previous empirical evidence on sex differences in youth adjustment (Chaplin & Aldao, 2013; Kuhnert et al., 2017). These sex differences in youths' externalizing problems and prosocial behaviors can be attributed to the interactions of biological sex distinctions (e.g., genetic expressions, sex hormones) and gender-specific socialization experiences (L. R. Brody, 2000; L. R. Brody & Hall, 2008; Chaplin & Aldao, 2013). Additionally, this study found no significant sex differences in youths' internalizing problems at both baseline and 1-may follow-up assessments. These non-significant sex differences in internalizing problems might be attributed to measurement biases induced by reports from the primary caregivers. As suggested by the literature on the assessment of youth psychopathology, internalizing problems are more difficult to detect among youth and adolescents, compared to externalizing problems (Acion et al., 2019; Hussong, Jones, Stein, Baucom, & Boeding, 2011). Youths' self-report internalizing problems can contribute unique information that is beyond parent-report assessments (Hope et al., 1999). However, as the current study employed a sample from a national data archive, only parent-report internalizing problems were available for analyses.

Biological sex differences in neurobiological sensitivity to family rearing environments. The findings of this study suggested that males exhibited higher levels of left amygdalar and hippocampal activations during positive emotional processing compared to females. However, the effect sizes of these sex differences were small (Cohen's $d < .10$). No other sex differences were detected in right amygdalar and hippocampal responses to positive or negative emotional stimuli. No significant sex differences were found in left amygdalar and hippocampal activations during negative emotional processing either. Furthermore, multiple group analyses (B. Muthén & Asparouhov, 2002) indicated no significant sex differences in the moderating effects of hippocampal or amygdalar activations on the links between family rearing environments and youth adjustment. Overall, the results of the current study suggest that the roles of the amygdala and hippocampus as indicators of neurobiological sensitivity to family rearing environments might be universal across male and female groups.

Limitations

There were several limitations of the current study that needed to be taken into consideration. First, the measurement tools employed by the ABCD study to assess family rearing environments, including family conflict and parental warmth, were shortened or trimmed to reduce the costs linked to assessment time and participant fatigue. For example, the parental acceptance scale from the CRPBI (E. S. Schaefer, 1965) was shortened from ten to five items. These shortened or trimmed measurement tools might lead to compromised construct validity. Additionally, youth adjustment was only assessed via reports from primary caregivers. This limitation in data availability might lead to measurement biases in the assessment of internalizing problems, which could hinder possible biological sex differences. The measurement biases in parent-report youth adjustment might also explain the relatively low internal reliability of the internalizing problems subscale. However, the significant correlations among family conflict, parental warmth, and youth adjustment might indicate good construct validity on shortened scales.

Second, the assessment of the family rearing environments in the ABCD study dataset could not reflect the full spectrum and multi-dimensional characteristics of family environments. Despite that family conflict and parental warmth assessed the negative and positive components of family rearing environments, respectively, they were not conceptually on the opposite poles of the interpersonal relationship quality among family members. Indeed, high levels of family conflict and parental warmth could occur in the same family rearing environments (Skopp, McDonald, Jouriles, & Rosenfield, 2007).

Third, data on the family rearing environments in the ABCD study were skewed to the positive extreme and exhibited small to moderate variance. For example, the mean score of family conflict was 2.05 (out of a range of 0 to 9; *SD* = 1.95), and the average of parental warmth was 2.78 (out of a range of 1 to 3; *SD* = .30). These two measures could not capture the full continuum of family rearing environments from positive to negative. However, using an assessment tool to measure the full spectrum of rearing environments is critical to examine neurobiological sensitivity patterns accurately and comprehensively (Boyce & Ellis, 2005; Ellis et al., 2005). Overall, the current study bore limitations in measurement designs, which might conceal potential biological sensitivity to context patterns. However, despite employing limited measurement tools, findings of this study have already revealed supports to the biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005). Optimizing the assessment methods to fully capture the continuum and dimensionality of family rearing environments should further confirm, rather than eliminate, these findings.

Fourth, the average behavioral performance (i.e., percentage of correct responses) during the EN-back task was 83.04%, which is lower than the accuracy reported in prior literature using the N-back task (A. Cohen, et al., 2016a). However, youth were 9 to 10 years old when they completed the EN-back task. Given that the EN-back task was difficult for such a young age group, and the task difficulty was not individualized to participants' performance levels, this accuracy was acceptable (Casey et al., 2018).

Lastly, the current dataset included a significant amount of missing data. The missing data on youth adjustment assessed at T3 (i.e., 1-year follow-up; 58.4% of participants) were mainly due to the partial T3 data release. The missing data on amygdalar and hippocampal activations during emotional processing (48.1% of participants) were caused by multiple quality-control reasons, including excessive movements, anatomical data quality issues, poor behavioral performance, and distorted fMRI images from all Philips MRI scanners. To address this issue, the current study employed the FIML algorithm to estimate missing data, which has been demonstrated to be effective and accurate (T. D. Little, Lang, Wu, & Rhemtulla, 2016).

Table 3.1 *ABCD Harmonized Imaging Scanning Parameters*

Scanner	**Siemens (Prisma VE11B-C)**			**Philips (Achieva dStream, Ingenia)**			**GE (MR750, DV25-26)**		
	T1	T2	fMRI	T1	T2	fMRI	T1	T2	fMRI
Matrix	256×256	256×256	90×90	256×256	256×256	90×90	256×256	256×256	90×90
Slices	176	176	60	225	256	60	208	208	60
FOV	256×256	256×256	216×216	256×256	256×256	216×216	256×256	256×256	216×216
%FOV phase	100%	100%	100%	93.75%	100%	100%	100%	100%	100%
Resolution (mm)	1.0×1.0×1.0	1.0×1.0×1.0	2.4×2.4×2.4	1.0×1.0×1.0	1.0×1.0×1.0	2.4×2.4×2.4	1.0×1.0×1.0	1.0×1.0×1.0	2.4×2.4×2.4
TR (ms)	2500	3200	800	6.31	2500	800	2500	3200	800
TE (ms)	2.88	565	30	2.9	251.7	30	2	60	30
TI (ms)	1060	NA	NA	1060	NA	NA	1060	NA	NA
Flip Angle (deg)	8	Variable	52	8	90	52	8	Variable	52
Parallel Imaging	2 ×	2 ×	Off	1.5×2.2	1.5×2.0	Off	2 ×	2 ×	Off
Multiband Acc.	Off	Off	6	Off	Off	6	Off	Off	6
Acquisition Time	7:12	6:35	--	5:38	2:53	--	6:09	5:50	--

Note. This information was obtained from Casey et al. (2018). FOV = Field-of-view, TR = Repetition time, TE = Echo time, TI = Inversion time, Multiband Acc. = Multiband acceleration.

Table 3.2 *Intra-Class Correlation Coefficients for Study Variables*

Variable	Intra-class correlation	95% *CI*
Family Conflict - T1	.0220	[.012, .045]*
Parental Acceptance - T1	.0040	[.001, .009]*
Prosocial Behaviors - T1	.0050	[.003, .013]*
Prosocial Behaviors - T3	.0030	[.000, .012]*
Internalizing Problems - T1	.0180	[.010, .039]*
Internalizing Problems - T3	.0170	[.008, .038]*
Externalizing Problems - T1	.0220	[.012, .045]*
Externalizing Problems - T3	.0150	[.007, .035]*
Left Hippocampus - Negative - T1	.0001	[-.001, .003]
Left Amygdala - Negative - T1	.0003	[-.001, .004]
Right Hippocampus-Negative - T1	-.0001	[-.001, .003]
Right Amygdala - Negative - T1	-.0009	[-.002, .001]
Left Hippocampus-Positive - T1	.0010	[-.001, .006]
Left Amygdala - Positive - T1	.0030	[.001, .010]*
Right Hippocampus - Positive - T1	.0010	[-.001,.005]
Right Amygdala - Positive - T1	.0005	[-.001, .004]

Note. T1 = Baseline assessments; T3 = 1-year follow-up assessments; Negative = Response to negative emotional stimuli; Positive = Response to positive emotional stimuli.

Table 3.3. *Summary of Participants' Emotional N-back Task Performance*

	Mean	*SD*	Min.	25%	Med.	75%	Max.
Total	83.04%	7.64%	61.25%	78.13%	83.75%	88.75%	99.38%
Total 2-back	79.51%	8.58%	61.25%	72.50%	80.00%	86.25%	100.00%
Total 0-back	86.56%	9.25%	61.25%	82.50%	88.75%	93.75%	100.00%
Positive emotions	84.43%	9.19%	50.00%	77.50%	85.00%	92.50%	100.00%
Positive emotions 2-back	80.74%	11.22%	15.00%	75.00%	80.00%	90.00%	100.00%
Positive emotions 0-back	84.66%	9.25%	42.50%	80.00%	85.00%	92.50%	100.00%
Negative emotions	81.24%	11.34%	30.00%	75.00%	80.00%	90.00%	100.00%
Negative emotions 2-back	83.56%	8.91%	47.50%	77.50%	85.00%	90.00%	100.00%
Negative emotions 0-back	80.86%	10.90%	40.00%	75.00%	80.00%	90.00%	100.00%
Neutral emotions	79.50%	10.27%	30.00%	72.50%	80.00%	87.50%	100.00%
Neutral emotions 2-back	75.20%	12.57%	5.00%	65.00%	75.00%	85.00%	100.00%
Neutral emotions 0-back	88.13%	11.78%	30.00%	80.00%	90.00%	95.00%	100.00%
Non-face Place	88.07%	11.86%	30.00%	80.00%	90.00%	95.00%	100.00%
Non-face Place 2-back	86.26%	11.70%	10.00%	80.00%	90.00%	95.00%	100.00%
Non-face Place 0-back	83.79%	13.57%	25.00%	75.00%	85.00%	95.00%	100.00%

Note. This table displays youths' rate of correct trails in the Emotional N-back task. *SD* = Standard deviation, Min. = Minimum, 25% = 25% percentile, Med. = Median, 75% = 75% percentile, Max. = Maximum.

Table 3.4 *Correlation and Descriptive Statistics of Study Variables (N = 11,875)*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1. Family Conflict T1	--																					
2. Parental Warmth T1	-.29**	--																				
3. Externalizing T1	.18**	-.10**	--																			
4. Internalizing T1	.09**	-.05**	.58**	--																		
5. Prosocial T1	-.09**	.13**	-.36**	-.16**	--																	
6. Externalizing T3	.16**	-.10**	.74**	.42**	-.30**	--																
7. Internalizing T3	.07**	-.06**	.43**	.69**	-.14**	.56**	--															
8. Prosocial T3	-.08**	.13**	-.33**	-.16**	.55**	-.37**	-.18**	--														
9. Left Hippocampus-Negative T1	-.01	-.01	-.01	-.01	.02	-.01	.02	.01	--													
10. Left Amygdala - Negative T1	-.01	-.01	-.01	-.01	.02	-.01	.03	.01	.58**	--												
11. Right Hippocampus-Negative T1	-.02	.00	-.01	-.02	.01	-.02	.00	.01	.65**	.45**	--											
12. Right Amygdala - Negative T1	-.02	.01	-.03**	-.02	.02	-.01	.02	.02	.44**	.50**	.54**	--										
13. Left Hippocampus-Positive T1	.00	.00	.00	.00	-.01	.01	.03	-.01	.46**	.31**	.31**	.21**	--									
14. Left Amygdala - Positive T1	.00	.01	.00	.01	.01	.01	.04*	.00	.27**	.44**	.21**	.24**	.58**	--								
15. Right Hippocampus-Positive T1	.00	.02	.00	-.01	.00	-.01	.01	.02	.32**	.23**	.44**	.26**	.67**	.47**	--							
16. Right Amygdala - Positive T1	-.01	.00	.00	.00	.01	.01	.03	-.01	.23**	.25**	.25**	.46**	.45**	.53**	.55**	--						
17. Youth sex	.06**	-.06**	.12**	.01	-.14**	.11**	.00	-.14**	.01	.02	.00	.01	.03*	.27*	.01	.01	--					
18. Race-White	-.05**	.03**	-.05**	.00	-.02	-.04**	.00	-.02	.01	.01	-.01	-.01	.01	.02	.01	.01	.02*	--				
19. Race-Black	.09**	-.02*	.06**	-.05**	-.02	.05**	-.05**	.00	-.01	-.02	.00	.01	-.03*	-.04**	-.01	-.01	-.02*	-.44**	--			
20. Race-Hispanic	-.01	.01	-.01	.03**	.04**	.01	.03	.04**	.00	.02	.00	.01	.00	.00	.00	.00	.00	-.53**	-.21**	--		
21. Household Income T1	-.11**	.05**	-.14**	-.09**	.01	-.13**	-.08**	-.01	.01	.01	-.01	.00	-.01	.01	-.02	-.02	.00	.37**	-.29**	-.22**	--	
22. Parental Marital Status	-.06**	.02**	-.13**	-.08**	.02	-.13**	-.08**	.00	-.01	.01	-.01	.02	.00	.01	-.01	.01	.02	.27**	-.34**	-.05**	.37**	--
Mean	2.05	2.78	4.45	5.05	5.25	3.98	5.01	5.18	.02	.05	.02	.06	-.02	-.01	-.02	.00	1.52	.52	.15	.20	103.09	.74
SD	1.95	.30	5.86	5.53	1.21	5.32	5.37	1.23	.30	.43	.32	.46	.31	.41	.31	.43	.50	.50	.36	.40	72.94	.44
Minimum	.00	1.00	.00	.00	.00	.00	.00	.00	-1.78	-2.87	-1.63	-3.17	-1.74	-2.57	-1.85	-2.48	1.00	.00	.00	.00	2.50	.00
Maximum	9.00	3.00	49.00	51.00	6.00	43.00	42.00	6.00	1.60	2.36	2.07	2.62	1.49	2.33	1.68	2.60	2.00	1.00	1.00	1.00	250.00	1.00
Skewness	.94	-1.82	2.30	1.94	-1.77	2.26	1.81	-1.58	-.07	-.32	.10	0.14	-.33	-.37	-.25	-.01	-.09	-.09	1.95	1.47	.59	-1.08
Kurtosis	.24	3.92	7.03	5.13	2.90	6.61	4.27	2.09	3.95	7.03	5.28	6.84	4.51	6.36	4.33	5.30	-1.99	-1.99	1.82	.16	-.55	-0.84

Note. T1 = Baseline assessment; T3 = 1-year follow-up assessment; Externalizing = Externalizing problems; Internalizing = Internalizing problems; Prosocial = Prosocial behaviors; Negative = Response to negative emotional stimuli; Positive = Response to positive emotional stimuli; *SD* = Standard deviation. Youth sex was coded as 1 = female and 2 = male; Race and parental marital status variables were coded as binary (0/1) variables; Household income was coded into $1,000. $^{*}p$ < .05, $^{**}p$ < .01.

Table 3.5. *Paired-Sample t-tests for the Mean Valence (Positive vs. Negative) Differences of Amygdalar and Hippocampal Responses to Emotional Stimuli*

Brain Hemisphere	ROI	Positive		Negative		$M_{\text{difference}}$	95% *CI* of $M_{\text{difference}}$	*t*	*df*	Cohen's *d*	*p*-value
		M	*SD*	*M*	*SD*						
Left	Hippocampus	-.0166	.3029	.0171	.3001	.0336	[.0258, .0414]	8.470***	6256	.107	<.001
	Amygdala	-.0107	.4093	.0460	.4199	.0556	[.0458, .0675]	10.230***	6259	.129	<.001
Right	Hippocampus	-.0180	.3065	.0196	.3149	.0366	[.0284, .0447]	8.828***	6260	.101	<.001
	Amygdala	.0025	.4294	.0634	.4535	.0610	[.0496, .0723]	10.533***	6257	.133	<.001

Note. ROI = Region of interests; *M* = Mean; *SD* = Standard Deviation; $M_{\text{difference}}$ = Mean difference; *CI* = Confidence interval; *df* = Degree of freedom. The unit of valence differences presented here is standardized activation coefficient β. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

Table 3.6. *The Direct Effects Models*

Family Environment Variables	Family Conflict				Parental Warmth			
Models	*B (SE)*	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Direct Effects								
Family Environment (T1) → EXT (T3)	.058 (.017)	.040	[.024, .092]**	.001	-.321 (.144)	-.031	[-.603, -.039]*	.025
Family Environment (T1) → INT (T3)	.197 (.116)	.024	[-.030, .424]	.089	-2.584 (1.174)	-.042	[-4.884, -.283]*	.028
Family Environment (T1) → Prosocial (T3)	-.036 (.005)	-.111	[-.046, -.026]***	<.001	.310 (.042)	.130	[.227, .392]***	<.001
Control								
EXT (T1) → EXT (T3)	.668 (.011)	.690	[.646, .690]***	<.001	.671 (.011)	.693	[.649, .692]***	<.001
Race – White → EXT (T3)	.065 (.045)	.034	[-.024, .153]	.151	.070 (.045)	.037	[-.017, .158]	.116
Race – Black → EXT (T3)	.016 (.057)	.006	[-.095, .127]	.778	.025 (.057)	.009	[-.086, .136]	.655
Race – Hispanic → EXT (T3)	.041 (.050)	.018	[-.056, .138]	.410	.045 (.049)	.020	[-.052, .142]	.359
Youth Sex → EXT (T3)	.058 (.023)	.031	[.013, .104]*	.011	.057 (.023)	.030	[.012, .103]*	.013
Income (T1) → EXT (T3)	-.029 (.015)	-.033	[-.058, .000]	.051	-.034 (.015)	-.038	[-.063, -.005]*	.023
PMarital → EXT (T3)	-.078 (.032)	-.038	[-.140, -.015]*	.015	-.073 (.032)	-.036	[-.136, -.011]*	.022
INT (T1) → INT (T3)	3.730 (.102)	.612	[3.531, 3.929]***	<.001	3.730 (.101)	.612	[3.532, 3.928]***	<.001
Race – White → INT (T3)	.119 (.253)	.011	[-.376, .614]	.638	.150 (.254)	.013	[-.347, .647]	.555
Race – Black → INT (T3)	-.635 (.325)	-.039	[-1.272, .002]	.051	-.591 (.325)	-.036	[-1.228, .045]	.069
Race – Hispanic → INT (T3)	.214 (.297)	.016	[-.368, .797]	.471	.252 (.300)	.019	[-.336, .841]	.400
Youth Sex → INT (T3)	.085 (.149)	.008	[-.206, .376]	.567	.072 (.147)	.006	[-.217, .360]	.627
Income (T1) → INT (T3)	-.073 (.098)	-.014	[-.265, .119]	.455	-.085 (.098)	-.017	[-.277, .106]	.382
PMarital → INT (T3)	-.514 (.218)	-.043	[-.942, -.086]*	.019	-.497 (.216)	-.042	[-.920, -.073]*	.022
Prosocial (T1) → Prosocial (T3)	.345 (.021)	.358	[.305, .385]***	<.001	.322 (.021)	.333	[.281, .363]***	<.001
Race – White → Prosocial (T3)	.029 (.011)	.066	[.007, .050]**	.009	.024 (.011)	.056	[.002, .046]*	.030
Race – Black → Prosocial (T3)	.011 (.015)	.018	[-.019, .042]	.460	.005 (.016)	.008	[-.026, .036]	.752
Race – Hispanic → Prosocial (T3)	.025 (.013)	.049	[.000, .049]	.052	.020 (.013)	.040	[-.005, .045]	.114
Youth Sex → Prosocial (T3)	-.050 (.006)	-.116	[-.063, -.038]***	<.001	-.051 (.006)	-.118	[-.064, -.038]***	<.001
Income (T1) → Prosocial (T3)	.013 (.004)	.065	[.005, .021]**	.003	.016 (.004)	.079	[.007, .024]***	<.001
PMarital → Prosocial (T3)	-.07 (.008)	-.015	[-.023, .010]	.413	-.009 (.008)	-.021	[-.026, .007]	.260
Model Fit Indices	$\chi^2(15) = 507.683$ ($p < .001$); CFI = .909; TLI = .800; RMSEA = .053; SRMR = .030				$\chi^2(15) = 494.735$ ($p < .001$); CFI = .907; TLI = .795; RMSEA = .052; SRMR = .030.			

Note. T1 = Baseline assessment; T3 = 1-year follow-up assessment; EXT = Externalizing problems; INT = Internalizing problems; Prosocial = Prosocial behaviors; Income = Household annual income; PMarital = Parental marital status. Youth sex was coded as 1 = female and 2 = male; Race and parental marital status variables were coded as binary (0/1) variables; Household income was coded into $1,000. **p* < .05, ***p* < .01, *** *p* < .001.

Table 3.7. *The Moderation Roles of Amygdalar Response to Negative Emotional Stimuli on the Links Between Family Conflict and Youth Adjustment*

Effects	Left Amygdala Model				Right Amygdala Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Direct Effects								
Fam. Conflict (T1) → EXT (T3)	.024 (.007)	.046	[.011, .037]***	<.001	.017 (.006)	.033	[.005, .030]**	.006
Fam. Conflict (T1) → INT (T3)	.010 (.007)	.020	[-.004, .024]	.145	.010 (.007)	.018	[-.004, .023]	.166
Fam. Conflict (T1) → Prosocial (T3)	-.013 (.002)	-.117	[-.017, -.009]***	<.001	-.013 (.002)	-.116	[-.016, -.009]***	<.001
Amygdala Act. (T1) → EXT (T3)	-.045 (.043)	-.019	[-.130, .040]	.304	-.005 (.037)	-.002	[-.077, .068]	.902
Amygdala Act. (T1) → INT (T3)	.088 (.046)	.038	[-.002, .178]	.056	.036 (.038)	.016	[-.039, .110]	.348
Amygdala Act. (T1) → Prosocial (T3)	-.006 (.013)	-.012	[-.032, .028]	.640	-.013 (.011)	-.027	[-.035, .009]	.255
Interaction Effects								
Fam. Conflict x Amygdala Act. → EXT (T3)	.049 (.023)	.040	[.004, .093]*	.031	.023 (.019)	.019	[-.014, .059]	.219
Fam. Conflict x Amygdala Act. → INT (T3)	.031 (.027)	.038	[-.022, .084]	.257	.015 (.020)	.013	[-.024, .054]	.452
Fam. Conflict x Amygdala Act. → Prosocial (T3)	.001 (.007)	.003	[-.013, .015]	.901	.005 (.005)	.022	[-.005, .016]	.308
Control								
EXT (T1) → EXT (T3)	.703 (.013)	.676	[.677, .728]***	<.001	.692 (.012)	.723	[.668, .716]***	<.001
Race – White → EXT (T3)	.047 (.045)	.023	[-.041, .135]	.294	.043 (.044)	.021	[-.043, .130]	.326
Race – Black → EXT (T3)	.004 (.060)	.001	[-.113, .122]	.945	-.006 (.059)	-.002	[-.120, .109]	.925
Race – Hispanic → EXT (T3)	.017 (.050)	.007	[-.081, .115]	.733	.023 (.049)	.010	[-.073, .120]	.633
Youth Sex → EXT (T3)	.076 (.025)	.037	[.027, .125]**	.002	.070 (.024)	.034	[.023, .118]**	.004
Income (T1) → EXT (T3)	-.051 (.017)	-.055	[-.084, -.018]**	.002	-.034 (.016)	-.035	[-.065, -.003]*	.033
PMarital → EXT (T3)	-.086 (.035)	-.039	[-.155, -.018]*	.014	-.087 (.034)	-.039	[-.153, -.020]*	.010
INT (T1) → INT (T3)	.717 (.014)	.649	[.689, .745]***	<.001	.676 (.012)	.688	[.652, .700]***	<.001
Race – White → INT (T3)	.031 (.045)	.015	[-.057, .120]	.488	.036 (.044)	.018	[-.049, .122]	.404
Race – Black → INT (T3)	-.113 (.059)	-.038	[-.228, .002]	.054	-.109 (.056)	-.037	[-.219, .001]	.052
Race – Hispanic → INT (T3)	.042 (.052)	.017	[-.060, .142]	.430	.044 (.050)	.018	[-.054, .141]	.378
Youth Sex → INT (T3)	.001 (.025)	.001	[-.048, .051]	.957	.000 (.025)	.000	[-.048, .048]	>.999
Income (T1) → INT (T3)	.003 (.016)	.004	[-.029, .035]	.840	.015 (.015)	.016	[-.015, .045]	.339
PMarital → INT (T3)	-.093 (.035)	-.043	[-.162, -.024]**	.009	-.082 (.034)	-.038	[-.148, -.016]*	.015
Prosocial (T1) → Prosocial (T3)	.343 (.020)	.353	[.303, .383]***	<.001	.310 (.017)	.352	[.276, .345]***	<.001
Race – White → Prosocial (T3)	.028 (.011)	.065	[.007, .050]**	.009	.029 (.011)	.066	[.008, .050]**	.008
Race – Black → Prosocial (T3)	.011 (.015)	.018	[-.019, .041]	.466	.011 (.015)	.018	[-.019, .042]	.464
Race – Hispanic → Prosocial (T3)	.024 (.013)	.047	[-.001, .048]	.058	.024 (.013)	.046	[-.001, .048]	.059
Youth Sex → Prosocial (T3)	-.050 (.006)	-.116	[-.063, -.038]***	<.001	-.049 (.006)	-.113	[-.062, -.036]***	<.001
Income (T1) → Prosocial (T3)	.013 (.004)	.063	[.004, .021]**	.003	.013 (.004)	.064	[.004, .021]**	.003
PMarital → Prosocial (T3)	-.007 (.008)	-.014	[-.023, .010]	.433	-.006 (.008)	-.013	[-.023, .010]	.468
Model Fit Indices	$\chi^2(6) = 61.456$ ($p < .001$); CFI = .991; TLI = .939; RMSEA = .028; SRMR = .012.				$\chi^2(6) = 32.643$ ($p < .001$); CFI = .996; TLI = .974; RMSEA = .019; SRMR = .010.			
Model Fit Improvement	$\Delta\chi^2(7) = 446.227$ ($p < .001$)				$\Delta\chi^2(7) = 485.040$ ($p < .001$)			

Note. T1 = Baseline assessment; T3 = 1-year follow-up assessment; Fam. = Family; Act. = Activation (to negative emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems; Prosocial = Prosocial behaviors; Income = Household annual income; PMarital = Parental marital status. Youth sex was coded as 1 = female and 2 = male; Race and parental marital status variables were coded as binary (0/1) variables; Household income was coded into $1,000. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 3.8. *The Moderation Roles of Amygdalar Response to Positive Emotional Stimuli on the Links Between Parental Warmth and Youth Adjustment*

Effects	Left Amygdala Model				Right Amygdala Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Direct Effects								
Warmth (T1) → EXT (T3)	-.013 (.048)	-.031	[-.197, -.010]*	.029	-.238 (.130)	-.024	[-.493, .017]	.067
Warmth (T1) → INT (T3)	-.079 (.050)	-.024	[-.177, .018]	.112	-.019 (.122)	.014	[-.267, .210]	.814
Warmth (T1) → Prosocial (T3)	.093 (.012)	.133	[.069, .117]***	<.001	.238 (.034)	.018	[.171, .306]***	<.001
Amygdala Act. (T1) → EXT (T3)	-.025 (.048)	-.010	[-.118, .069]	.608	-.024 (.036)	.016	[-.093, .046]	.507
Amygdala Act. (T1) → INT (T3)	.054 (.047)	.022	[-.038, .146]	.250	.016 (.039)	.019	[-.061, .093]	.679
Amygdala Act. (T1) → Prosocial (T3)	-.014 (.011)	-.026	[-.034, .007]	.199	-.010 (.009)	.021	[-.028, .009]	.293
Interaction Effects								
Warmth x Amygdala Act. → EXT (T3)	-.114 (.123)	-.015	[-.355, .126]	.352	.264 (.360)	.011	[-.441, .970]	.463
Warmth x Amygdala Act. → INT (T3)	.080 (.126)	.010	[-.168, .328]	.527	-.334 (.481)	-.015	[-1.278, .609]	.487
Warmth x Amygdala Act. → Prosocial (T3)	-.004 (.035)	-.022	[-.072, .065]	.919	.085 (.113)	.018	[-.137, .306]	.455
Control								
EXT (T1) → EXT (T3)	.706 (.013)	.681	[.680, .733]***	<.001	.629 (.010)	.704	[.609, .649]***	<.001
Race – White → EXT (T3)	.052 (.045)	.025	[-.037, .140]	.252	.071 (.045)	.036	[-.017, .159]	.116
Race – Black → EXT (T3)	.012 (.060)	.004	[-.105, .130]	.838	.015 (.057)	.005	[-.097, .127]	.789
Race – Hispanic → EXT (T3)	.018 (.050)	.008	[-.080, .116]	.713	.053 (.050)	.023	[-.045, .151]	.289
Youth Sex → EXT (T3)	.073 (.025)	.036	[.024, .123]**	.004	.061 (.023)	.031	[.015, .106]**	.009
Income (T1) → EXT (T3)	-.055 (.017)	-.058	[-.088, -.022]**	.001	-.022 (.015)	-.024	[-.051, .007]	.142
PMarital → EXT (T3)	-.085 (.035)	-.039	[-.154, -.016]*	.016	-.076 (.032)	-.037	[-.139, -.013]*	.018
INT (T1) → INT (T3)	.717 (.014)	.649	[.689, .745]***	<.001	.569 (.010)	.654	[.550, .588]***	<.001
Race – White → INT (T3)	.034 (.045)	.017	[-.054, .123]	.449	.047 (.042)	.026	[-.035, .128]	.264
Race – Black → INT (T3)	-.104 (.058)	-.035	[-.218, .011]	.076	-.092 (.055)	.035	[-.198, .015]	.093
Race – Hispanic → INT (T3)	.045 (.052)	.019	[-.056, .147]	.382	.042 (.047)	.020	[-.050, .135]	.369
Youth Sex → INT (T3)	.001 (.025)	.000	[-.049, .051]	.978	-.009 (.022)	-.005	[-.053, .035]	.681
Income (T1) → INT (T3)	.000 (.016)	.000	[-.032, .032]	.989	.023 (.014)	.028	[-.005, .050]	.102
PMarital → INT (T3)	-.086 (.035)	-.040	[-.155, -.016]*	.016	-.069 (.030)	-.036	[-.127, -.011]*	.019
Prosocial (T1) → Prosocial (T3)	.319 (.021)	.329	[.278, .360]**	<.001	.254 (.017)	.323	[.220, .288]***	<.001
Race – White → Prosocial (T3)	.024 (.011)	.055	[.002, .046]*	.030	.019 (.009)	.050	[.001, .038]*	.040
Race – Black → Prosocial (T3)	.004 (.016)	.007	[-.026, .035]	.786	.001 (.014)	.001	[-.027, .029]	.960
Race – Hispanic → Prosocial (T3)	.021 (.013)	.040	[-.004, .046]	.104	.016 (.011)	.035	[-.005, .038]	.144
Youth Sex → Prosocial (T3)	-.051 (.006)	-.116	[-.063, -.038]***	<.001	-.043 (.006)	-.110	[-.054, -.032]***	<.001
Income (T1) → Prosocial (T3)	.016 (.004)	.079	[.007, .024]***	<.001	.014 (.004)	.080	[.007, .022]***	<.001
PMarital → Prosocial (T3)	-.010 (.008)	-.022	[-.027, .007]	.239	-.008 (.008)	-.020	[-.023, .007]	.277
Model Fit Indices	$\chi^2(6) = 63.483$ ($p < .001$); CFI = .990; TLI = .937; RMSEA = .028; SRMR = .013.				$\chi^2(6) = 36.612$ ($p < .001$); CFI = .995; TLI = .965; RMSEA = .021; SRMR = .010.			
Model Fit Improvement	$\Delta\chi^2(7) = 441.252$ ($p < .001$)				$\Delta\chi^2(7) = 458.123$ ($p < .001$)			

Note. T1 = Baseline assessment; T3 = 1-year follow-up assessment; Warmth = Parental warmth; Act. = Activation (to positive emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems; Prosocial = Prosocial behaviors; Income = Household annual income; PMarital = Parental marital status. Youth sex was coded as 1 = female and 2 = male; Race and parental marital status variables were coded as binary (0/1) variables; Household income was coded into $1,000. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 3.9. *The Moderation Roles of Hippocampal Response to Negative Emotional Stimuli on the Links Between Family Conflict and Youth Adjustment*

Effects	Left Hippocampus Model				Right Hippocampus Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Direct Effects								
Fam. Conflict (T1) → EXT (T3)	.017 (.006)	.032	[.005, .030]**	.007	.017 (.006)	.032	[.004, .029]**	.008
Fam. Conflict (T1) → INT (T3)	.009 (.007)	.017	[-.005, .022]	.209	.009 (.007)	.018	[-.004, .023]	.186
Fam. Conflict (T1) → Prosocial (T3)	-.013 (.002)	-.115	[-.016, -.009]***	<.001	-.013 (.002)	-.116	[-.016, -.009]***	<.001
Hippocampus Act. (T1) → EXT (T3)	-.100 (.057)	-.029	[-.212, .012]	.081	-.147 (.062)	-.044	[-.268, -.026]*	.018
Hippocampus Act. (T1) → INT (T3)	.003 (.057)	.001	[-.107, .114]	.953	-.043 (.064)	-.013	[-.168, .083]	.503
Hippocampus Act. (T1) → Prosocial (T3)	-.013 (.017)	-.018	[-.047, .021]	.455	.004 (.009)	-.031	[-.052, .010]	.183
Interaction Effects	.							
Fam. Conflict x Hippocampus Act. → EXT (T3)	.032 (.029)	.019	[-.025, .090]	.271	.010 (.026)	.006	[-.042, .061]	.708
Fam. Conflict x Hippocampus Act. → INT (T3)	.066 (.031)	.039	[.005, .128]*	.034	-.003 (.030)	-.002	[-.062, .055]	.914
Fam. Conflict x Hippocampus Act. → Prosocial (T3)	.003 (.009)	.007	[-.015, .021]	.777	.004 (.009)	.010	[-.014, .021]	.683
Control								
EXT (T1) → EXT (T3)	.693 (.012)	.723	[.669, .717]***	<.001	.693 (.012)	.723	[.669, .717]***	<.001
Race – White → EXT (T3)	.043 (.044)	.020	[-.044, .129]	.332	.043 (.044)	.021	[-.043, .129]	.326
Race – Black → EXT (T3)	-.007 (.058)	-.002	[-.121, .108]	.911	-.003 (.058)	-.001	[-.117, .111]	.958
Race – Hispanic → EXT (T3)	.023 (.049)	.010	[-.073, .119]	.635	.025 (.049)	.010	[-.071, .121]	.606
Youth Sex → EXT (T3)	.071 (.024)	.034	[.024, .118]**	.003	.071 (.024)	.034	[.024, .118]**	.003
Income (T1) → EXT (T3)	-.034 (.016)	-.035	[-.065, -.003]*	.034	-.035 (.016)	-.036	[-.066, -.004]*	.028
PMarital → EXT (T3)	-.086 (.034)	-.039	[-.152, -.021]*	.010	-.085 (.034)	-.038	[-.150, -.019]*	.012
INT (T1) → INT (T3)	.676 (.012)	.687	[.562, .700]**	<.001	.676 (.012)	.688	[.652, .701]***	<.001
Race – White → INT (T3)	.038 (.044)	.019	[-.048, .123]	.386	.036 (.044)	.010	[-.049, .122]	.406
Race – Black → INT (T3)	-.109 (.056)	-.036	[-.218, .001]	.053	-.017 (.056)	-.036	[-.217, .003]	.056
Race – Hispanic → INT (T3)	.044 (.050)	.018	[-.054, .142]	.378	.044 (.050)	.019	[-.053, .142]	.372
Youth Sex → INT (T3)	.002 (.024)	.001	[-.046, .050]	.934	.001 (.024)	.001	[-.047, .049]	.958
Income (T1) → INT (T3)	.014 (.015)	.015	[-.016, .044]	.360	.014 (.015)	.015	[-.016, .044]	.367
PMarital → INT (T3)	-.081 (.034)	-.037	[-.147, -.015]*	.016	-.080 (.034)	-.037	[-.146, -.014]*	.017
Prosocial (T1) → Prosocial (T3)	.310 (.018)	.352	[.276, .345]***	<.001	.310 (.017)	.352	[.276, .344]***	<.001
Race – White → Prosocial (T3)	.028 (.011)	.065	[.008, .049]**	.009	.028 (.011)	.065	[.007, .050]**	.008
Race – Black → Prosocial (T3)	.011 (.015)	.017	[-.019, .041]	.481	.011 (.015)	.018	[-.019, .042]	.461
Race – Hispanic → Prosocial (T3)	.023 (.013)	.046	[-.001, .048]	.063	.024 (.013)	.046	[-.001, .048]	.059
Youth Sex → Prosocial (T3)	-.050 (.006)	-.114	[-.062, -.037]***	<.001	-.050 (.006)	-.114	[-.062, -.037]***	<.001
Income (T1) → Prosocial (T3)	.013 (.004)	.066	[.005, .022]**	.002	.013 (.004)	.065	[.005, .021]**	.003
PMarital → Prosocial (T3)	-.006 (.008)	-.014	[-.023, .010]	.440	-.006 (.008)	-.014	[-.023, .010]	.452
Model Fit Indices	$\chi^2(6) = 31.750$ ($p < .001$); CFI = .996; TLI = .975; RMSEA = .019; SRMR = .010.				$\chi^2(6) = 31.771$ ($p < .001$); CFI = .996; TLI = .974; RMSEA = .019; SRMR = .010.			
Model Fit Improvement	$\Delta\chi^2(7) = 475.933$ ($p < .001$)				$\Delta\chi^2(7) = 475.912$ ($p < .001$)			

Note. T1 = Baseline assessment; T3 = 1-year follow-up assessment; Fam. = Family; Act. = Activation (to negative emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems; Prosocial = Prosocial behaviors; Income = Household annual income; PMarital = Parental marital status. Youth sex was coded as 1 = female and 2 = male; Race and parental marital status variables were coded as binary (0/1) variables; Household income was coded into $1,000. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 3.10. *The Moderation Roles of Hippocampal Response to Positive Emotional Stimuli on the Links Between Parental Warmth and Youth Adjustment*

Effects	Left Hippocampus Model				Right Hippocampus Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Direct Effects								
Warmth (T1) → EXT (T3)	-.177 (.102)	-.024	[-.377, .023]	.082	-.233 (.132)	-.024	[-.491, .025]	.077
Warmth (T1) → INT (T3)	-.040 (.094)	-.006	[-.226, .145]	.670	-.033 (.122)	-.004	[-.273, .206]	.768
Warmth (T1) → Prosocial (T3)	.181 (.027)	.121	[.128, .234]***	<.001	.239 (.034)	.123	[.172, .307]***	<.001
Hippocampus Act. (T1) → EXT (T3)	-.027 (.053)	-.009	[-.131, ,076]	.606	-.067 (.053)	-.021	[-.172, .037]	.206
Hippocampus Act. (T1) → INT (T3)	.038 (.049)	.013	[-.058, .135]	.436	.022 (.049)	.008	[-.074, .119]	.647
Hippocampus Act. (T1) → Prosocial (T3)	-.008 (.013)	-.013	[-.033, .017]	.515	-.018 (.013)	-.028	[-.043, .249]	.180
Interaction Effects								
Warmth x Hippocampus Act. → EXT (T3)	-.524 (.441)	-.020	[-1.389, .341]	.235	-.091 (.625)	-.003	[-1.316, 1.518]	.884
Warmth x Hippocampus Act. → INT (T3)	-.914 (.417)	-.037	[-1.732, -.096]*	.028	-.551 (.579)	-.017	[-1.686, .941]	.342
Warmth x Hippocampus Act. → Prosocial (T3)	-.040 (.114)	-.007	[-.263, .183]	.727	-.144 (.190)	-.021	[-.516, .346]	.450
Control								
EXT (T1) → EXT (T3)	.629 (.010)	.704	[.609, .649]***	<.001	.629 (.010)	.704	[.609, .649]***	<.001
Race – White → EXT (T3)	.073 (.045)	.037	[-.015, .160]	.103	.070 (.045)	.036	[-.017, .158]	.116
Race – Black → EXT (T3)	.014 (.057)	.005	[-.097, .126]	.804	.014 (.057)	.005	[-.098, .125]	.810
Race – Hispanic → EXT (T3)	.055 (.050)	.024	[-.043, .152]	.271	.054 (.050)	.024	[-.044, .151]	.281
Youth Sex → EXT (T3)	.060 (.023)	.031	[.014, .106]*	.010	.060 (.023)	.031	[.014, .106]*	.010
Income (T1) → EXT (T3)	-.020 (.015)	-.023	[-.050, .009]	.169	-.021 (.015)	-.024	[-.050, .008]	.156
PMarital → EXT (T3)	-.080 (.032)	-.039	[-.143, -.017]*	.013	-.079 (.032)	-.038	[-.141, -.016]*	.014
INT (T1) → INT (T3)	.569 (.010)	.654	[.550, .588]***	<.001	.569 (.010)	.654	[.550, .588]***	<.001
Race – White → INT (T3)	.050 (.042)	.028	[-.032, .132]	.230	.048 (.042)	.027	[-.034, .130]	.250
Race – Black → INT (T3)	-.091 (.054)	-.034	[-.197, .016]	.096	-.090 (.054)	-.034	[-.197, .016]	.097
Race – Hispanic → INT (T3)	.045 (.047)	.021	[-.048, .137]	.345	.044 (.047)	.021	[-.049, .136]	.354
Youth Sex → INT (T3)	-.011 (.022)	-.006	[-.055, .033]	.618	-.009 (.022)	-.005	[-.053, .035]	.681
Income (T1) → INT (T3)	.023 (.014)	.028	[-.004, .051]	.093	.023 (.014)	.028	[-.005, .050]	.102
PMarital → INT (T3)	-.071 (.030)	-.037	[-.129, -.012]*	.017	-.069 (.030)	-.036	[-.127, -.011]*	.020
Prosocial (T1) → Prosocial (T3)	.255 (.017)	.324	[.221, .288]***	<.001	.254 (.017)	.322	[.220, .287]***	<.001
Race – White → Prosocial (T3)	.020 (.009)	.050	[.001, .038]*	.039	.019 (.009)	.050	[.001, .038]*	.040
Race – Black → Prosocial (T3)	.001 (.014)	.001	[-.027, .029]	.968	.000 (.014)	.001	[-.028, .028]	.977
Race – Hispanic → Prosocial (T3)	.016 (.011)	.036	[-.005, .038]	.142	.017 (.011)	.036	[-.005, .038]	.135
Youth Sex → Prosocial (T3)	-.043 (.006)	-.111	[-.054, -.032]***	<.001	-.043 (.006)	-.111	[-.054, -.032]***	<.001
Income (T1) → Prosocial (T3)	.015 (.004)	.081	[.007, .022]**	<.001	.015 (.004)	.082	[.007, .022]***	<.001
PMarital → Prosocial (T3)	-.009 (.008)	-.022	[-.024, .006]	.244	-.009 (.008)	-.023	[-.024, .006]	.220
Model Fit Indices	$\chi^2(6)$ = 35.928 (*p* < .001); CFI = .995; TLI = .966; RMSEA = .20; SRMR = .010.				$\chi^2(6)$ = 36.538 (*p* < .001); CFI = .996; TLI = .965; RMSEA = .021; SRMR = .010.			
Model Fit Improvement	$\Delta\chi^2(7)$ =458.807 (*p* < .001)				$\Delta\chi^2(7)$ = 458.197 (*p* < .001)			

Note. T1 = Baseline assessment; T3 = 1-year follow-up assessment; Warmth = Parental warmth; Act. = Activation (to positive emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems; Prosocial = Prosocial behaviors; Income = Household annual income; PMarital = Parental marital status. Youth sex was coded as 1 = female and 2 = male; Race and parental marital status variables were coded as binary (0/1) variables; Household income was coded into $1,000. **p* < .05, ***p* < .01, *** *p* < .001.

Table 3.11. *Paired-Sample t-tests for the Mean Hemisphere (Right vs. Left) Differences of Amygdalar and Hippocampal Responses to Negative and Positive Emotional Stimuli*

Emotional Stimuli	ROI	Left		Right		$M_{\text{difference}}$	95% *CI* of $M_{\text{difference}}$	*t*	*df*	Cohen's *d*	*p*-value
		M	*SD*	*M*	*SD*						
Negative	Hippocampus	.0177	.3011	.0197	.3129	-.0019	[-.0083, .0044]	-.594	6261	-.0208	.520
	Amygdala	.0461	.4216	.0635	.4526	-.0174	[-.0282, -.0066]	-3.155**	6261	-.0248	.002
Positive	Hippocampus	-.0161	.3017	-.0182	.3063	.0022	[-.0040, .0083]	.690	6260	.0202	.490
	Amygdala	-.0090	.4075	.0023	.4278	-.0114	[-.0214, -.0013]	-2.222*	6259	-.0240	.026

Note. ROI = Region of interests; *M* = Mean; *SD* = Standard Deviation; $M_{\text{difference}}$ = Mean difference; *CI* = Confidence interval; *df* = Degree of freedom. The unit of hemisphere differences presented here is standardized activation coefficient β. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 3.12. *Independent Sample t-Tests for the Sex Differences of Study Variables*

Variables	Female		Male		$M_{\text{difference}}$	95% *CI* of $M_{\text{difference}}$	*t*	*df*	Cohen's *d*	*p*-value
	M	*SD*	*M*	*SD*						
Household Income	102.7289	73.0684	103.4493	72.8162	-.7204	[-3.47, 2.02]	-.5141	10852	-.0096	.607
Family Conflict	1.9243	1.9313	2.1572	1.9680	-.2329	[-.30, -.16]	-6.4967***	11792	-.1194	< .001
Parental Warmth	2.7989	.2936	2.7639	0.3131	.0350	[.02, .05]	6.2703***	11830	.1150	< .001
Externalizing T1	3.7497	5.1684	5.1000	6.3688	-1.3504	[-1.56, -1.14]	-12.7249***	11690	-.2328	< .001
Internalizing T1	5.0106	5.5061	5.0779	5.5483	-.0673	[-.27, .13]	-.6628	11862	-.0127	.508
Prosocial T1	5.4273	1.0856	5.0889	1.2990	.3384	[.30, .38]	15.4287***	11745	.2827	< .001
Externalizing T3	3.3476	4.7727	4.5569	5.7255	-1.2093	[-.15, -.92]	-8.0847***	4891	-.2296	< .001
Internalizing T3	5.0114	5.2643	5.0151	5.4736	-.0037	[-.30, .30]	-.0243	4935	-.0019	.981
Prosocial T3	5.3690	1.1145	5.0142	1.3034	.3548	[.29, .42]	10.3025***	4910	.2932	< .001
Left Hippocampus-Negative T1	.0151	.2839	.0186	.3223	-.0035	[-.02, .01]	-.4618	6237	-.0115	.642
Left Amygdala - Negative T1	.0396	.4021	.0536	.4511	-.0140	[-.04, .01]	-1.3008	6246	-.0325	.194
Right Hippocampus-Negative T1	.0196	.2934	.0193	.3398	.0003	[-.02, .02]	.0356	6215	.0009	.972
Right Amygdala - Negative T1	.0594	.4427	.0666	.4763	-.0072	[-.03, .02]	-.6232	6275	-.0157	.533
Left Hippocampus-Positive T1	-.0259	.2909	-.0064	.3191	-.0195	[-.03, -.004]	-2.5353*	6262	-.0639	.011
Left Amygdala - Positive T1	-.0201	.3964	.0020	.4313	-.0222	[-.04, -.002]	-2.1237*	6265	-.0531	.034
Right Hippocampus-Positive T1	-.0197	.2924	-.0162	.3254	-.0035	[-.02, .01]	-.4481	6253	-.0113	.654
Right Amygdala - Positive T1	-.0033	.4143	.0086	.4529	-.0119	[-.03, .01]	-1.0890	6264	-.0274	.276

Note. ROI = Region of interests; *M* = Mean; *SD* = Standard Deviation; $M_{\text{difference}}$ = Mean difference; *CI* = Confidence interval; *df* = Degree of freedom; T1 = Baseline assessments; T3 = 1-year follow-up. **p* < .05, ***p* < .01, *** *p* < .001.

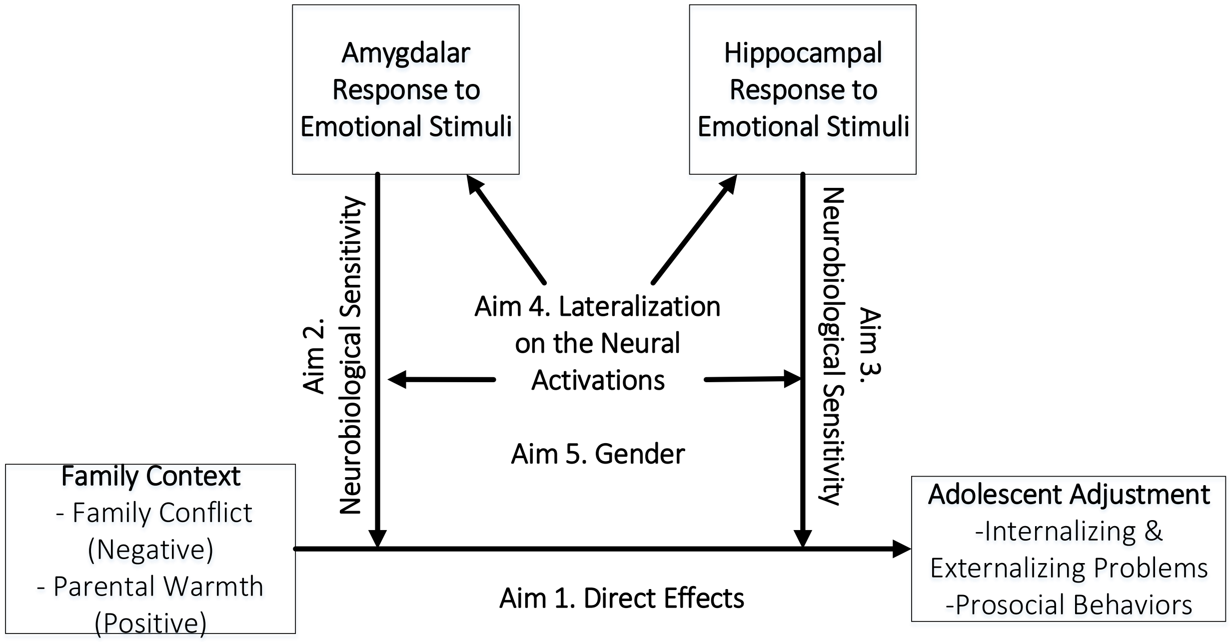

Figure 3.1. Summary of Aims and Hypotheses of the Current Study.

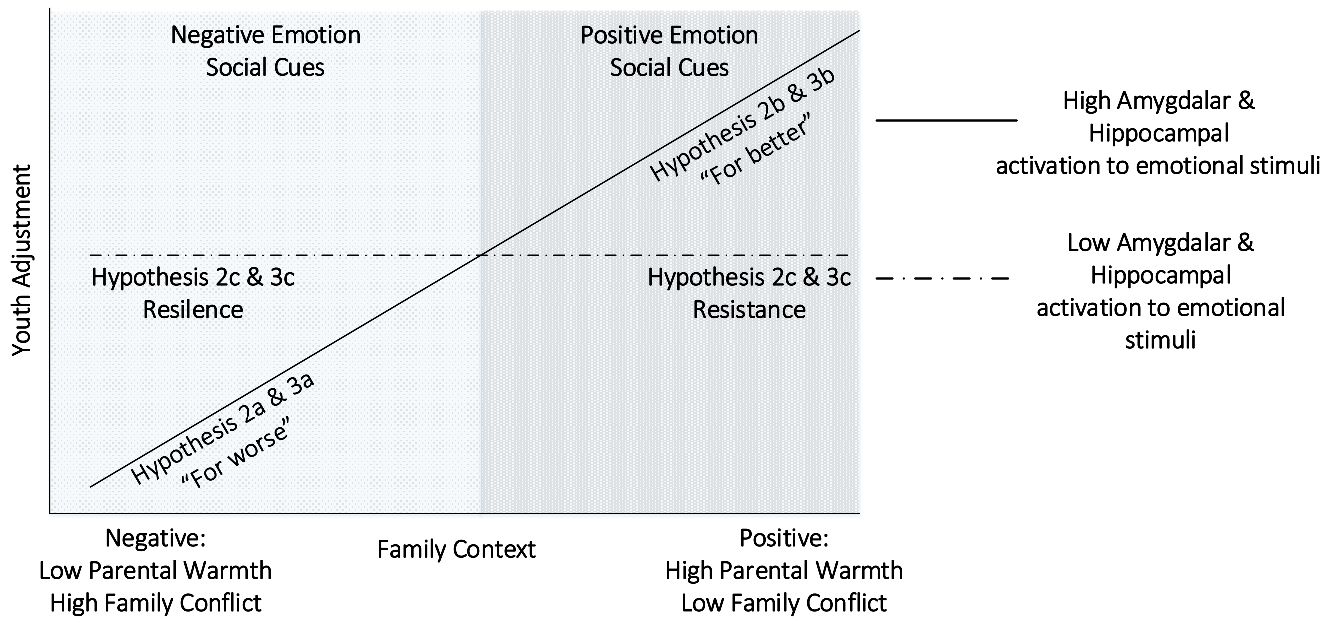

Figure 3.2. Elucidation of Neurobiological Sensitivity Hypotheses on the Moderating Effects of Amygdalar and Hippocampal Activations to Emotional Stimuli on the Associations Between Positive and Negative Family rearing environments and Youth Adjustment.

Note. The solid line indicates youth with high amygdalar and hippocampal activation during emotional processing who will exhibit a "for better and for worse" pattern under the influences of family environments. The dash-dot line represents youth with low amygdalar and hippocampal activation, who were hypothesized to be resilient to the harmful impact of adverse family rearing environments. These youth were also hypothesized to be resistant to the positive influences of supportive family relationships.

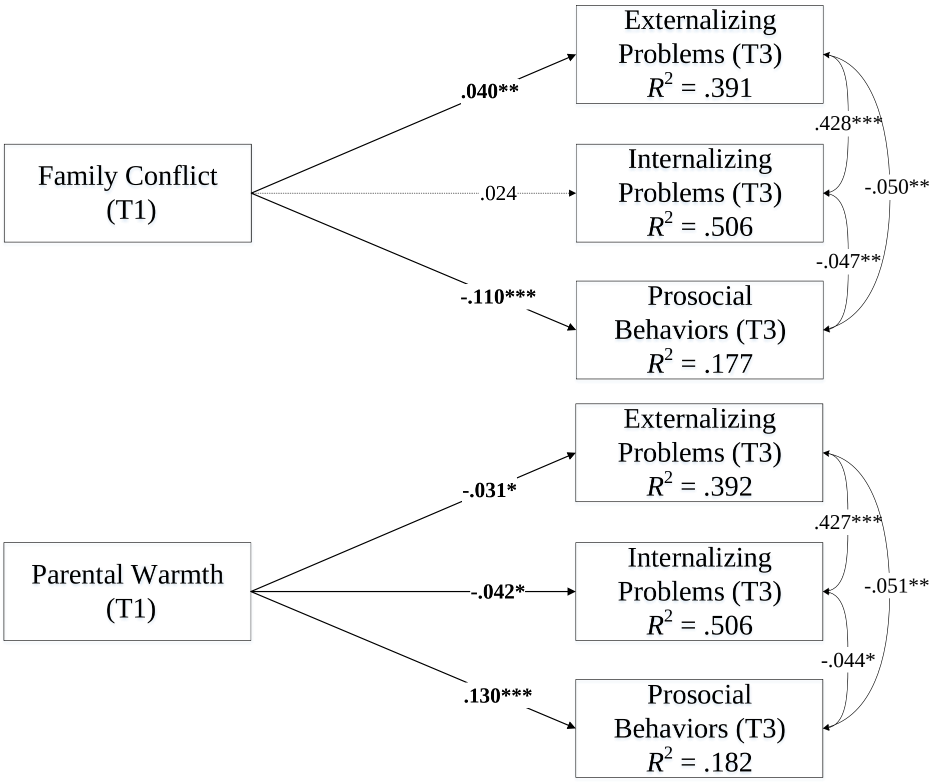

Figure 3.3. The Direct Effects of Family Conflict and Parental Warmth on Youth Externalizing, Internalizing Problems and Prosocial Behaviors.

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations, while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. This model controlled for youth biological sex, race/ethnicity, household income, and parental marital status. Model fit was acceptable: For the family conflict model: $\chi^2(15) = 507.683$ ($p < .001$); CFI = .909; TLI = .800; RMSEA = .053; SRMR = .030; for the parental warmth model: $\chi^2(15) = 494.735$ ($p < .001$); CFI = .907; TLI = .795; RMSEA = .052; SRMR = .030. Covariates are omitted for figure clarity. *$p < .05$, **$p < .01$, *** $p < .001$.

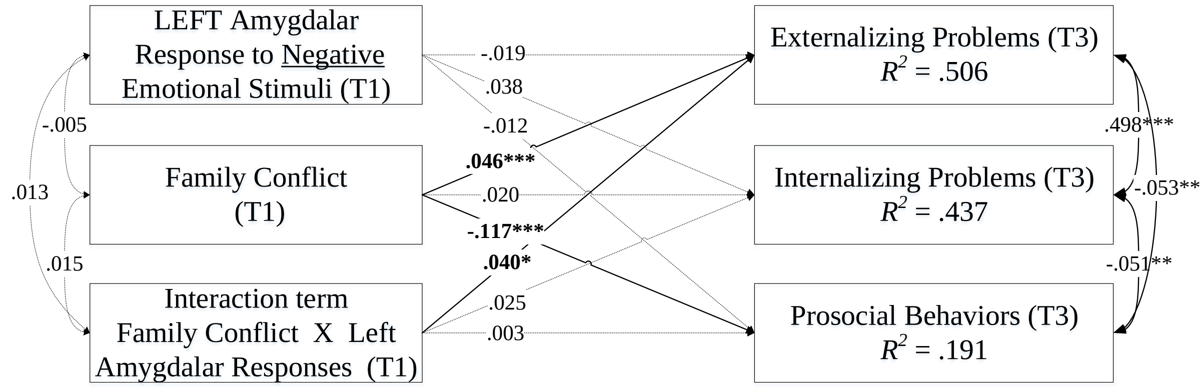

Figure 3.4. The Structural Equation Model of the Associations among Family Conflict, Left Amygdalar Responses to Negative Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 61.456$ ($p < .001$); CFI = .991; TLI = .939; RMSEA = .028; SRMR = .012. *$p < .05$, **$p < .01$, *** $p < .001$.

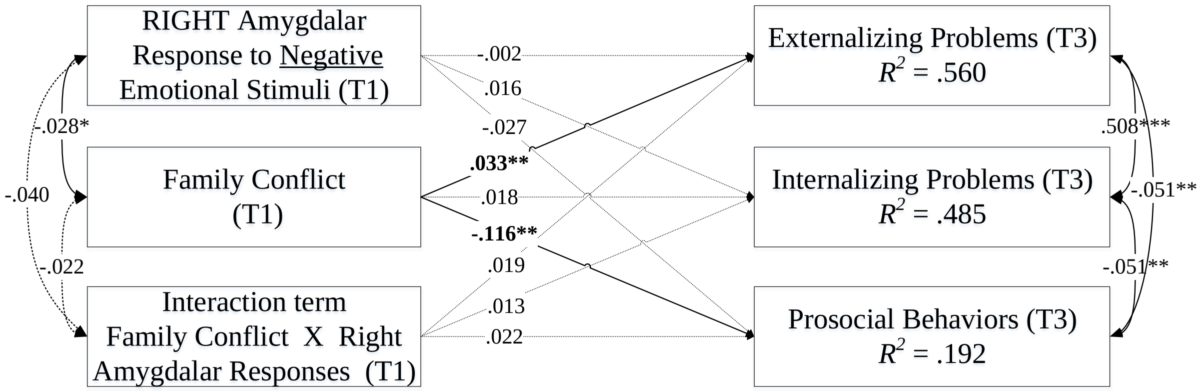

Figure 3.5 The Structural Equation Model of the Associations among Family Conflict, Right Amygdalar Responses to Negative Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 32.643$ ($p < .001$); CFI = .996; TLI = .974; RMSEA = .019; SRMR = .010. *$p < .05$, **$p < .01$, *** $p < .001$.

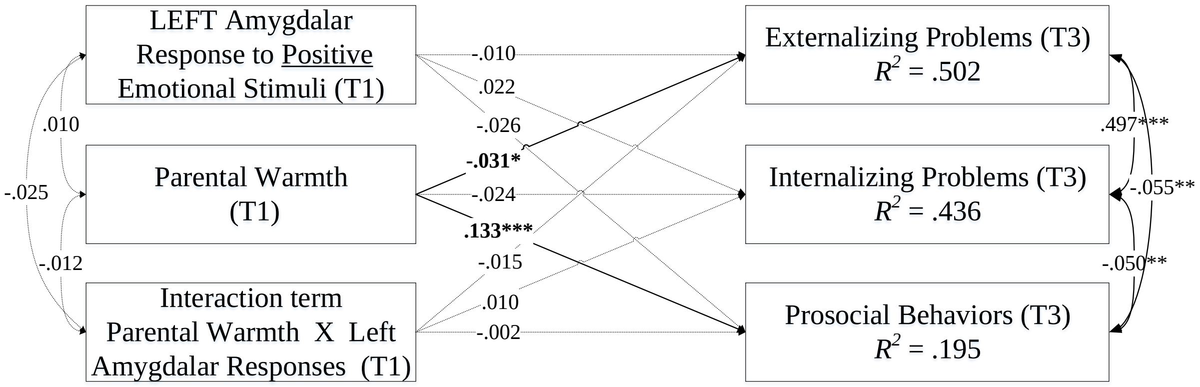

Figure 3.6. The Structural Equation Model of the Associations among Parental Warmth, Left Amygdalar Responses to Positive Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 63.483$ ($p < .001$); CFI = .990; TLI = .937; RMSEA = .028; SRMR = .013. *$p < .05$, **$p < .01$, *** $p < .001$.

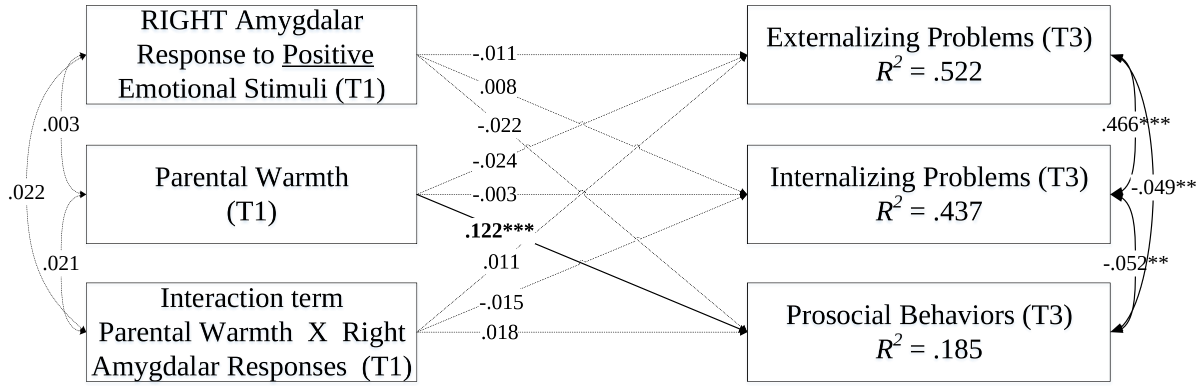

Figure 3.7. The Structural Equation Model of the Associations among Parental Warmth, Right Amygdalar Responses to Positive Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 36.612$ ($p < .001$); CFI = .995; TLI = .965; RMSEA = .021; SRMR = .010. **p* < .05, ***p* < .01, *** *p* < .001.

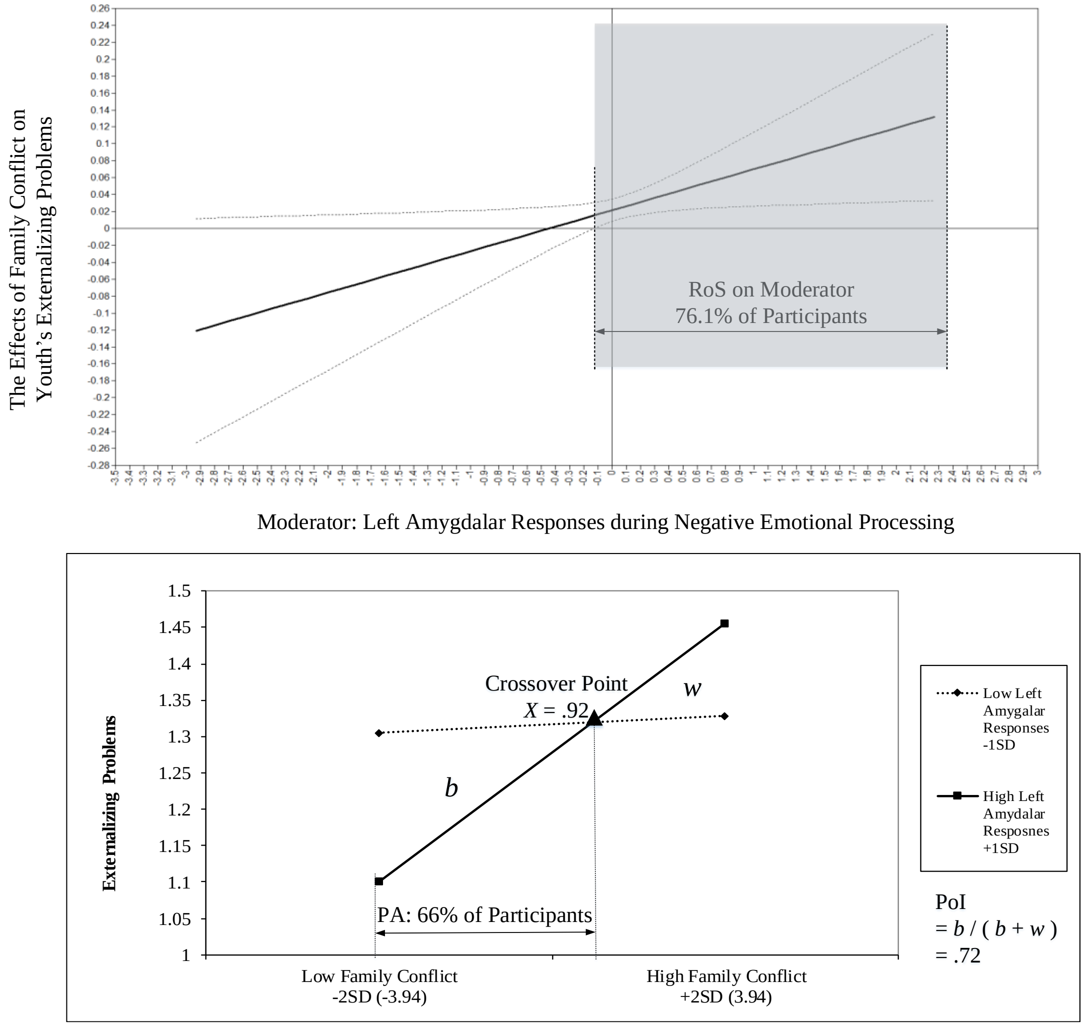

Figure 3.8. The Interpretation of the Significant Moderation Effect of Left Amygdalar Activations during Negative Emotional Stimuli on the Associations Between Family Conflict and Youth Externalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = "for better"; *w* = "for worse". The dotted line indicates youth with low left amygdalar responses during negative emotional stimuli, while the solid line indicates youth with high left amygdalar responses during negative emotional stimuli.

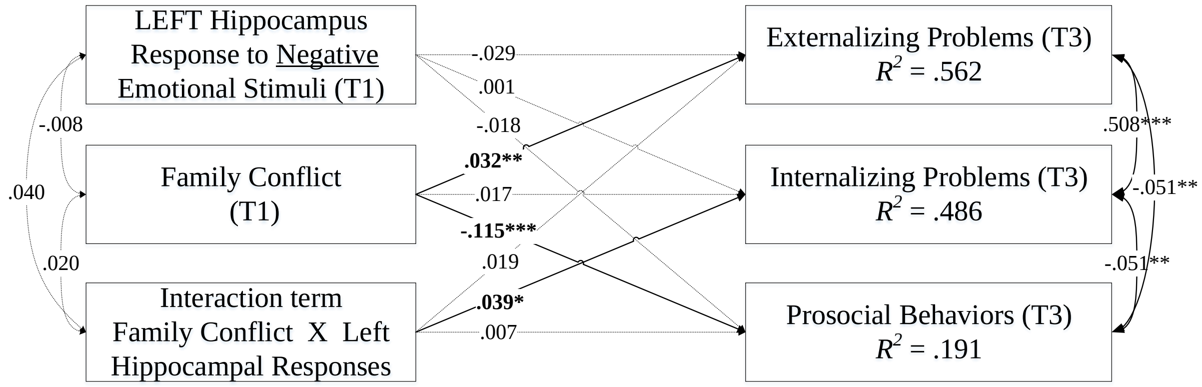

Figure 3.9 The Structural Equation Models of Associations among Family Conflict, Left Hippocampal Responses to Negative Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 31.750$ ($p < .001$); CFI = .996; TLI = .975; RMSEA = .019; SRMR = .010. *$p < .05$, **$p < .01$, *** $p < .001$.

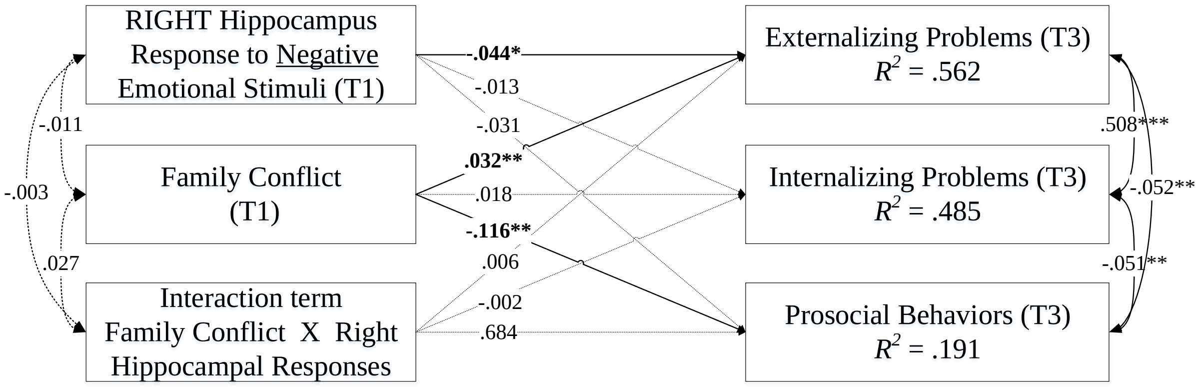

Figure 3.10 The Structural Equation Models of Associations among Family Conflict, Right Hippocampal Responses to Negative Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 31.771$ ($p < .001$); CFI = .996; TLI = .974; RMSEA = .019; SRMR = .010. *$p < .05$, **$p < .01$, *** $p < .001$.

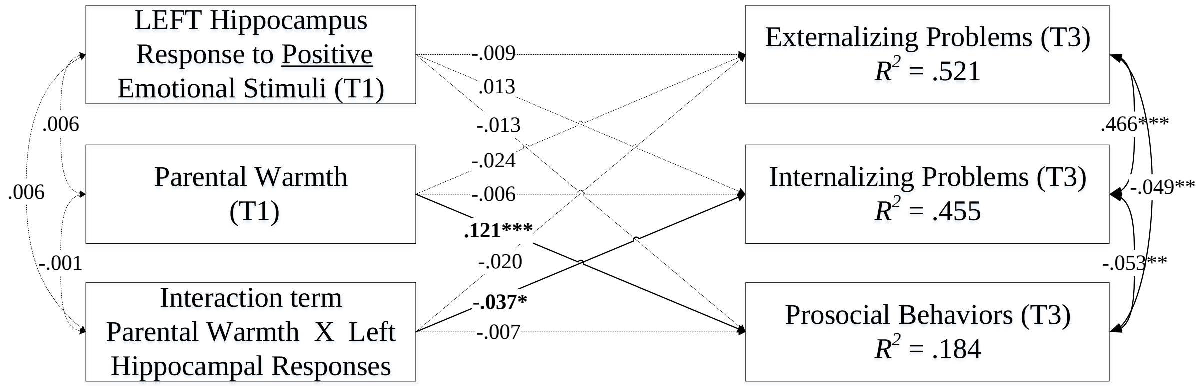

Figure 3.11 The Structural Equation Model of Associations among Parental Warmth, Left Hippocampal Responses to Positive Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 35.928$ ($p < .001$); CFI = .995; TLI = .966; RMSEA = .20; SRMR = .010. *$p < .05$, **$p < .01$, *** $p < .001$.

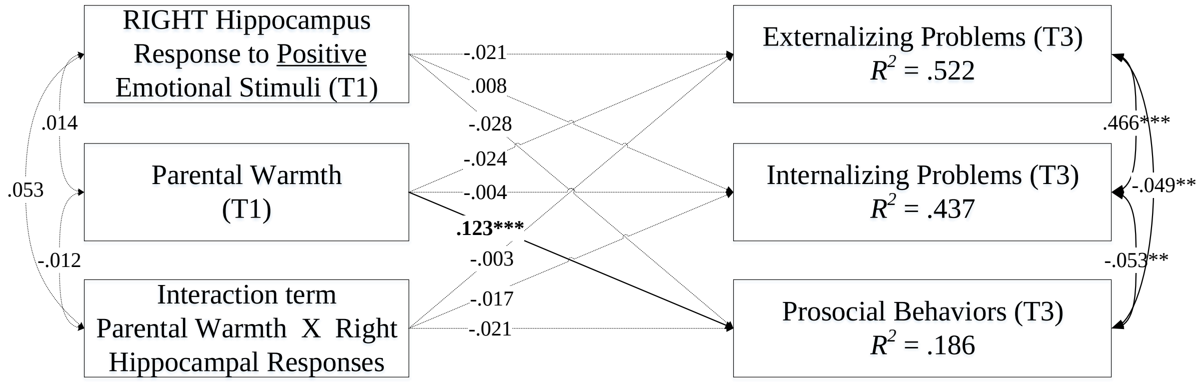

Figure 3.12 The Structural Equation Model of Associations among Parental Warmth, Right Hippocampal Responses to Positive Emotional Stimuli, and Youth Adjustment Outcomes

Note. T1 = Baseline assessments; T3 = 1-year follow-up; R^2 = Proportion of variance explained in this model. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. Models controlled for youth biological sex, race/ethnicity, household income, and the parental marital status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(6) = 36.538$ ($p < .001$); CFI = .996; TLI = .965; RMSEA = .021; SRMR = .010. **p* < .05, ***p* < .01, *** *p* < .001.

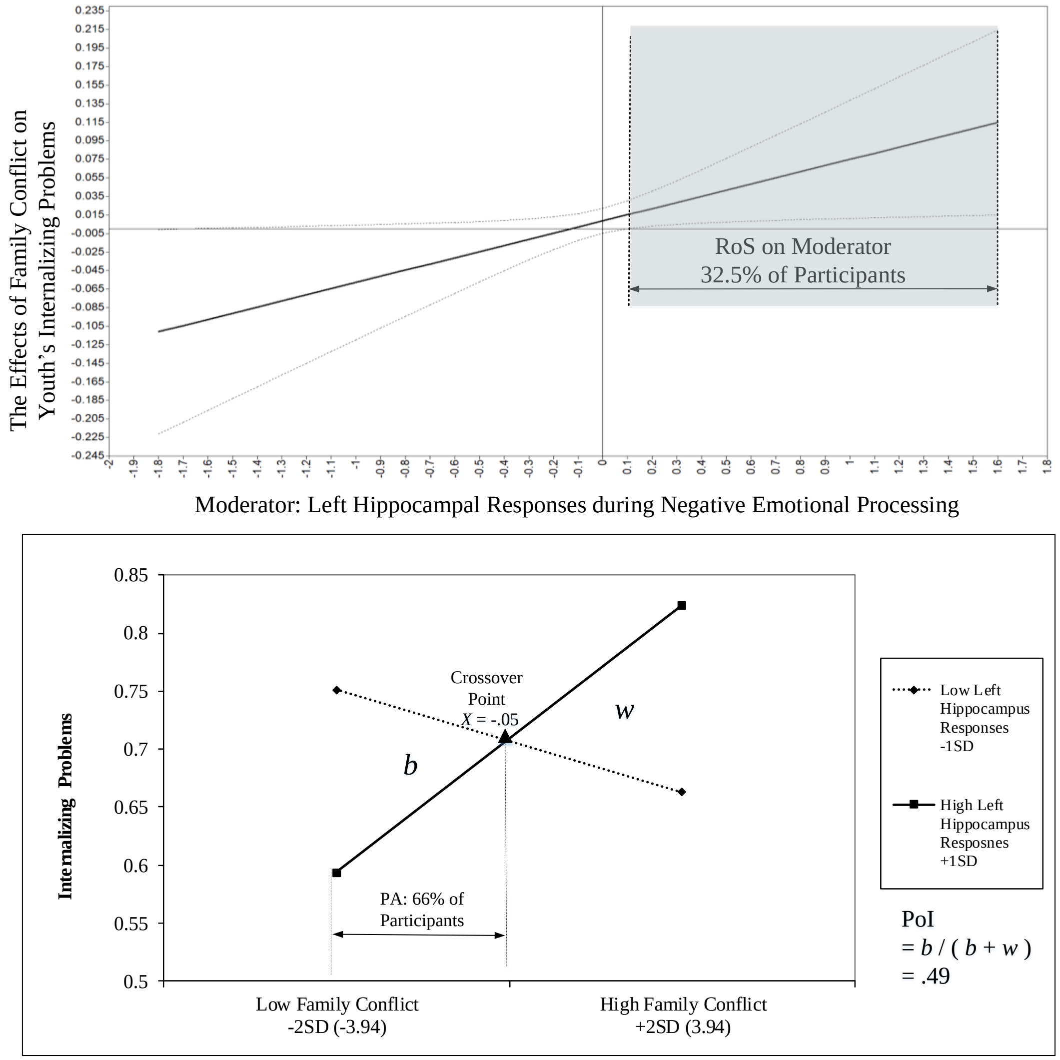

Figure 3.13. The Interpretation of the Significant Moderation Effect of Left Hippocampal Activations during Negative Emotional Stimuli on the Associations Between Family Conflict and Youth Internalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = “for better”; *w* = “for worse”. The dotted line indicates youth with low left hippocampal responses during negative emotional stimuli, while the solid line indicates youth with high left hippocampal responses during negative emotional stimuli.

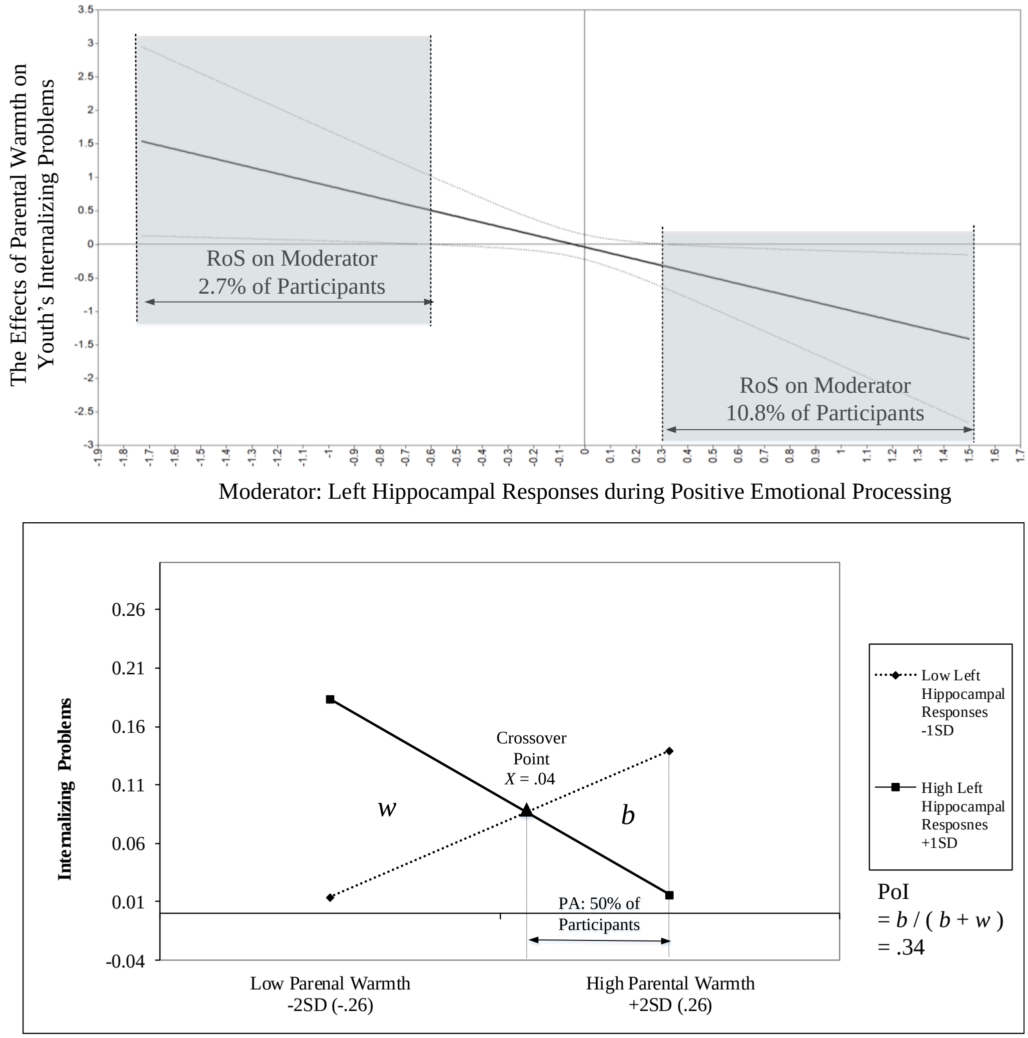

Figure 3.14. The Interpretation of the Significant Moderation Effect of Left Hippocampal Activations during Positive Emotional Stimuli on the Associations Between Parental Warmth and Youth Internalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = "for better"; *w* = "for worse". The dotted line indicates youth with low left hippocampal responses during positive emotional stimuli, while the solid line indicates youth with high left hippocampal responses during positive emotional stimuli.

CHAPTER 4

FAMILY FUNCTIONING AND ADOLESCENT BEHAVIORAL PROBLEMS: THE MODERATING ROLES OF THE AMYGDALAR AND HIPPOCAMPAL RESPONSES DURING EMOTIONAL PROCESSING[2]

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[2] Liu, Sihong. To be submitted to *Developmental Cognitive Neuroscience*

Abstract

The quality of interpersonal relationships in family environments (i.e., family functioning) is a robust predictor of youth's development of problem behaviors. However, youth often do not respond equally even to similar family rearing environments. Recent developmental and neurobiological research suggests that the variability in youths' responses to family influences stems in part from individual differences in neurobiological interactions with rearing environments. The current study aimed to examine the neural architecture underlying youths' differential responses to the influences of family functioning, with the amygdala and hippocampus as key neurobiological foci. Specifically, this study investigated how amygdalar and hippocampal activations during negative and positive emotional processing moderated the associations between family functioning and youths' internalizing and externalizing problems. Data were obtained from a sample of rural adolescents (aged 12-14, *N* = 123) and their primary caretakers recruited from Georgia, with multi-method and multi-reporter assessments. Family functioning was assessed using a comprehensive measurement tool that captured the full spectrum (from negative to positive) and dimensionality of rearing environments. Youths' amygdalar and hippocampal responses to emotional stimuli were obtained during an fMRI Emotional N-back paradigm. Results suggested that balanced family functioning was significantly associated with reduced problem behaviors. Further, left amygdalar responses to positive and negative emotional stimuli, as well as left hippocampal responses to positive emotional processing, significantly exacerbated the effects of family functioning on youth adjustment. The findings of this study indicated that heightened amygdalar and hippocampal activations during emotional processing were associated with youths' elevated sensitivity to the influences of family functioning.

Introduction

Family functioning, defined as the patterns of interpersonal relationships in the family rearing environments (Olson, 2000), is a robust predictor of adolescents' developmental adjustment (Oshri et al., 2015b; Rabinowitz, Osigwe, Drabick, & Reynolds, 2016). Youth who are reared in dysfunctional families are more likely to develop psychopathology and problem behaviors (Joh, Kim, Park, & Kim, 2013; Oshri et al., 2015b). Nevertheless, youth do not respond equally to family environments with similar quality of interpersonal relationships. As suggested by the multifinality concept of the developmental psychopathology perspective (Cicchetti & Rogosch, 1996), similar caregiving quality can result in different developmental outcomes. For example, when exposed to adverse family environments, some adolescents are at elevated risk for developing psychopathology and risk behaviors, whereas other youth exhibit resilience (i.e., a developmental process that promotes youths' developmental competence under adverse environments; Luthar, 2006; A. S. Masten & Obradović, 2006). Similarly, under the influence of supportive family environments, some youth develop more adaptive responses than others (G. H. Brody, Yu, Chen, Kogan, et al., 2013).

This multifinality in youths' differential responses to family rearing environments stems partially from the interactions between neurobiological factors and caregiving experiences (Boyce & Ellis, 2005; Cicchetti & Rogosch, 1996). Therefore, the differential responsivity to the influences of rearing environments is referred to as *neurobiological sensitivity* (Ellis et al., 2011; Schriber & Guyer, 2016). The individual heterogeneity in neurobiological sensitivity renders some youth to be more responsive than others to similar rearing environments (Ellis et al., 2011). Thus, youth with elevated neurobiological sensitivity are either more vulnerable to the harmful impacts of adverse rearing experiences or benefiting more from positive caregiving experiences (Boyce & Ellis, 2005). Further, youths' neurobiological sensitivity processes are regulated by the brain (Ellis et al., 2011). In particular, the social-affective brain circuitry plays a critical role in neurobiological sensitivity because it modulates youths’ emotional and stress reactivity (Casey et al., 2008; Nelson et al., 2005; Steinberg, 2008). However, empirical research that tests the associations between the social-affective brain circuitry and youths’ neurobiological sensitivity to family influences is scarce (Schriber & Guyer, 2016). The current study aimed to fill this gap. The key foci of the social-affective brain circuitry examined in this study involved two major subcortical regions, including the amygdala and hippocampus. In particular, this study investigated how amygdalar and hippocampal activations during positive and negative emotional stimuli moderated the links between family functioning and youths’ problem behaviors.

Family Functioning and Youth Adjustment

Family functioning refers to the quality of interactions among family members (Olson, 2000). Family functioning is often embedded in a multidimensional system that comprises multiple characteristics and dynamic interpersonal relationships (Patterson, 2002). Olson’s (2011) circumplex model of marital and family systems captures this multidimensional structure of family functioning. This circumplex model suggests that the quality of family interactions can be characterized as a whole unit via three core elements, including family cohesion, flexibility, and communication (Olson, 2011). The cohesion dimension is defined as the emotional bonding among family members, which includes two balanced (i.e., separated and connected) and two unbalanced (i.e., disengaged and enmeshed) levels (Olson, 2000). Flexibility is a dimension that reflects the quality of family organization and leadership presented in relationship rules and roles. Family flexibility also incorporates two balanced (i.e., flexible and structured) and two unbalanced (i.e., rigid and chaotic) levels (Olson, 2000). Communication is a facilitating dimension that is critical for the formation of the other two components (i.e., cohesion and flexibility; Olson & Gorall, 2006).

Balanced family functioning is characterized by the balanced levels of cohesion and flexibility and effective communication skills (Olson, 2011). In contrast, unbalanced levels of cohesion and flexibility and poor communication indicate an unbalanced and dysfunctional family rearing environments (Olson, 2011). Family functioning bears extensive implications for youths' socioemotional and behavioral adjustment (Leve, Kim, & Pears, 2005; Oshri et al., 2015b). For example, Joh et al. (2013) suggest that balanced levels of family cohesion and flexibility are linked to youths' reduced problem behaviors. Similarly, Hollenstein, Granic, Stoolmiller, and Snyder (2004) reports that unbalanced family functioning, as reflected by rigidity among family members, is associated with youths' increased risk for developing internalizing and externalizing problems. Additionally, Balanced family functioning can also protect youth from developing problem behaviors under the influences of early life stress (Oliva, Jiménez, & Parra, 2009; Oshri et al., 2015a).

Adolescents' Differential Responses to Family Impacts and Neurobiological Sensitivity

Research in human development widely acknowledges the individual heterogeneity in whether, how, and how much youth are affected by the rearing environment (Schriber & Guyer, 2016). Despite the well-documented links between family dysfunction and youth's behavioral problems (Joh et al., 2013; Leve et al., 2005), such individual variability exists in youths' developmental responses to similar family environments (Luthar, 2006; A. S. Masten & Obradović, 2006). Some youth are more responsive to the influences of positive and negative rearing environments than others. Further, youths' heterogeneous responses to similar caregiving experiences have been attributed to differences in biological sensitivity to environmental influences (Ellis et al., 2011). Specifically, youths' differential behavioral reactions to positive or negative rearing environments are induced by the interactions between specific neurobiological factors and caregiving experiences (i.e., neurobiological sensitivity). To explain why youth respond differently even to similar rearing environments, theories on youths' differential responses to environmental influences progress from the *diathesis-stress* to the *biological sensitivity to context* and the *vantage sensitivity* models. These three models capture different ranges of the youth development spectrum.

The diathesis-stress hypothesis focuses on developmental vulnerability under early life stress (i.e., the negative extreme). This model suggests that specific neurobiological indicators enable some youth to be more likely to develop maladaptive outcomes under the impacts of early adversity (Monroe & Simons, 1991). However, an accumulation of empirical evidence suggests that these vulnerable individuals might instead also develop more positive outcomes when put in an advantageous environment (Obradović et al., 2010; Oshri, Hallowell, et al., 2019). Youths' vulnerability under adverse environments can be viewed instead as sensitivity or developmental plasticity (Schriber & Guyer, 2016). Therefore, the biological sensitivity to context model is proposed, which extends the diathesis-stress theory by introducing the notion that neurobiological factors can also intensify youths' positive outcomes under the influences of supportive family environments (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, the biological sensitivity to context theory purports that specific sensitivity indicators render some youth to be more responsive to the impacts of both negative and positive caregiving experiences. These neurobiological factors can interact with adverse rearing environments and predispose youth at elevated risk for developing maladaptive outcomes. However, in interaction with supportive environments, the same neurobiological architecture can also serve as a potentiating factor that exacerbates positive youth development (Boyce & Ellis, 2005; Ellis et al., 2005). Meanwhile, the vantage sensitivity hypothesis emphasizes positive youth development under advantageous environments (i.e., the positive extreme). This vantage sensitivity model suggests that some youth with vantage sensitivity indicators are more likely to benefit from a supportive and nurturing rearing environment (Pluess, 2017; Pluess & Belsky, 2013).

Overall, the diathesis-stress, biological sensitivity to context, and vantage sensitivity models converge to suggest that certain sensitivity indicators, which are embedded in neurobiological processes (i.e., neurobiological sensitivity indicators), render some youth to be more responsive to the impacts of either positive or negative environmental influences (Ellis et al., 2011; Jolicoeur-Martineau et al., 2017). Adolescents with heightened neurobiological sensitivity might develop more maladaptive outcomes under dysfunctional family experiences. However, they can also obtain more benefits from balanced family functioning (Boyce & Ellis, 2005). In contrast, other youth who lack these sensitivity indicators are not responsive to environmental influences. For these less responsive youth, there is no significant association between rearing experiences and their developmental outcomes (Boyce & Ellis, 2005). In other words, adolescents vary in the degree to which they tune to family environmental influences (Nelson & Guyer, 2011).

Social-Affective Brain Circuitry and Adolescent Neurobiological Sensitivity

From an evolutionary developmental perspective, youths' differential sensitivity to family environments is calibrated for better adaptation to early rearing experiences (Nelson & Guyer, 2011). This calibration to early experiences is achieved through modifying neural structures and functions (Nelson & Guyer, 2011; Schriber & Guyer, 2016). Childhood and adolescence are characterized by significant neural plasticity, namely, the ability of neural characteristics to change in response to environmental inputs (Guyer et al., 2018). This neural plasticity enables youth to calibrate their brain structures and functions in response to alterations in the caregiving environment (Guyer et al., 2018). However, there is sparse research that examines the associations between neural mechanisms and adolescents' differential sensitivity to the impacts of family functioning (Schriber & Guyer, 2016). To fill this gap, it is necessary first to identify the neural functions that can serve as candidate sensitivity indicators.

Recent adolescent neurodevelopmental models provide a theoretical basis to identify neural structures and functions as candidate sensitivity indicators. These models include the dual system theory (Casey et al., 2008; Steinberg, 2008), the triadic model (M. Ernst, 2014), and the social re-orientation framework (Nelson et al., 2005). According to these theories, the functions of adolescents' social-affective brain circuitry may play a critical role in regulating the differential responses to family environmental influences (Schriber & Guyer, 2016). Specifically, the social-affective brain circuitry modulates youths' emotional and stress reactivity, which assists in the calibration of behavioral reactions in response to social cues (Guyer et al., 2011). The heightened reactivity of the social-affective brain circuity indicates youths' elevated emotional and stress responses to social stimuli (Casey et al., 2008; M. Ernst, 2014; Guyer et al., 2011; Steinberg, 2008). These increased responses to emotional and stressful situations allow adolescents to initiate more intense behavioral reactions, and thus develop corresponding adaptive or maladaptive outcomes under positive or negative rearing environments (Schriber & Guyer, 2016). Therefore, the functions of the social-affective brain circuitry could serve as a potential indicator of youths' neurobiological sensitivity under the influences of family functioning (Schriber et al., 2017; Schriber & Guyer, 2016).

The social-affective brain circuitry includes the amygdala and hippocampus, two major subcortical brain regions. The amygdala plays a crucial role in regulating youths' emotional processing in response to both positive and negative social cues (Garavan et al., 2001). The hippocampus is involved in the consolidation of emotional memories (Manns et al., 2003) and regulating acute stress responses (Mizoguchi et al., 2003). Both the amygdala and hippocampus, in general, mediate adolescents' emotional and stress reactivity. Therefore, the functions of the amygdala and hippocampus may be connected with youths' differential sensitivity to the influences of family rearing environments (McLaughlin et al., 2014; Schriber et al., 2017; Schriber & Guyer, 2016; Whittle et al., 2011; Yap et al., 2008). Heightened amygdalar and hippocampal responses to emotional stimuli may suggest elevated neural engagement in emotion and stress regulation, which interacts with positive or negative rearing environments and induced youths' adaptive or maladaptive responses (Schriber & Guyer, 2016; Whittle et al., 2011; Yap et al., 2008).

Neuroimaging research documents significant lateralization (i.e., the tendency for certain brain functions to be specialized to one brain hemisphere) in amygdalar and hippocampal activations in response to emotional stimuli (Baas et al., 2004; Sergerie et al., 2008; Wager et al., 2003). For example, when presented with positive or negative emotional stimuli, activations were more frequently reported on the left than the right amygdala (Baas et al., 2004; Sergerie et al., 2008). However, the magnitude of activation effect sizes was found to be similar across the left and right brain hemispheres (Baas et al., 2004; Sergerie et al., 2008). The lateralized amygdala activities may be attributed to the global/local hemisphere biases (Cahill, 2003; Gläscher & Adolphs, 2003). Specifically, the left amygdala is suggested to be more involved in local (i.e., fine-grained details) and elaborate information processing. In contrast, the right amygdala is more engaged in global (i.e., holistic) and autonomic processing (Cahill, 2003; Gläscher & Adolphs, 2003). In an emotional processing task such as the Emotional N-back, research participants typically are required to focus on details of facial expressions (Tottenham, Tanaka, et al., 2009). Thus, the left amygdala may be more engaged in these emotional processing tasks (Baas et al., 2004).

Furthermore, the hippocampus also shows significant lateralization in its functions of memory consolidation. Hippocampal functions in spatial memory consolidation are found to be localized on the right brain hemisphere. In contrast, the functions in verbal memory consolidation are suggested to be localized on the left hippocampus (Burgess et al., 2002; Klur et al., 2009; Maguire, 2001). The left hippocampus is also shown to be more closely connected to stress-related psychopathology compared to the right hippocampus (MacMaster & Kusumakar, 2004; Schriber et al., 2017; Vythilingam et al., 2002; Whittle et al., 2013). Overall, when testing the roles of the amygdala and hippocampus as neurobiological sensitivity indicators, it is necessary to take the lateralization of amygdalar and hippocampal activations into consideration.

The Present Study

The current study aimed to examine the neural mechanisms underlying adolescents' differential responses to the influences of family functioning. The aims and hypotheses of this study were four-fold and presented in Figure 4.1. First, the direct effects of family functioning, reflected in family cohesion, flexibility, and communication skills, on youths' internalizing and externalizing problems were examined. It was hypothesized that balanced family functioning (i.e., balanced family cohesion and flexibility and effective communication skills) would be associated with youths' decreased problem behaviors. Similarly, dysfunctional family rearing environments were hypothesized to be linked to increased problem behaviors (hypothesis 1).

Then, the moderating effects of amygdalar and hippocampal responses to positive and negative emotional stimuli on the relations between family functioning and youth adjustment were tested. It was hypothesized that the amygdalar (hypothesis 2) and hippocampal (hypothesis 3) activations would amplify the connections between family functioning and youth adjustment, following a biological sensitivity to context pattern (as presented in Figure 4.2). In particular, higher amygdalar and hippocampal activations during emotional processing would amplify the associations between unbalanced family functioning and increased problem behaviors (hypotheses 2a and 3a). Higher neural activations were also hypothesized to intensify the impact of balanced family functioning on reduced internalizing and externalizing problems (hypotheses 2b and 3b). For youth with low levels of amygdalar activations during emotional processing, it was hypothesized that their developmental outcomes were not significantly affected by family functioning (hypotheses 2c and 3c).

Lastly, the lateralization of amygdalar and hippocampal activations during emotional processing and their roles as neurobiological sensitivity indicators were investigated. This study hypothesized that the left amygdala and hippocampus would exhibit higher levels of activations in response to emotional stimuli (hypothesis 4a). Additionally, it was hypothesized that left amygdalar and hippocampal would exhibit a stronger moderating effect on the associations between family functioning and youth adjustment, compared to the right amygdala and hippocampus (hypothesis 4b).

Methods

Participants

Data of this study were drawn from a sample of adolescents and their primary caregivers who were enrolled in the Development and Risk and Resilience in Rural Youth (DORRY) project. The DORRY project targeted adolescents and their primary caregivers resided in rural Georgia. During the baseline data collection, adolescents aged 12-14 years old and their primary caregivers were recruited. For families with more than one eligible child, only one parent-child dyad participated in the study.

The current study included 123 dyads of youth and their primary caregivers whose data were obtained from January 2019 to February 2020. Youth were 12 to 14 years old during the baseline data collection (*M* = 12.90, *SD* = .81). The biological sex distribution of youth was approximately even, with 55% (*n* = 67) females and 45% (*n* = 56) males. For race/ethnicity distribution, there were 77% (*n* = 95) White, 12% (*n* = 15) Black, 5% (*n* = 6) Hispanic/Latino(a), and 6% (*n* = 7) Other. For primary caregivers, most of them (94%, *n* = 115) were female. There were 70% (*n* = 86) of primary caregivers who were married. Large variances existed in participants' sociodemographic characteristics. For example, there were 48% of primary caregivers with an education level below a Bachelor's degree. Most (80%) primary caregivers were employed. Additionally, there were 46% of families having an annual household income below 200% federal poverty level (i.e., $51,500 for a family with four people).

Procedures

This study was reviewed and approved by the University of Georgia Institutional Review Board for ethical conduct. Participants were recruited from rural counties in Georgia, U.S. through non-university-affiliated community recruiters, flyers, social media advertisements, and the assistance of school districts. Interested primary caregivers completed an online information form and were then reached out by a research assistant to complete a brief phone screening to determine their eligibility.

During the baseline data collection, the MRI scanner was changed. Therefore, the neuroimaging data of 29 participants were obtained from a different MRI scanner. The MRI and fMRI data acquisition protocols, including all scanning parameters, were synchronized between two scanners. Additionally, scanner differences were controlled for in the analyses (Stonnington et al., 2008). The first data collection session was a two-hour home visit, during which parents and youth provided their informed consent and assent, respectively, and completed a series of surveys and computer-based tasks. Trained research assistants also conducted home and neighborhood environment observations. At the end of the home visit session, youth were compensated with $20, and parents were compensated with $30.

One week after the home-visit session, participants were invited to participate in the second session at the Bio-Imaging Research Center at the University of Georgia, during which the youth and primary caregiver completed a three-hour data collection, including interactional tasks, psychophysiological assessments, computer-based tasks, and one 60-minute neuroimaging scanning. Before the neuroimaging procedure, a pre-scan session (30 minutes) was administered to participants, which included an MRI contraindication rescreening, MRI simulation and motion compliance training, and practice on the two fMRI tasks (MID and EN-Back). The scanning consisted of a fixed order of a localizer, the 3D T1-weighted scanning (5 minutes), a resting-state fMRI (1 run, 10 minutes), the monetary incentive delay task (2 runs, 10 minutes), the Emotional NBack (EN-Back) task (2 runs, 10 minutes), the 3D T2-weighted scanning (DTI, 8.5 minutes), and the phase map (2 runs, 3 minutes and 10 seconds). After the lab visit session, the youth got $40, and parents got $100 for incentives. The primary caregiver also provided consent to be reached out again and contact information for follow-up data collection.

Measures

Family functioning. Family functioning was assessed through the parent-report Family Adaptability and Cohesion Evaluation Scale (FACES) IV Package (Olson et al., 2006). FACES IV measures different dimensions of family functioning comprehensively, including cohesion, flexibility, and communication. Based on the Circumplex Model of Marital and Family Systems, family cohesion and flexibility are two core dimensions of the family functioning, whereas family communication is a facilitating dimension that captures how family members alter their cohesion and flexibility (Olson & Gorall, 2006).

In the Circumplex Model of Marital and Family Systems, balanced family functioning refers to healthy interpersonal relationships in the family environment. In contrast, unbalanced family functioning reflects dysfunctional and unhealthy caregiving environments. Family cohesion was captured through three subscales, including cohesion ($\alpha = .77$), enmeshed ($\alpha = .58$), and disengaged ($\alpha = .64$). Family flexibility was also measured via three subscales, including flexibility ($\alpha = .66$), chaotic ($\alpha = .71$), and rigid ($\alpha = .66$). Each subscale consisted of seven items that ask the parent to rate how much they agree with the statements of their families, with responses ranging from 1 (strongly agree) to 5 (strongly agree). Examples questions included “Family members are involved in each other’s lives” and “My family is able to adjust to change when necessary.” A ratio score of balanced/unbalanced scales was created for cohesion and flexibility dimensions, respectively. Higher ratio scores indicating more balanced family cohesion and flexibility dimensions.

Family communication was measured via ten items from the FACES IV package (Olson et al., 2006). Example questions included “Family members are very good listeners” and “Family members express affection to each other.” The raw score of family communication was calculated by summing the ten items (α = .84). Higher percentile scores (minimum = 0, maximum = 100) indicated better family communication skills. A composite score of family functioning was obtained by taking the mean of standardized cohesion ratio, standardized flexibility ratio, and communication percentile scores.

Youth Internalizing and externalizing problems. Youths' internalizing and externalizing problems were assessed by the Child Behavior Checklist (CBCL; Achenbach et al., 2001) reported by primary caregivers. This scale has been widely used and well-validated to measure adolescents' behavior problems. In CBCL, parents were instructed to answer how much a series of behavior problems fit their children using a 3-point scale with responses ranging from 0 (not true) to 2 (very often true). The internalizing problems were quantified using three syndrome scales: anxious/depressed (13 items), withdrawn (8 items), and somatic complaints (11 items). The externalizing problems were quantified with two syndrome scales: rule-breaking (10 items) and aggressive behaviors (17 items). In the current study, raw scores of internalizing (α = .71, minimum = 0, maximum = 64) and externalizing (α = .71, minimum = 0, maximum = 54) problems were used in analyses.

Demographics and socioeconomic status (SES). Primary caregivers reported on youths' and their demographics, including biological sex, race/ethnicity, age, and SES. Youths' biological sex was coded as 1 for females and 0 for males. Parental highest education was coded into an ordinal variable that included five values: "<high school diploma," "high school diploma/ GED," "some college," "Bachelor," and "post-graduate degree." Primary caregivers also reported their employment status, which was coded as 1 for employed and -1 for unemployed. Participants reported their household income on ten ranges (e.g., < $5,000, $5,001 to $10,000) based on the total combined family income in the past 12 months. The household income variable was then recoded into the median of each category. The household income and primary caregiver highest education variables were standardized. Then, to obtain the household SES variable, a composite score was calculated by summarizing parental employment status, standardized household income, and standardized primary caregiver education variables.

Amygdala and hippocampus activation assessment.

Paradigm. Amygdalar and hippocampal responses to positive and negative emotional stimuli were assessed during the EN-back task. The EN-back paradigm used in the current study was adopted from the ABCD study (Barch et al., 2013; A. Cohen et al., 2016a). This EN-back was a block-design task that engaged youths' working memory and emotional reactivity processes, which included two imaging runs with each lasting five minutes. The working memory component of this task involved two memory loading conditions, including the 2-back and 0-back conditions. For the 0-back condition, a target picture was presented to youth at the beginning. Youth were asked to respond "match" when the current stimulus matched the target picture. For the 2-back condition, youth responded "match" when the current stimulus matched the picture that was shown two trials before. The emotional reactivity component of the EN-back task contained four emotional stimuli, including negative (i.e., fearful), positive (i.e., happy), and neutral facial-expressions, and non-facial expressions (Casey et al., 2018; Tottenham, Tanaka, et al., 2009).

Each run of the EN-back task consisted of eight blocks: four 0-back and four 2-back blocks. Each memory load condition included one positive, one negative, one neutral, and one non-facial expression block. Additionally, there were also four fixations that each lasted for 15 seconds in each run. Each block started with a 2.5-second cue indicating whether to perform the 2-back or 0-back condition. Then, ten stimuli (i.e., ten trials) were presented with each trial, including a 2-second duration of the stimuli and a 500-millisecond interstimulus interval. Each block included two targets (i.e., matched stimulus), two to three non-target lures, and the rest as non-lures (i.e., stimuli only presented once). At the end of each block, a 500-millisecond cross was presented to alert youth of the task switch. Therefore, each block lasted for 28 seconds. In total, 160 stimuli were presented during the EN-back task across the two runs, with 96 unique stimuli of four different emotional stimulus types (i.e., none, neutral, negative, and positive). Each emotional stimulus type includes 24 unique stimuli.

Paradigm behavioral assessment. Youths' behavioral performance measures on the EN-back task were calculated, including the number, rate, and the reaction time of correct trials. The rate of correct trails (i.e., performance accuracy) was obtained by calculating the percentage of correct trails. The reaction time was obtained by calculating the mean reaction time of correct trials. These behavioral performance metrics were calculated by condition (i.e., 2-back and 0-back conditions), stimuli type (i.e., none, neutral, positive, and negative), and combinations of the two (e.g., 2-back positive stimuli, 2-back negative stimuli, etc.). In the present study, participants' accuracy (i.e., the percentage of correct trials) was used as the major indicator of the EN-back behavioral performance (Casey et al., 2018). Specifically, the percentage of correct trials were obtained for the total 160 trails, for 0-back and 2-back conditions separately, for the four different emotional types (i.e., positive, negative, neutral, and places) separately, and for the working memory condition-specific and stimuli-specific blocks (e.g., 2-back negative emotions; 2-back positive emotions, etc.). The fMRI data of participants who exhibited lower than 60% accuracy on the 2-back or 0-back tasks (*n* = 2) were excluded from the analyses.

MRI acquisition. Whole-brain fMRI was conducted using the General Electric 16-channel fixed-site Signa HDx 3.0 Tesla MRI scanner in the Bio-Imaging Research Center at the

University of Georgia. A standard adult-size three-axis local gradient head coil equipped with the elliptical end-capped quadrature radiofrequency coil was employed to obtain the fMRI data. The EN-back task was adopted from the ABCD study paradigm (Casey et al., 2018; A. Cohen et al., 2016a) and programmed in E-Prime Professional 2.0 version (Psychology Software Tools, PA) on a desktop computer. No modification was made to the EN-back paradigm. While lying supine on the scanner table, the EN-back visual stimuli were presented through an LED goggle system. The youth responded to the task using an MRI-compatible response box. Earplugs and headphones were used to attenuate the scanner noise and protect youths' hearing. Pads and cushions were placed between the youth's head and the head coil to reduce motion artifact. During the scanning, youth kept in continuous visual and verbal contact with the experimenter through an observation window and headphones. Parents also observed the entire scanning session from a seat near the observation window. Structural images were obtained for anatomical reference using a whole-brain high-resolution T1-weighted, fast-spoiled gradient echo scan (TR = 8.2 ms; TE = 25 ms; FOV = 256 × 256 mm; matrix = 64 × 64; 160 contiguous 1 mm axial slices; voxel size, 1 mm^3). fMRI data were acquired via a single-shot, gradient-echo echoplanar pulse sequence (TR = 2,000 ms; TE = 25 ms; FOV = 225 × 225 mm; matrix = 256 × 256 mm). Contiguous 3.5mm thick axial slices were acquired to provide whole-brain coverage in 3.5mm^3 voxels.

Imaging processing. The neuroimaging data processing was conducted using the Analysis of NeuroImages (AFNI) software (R. W. Cox, 1996). Data pre-processing followed established procedures (e.g., Oshri, Hallowell, et al., 2019; Philip et al., 2016). This procedure included removing outliers, skull stripping, alignment to T1 anatomical data, and censoring TRs exhibiting excessive movement (> 0.4mm). In the fMRI data processing, each frame was registered to the first using AFNI'S 3dvolreg (R. W. Cox, 1996; Hagler Jr et al., 2019). This process also provided head motion time courses, which were further incorporated into task-based fMRI analyses. Datasets were transformed into Talairach space (Talairach & Tournoux, 1988). Participants who exhibited movement with greater than 25% censored TRs were excluded from further analyses. Spatial blurring over a 7mm (i.e., twice the voxel size) radius was applied using a Gaussian kernel to compensate for typical variations in functional neuroanatomy across participants. The three initial TRs of each imaging run were removed before analyses.

A voxelwise General Linear Model (GLM) procedure was implemented to quantify brain response for each emotional stimuli (i.e., positive and negative) at the individual level using AFNI (R. W. Cox, 1996). Across the two runs of the EN-back task, there were four blocks for each emotional stimuli (A. Cohen et al., 2016a). The time courses of each emotional stimuli convolved with a hemodynamic function. Motion estimates were included as independent variables (Power et al., 2014). The blocks with positive and negative emotional stimuli were introduced into the models as predictors. The blocks with neutral emotional stimuli were modeled as a linear contrast baseline. Instruction screens, fixation, and blocks with non-facial stimuli were left as resting states. Linear contrasts of positive vs. neutral faces and negative vs. neutral faces were the focus of this investigation of adolescents' emotional reactivity towards positive and negative facial expressions. Specifically, for each voxel of every participant, a GLM was conducted. In the GLM, the temporal pattern of the positive or negative emotion block and covariates (e.g., head movement values) were modeled as independent variables. The blood-oxygen-level-dependent (BOLD) signals over time were modeled as the dependent variable.

Group summary maps were created to evaluate the validity BOLD response to the emotional reactivity component of the EN-back task. To create group summary maps of the neural activations in response to negative and positive emotional stimuli (in contrast to neutral stimuli), the parameter estimates of positive/negative stimulus effects were compared to a hypothetical mean of zero for each voxel using pooled-variance one-sample Student's t-tests. A family-wise error-corrected (FWE) rate of $p < .05$ with a minimum cluster size of 15 adjacent voxels were used to allow for the separation of distinct clusters into individual ROIs. Additionally, a group summary map of the neural activations in response to negative emotional stimuli, in contrast to positive stimuli, was created to explore the valence (negative vs. positive) differences in neural activity, using the same methods.

In the current study, four regions of interest (ROIs) were examined, including the left and right amygdala and hippocampus (see Table 4.1 for coordinates). For each subcortical ROI, the average GLM standardized beta coefficients across voxels were computed for each emotional condition. The mean BOLD response in each ROI was used to test hypotheses.

Imaging quality control. In the current study, neuroimaging data were obtained from 80 adolescents. The lack of neuroimaging data for the rest 43 participants was due to MRI ineligibility (mostly caused by metal in the body such as braces; $n = 26$), attrition (i.e., participants refused to return for the MRI scanning sessions; $n = 8$), and incomplete data collection ($n = 9$). Out of the 80 adolescents, fMRI data of 12 participants were excluded from analyses. This exclusion included 3.75% ($n = 3$) participants whose EN-back data were not obtained, 2.50% ($n = 2$) participants with poor anatomical data quality, 5.00% ($n = 4$) youth who had excessive head motions, and 3.75% ($n = 3$) who did not meet the behavioral performance criteria (i.e., both 2-back and n-back total accuracy > 60%). Therefore, there were 68 adolescents' fMRI data for the EN-back task included in the final analyses. Independent sample t-tests and chi-square tests were conducted to compare youth whose fMRI data were excluded due to quality control reasons with other youth whose fMRI data were included in analyses.

Analytical Plan

Preliminary data analysis (basic descriptive and correlation analyses of study variables) was conducted in IBM SPSS software version 25.0 (Armonk, NY). Paired-sample t-tests were also conducted in SPSS to compare the mean difference of left and right amygdalar and hippocampal responses to positive and negative emotional stimuli. Then, Study hypotheses were tested using structural equation models (SEM) with maximum likelihood estimation with robust standard errors (Yuan & Bentler, 2000) in Mplus Version 7.4 (L. Muthén & Muthén, 2012). In all the SEM models, youths' biological sex and household SES were controlled for (i.e., added as independent variables that predicted youth adjustment), and covaried with family functioning.

First, direct effect models were constructed to examine the associations between family functioning and adolescents' internalizing and externalizing problems. Next, the moderating effects of amygdalar and hippocampal responses to positive and negative emotional social cues on the associations between family functioning and youth adjustment were tested. Given that internalizing and externalizing problems captured distinctive individual variability (r = .588, indicating 65.43% unshared variance), internalizing and externalizing problems were modeled separately in SEM to increase statistical power. The left and right amygdalar and hippocampal responses to positive and negative social cues were also tested separately as moderators in order to examine the region-, laterality-, valence-, and outcome-specific effects. Therefore, a total of 16 models were tested in this study.

In each model, to examine the moderating role of brain responses on the associations between family functioning and youth behavioral problems, an interaction term of the neural responses and the family functioning was created and added to the model. A significant linear association between the interaction term and youth outcome indicated a significant moderating effect of the neural activations on the link between family functioning and youth adjustment.

Significant moderating effects were interpreted using the Johnson-Neyman plot (P. O. Johnson & Neyman, 1936) and the simple slope method (Aiken et al., 1991; Dawson, 2014). The proportion of interaction (PoI) and the proportion affected (PA) tests were adopted to distinguish the neurobiological sensitivity patterns (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012). The PoI test divided youths' differential responses to rearing environments into two regions, including the "for better" and the "for worse" regions. The "for worse" metaphor referred to youth who were biologically vulnerable to the influences of adverse caregiving experiences (Boyce & Ellis, 2005). The "for better" metaphor, in contrast, indicated youth who were neurobiologically predisposed to develop more adaptive responses to positive rearing environments (Boyce & Ellis, 2005). PoI stands for the proportion of "for better" region compared to "for worse" region, with a value close to .50 meeting the criteria for the biological sensitivity to context, a value close to 0.00 suggesting diathesis-stress, and a value close to 1.00 suggesting vantage sensitivity patterns. Del Giudice (2017) purports a PoI value between .20 and .80 as the criteria for potential biological sensitivity to context pattern. Additionally, PA is defined as the proportion of the population that was differentially affected by the moderator. Roisman et al. (2012) purport an acceptable PA index of over 16% as the criteria for the biological sensitivity to context pattern.

To address the missing data issue, Little's Missing Completely at Random (MCAR) test was conducted to examine the missing data pattern (R. J. Little & Rubin, 2002). The full-informative maximum likelihood (FIML) algorithm was used to estimate missing data in Mplus.

The model fit was assessed using indices, including the chi-square test, the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR). Satisfactory model fit was determined based on criteria including an insignificant chi-square test, a minimum value of .95 for CFI and TLI, a minimum value of .08 for SRMR, and a minimum value of .06 for RMSEA (Hu & Bentler, 1999).

Results

Descriptive and Correlation Analyses

Table 4.2 presented adolescents' behavioral performance in the EN-back task, assessed through the percentage of correct trials. Overall, performance levels indicated that most participants understood and could perform the task well across the two runs. The average accuracy rates (i.e., percentage of correct trials) were 88.33% for 2-back and 93.59% for 0-back conditions. Table 4.3 presented the descriptive statistics and correlation analyses of study variables. Families overall exhibited high levels of cohesion (M_{ratio} = 4.27; *SD* = 1.45), flexibility (M_{ratio} = 2.27; *SD* = .72), and communication ($M_{percentile}$ = 70.85; *SD* = 18.62) with moderate variance. In terms of family satisfaction, there were 36.5% primary caregivers who reported very low or low satisfaction towards their family rearing environments, 23.5% who reported moderate levels of family satisfaction, and 40.0% who reported high or very high family satisfaction levels.

Independent sample t-tests and chi-square tests were conducted to compare youth whose fMRI data were excluded due to quality control reasons with other youth whose fMRI data were included in analyses. Results suggested that youth whose fMRI data were excluded exhibited significantly higher levels of internalizing ($t(120) = 2.38$, $M_{\text{difference}} = 2.42$, 95%*CI* of $M_{\text{difference}}$

[.41, 4.43], $p < .05$) and externalizing ($t(89.20) = 2.12$, $M_{\text{difference}} = 2.16$, 95%*CI* of $M_{\text{difference}}$ [.13, 4.19], $p < .05$) problems. No other statistically significant differences were found in terms of youths' demographics, household SES, and family functioning variables.

Study variables were correlated in the expected directions. Specifically, family cohesion, flexibility, and communication dimensions were positively correlated with each other (*r* ranged from .52 to .53, $p < .001$). Higher levels of family cohesion, flexibility, and communication levels were associated with youths' reduced internalizing and externalizing problems (*r* ranged from -.36 to -.12). Youths' internalizing and externalizing problems were moderately correlated ($r = .53$, 95% *CI*[.35, .71], $p < .001$). The left and right amygdalar and hippocampal activations during positive and negative emotional stimuli were overall positively correlated. Specifically, under emotional stimuli with the same valence (positive or negative), amygdalar and hippocampal responses were positively and significantly correlated (*r* ranged from .34 to .92, $p < .001$). Additionally, for each ROI, neural activations under different emotional stimuli exhibited positive and significant correlations (*r* ranged from .47 to .66, $p < .001$). Higher levels of household income were positively associated with parental education level ($r = .51$, 95% *CI*[-.66, -.08], $p < .05$) and more balanced family cohesion ($r = .24$, 95% *CI*[.33, .69], $p < .001$). Household income was also negatively correlated with right amygdalar responses to negative emotional stimuli ($r = -.26$, 95% *CI*[-.50, -.02], $p < .05$). The education levels of primary caregivers were positively associated with parental employment status ($r = .34$, 95% *CI*[.16, .52], $p < .001$). Youths' externalizing problems were significantly associated with elevated activations in the left amygdala ($r = .26$, 95% *CI*[.02, .50], $p < .05$) and right hippocampus ($r = .24$, 95% *CI*[.01, .48], $p < .05$) in responses to negative emotional stimuli.

Neural Activity During the EN-back Task

Figure 4.3 presented the group-level neural activations associated with *negative* emotional stimuli in contrast to *neutral* facial expressions. The upper panel of Table 4.4 displayed the cluster size and coordinates of brain regions that exhibited significant neural activity during the negative emotional processing, above and beyond the neutral emotional stimuli. Results suggested that the left and right amygdala exhibited significant group-level activations in response to negative emotional stimuli. However, the hippocampus did not show significant activations after corrections based on the FWE threshold ($p < .05$). Additionally, the parahippocampal gyrus, lingual gyrus, fusiform gyrus, inferior occipital gyrus, right superior and middle temporal gyrus, and left inferior frontal gyrus showed significant activations in response to negative emotional stimuli. Further, the inferior parietal lobule, right middle frontal gyrus, precentral gyrus, left cingulate gyrus, and right superior parietal lobule presented significant deactivations in response to negative emotional stimuli.

Figure 4.4 presented the group-level neural activations associated with *positive* emotional stimuli in contrast to *neutral* facial expressions. The middle panel of Table 4.4 displayed the cluster size and coordinates of brain regions that exhibited significant neural activity during the positive emotional processing, above and beyond the neutral emotional stimuli. Results suggested that the left and right amygdala and hippocampus did not exhibit significant group-level activations in response to positive emotional stimuli at the FWE threshold ($p < .05$). However, the lingual gyrus and right posterior cingulate showed significant activations in response to negative emotional stimuli. Further, the middle frontal gyrus, superior frontal gyrus, right inferior parietal lobule, and left supramarginal gyrus presented significant deactivations in response to negative emotional stimuli.

To explore the differences in neural activations during positive and negative emotional processing, Figure 4.5 presented the group-level neural activations associated with *negative* emotional stimuli in contrast to *positive* facial expressions. The bottom panel of Table 4.4 displayed the cluster sizes and coordinates of brain regions that exhibited significant neural activity during the negative emotional processing, above and beyond the positive emotional stimuli. At the FWE threshold ($p < .05$), results showed that brain regions including the declive gyrus, fusiform gyrus, inferior frontal gyrus, precuneus, left middle frontal gyrus, and left superior frontal gyrus exhibited heightened activations during negative emotional processing, compared to positive emotional processing.

Whole-brain voxelwise paired-sample t-tests were conducted to explore the valence (positive vs. negative emotional stimuli) differences in amygdalar and hippocampal activations during emotional processing (Table 4.5). Results suggested that left amygdala exhibited higher levels of activation during negative emotional processing compared to positive emotional processing ($t(67) = 2.37$, $M_{\text{difference}} = 1.76$, 95%*CI* of $M_{\text{difference}}$ [.27, .32], $p < .05$). However, the hippocampus and right amygdala showed no statistically significant differences in their activations as responses to emotional stimuli of different valences.

Hypothesis 1: The Direct Effects of Family Functioning on Youth Adjustment

A structural equation model was tested to examine the direct effects of family functioning on youth internalizing and externalizing problems (Table 4.6 and Figure 4.6). The model exhibited good fit indices: $\chi^2(2) = 3.77$ ($p = .93$); CFI = .96; TLI = .86; RMSEA = .09; SRMR = .04. Results indicated that balanced family functioning, as reflected by healthy family cohesion and flexibility, as well as effective communication, was significantly associated with lower levels of internalizing ($\beta = -.31$, 95%*CI* [-3.33, -.68], $p < .01$) and externalizing ($\beta = -.32$, 95%*CI*

[-2.87, -1.10], *p* < .001) problems among adolescents. This model controlled for youths' biological sex and household SES. Youths' biological sex was found to be not significantly associated with youths' internalizing (β = -.02, 95%*CI* [-2.14, 1.67], *p* = .81) or externalizing (β = -.14, 95%*CI* [-3.31, .28], *p* = .10) problems. Similarly, household SES was not linked to internalizing (β = .09, 95%*CI* [-.26, .78], *p* = .32) or externalizing (β = -.03, 95%*CI* [-.63, .46], *p* = .76) problems among youth.

Hypothesis 2: The Amygdalar Activations as Moderators on the Associations Between Family Functioning and Youth Adjustment

Negative emotional stimuli. Table 4.7, Figure 4.7, and Figure 4.8 presented the results of the SEM models with amygdalar responses to *negative* emotional stimuli as moderators on the associations between family functioning and youth adjustment.

Left amygdalar activations as moderator. The left column of Table 4.7 and Figure 4.7 showed the SEM models in which *left amygdalar* activations in response to *negative* emotional stimuli interact with family functioning and impact youths' internalizing and externalizing problems, respectively. Both models exhibited excellent fit indices (Externalizing model: $\chi^2(4)$ = 4.44 (*p* = .96); CFI = .98; TLI = .97; RMSEA = .03; SRMR = .04; Internalizing model: $\chi^2(4)$ = 1.69 (*p* = .79); CFI = 1.00; TLI = 1.18; RMSEA = .00; SRMR = .03). In the model that tested externalizing problems as outcomes, balanced family functioning was negative associated with youths' externalizing problems (β = -.24, 95%*CI* [-2.52, -.50], *p* < .01). The interaction term of family functioning and left amygdalar activations was also significantly linked to youths' externalizing problems (β = -.29, 95%*CI* [-.54, -.03], *p* < .05). In the model that tested internalizing problems as outcomes, balanced family functioning was negatively associated with youths' internalizing problems (β = -.24, 95%*CI* [-2.76, -.31], *p* < .05). The interaction term of family functioning and left amygdalar activations was also significantly linked to youths' internalizing problems ($\beta = -.24$, 95%*CI* [-.49, -.01], $p < .05$). Overall, these two models suggested that left amygdalar responses to negative emotional stimuli significantly exacerbated the impact of unbalanced family functioning on youths' internalizing and externalizing problems.

Figure 4.9 and Figure 4.10 displayed the interpretation of the moderating effect of left amygdalar responses to negative emotional stimuli on the associations between family functioning and youths' externalizing and internalizing problems, respectively. The upper panel of these two figures presented the Johnson-Neyman plots (P. O. Johnson & Neyman, 1936), in which the X-axis indicated the moderator (i.e., left amygdalar activations) and the Y-axis represented the main effects of family functioning on youth's externalizing/internalizing problems. The Johnson-Neyman plots showed that for youth with relatively high left amygdalar activations (64.70% of participants for the internalizing problems model, 63.20% of participants for the model of the externalizing problem), balanced family functioning was negatively related to youth's problem behaviors. For youth with low amygdalar activations (35.30% of participants for the internalizing problems model, 36.80% of participants for the model of the externalizing problem), the effects of family functioning on youths' externalizing and internalizing problems were not statistically significant.

The lower panel of Figure 4.9 and Figure 4.10 displayed the simple slope figures and the differential susceptibility pattern critical tests (Del Giudice, 2017; Roisman et al., 2012). Accordingly, among participants with higher left amygdalar activations during negative emotional stimuli (presented by the solid line), unbalanced family functioning was associated with elevated externalizing and internalizing problems among youth. Among participants with lower left amygdalar activations (presented by the dashed line), family functioning was not significantly linked to youth’s externalizing or internalizing problems. For the model of the externalizing problems, a PoI of .73 and a PA of 29.60% were calculated. For the internalizing model, a PoI of .76 and a PA of 27.00% were also obtained from the interpretations (Del Giudice, 2017; Roisman et al., 2012). Overall, these effects supported the biological sensitivity to context model (Boyce & Ellis, 2005; Del Giudice, 2017; Ellis et al., 2005; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012). Specifically, with high left amygdalar activations during negative emotional processing, youth who reported unbalanced family functioning exhibited more externalizing and internalizing problems. However, high levels of left amygdalar activations also intensified reduced problem behaviors among youth who reported balanced family functioning.

Right amygdalar activations as moderator. The right column of Table 4.7 and Figure 4.8 showed the SEM models in which right amygdalar activations in response to *negative* emotional stimuli interact with family functioning and impact youths’ internalizing and externalizing problems, respectively. Both models were just-identified (Brown, 2015). Balanced family functioning was negatively associated with youths’ externalizing problems (β = -.29, 95%*CI* [-2.86, -.81], *p* < .001) and internalizing problems (β = -.28, 95%*CI* [-3.27, -.40], *p* < .05). High levels of right amygdalar activations during negative emotional processing were not significantly associated with youths’ externalizing (β = .13, 95%*CI* [-.09, .28], *p* = .30) or internalizing (β = .20, 95%*CI* [-.01, .30], *p* = .06) problems. The interaction term of family functioning and left amygdalar activations was not significantly linked to youths’ externalizing (β = -.18, 95%*CI* [-.41, .07], *p* = .16) or internalizing problems (β = -.11, 95%*CI* [-.29, .07], *p* = .24). Overall, these two models suggested that the interaction between right amygdalar responses to negative emotional stimuli and family functioning did not influence youth adjustment significantly.

Positive emotional stimuli. Table 4.8, Figure 4.11, and Figure 4.112 presented the results of the SEM models with amygdalar responses to *positive* emotional stimuli as moderators on the associations between family functioning and youth adjustment.

Left amygdalar activations as moderator. The left column of Table 4.8 and Figure 4.11 showed the SEM models in which *left amygdalar* activations in response to *positive* emotional stimuli interact with family functioning and impact youths' internalizing and externalizing problems, respectively. Both models exhibited excellent fit indices (Externalizing model: $\chi^2(4)$ = 3.74 (p = .91); CFI = 1.00; TLI = 1.01; RMSEA = .00; SRMR = .04; Internalizing model: $\chi^2(4)$ = 3.597 (p = .98); CFI = 1.00; TLI = 1.03; RMSEA = .00; SRMR = .04). Balanced family functioning was negatively associated with youths' externalizing problems (β = -.27, 95%*CI* [-2.67, -.78], p < .001) and internalizing problems (β = -.29, 95%*CI* [-3.13, -.61], p < .01). High levels of left amygdalar activations during positive emotional processing were associated with youths' elevated externalizing (β = .25, 95%*CI* [.02, .34], p < .05) but not internalizing (β = .11, 95%*CI* [-.05, .21], p = .22) problems. The interaction term of family functioning and left amygdalar activations was significantly linked to youths' externalizing (β = -.33, 95%*CI* [-63, -.09], p < .05) and internalizing problems (β = -.33, 95%*CI* [-.67, -.10], p < .01). Overall, these two models suggested that left amygdalar responses to positive emotional stimuli significantly exacerbated the associations between family functioning and youth adjustment.

Figure 4.13 and Figure 4.14 displayed the interpretation of the moderating effect of left amygdalar responses to positive emotional stimuli on the associations between family functioning and youths' externalizing and internalizing problems, respectively. The upper panel of these two figures presented the Johnson-Neyman plots (P. O. Johnson & Neyman, 1936), in which the X-axis indicated the moderator (i.e., left amygdalar activations) and the Y-axis represented the main effects of family functioning on youth’s externalizing/internalizing problems. The Johnson-Neyman plots showed that for youth with relatively high left amygdalar activations in response to positive emotional stimuli (61.80% of participants), balanced family functioning was negatively related to youth’s externalizing and internalizing problems. For youth with low amygdalar activations (38.20% of participants), the effects of family functioning on youths’ externalizing and internalizing problems were not statistically significant.

The lower panels of Figure 4.13 and Figure 4.14 displayed the simple slope figures and the differential susceptibility pattern critical tests (Del Giudice, 2017; Roisman et al., 2012). Accordingly, among participants with higher left amygdalar activations during positive emotional processing (presented by the solid line), unbalanced family functioning was associated with elevated externalizing and internalizing problems. Balanced family functioning was linked to reduced problem behaviors among youth. Among participants with lower left amygdalar activations (presented by the dashed line), family functioning was not significantly linked to youth’s externalizing or internalizing problems. For the model of the externalizing problems, a PoI of .73 and a PA of 29.60% were calculated. For the internalizing model, a PoI of .62 and a PA of 37.40% were also obtained from the interpretations (Del Giudice, 2017; Roisman et al., 2012). Overall, these effects supported the biological sensitivity to context model (Boyce & Ellis, 2005; Del Giudice, 2017; Ellis et al., 2005; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012). Specifically, youth who had high left amygdalar activations during positive emotional processing and reported unbalanced family functioning exhibited more externalizing and internalizing problems. In contrast, with the same high levels of amygdalar activations, youth also showed reduced problem behaviors in response to balanced family environments.

Right amygdalar activations as moderator. The right column of Table 4.8 and Figure 4.12 showed the SEM models in which right amygdalar activations in response to *positive* emotional stimuli interacted with family functioning and impact youths' internalizing and externalizing problems, respectively. Both models were just-identified (Brown, 2015). Balanced family functioning was negatively associated with youths' externalizing problems (β = -.32, 95%*CI* [-3.01, -.10], *p* < .001) and internalizing problems (β = -.31, 95%*CI* [-3.38, -.68], *p* < .01). However, left amygdalar activations during negative emotional processing were not associated with youths' externalizing (β = .13, 95%*CI* [-.08, 28], *p* = .27) or internalizing (β = .04, 95%*CI* [-.14, .19], *p* = .71) problems significantly. The interaction term of family functioning and left amygdalar activations was not significantly linked to youths' externalizing (β = -.23, 95%*CI* [-.48, .01], *p* = .06) or internalizing problems (β = -.15, 95%*CI* [-.36, .03], *p* = .10) either. Overall, these two models suggested that right amygdalar responses to positive emotional stimuli did not interact with family functioning and influence youth adjustment significantly.

Hypothesis 3: The Hippocampal Activations as Moderators on the Associations Between Family Functioning and Youth Adjustment

Negative emotional stimuli. Table 4.9, Figure 4.15, and Figure 4.16 presented the results of the SEM models with hippocampal responses to *negative* emotional stimuli as moderators on the associations between family functioning and youth adjustment.

Left hippocampal activations as moderator. The left column of Table 4.9 and Figure 4.15 showed the SEM models in which *left hippocampal* activations in response to *negative* emotional stimuli interact with family functioning and impact youths' internalizing and externalizing problems. Both models exhibited acceptable fit indices (Externalizing model: $\chi^2(4)$

= 4.70 (*p* = .83); CFI = .94; TLI = .93; RMSEA = .04; SRMR = .04; Internalizing model: just-identified; $\chi^2(0)$ = .00 (*p* = 1.00); CFI = 1.00; TLI = 1.00; RMSEA = .00; SRMR = .00). Family functioning was found to be significantly and negatively related to youths' internalizing (β = -.32, 95%*CI* [-3.55, -.58], *p* < .01) and externalizing (β = -.27, 95%*CI* [-2.96, -.43], *p* < .01)\ problems. Left hippocampal activations during negative emotional processing were not significantly associated with youths' externalizing (β = .17, 95%*CI* [-.11, .46], *p* = .23) and internalizing problems (β = .09, 95%*CI* [-.17, .37], *p* = .46). The interaction term of family functioning and left hippocampal activations was not significantly linked to youths' externalizing (β = -.25, 95%*CI* [-.66, .07], *p* = .1) or internalizing (β = -.09, 95%*CI* [-.23, .44], *p* = .53) problems either. Overall, these two models suggested that left hippocampal responses to negative emotional stimuli did not significantly moderated the impact of family functioning on youths' internalizing and externalizing problems.

Right hippocampal activations as moderator. The right column of Table 4.9 and Figure 4.16 showed the SEM models in which right hippocampal activations in response to *negative* emotional stimuli interact with family functioning and impact youths' internalizing and externalizing problems. Both models were just-identified (Brown, 2015). Family functioning was found to be significantly and negatively related to youths' internalizing (β = -.28, 95%*CI* [-3.32, -.27], *p* < .05) and externalizing (β = -.26, 95%*CI* [-2.77, -.48], *p* < .01) problems. Right hippocampal activations during negative emotional processing were significantly and positively associated with youths' externalizing (β = .36, 95%*CI* [.12, .62], *p* < .01) and internalizing problems (β = .23, 95%*CI* [.01, .50], *p* < .05). The interaction term of family functioning and right hippocampal activations was not significantly linked to youths' externalizing (β = -.16, 95%*CI* [-.53, .13], *p* = .24) or internalizing (β = -.09, 95%*CI* [-.43, .19], *p* = .45) problems either.

Overall, these two models suggested that right hippocampal responses to negative emotional stimuli did not significantly moderated the impact of family functioning on youths' internalizing and externalizing problems.

Positive emotional stimuli. Table 4.10, Figure 4.17, and Figure 4.18 presented the results of the SEM models with hippocampal responses to *positive* emotional stimuli as moderators on the associations between family functioning and youth adjustment.

Left hippocampal activations as moderator. The left column of Table 4.10 and Figure 4.17 showed the SEM models in which *left hippocampal* activations in response to *positive* emotional stimuli interact with family functioning and impact youths' internalizing and externalizing problems, respectively. Both models exhibited excellent fit indices (Externalizing model: $\chi^2(4) = 4.44$ (*p* = .88); CFI = .98; TLI = .97; RMSEA = .03; SRMR = .04; Internalizing model: $\chi^2(4) = 4.44$ (*p* = .89); CFI = .98; TLI = .98; RMSEA = .02; SRMR = .04). Balanced family functioning was negatively associated with youths' externalizing problems (β = -.26, 95%*CI* [-2.68, -.56], *p* < .01) and internalizing problems (β = -.29, 95%*CI* [-3.209, -.52], *p* < .01). High levels of left hippocampal activations during positive emotional processing were associated with youths' elevated internalizing (β = .26, 95%*CI* [.07, .55], *p* < .05) but not externalizing (β = .16, 95%*CI* [-.02, .37], *p* =.07) problems. The interaction term of family functioning and left hippocampal activations was significantly linked to youths' externalizing (β = -.40, 95%*CI* [-.98, -.25], *p* < .01) but not internalizing problems (β = -.17, 95%*CI* [-.63, .08], *p* = .12). Overall, left hippocampal responses to positive emotional stimuli significantly intensified the associations between family functioning and youth externalizing problems.

Figure 4.19 displayed the interpretation of the moderating effect of left hippocampal responses to positive emotional stimuli on the associations between family functioning and youths' externalizing problems. The upper panel of this figure presented the Johnson-Neyman plot (P. O. Johnson & Neyman, 1936), in which the X-axis indicated the moderator (i.e., left hippocampal activations) and the Y-axis represented the main effects of family functioning on youth's externalizing problems. The Johnson-Neyman plots showed that for youth with relatively high left hippocampal activations in response to positive emotional stimuli (60.30% of participants), balanced family functioning was related to youth's reduced externalizing problems. For youth with moderate hippocampal activations (35.3% of participants), the effect of family functioning on youths' externalizing problems was not statistically significant. For a small percentage of youth with extremely low hippocampal activations (4.40% of participants), this effect was significantly positive.

The lower panel of Figure 4.17 presented the simple slope figure and the differential susceptibility pattern critical tests (Del Giudice, 2017; Roisman et al., 2012). Accordingly, among participants with higher left hippocampal activations during positive emotional processing (presented by the solid line), unbalanced family functioning was associated with elevated externalizing problems. Balanced family functioning was linked to reduced problem behaviors among youth. A PoI of .66 and a PA of 34.80% were calculated from the interpretations (Del Giudice, 2017; Roisman et al., 2012). Overall, this effect supported the biological sensitivity to context model (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012). Specifically, high left hippocampal activations during positive emotional processing exacerbated the development of externalizing problems for youth who reported unbalanced family environments. However, with the same high levels of hippocampal activations, youth also showed reduced problem behaviors as adaptive responses to balanced family functioning. Additionally, for a small proportion of youth with high levels of left hippocampal deactivations (i.e., low levels of hippocampal activations), a “steeling” effect was revealed. This “steeling” effect suggested that dysfunctional family rearing environments were linked to youths’ reduced problem behaviors (R. T. Liu, 2015; Repetti & Robles, 2016; Rutter, 2006).

Right hippocampal activations as moderator. The right column of Table 4.10 and Figure 4.18 showed the SEM models in which right hippocampal activations in response to *positive* emotional stimuli interact with family functioning and impact youths’ internalizing and externalizing problems. Both models exhibited acceptable fit indices (Externalizing model: $\chi^2(4) = 4.79$ ($p = .90$); CFI = .94; TLI = .92; RMSEA = .04; SRMR = .04; Internalizing model: $\chi^2(4) = .4.67$ ($p = .89$); CFI = .94; TLI = .93; RMSEA = .04; SRMR = .04). Family functioning was found to be significantly and negatively related to youths’ externalizing (β = -.32, 95%*CI* [-2.98, -1.04], $p < .001$) and internalizing (β = -.31, 95%*CI* [-3.43, -.63], $p < .01$) problems. Higher levels of right hippocampal activations during positive emotional processing were significantly associated with youths’ increased internalizing (β = .21, 95%*CI* [.02, .42], $p < .05$) and externalizing (β = .26, 95%*CI* [.02, .50], $p < .05$) problems. The interaction term of family functioning and left hippocampal activations was not significantly linked to youths’ externalizing (β = -.04, 95%*CI* [-.43, .32], $p = .77$) or internalizing (β = -.10, 95%*CI* [-.45, .16], $p = .34$) problems. Overall, these two models suggested that right hippocampal responses to positive emotional stimuli did not significantly moderated the impact of family functioning on youths’ internalizing and externalizing problems.

Hypothesis 4: Lateralization of Amygdalar and Hippocampal Activations

Table 4.11 presented the paired-sample t-tests on the mean differences between the left and right amygdalar and hippocampal activations during positive and negative emotional processing. Results suggested no statistically significant mean differences in amygdalar and hippocampal responses to either negative (amygdala: $t(67) = -1.49$, $p = .14$; hippocampus: $t(67) = -.46$, $p = .65$) or positive (amygdala: $t(67) = -1.68$, $p = .10$; hippocampus: $t(4967 = .24$, $p = .81$) emotional stimuli. Therefore, amygdalar and hippocampal activations were similar in magnitude across the brain hemispheres. Additionally, only the left amygdala and hippocampus were found to significantly exacerbate the associations between family rearing environments and youth adjustment. No significant moderation effect for right amygdalar or hippocampal functions was identified. Therefore, left amygdalar and hippocampal activations during emotional processing are more salient for regulating youths' neurobiological sensitivity to the influences of family functioning, compared to the right amygdala and hippocampus.

Discussion

Family functioning is critical for shaping youths' socioemotional development. However, youth who are exposed to family rearing environments with similar quality of interpersonal relationships may respond to these environments in different ways (Luthar, 2006; A. S. Masten & Obradović, 2006). Some youth are either more vulnerable to the harmful impacts of dysfunctional family environments or benefiting more from balanced family functioning. Others may not be significantly influenced by rearing environments (Boyce & Ellis, 2005). This variability in youths' differential responses to rearing environments is embedded in youths' biological sensitivity to family effects (Ellis & Boyce, 2008). The biological sensitivity to context theory suggests that these individual differences in response to similar rearing environments may stem from individual variability in neural processes (Boyce & Ellis, 2005; Ellis et al., 2011). For example, the functions of the social-affective brain circuitry mediate youths' differential responses to rearing environments (Schriber & Guyer, 2016). Yet, scarce research empirically examined the neural mechanisms underlying youths' differential sensitivity to family rearing environments (Schriber & Guyer, 2016). The current study aimed to explain the neural architecture underpinning the individual differences in youths' responses to the influences of family functioning. Specifically, this study examined the moderating effects of amygdalar and hippocampal responses to positive and negative emotional stimuli on the associations between family functioning and youth adjustment.

Data of this study were drawn from a sample of 123 rural adolescents and their primary caregivers recruited from Georgia. A multidimensional measure of family functioning (i.e., FACES IV package; Olson et al., 2006) that could capture the full spectrum of youths' rearing environment quality was adopted to assess family rearing environments. Results of the present study overall supported the study hypotheses. First, balanced family functioning was significantly associated with youths' reduced internalizing and externalizing problems. Second, left amygdalar responses to positive and negative emotional stimuli were found to intensify the effects of family functioning on youths' externalizing and internalizing problems (Boyce & Ellis, 2005; Ellis et al., 2005). Similarly, left hippocampal activations during positive emotional stimuli significantly interacted with family functioning and affected youths' externalizing problems (Boyce & Ellis, 2005; Ellis et al., 2005). Lastly, the amygdala and hippocampus did not present significant lateralization effects in neural activations. However, only the amygdala and hippocampus on the left hemisphere, rather than the right brain hemisphere, were found to significantly intensify the associations between family functioning and youth adjustment.

Neural Activity During the EN-back Task

The current study found that youth exhibited statistically significant high levels of left and right amygdalar activations during negative emotional processing, but not positive emotional processing. Additionally, neural activations were not identified in the hippocampus in response to either positive or negative emotional stimuli. The activations of the amygdala during negative emotional processing validated the emotional reactivity component of the EN-back task (Casey et al., 2018; A. Cohen et al., 2016a). Further, the left amygdala exhibited higher levels of activations in response to negative stimuli, compared to positive stimuli. This valence difference in amygdalar activations suggests that the amygdala is more engaged in processing negative emotions, such as fear, among this sample of rural adolescents. Adolescence is a developmental period when youth are especially sensitive to negative social feedback (Silva et al., 2016; Somerville, 2013; Tottenham & Galván, 2016). Therefore, the heightened amygdalar activations to negative emotional stimuli might may adolescents' elevated susceptibility to the influences of negative social and family environments (Monk et al., 2008).

The current study found that adolescents did not exhibit significant hippocampal activations during emotional stimuli, which might be due to the emotional-cognitive design of the EN-back task (A. Cohen et al., 2016a; Schweizer et al., 2019). The EN-back paradigm includes two components: working memory and emotional reactivity (Casey et al., 2018; A. Cohen et al., 2016a). It is important to note that the emotional component in this paradigm reflects youths' emotional reactivity when they are completing cognitive tasks (0-back or 2-back), rather than unconditioned and uninstructed emotional reactivity (A. Cohen et al., 2016a). It is possible that some brain regions that are responsible for emotional and stress regulation, such as the hippocampus, are suppressed when youth exercise cognitive abilities to complete the 0-back and 2-back tasks (Stretton et al., 2012). For example, Stretton et al. (2012) report that hippocampal activities are progressively suppressed with the increase of loads in the working memory task (i.e., from 0-back to 2-back tasks). The suppressed hippocampal activities result in the non-significant neural activations in the hippocampus when youth complete the EN-back task.

Except for the amygdala, youth also exhibited neural activity on other brain regions during the EN-back task. For example, in response to emotional stimuli, the lingual gyrus, fusiform gyrus, middle temporal gyrus, and inferior occipital gyrus showed significant neural activations. These regions are relevant to visual information processing (McCarthy, Puce, Belger, & Allison, 1999). Particularly, the fusiform gyrus is specialized in facial recognition and the perception of emotions in facial stimuli (Radua et al., 2010). The middle temporal gyrus is also connected with facial recognition (Acheson & Hagoort, 2013). Additionally, in response to positive emotional stimuli, the right posterior cingulate displayed significant activations, which corresponds with its functions in identifying emotional salience (Maddock, Garrett, & Buonocore, 2001, 2003). Overall, the activations of these brain regions correspond to the stimuli of emotional facial expressions employed in the EN-back task (A. Cohen et al., 2016a; Tottenham, Tanaka, et al., 2009).

There were several brain regions that showed significant deactivations in the emotional contrasts of the EN-back task. These regions included the middle frontal gyrus, inferior parietal lobule, superior frontal gyrus, and precentral gyrus. The middle frontal gyrus is shown to be engaged in attention shifting and orientation (Japee, Holiday, Satyshur, Mukai, & Ungerleider, 2015). The inferior parietal lobule is involved in visuospatial processing (Sturm, Haase, & Levenson, 2016). The precentral gyrus plays an important role in controlling voluntary motor movements (Banker & Tadi, 2019). Additionally, the superior frontal gyrus is suggested to be involved in self-awareness (Goldberg, Harel, & Malach, 2006) and laughter (Fried, Wilson, MacDonald, & Behnke, 1998). The deactivations of these regions in the emotional contrasts of the EN-back task reflect the suppression of these relevant cognitive and sensory functions by the activations of regions responsible for emotional processing (Schweizer et al., 2019).

Hypothesis 1: The Direct Effects of Family Functioning on Youth Adjustment

This study supported the first study hypothesis by suggesting that balanced family functioning was associated with youths’ lower levels of internalizing and externalizing problems. This finding corroborates the circumplex model of marital and family systems (Olson & Gorall, 2006). It suggests a consistent and robust influence of the dynamic interpersonal relationships in the family rearing environments on youth adaptation (Joh et al., 2013; Oshri et al., 2015b; Rabinowitz et al., 2016). According to this circumplex model (Olson, 2011), the functioning of family systems consists of three dimensions, including cohesion, flexibility, and communication. Family cohesion reflects the emotional bonding and boundaries among family members, in particular, how the family system balances the togetherness versus separateness of their members (Olson, 2000). As a spectrum, balanced (i.e., moderate) levels of family cohesion suggest harmonious interactions and commitment to supporting other family members (Rabinowitz et al., 2016). The low (i.e., disengaged) and high (i.e., enmeshed) extremes of cohesion both indicate unhealthy interpersonal relationships (Olson & Gorall, 2006). Specifically, in disengaged family rearing environments, family members experience a lack of belongingness and are less dedicated to supporting each other. Thus, disengaged family environments might induce youths’ heightened levels of emotional distress and behavioral problems (Carthy, Horesh, Apter, Edge, & Gross, 2010; Lucia & Breslau, 2006). On the other hand, when placed in enmeshed family environments, family members exhibit extreme levels of emotional closeness, dependency, and demanding loyalty (Olson, 2000). The lack of personal separateness and private space in enmeshed family systems also increases youths’ risk for developing psychopathology and problem behaviors, such as internalizing and externalizing problems (Joh et al., 2013; Rabinowitz et al., 2016).

Family flexibility refers to the degree to which family members stick to family roles and rules, discipline, and negotiation styles (Olson, 2000). Similar to cohesion, family flexibility is also a continuum with moderate levels indicating healthy family functioning. Within structured or flexible family environments, democratic and egalitarian disciplines are usually endorsed, and negotiations about family rules are open to all family members (Olson & Gorall, 2006). Youth reared in such a family rearing environments are more likely to experience balanced and stable interpersonal relationships, which promotes youths' emotional security and enables them to achieve positive developmental outcomes (Hollenstein et al., 2004). In contrast, the low extreme of the flexibility spectrum is rigid family relationships (Olson et al., 2006). Family rigidity limits youths' opportunities to regulate their behaviors using newly-obtained emotional regulation skills, and thus put them at high risk for psychopathology (Hollenstein et al., 2004). The high extreme of flexibility spectrum, on the opposite side, is chaotic family relationships characterized by high levels of noises, crowding, and disorganization (Evans, Eckenrode, & Marcynyszyn, 2010; Olson & Gorall, 2006). Youth reared in chaotic family rearing environments experience low levels of predictability and safety. Thus, they are more likely to develop maladaptive outcomes as an evolutionary survival strategy (Evans et al., 2010; Hardaway, Wilson, Shaw, & Dishion, 2012).

The third element of family functioning is communication, which is a facilitating dimension that coordinates the changes of cohesion and flexibility (Olson et al., 2006). Communication involves family members' listening and speaking skills, self-disclosure, clarity, and respect (Olson, 2011). Family systems with balanced functioning tend to also exhibit high levels of good communication. In contrast, dysfunctional family rearing environments are usually coupled with poor communication skills among family members (Koerner & Mary Anne, 2002; Olson, 2011). Therefore, effective communication skills can promote cohesive and flexible family functioning and further facilitate youths' positive developmental outcomes (Elgar, Craig, & Trites, 2013; Olson & Gorall, 2006). Overall, this study supports the circumplex model (Olson & Gorall, 2006) by showing that family functioning, characterized by family cohesion, flexibility, and communication, was linked to youths' development of internalizing and externalizing problems.

Hypothesis 2: The Amygdalar Activations as Moderators on the Associations Between Family Functioning and Youth Adjustment

In line with the second hypothesis, amygdalar responses to positive and negative emotional stimuli were found to serve as neurobiological sensitivity indicators. Specifically, left amygdalar activations during negative and positive emotional stimuli significantly exacerbated the effect of family functioning on youths' internalizing and externalizing problems. Therefore, left amygdalar responses to emotional stimuli can serve as neurobiological sensitivity indicators for youth. Further, the moderating effects of left amygdalar activations on the links between family functioning and youth adjustment generally supported the biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, youth who had elevated amygdalar responses to emotional stimuli and reported dysfunctional family environments exhibited more developmental vulnerability. In contrast, with the same high amygdalar activations, youth also showed elevated developmental competence (reduced problem behaviors) in response to balanced and healthy caregiving environments.

These findings on amygdalar activations as neurobiological sensitivity indicators are aligned with amygdalar functions in emotional processing in response to both negative and positive stimuli (Hamann & Mao, 2002; LeDoux, 1998). The amygdala is involved in detecting negative or positive emotional stimuli induced by adverse or supportive caregiving experiences. Subsequently, the amygdala can facilitate youth to initiate behavioral reactions as responses to positive or negative emotional stimuli (Cahill, Babinsky, Markowitsch, & McGaugh, 1995; LeDoux, 1998; Weymar & Schwabe, 2016; Windle et al., 2018). Therefore, high levels of amygdalar activations under emotional processing are associated with heightened sensitivity to family rearing environments. In contrast, blunted amygdalar responses to emotional stimuli are linked to youths' reduced responsivity to family influences.

Hypothesis 3: The Hippocampal Activations as Moderators on the Associations Between Family Functioning and Youth Adjustment

In support of the third hypothesis, this study also found that left hippocampal responses to *positive* emotional stimuli significantly exacerbated the influences of family functioning on youths' externalizing problems. This significant moderating effect followed a biological sensitivity to context pattern (Boyce & Ellis, 2005; Ellis et al., 2005). This role of the left hippocampus as a neurobiological sensitivity indicator can be attributed to its functions in emotional regulation, especially in consolidating the emotional elements of explicit memories (Burgess et al., 2002). Heightened hippocampal activations during emotional stimuli may indicate youths' elevated emotional engagement during their interactions with primary caregivers. This heightened emotional engagement enables youth to initiate differential behavioral reactions in response to different family rearing environments (Burgess et al., 2002; Richter-Levin & Akirav, 2000). Specifically, youth with elevated hippocampal responses could develop more positive outcomes in response to a supportive rearing environment. However, with the same high hippocampal activations, youth who reported unbalanced family functioning were also at increased risk of developing problem behaviors.

Further, for a small proportion of adolescents with extremely high hippocampal deactivations (i.e., negative activation coefficient betas) during positive emotional processing, the dysfunctional family rearing environments were linked to youths' reduced externalizing problems. This finding reflects a "steeling" effect. The "steeling" effect indicates that experiences of moderate levels of early life stress promote youths' positive developmental outcomes via strengthening their resilience and resistance to future life stress (R. T. Liu, 2015; Repetti & Robles, 2016; Rutter, 2006). During emotional processing, hippocampal deactivations may indicate the suppression of the social-affective brain circuitry by the activations of the cognitive control system. The cognitive control system regulates youths' development of self-regulation abilities and executive functioning. Adequate levels of executive functioning are critical for developing resilience and "steeling" effect when adolescents were exposed to early adversity (Davidovich et al., 2016; Finch & Obradović, 2017).

Hippocampal models did not support the role of left hippocampal responses to *negative* emotional stimuli as neurobiological sensitivity indicators. Specifically, the moderating effect of left hippocampal activations during negative emotional processing on the associations between family functioning on youth adjustment was statistically non-significant. This null result may be due to the small magnitude of activations on the left hippocampus during negative emotional processing. As presented in Figure 4.3, the amygdala exhibited statistically stronger group-level activations in response to negative stimuli compared to the hippocampus. The lower levels of activations on the left hippocampus during responses to negative facial expressions may conceal the possible neurobiological sensitivity to context patterns.

Hypothesis 4: Lateralization of Amygdalar and Hippocampal Activations During Emotional Processing

The results of the current study did not find statistically significant lateralization in amygdalar and hippocampal activations in response to negative or positive emotional stimuli. This result is aligned with findings from two meta-analyses studies, which suggest the brain hemisphere differences in the effect sizes of amygdalar and hippocampal activations during emotional processing to be statistically small and non-significant (Fusar-Poli et al., 2009; Sergerie et al., 2008). Further, the current study supported hypothesis 4b by showing that only the left amygdala and hippocampus significantly exacerbated the influence of family functioning on youth adjustment. Therefore, the neurobiological sensitivity indicators were found to be lateralized to the left brain hemisphere. For amygdalar functions, the left hemisphere is more engaged in detailed analyses and evaluation of emotional information. In contrast, the right amygdala is responsible for immediate and autonomic stimuli detection (Gläscher & Adolphs, 2003; Markowitsch, 1999; Wright et al., 2003). It is possible that left amygdalar functions are more engaged when youth detect threats or advantageous elements from their rearing environments and initiate corresponding behavioral reactions. This temporal dynamic in amygdalar activations enables the left amygdala to be a more salient neurobiological sensitivity indicator, which exhibited stronger moderating effects on the associations between family functioning and youth adjustment, compared to the right amygdala (Cahill, 2003; Canli et al., 2000).

For the hippocampus, ample empirical evidence documents that the associations among hippocampal functions, early life experiences, and the development of psychopathology are specific to the left brain hemisphere (MacMaster & Kusumakar, 2004; Schriber et al., 2017; Vythilingam et al., 2002; Whittle et al., 2013). The left hippocampus may be more reactive to early rearing environments because of its higher concentration and expression of corticosteroid receptors and increased engagement in HPA-axis regulation compared to the right hippocampus (Hou et al., 2013; Madsen et al., 2012). Therefore, in response to emotional stimuli, left hippocampal activations may be a more salient neurobiological sensitivity indicator, which showed stronger moderating effects on the associations between family functioning and youth adjustment, compared to the right hippocampus.

Limitations

There were several limitations to the current study. First, this study targeted families in rural areas of the Southeastern U.S., which might limit the generalizability of the findings to youth from urban areas, other regions of the U.S., and other countries. However, research on neurobiological sensitivity hypotheses among rural youth is scarce, despite the fact that rural adolescents are at high risk for developing psychopathology and risk behaviors (Evans, Vermeylen, Barash, Lefkowitz, & Hutt, 2009). Second, the data employed by this study are cross-sectional. Thus, the temporal precedence among family functioning, youth adjustment, and neural mechanisms could not be determined. However, the DORRY study is a 5-year longitudinal study with on-going data collection, and two more time-points of data on youth behaviors and neural mechanisms (via MRI and fMRI) will be obtained. Once the data collection on the second time-point is completed, analyses will be re-conducted to confirm the study findings. Additionally, the internal reliability of the enmeshed subscale in the FACES-IV package was ($\alpha = .58$), which might limit the reliability of the cohesion dimension of family functioning. Lastly, the modest sample size ($N = 123$) limited the power to test the study hypotheses. Despite these limitations, this study included significant methodological strengths. These strengths included the use of a detailed measurement tool (i.e., FACES IV; Olson, Gorall, & Tiesel, 2004) to capture the full spectrum and dimensionality of family functioning, using fMRI to assess adolescents' neural mechanisms in response to emotional stimuli, and the sampling of understudied populations.

Table 4.1 *Regions of Interests (ROIs) Coordinates*

ROI	Sphere	Center of Mass Coordinates		
		X	Y	Z
Left Amygdala	5	23	5	-15
Right Amygdala	5	-23	5	-15
Left Hippocampus	5	30	24	-9
Right Hippocampus	5	-30	24	-9

Note. Coordinates are defined in Talairach space (RAI).

Table 4.2. *Summary of Participants' Emotional N-back Task Performance*

	Mean	*SD*	Min.	25%	Med.	75%	Max.
Total	89.81%	6.43%	65.26%	86.22%	91.25%	94.36%	100.00%
Total 2-back	87.01%	8.16%	60.00%	82.65%	89.31%	92.50%	100.00%
Total 0-back	92.78%	6.44%	63.75%	90.00%	94.94%	96.25%	100.00%
Positive emotions	89.40%	8.11%	65.00%	85.00%	92.50%	95.00%	100.00%
Positive emotions 2-back	85.81%	11.67%	45.00%	80.00%	90.00%	95.00%	100.00%
Positive emotions 0-back	93.21%	8.38%	65.00%	90.00%	95.00%	100.00%	100.00%
Negative emotions	90.22%	7.21%	65.00%	87.50%	92.50%	95.00%	100.00%
Negative emotions 2-back	86.91%	10.22%	45.00%	80.00%	90.00%	95.00%	100.00%
Negative emotions 0-back	93.73%	7.85%	60.00%	90.00%	95.00%	100.00%	100.00%
Neutral emotions	91.34%	8.80%	57.50%	90.00%	95.00%	97.50%	100.00%
Neutral emotions 2-back	89.26%	10.45%	60.00%	85.00%	92.50%	95.00%	100.00%
Neutral emotions 0-back	93.58%	10.65%	50.00%	95.00%	95.00%	100.00%	100.00%
Non-face Place	86.19%	9.16%	42.50%	82.50%	87.50%	92.50%	100.00%
Non-face Place 2-back	84.04%	12.13%	40.00%	80.00%	85.00%	93.75%	100.00%
Non-face Place 0-back	88.51%	11.48%	45.00%	85.00%	90.00%	100.00%	100.00%

Note. Youths' behavioral performance was reflected by their percentage of correct responses. *SD* = Standard deviation, Min. = Minimum, 25% = 25% percentile, Med. = Median, 75% = 75% percentile, Max. = Maximum.

Table 4.3 *Correlation and Descriptive Statistics of Study Variables (N = 123)*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. FACES Cohesion	--																
2. FACES Flexibility	$.52^{**}$	--															
3. FACES Communication	$.52^{**}$	$.53^{**}$	--														
4. Internalizing Problems	-.12	$-.25^{**}$	$-.36^{**}$	--													
5. Externalizing Problems	-.12	$-.30^{**}$	$-.35^{**}$	$.53^{**}$	--												
6. Left Amygdala-Negative	$-.26^{*}$	-.11	-.18	$.26^{*}$	$.27^{*}$	--											
7. Right Amygdala-Negative	-.18	-.08	-.13	.21	.19	$.92^{**}$	--										
8. Left Hippocampus-Negative	-.10	.00	-.08	.11	.15	$.73^{**}$	$.78^{**}$	--									
9. Right Hippocampus-Negative	-.08	-.11	-.18	$.24^{*}$	$.35^{**}$	$.61^{**}$	$.66^{**}$	$.58^{**}$	--								
10. Left Amygdala-Positive	-.14	-.04	-.02	.20	$.32^{**}$	$.66^{**}$	$.51^{**}$	$.35^{**}$	$.36^{**}$	--							
11. Right Amygdala-Positive	-.07	-.01	.01	.07	.16	$.68^{**}$	$.66^{**}$	$.48^{**}$	$.35^{**}$	$.81^{**}$	--						
12. Left Hippocampus-Positive	-.04	.04	.00	.23	.17	$.55^{**}$	$.50^{**}$	$.54^{**}$	$.34^{**}$	$.78^{**}$	$.75^{**}$	--					
13. Right Hippocampus-Positive	.00	.00	-.05	.21	.23	$.38^{**}$	$.34^{**}$	.08	$.47^{**}$	$.71^{**}$	$.64^{**}$	$.59^{**}$	--				
14. Youth biological sex	-.05	.03	-.07	-.03	-.13	-.09	-.10	-.03	-.17	.03	-.03	-.06	-.09	--			
15. Household Income	$.24^{*}$	-.01	.02	.05	.02	-.19	$-.26^{*}$	-.18	-.18	-.14	-.22	-.10	-.12	-.10	--		
16. Parental Education	.17	.04	.06	.02	-.08	-.19	-.17	-.13	-.16	-.18	-.05	-.04	-.04	-.13	$.51^{**}$	--	
17. Parental Employment	.06	.09	.02	.05	.02	.03	-.01	.01	.00	-.01	.03	-.08	.01	-.10	.05	$.34^{**}$	--
Mean	4.27	2.27	70.85	5.25	5.39	2.34	2.90	1.09	1.35	.59	1.50	1.02	.88	.54	65.57	4.63	.59
SD	1.45	.72	18.62	5.69	5.48	7.24	7.64	5.21	5.14	7.44	7.03	4.78	5.43	.50	30.77	1.11	.81
Minimum	1.19	.69	14.00	.00	.00	-27.74	-22.29	-14.02	-13.52	-23.46	-14.07	-10.42	-10.76	.00	7.50	2.00	-1.00
Maximum	7.64	4.52	97.00	30.00	32.00	27.73	25.37	15.96	17.27	30.46	25.11	16.49	22.48	1.00	100.00	6.00	1.00
Skewness	.14	.99	-.84	1.74	1.81	.05	.43	.08	.31	.99	1.34	.65	1.31	-.18	-.29	-.32	-1.48
Kurtosis	-.85	1.36	.40	3.69	4.64	6.46	2.93	1.32	1.88	5.21	3.40	1.64	3.69	-2.00	-1.40	-.71	.19

Note. Negative = Response to negative emotional stimuli; Positive = Response to positive emotional stimuli; *SD* = Standard deviation. Youth biological sex was coded as 0 = males and 1 = females; Household income was coded into $1,000; Parental employment was coded into binary variable (-1 for unemployed and 1 for employed). *$p < .05$, **$p < .01$.

Table 4.4. *Cluster Size and Coordinates of Neural Activations*

Region	#Voxels	X	Y	Z	Direction
Negative vs. Neutral Contrast					
Primary Visual Cortex	2498	-1.2	76.1	-3.2	Activation
Right Inferior Parietal Lobule	471	-46.2	47.1	41.7	Deactivation
Right Middle Frontal Gyrus	307	-32.5	-23.9	39.8	Deactivation
Left Inferior Parietal Lobule	99	47.3	49.8	46.6	Deactivation
Left Middle Frontal Gyrus	98	35.0	-31.8	32.4	Deactivation
Right Superior Temporal Gyrus	95	-44.5	-12.1	-20.7	Activation
Left Precentral Gyrus	52	24.7	4.0	56.6	Deactivation
Right Middle Temporal Gyrus	51	-44.8	44.1	4.6	Activation
Right Amygdala	35	-26.5	6.5	-13.8	Activation
Left Cingulate Gyrus	30	-0.5	28.9	27.8	Deactivation
Left Inferior Frontal Gyrus	26	28.6	-12.9	-18.1	Activation
Right Precentral Gyrus	17	-53.4	-2.7	11.8	Deactivation
Left Amygdala	16	33.7	10.2	-14.3	Activation
Right Superior Parietal Lobule	16	-18.2	63.1	56.7	Deactivation
Positive vs. Neutral Contrast					
Right Middle Frontal Gyrus & Right Inferior Parietal Lobule	1971	-33.5	28.9	39.2	Deactivation
Right Lingual Gyrus	653	-24.9	70.3	-2.7	Activation
Left Middle Frontal Gyrus	588	33.3	-7.9	43.8	Deactivation
Left Supramarginal Gyrus	489	45.1	51.8	33.6	Deactivation
Left Lingual Gyrus	463	25.6	72.4	-1.2	Activation
Right Superior Frontal Gyrus	57	-25.0	-54.0	10.4	Deactivation
Left Superior Frontal Gyrus	41	27.6	-51.9	7.6	Deactivation
Right Posterior Cingulate	15	-15.9	48.2	11.7	Activation
Negative vs. Positive Contrast					
Declive & Fusiform Gyrus	2331	33.2	74.5	-18.0	Activation
Left Inferior Frontal Gyrus	153	43.8	-16.5	-7.5	Activation
Right Inferior Frontal Gyrus	153	-50.8	-20.0	24.0	Activation
Left Precuneus	42	1.8	57.0	48.5	Activation
Left Middle Frontal Gyrus	30	43.8	-16.5	27.5	Activation
Left Superior Frontal Gyrus	28	1.8	-9.5	55.5	Activation
Right Precuneus	17	-22.8	57.0	52.0	Activation

Note. #Voxels = Number of voxels. A minimum cluster size of 15 adjacent voxels and a family-wise error-corrected rate of $p < .05$ were used to allow for the separation of distinct clusters into individual ROIs. Brain regions were defined in the Talairach-Daemon atlas.

Table 4.5. *Paired-Sample t-tests for the Mean Valence (Positive vs. Negative) Differences of Amygdalar and Hippocampal Responses to Emotional Stimuli*

Brain Hemisphere	ROI	Positive		Negative		$M_{\text{difference}}$	95% CI of $M_{\text{difference}}$	t	df	Cohen's d	p-value
		M	SD	M	SD						
Left	Amygdala	.591	7.440	2.337	7.238	1.746	[.273, .320]*	2.366	67	.297	.021
	Hippocampus	1.019	4.778	1.087	5.207	.068	[-1.093, 1.229]	.118	67	.016	.907
Right	Amygdala	1.493	7.039	2.898	7.636	1.402	[-.079, 2.883]	1.890	67	.231	.063
	Hippocampus	.884	5.430	1.351	5.141	.466	[-.853, 1.787]	.706	67	.086	.483

Note. ROI = Region of interests; M = Mean; SD = Standard Deviation; $M_{\text{difference}}$ = Mean difference; CI = Confidence interval; df = Degree of freedom. The unit of valence differences presented here is standardized activation coefficient β. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 4.6. *The Direct Effects Model*

Effects	*B (SE)*	β	95%*CI of B*	*p*-value
Direct Effects				
Family Functioning → INT	-2.005 (.677)	-.307	[-3.331, -.679]**	.003
Family Functioning → EXT	-1.986 (.452)	-.316	[-2.871, -1.100]***	<.001
Control				
Youth Sex → INT	-.232 (.971)	-.020	[-2.136, 1.672]	.811
Youth Sex → EXT	-1.515 (.917)	-.138	[-3.313, .283]	.099
SES → INT	.263 (.265)	.093	[-.256, .782]	.321
SES → EXT	-.085 (.276)	-.031	[-.627, .457]	.758

Note. EXT = Externalizing problems; INT = Internalizing problems; SES = Socioeconomic status, which was computed as a composite score of household income, parental education, and parental employment status. Youth biological sex was coded as 0 = male and 1 = female. Model fit is excellent: $\chi^2(2) = 3.768$ ($p = .927$); CFI = .958; TLI = .855; RMSEA = .085; SRMR = .040.

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

Table 4.7. *The Moderation Roles of Amygdalar Response to Negative Emotional Stimuli on the Links Between Family Functioning and Youth Adjustment*

Effects	Left Amygdala Model				Right Amygdala Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Externalizing Problems Model								
Direct Effects								
Family Functioning → EXT	-1.508 (.517)	-.240	[-2.522, -.495]**	.004	-1.835 (.524)	-.292	[-2.863, -.807]***	<.001
Amygdala Act. → EXT	.121 (.082)	.162	[-.040, .282]	.141	.095 (.093)	.132	[-.086, .277]	.304
Interaction Effects								
Family Functioning x Amygdala Act. → EXT	-.284 (.131)	-.292	[-.541, -.028]*	.030	-.171 (.122)	-.182	[-.410, .067]	.159
Control								
Youth Sex → EXT	-1.358 (.880)	-.124	[-3.082, .366]	.123	-1.211 (.930)	-.110	[-3.034, .612]	.193
SES → EXT	-.018 (.255)	-.006	[-.518, .482]	.945	.017 (.288)	.006	[-.548, .581]	.954
Model Fit Indices	$\chi^2(4) = 4.436$ ($p = .963$); CFI = .976; TLI = .970; RMSEA = .030; SRMR = .040				$\chi^2(0) = .000$ ($p = 1.000$); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000			
Internalizing Problems Model								
Direct Effects								
Family Functioning → INT	-1.535 (.626)	-.235	[-2.762, -.308]*	.014	-1.837 (.733)	-.282	[-3.273, -.400]*	.012
Amygdala Act. → INT	.118 (.073)	.150	[-.026, .261]	.107	.146 (.077)	.195	[-.006, .297]	.060
Interaction Effects								
Family Functioning x Amygdala Act. → INT	-.246 (.123)	-.239	[-.487, -.005]*	.046	-.110 (.094)	-.112	[-.294, .073]	.239
Control								
Youth Sex → INT	-.257 (.953)	-.023	[-2.125, 1.611]	.787	.092 (.976)	.008	[-1.821, 2.004]	.925
SES → INT	.009 (.016)	.049	[-.022, .040]	.561	.385 (.255)	.137	[-.115, .885]	.131
Model Fit Indices	$\chi^2(4) = 1.694$ ($p = .792$); CFI = 1.000; TLI = 1.178; RMSEA = .000; SRMR = .027				$\chi^2(0) = .000$ ($p = 1.000$); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000			

Note. Act. = Activation (to negative emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems. Youth sex was coded as 0 = male and 1 = female. **p* < .05, ***p* < .01, *** *p* < .001.

Table 4.8. *The Moderation Roles of Amygdalar Response to Positive Emotional Stimuli on the Links Between Family Functioning and Youth Adjustment*

Effects	Left Amygdala Model				Right Amygdala Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
<u>Externalizing Problems Model</u>								
Direct Effects								
Family Functioning → EXT	-1.723 (.483)	-.274	[-2.669, -.777]***	<.001	-2.003 (.514)	-.319	[-3.010, -.996]***	<.001
Amygdala Act. → EXT	.179 (.083)	.251	[.017, .342]*	.031	.101 (.091)	.130	[-.077, .278]	.267
Interaction Effects								
Family Functioning x Amygdala Act. → EXT	-.364 (.143)	-.333	[-.633, -.085]*	.011	-.238 (.125)	-.226	[-.484, .008]	.058
Control								
Youth Sex → EXT	-1.477 (.857)	-.135	[-3.156, .202]	.085	-1.074 (.927)	-.098	[-2.891, .744]	.247
SES → EXT	-.012 (.236)	-.004	[-.474, .451]	.961	-.015 (.271)	-.005	[-.546, .516]	.957
Model Fit Indices	$\chi^2(4) = 3.739$ (p = .910); CFI = 1.000; TLI = 1.014; RMSEA = .000; SRMR = .036				$\chi^2(0) = .000$ (p = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000			
<u>Internalizing Problems Model</u>								
Direct Effects								
Family Functioning → INT	-1.870 (.644)	-.287	[-3.133, -.608]**	.004	-2.029 (.687)	-.311	[-3.375, -.684]**	.003
Amygdala Act. → INT	.080 (.065)	.105	[-.046, .207]	.215	.030 (.080)	.037	[-.126, .186]	.708
Interaction Effects								
Family Functioning x Amygdala Act. → INT	-.388 (.146)	-.331	[-.674, -.102]**	.008	-.163 (.099)	-.147	[-.358, .032]	.102
Control								
Youth Sex → INT	-.259 (.935)	-.023	[-2.092, 1.575]	.782	.039 (.969)	.003	[-1.860, 1.937]	.968
SES → INT	.268 (.233)	.095	[-.189, .725]	.250	.293 (.259)	.104	[-.215, .801]	.259
Model Fit Indices	$\chi^2(4) = 3.597$ (p = .983); CFI = 1.000; TLI = 1.027; RMSEA = .000; SRMR = .036				$\chi^2(0) = .000$ (p = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000			

Note. Act. = Activation (to negative emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems. Youth sex was coded as 0 = male and 0 = female. *$p < .05$, **$p < .01$, *** $p < .001$.

Table 4.9. *The Moderation Roles of Hippocampal Response to Negative Emotional Stimuli on the Links Between Family Functioning and Youth Adjustment*

Effects	Left Hippocampal Model				Right Hippocampal Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Externalizing Problems Model								
Direct Effects								
Family Functioning → EXT	-1.697 (.646)	-.270	[-2.962, -.431]**	.009	-1.621 (.583)	-.258	[-2.765, -.478]**	.005
Hippocampal Act. → EXT	.175 (.146)	.166	[-.111, .460]	.230	.369 (.128)	.357	[.119, .620]**	.004
Interaction Effects								
Family Functioning x Hippocampal Act. → EXT	-.298 (.186)	-.252	[-.663, .067]	.110	-.200 (.170)	-.155	[-.533, .134]	.240
Control								
Youth Sex → EXT	-1.398 (.884)	.128	[-3.132, .335]	.114	-.685 (.837)	-.063	[-2.325, .955]	.413
SES → EXT	.060 (.277)	.022	[-.483, .603]	.830	.039 (.252)	.014	[-.455, .532]	.878
Model Fit Indices	χ^2(4) = 4.695 (*p* = .829); CFI = .942; TLI = .928; RMSEA = .038; SRMR = .037				χ^2(0) = .000 (*p* = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000			
Internalizing Problems Model								
Direct Effects								
Family Functioning → INT	-2.059 (.760)	-.316	[-3.548, -.579]**	.007	-1.796 (.779)	-.276	[-3.322, -.270]*	.021
Hippocampal Act. → INT	.102 (.138)	.093	[-.169, .373]	.461	.257 (.125)	.232	[.012, .502]*	.040
Interaction Effects								
Family Functioning x Hippocampal Act. → INT	.107 (.172)	.086	[-.229, .444]	.532	-.121 (.159)	-.090	[-.434, .191]	.446
Control								
Youth Sex → INT	-.259 (.964)	-.023	[-2.149, 1.631]	.788	.302 (.978)	.027	[-1.615, 2.219]	.758
SES → INT	.278 (.265)	.099	[-.241, .797]	.293	.358 (.247)	.127	[-.125, .842]	.146
Model Fit Indices	χ^2(0) = .000 (*p* = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000				χ^2(0) = .000 (*p* = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000			

Note. Act. = Activation (to negative emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems. Youth sex was coded as 0 = male and 1 = female. **p* < .05, ***p* < .01, *** *p* < .001.

Table 4.10. *The Moderation Roles of Hippocampal Response to Positive Emotional Stimuli on the Links Between Family Functioning and Youth Adjustment*

Effects	Left Hippocampal Model				Right Hippocampal Model			
	B (SE)	β	95%*CI of B*	*p*-value	*B (SE)*	β	95%*CI of B*	*p*-value
Externalizing Problems Model								
Direct Effects								
Family Functioning → EXT	-1.615 (.541)	-.257	[-2.675, -.555]**	.003	-2.008 (.497)	-.320	[-2.982, -1.035]***	<.001
Hippocampal Act. → EXT	.177 (.098)	.156	[-.015, .370]	.071	.262 (.123)	.264	[.020, .504]*	.034
Interaction Effects								
Family Functioning x Hippocampal Act. → EXT	-.613 (.187)	-.397	[-.981, -.246]**	.001	-.057 (.191)	-.041	[-.431, .318]	.767
Control								
Youth Sex → EXT	-1.340 (.844)	-.122	[-2.993, .313]	.112	-1.313 (.888)	-.120	[-3.053, .428]	.139
SES → EXT	.001 (.238)	.000	[-.466, .468]	.997	-.038 (.275)	-.014	[-.578, .501]	.889
Model Fit Indices	$\chi^2(4) = 4.439$ (*p* = .881); CFI = .979; TLI = .974; RMSEA = .030; SRMR = .038				$\chi^2(4) = 4.794$ (*p* = .901); CFI = .938; TLI = .922; RMSEA = .040; SRMR = .044			
Internalizing Problems Model								
Direct Effects								
Family Functioning → INT	-1.908 (.706)	-.293	[-3.291, -.524]**	.007	-2.027 (.714)	-.311	[-3.428, -.627]**	.005
Hippocampal Act. → INT	.307 (.123)	.260	[.066, .548]*	.012	.219 (.104)	.210	[.015, .424]*	.036
Interaction Effects								
Family Functioning x Hippocampal Act. → INT	-.279 (.181)	-.167	[-.634, .075]	.123	-.148 (.155)	-.104	[-.452, .156]	.339
Control								
Youth Sex → INT	-.138 (.943)	-.012	[-1.986, 1.711]	.884	-.063 (.952)	-.006	[-1.930, 1.804]	.947
SES → INT	.346 (.253)	.123	[-.150, .842]	.172	.260 (.270)	.092	[-.269, .788]	.336
Model Fit Indices	$\chi^2(4) = 4.236$ (*p* = .888); CFI = .983; TLI = .978; RMSEA = .022; SRMR = .037				$\chi^2(4) = 4.667$ (*p* = .893); CFI = .941; TLI = .926; RMSEA = .037; SRMR = .043			

Note. Act. = Activation (to negative emotional stimuli); EXT = Externalizing problems; INT = Internalizing problems. Youth sex was coded as 0 = male and 1 = female. **p* < .05, ***p* < .01, *** *p* < .001.

Table 4.11. *Paired-Sample t-tests for the Lateralization of Amygdalar and Hippocampal Responses to Negative and Positive Emotional Stimuli*

Emotional Stimuli	ROI	Left		Right		$M_{\text{difference}}$	95% *CI* of $M_{\text{difference}}$	*t*	*df*	Cohen's *d*	*p*-value
		M	*SD*	*M*	*SD*						
Negative	Hippocampus	1.088	5.208	1.351	5.141	-.263	[-1.413, .887]	-.456	67	.055	.650
	Amygdala	2.338	7.238	2.898	7.637	-.561	[-1.310, .189]	-1.493	67	.181	.140
Positive	Hippocampus	1.019	4.778	.884	5.430	.136	[-.986, 1.257]	.241	67	.029	.810
	Amygdala	.591	7.440	1.496	7.029	-.905	[-1.979, .169]	-1.682	67	.204	.097

Note. ROI = Region of interests; *M* = Mean; *SD* = Standard Deviation; $M_{\text{difference}}$ = Mean difference; *CI* = Confidence interval; *df* = Degree of freedom. The unit of hemisphere differences presented here is standardized activation coefficient β. $^*p < .05$, $^{**}p < .01$, $^{***}p < .001$.

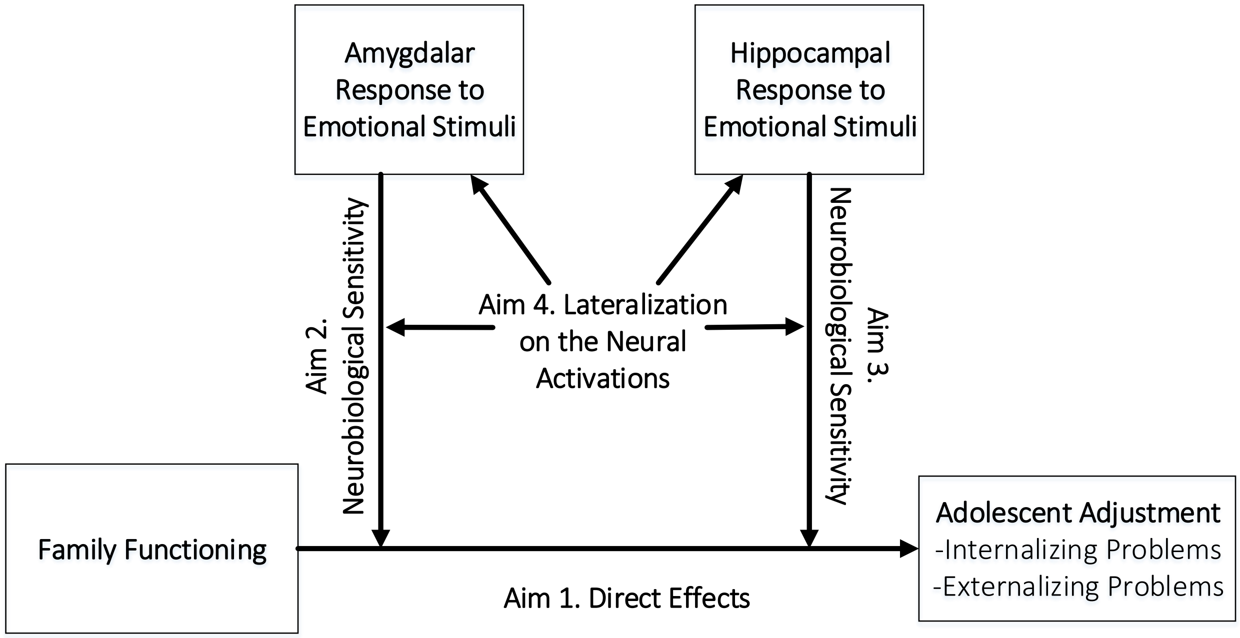

Figure 4.1. Summary of Aims and Hypotheses of the Current Study.

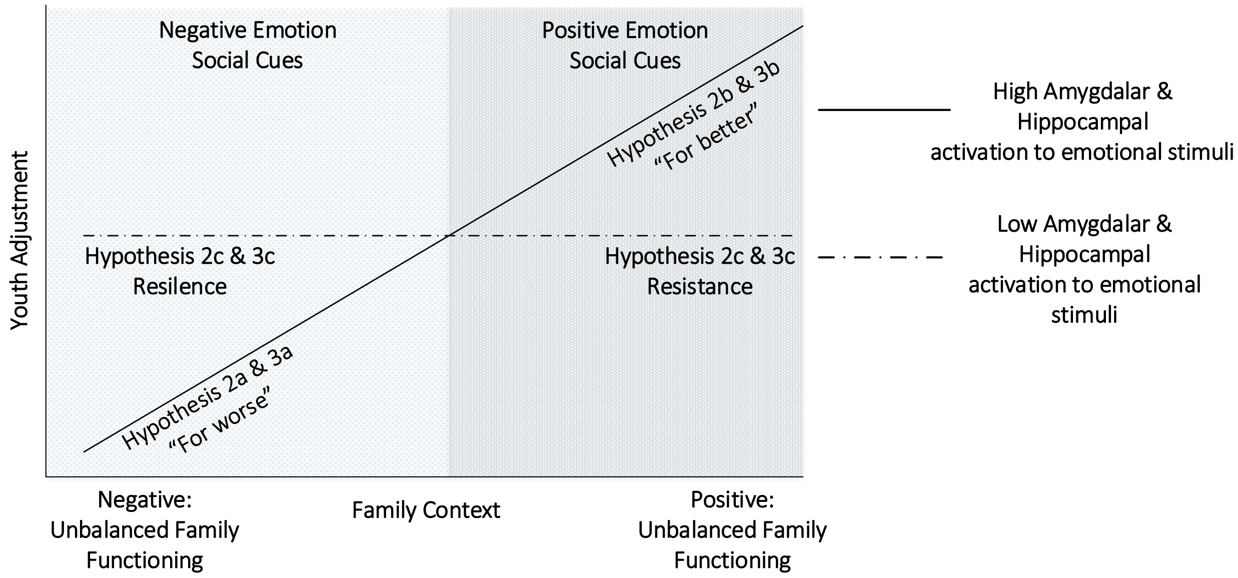

Figure 4.2. Elucidation of Neurobiological Sensitivity Hypotheses on the Moderating Effects of Amygdalar and Hippocampal Activations to Emotional Stimuli on the Associations Between Family Functioning and Youth Adjustment.

Note. The solid line indicates youth with high amygdalar and hippocampal activation during emotional processing who will exhibit a "for better and for worse" pattern under the influences of family environments. The dash-dot line represents youth with low amygdalar and hippocampal activation, who will be resilient to the harmful influences of unbalanced family functioning and resistant to the positive influences of balanced family functioning.

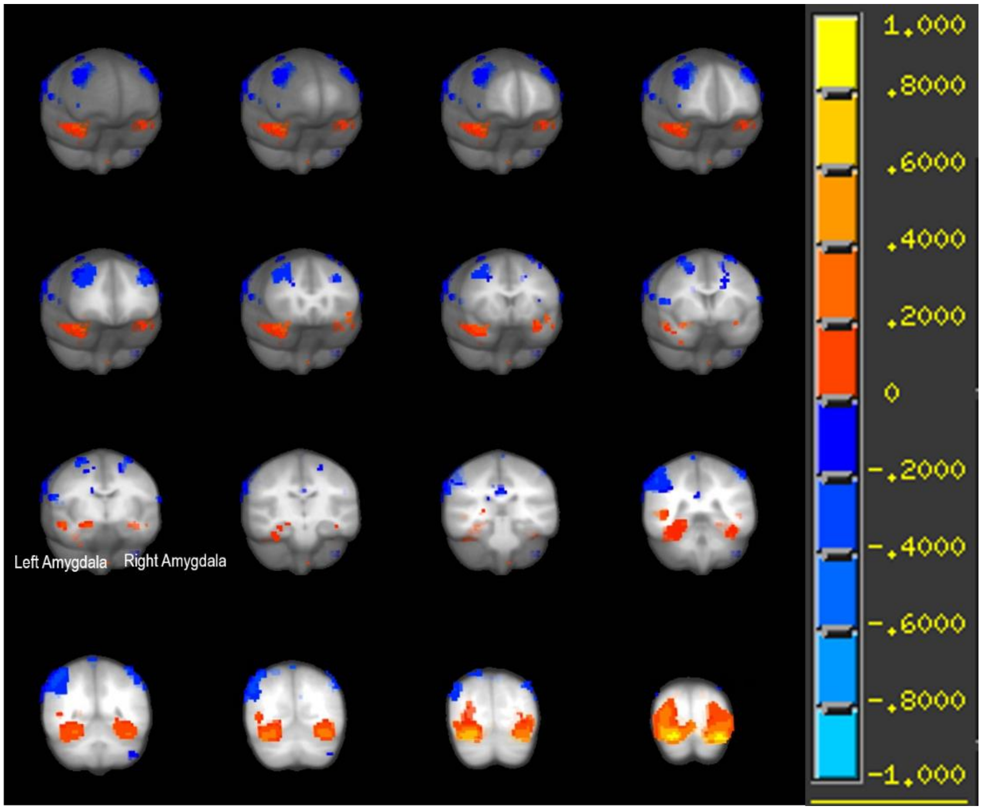

Figure 4.3. Neural Activity Associated with Negative vs. Neutral Stimuli in the Emotional N-Back Task.

Note. This figure presents the group-level activations with negative vs. neutral contrast during the emotional N-back task. Coronal slices show three-dimensional brain regions that exhibited significant activity during the negative emotional stimuli above and beyond that exhibited during the neutral emotional stimuli. Talairach Y-plan: -75 to +85 in 19mm slices. The scale of activations is presented in percent signal change (maximum = 1, minimum = -1). A minimum cluster size of 15 adjacent voxels and a family-wise error-corrected rate of $p < .05$ were used to allow for the separation of distinct clusters into individual ROIs. Underlying anatomical image of the average of 68 participants were used for this figure. Warm colors (i.e., red, orange, and yellow) represent neural activations, while cold colors (i.e., blue) represent deactivations.

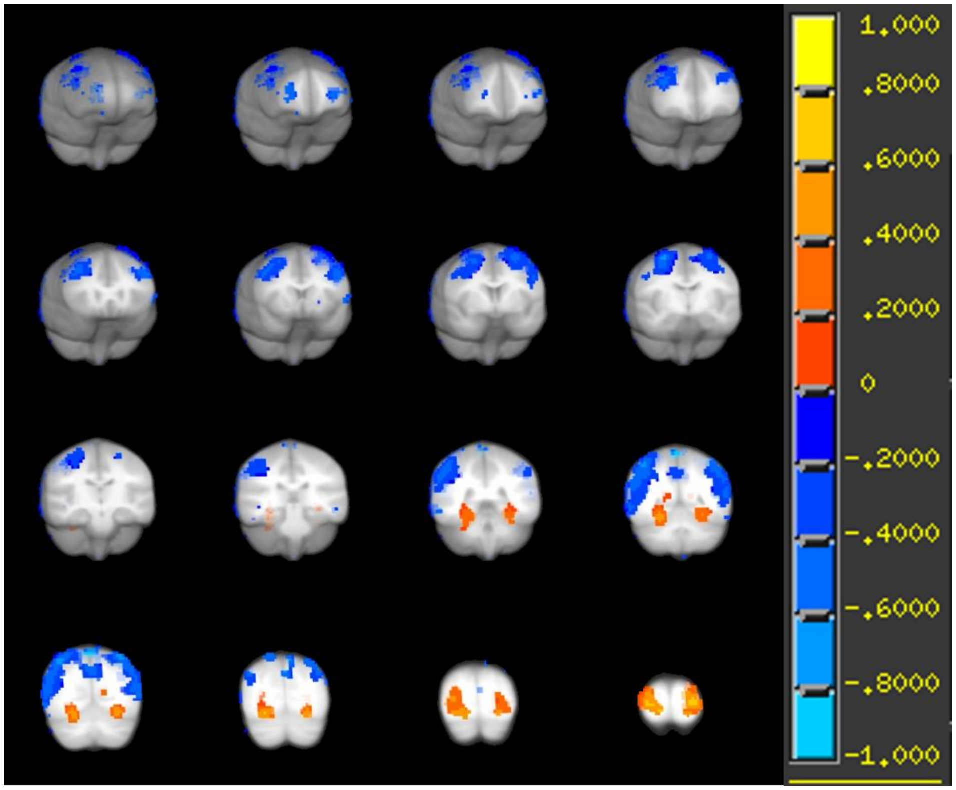

Figure 4.4. Neural Activity Associated with Positive vs. Neutral Stimuli in the Emotional N-Back Task.

Note. This figure presents the group-level activations with positive vs. neutral contrast during the emotional N-back task. Coronal slices show three-dimensional brain regions that exhibited significant activity during the positive emotional stimuli above and beyond that exhibited during the neutral emotional stimuli. Talairach Y-plan: -75 to +85 in 19mm slices. The scale of activations is presented in percent signal change (maximum = 1, minimum = -1). A minimum cluster size of 15 adjacent voxels and a family-wise error-corrected rate of $p < .05$ were used to allow for the separation of distinct clusters into individual ROIs. Underlying anatomical image of the average of 68 participants were used for this figure. Warm colors (i.e., red, orange, and yellow) represent neural activations, while cold colors (i.e., blue) represent deactivations.

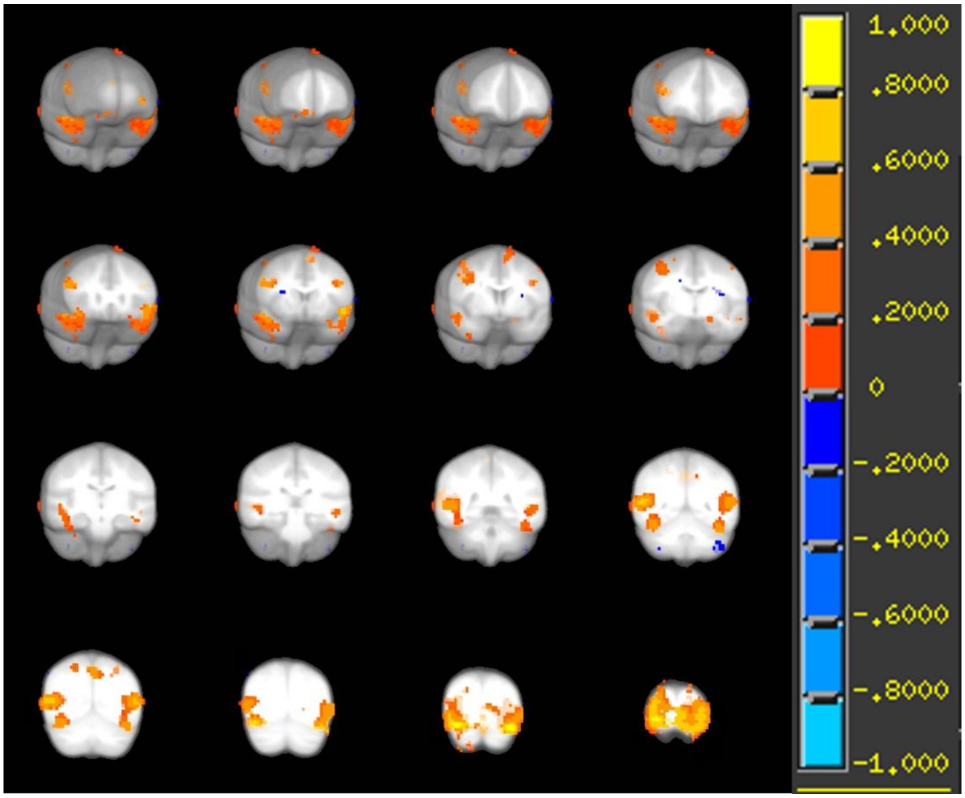

Figure 4.5. Neural Activity Associated with Negative vs. Positive Stimuli in the Emotional N-Back Task.

Note. This figure presents the group-level activations with negative vs. positive contrast during the emotional N-back task. Coronal slices show three-dimensional brain regions that exhibited significant activity during the negative emotional stimuli above and beyond that exhibited during the positive emotional stimuli. Talairach Y-plan: -75 to +85 in 19mm slices. The scale of activations is presented in percent signal change (maximum = 1, minimum = -1). A minimum cluster size of 15 adjacent voxels and a family-wise error-corrected rate of $p < .05$ were used to allow for the separation of distinct clusters into individual ROIs. Underlying anatomical image of the average of 68 participants were used for this figure. Warm colors (i.e., red, orange, and yellow) represent neural activations, while cold colors (i.e., blue) represent deactivations.

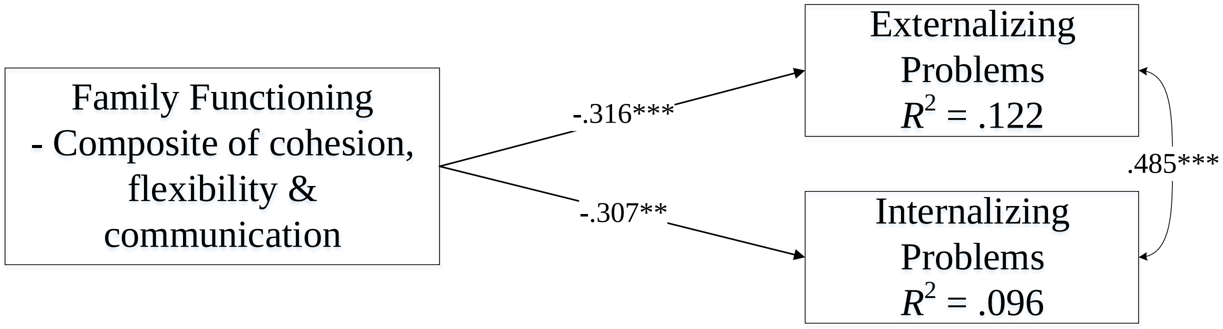

Figure 4.6. The Direct Effects of Family Functioning on Youth Externalizing and Internalizing Problems.

Note. R^2 = Proportion of variance explained in this model. Standardized coefficients are presented in this figure. This model controlled for youth biological sex and socioeconomic status. Covariates are omitted for figure clarity. Model fit was good: $\chi^2(2) = 3.768$ ($p = .927$); CFI = .958; TLI = .855; RMSEA = .085; SRMR = .040. *$p < .05$, **$p < .01$, *** $p < .001$.

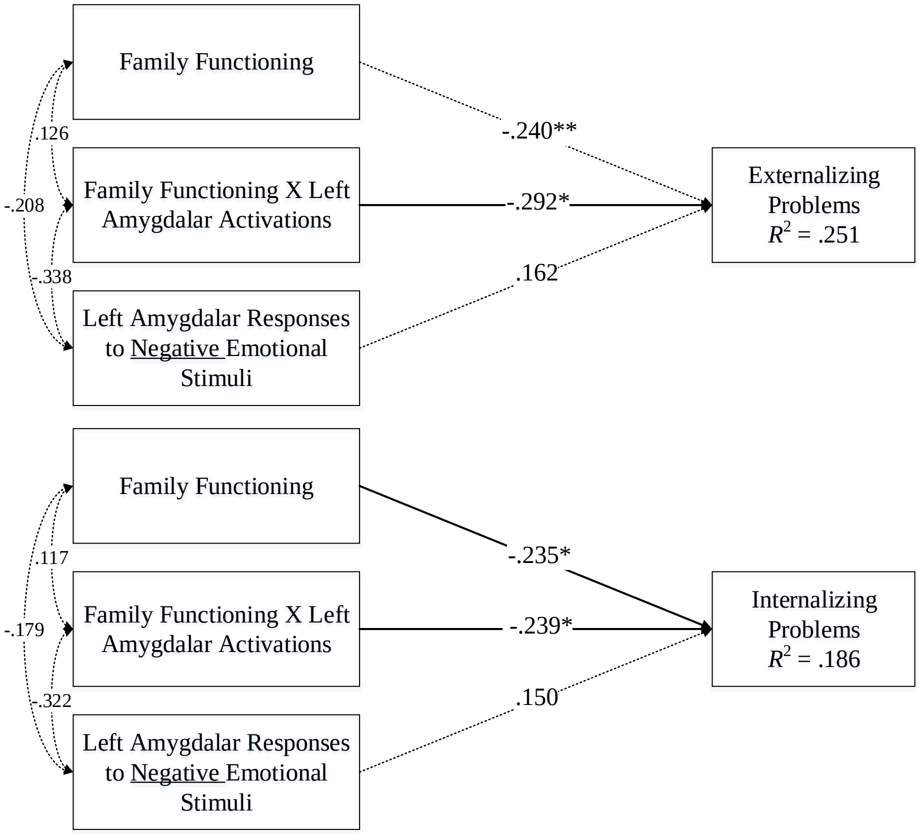

Figure 4.7. The Structural Equation Model of the Associations among Family Functioning, Left Amygdalar Responses to Negative Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit was good: Externalizing model: $\chi^2(4) = 4.436$ ($p = .963$); CFI = .976; TLI = .970; RMSEA = .030; SRMR = .040; Internalizing model: $\chi^2(4) = 1.694$ ($p = .792$); CFI = 1.000; TLI = 1.178; RMSEA = .000; SRMR = .027. *$p < .05$, **$p < .01$, *** $p < .001$.

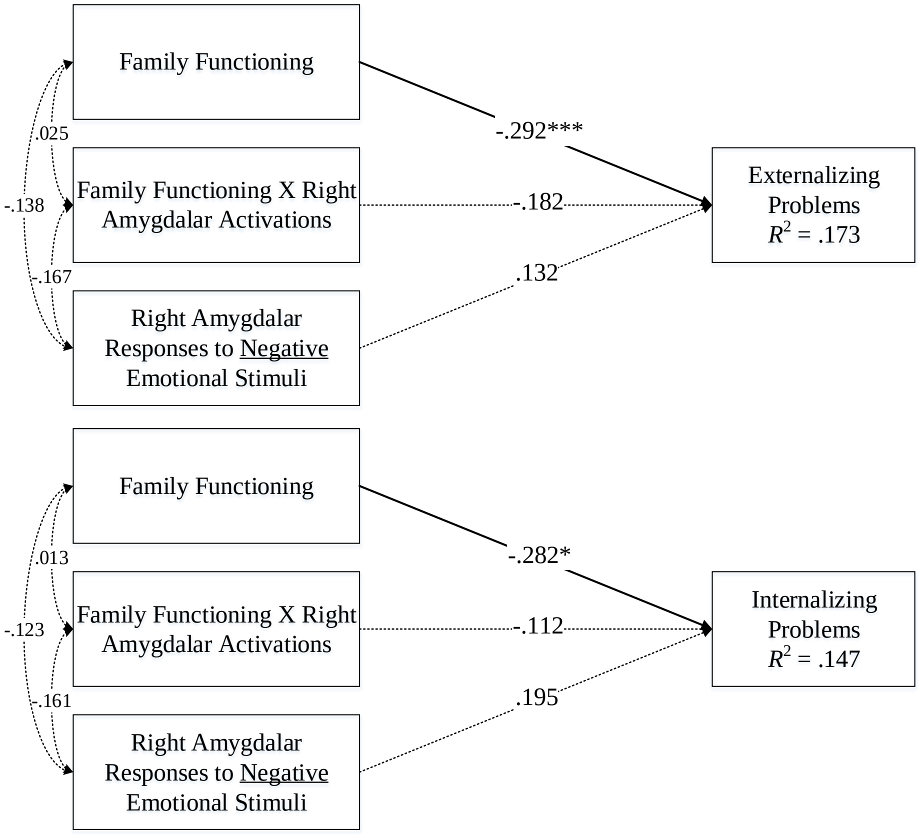

Figure 4.8. The Structural Equation Model of the Associations among Family Functioning, Right Amygdalar Responses to Negative Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Externalizing model: $\chi^2(0) = .000$ (p = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000; Internalizing model: $\chi^2(0)$ = .000 (p = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000. *$p < .05$, **$p < .01$, *** $p < .001$.

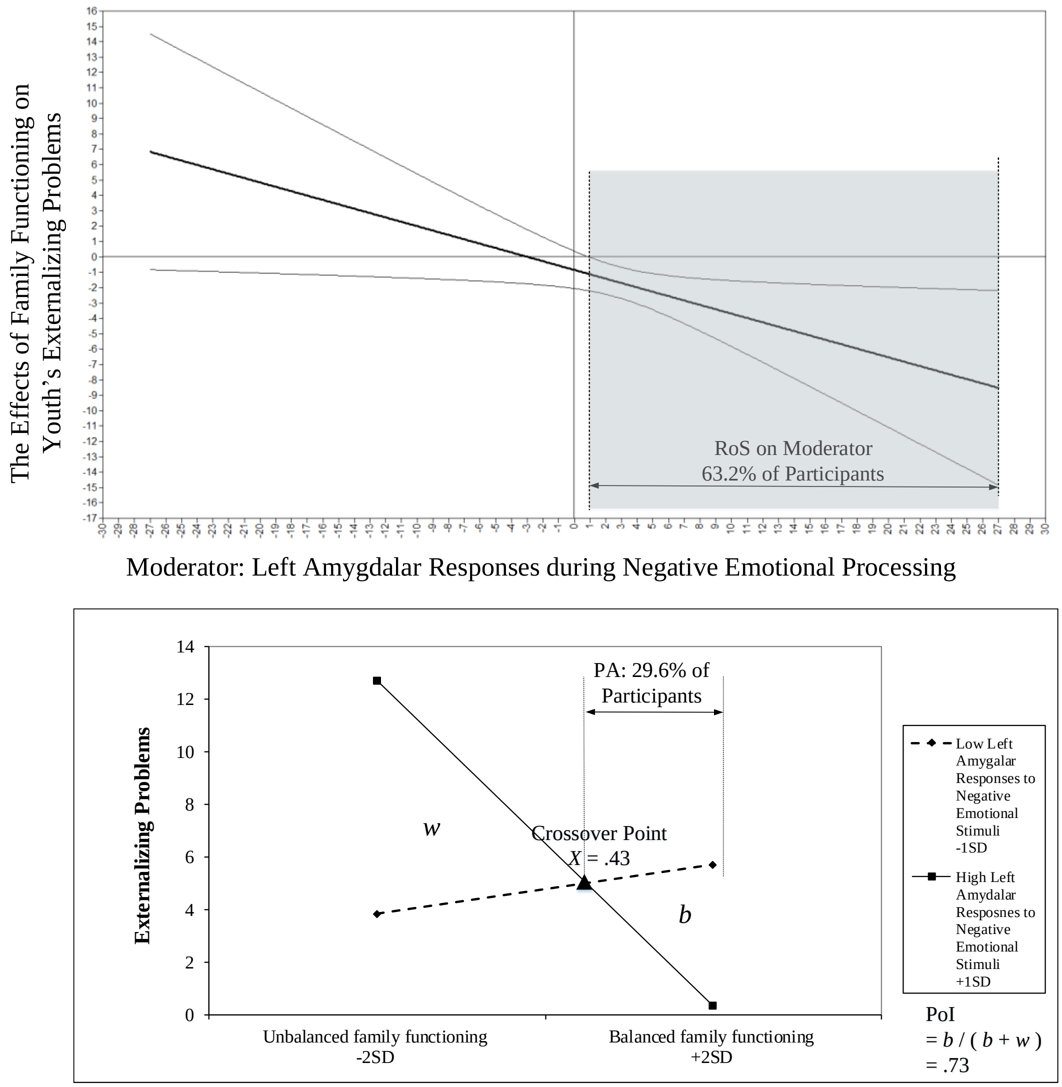

Figure 4.9. The Interpretation of the Significant Moderation Effect of Left Amygdalar Activations during Negative Emotional Stimuli on the Associations Between Family Functioning and Youth Externalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = "for better"; *w* = "for worse". The dashed line indicates youth with low left amygdalar responses during negative emotional stimuli, while the solid line indicates youth with high left amygdalar responses during negative emotional stimuli.

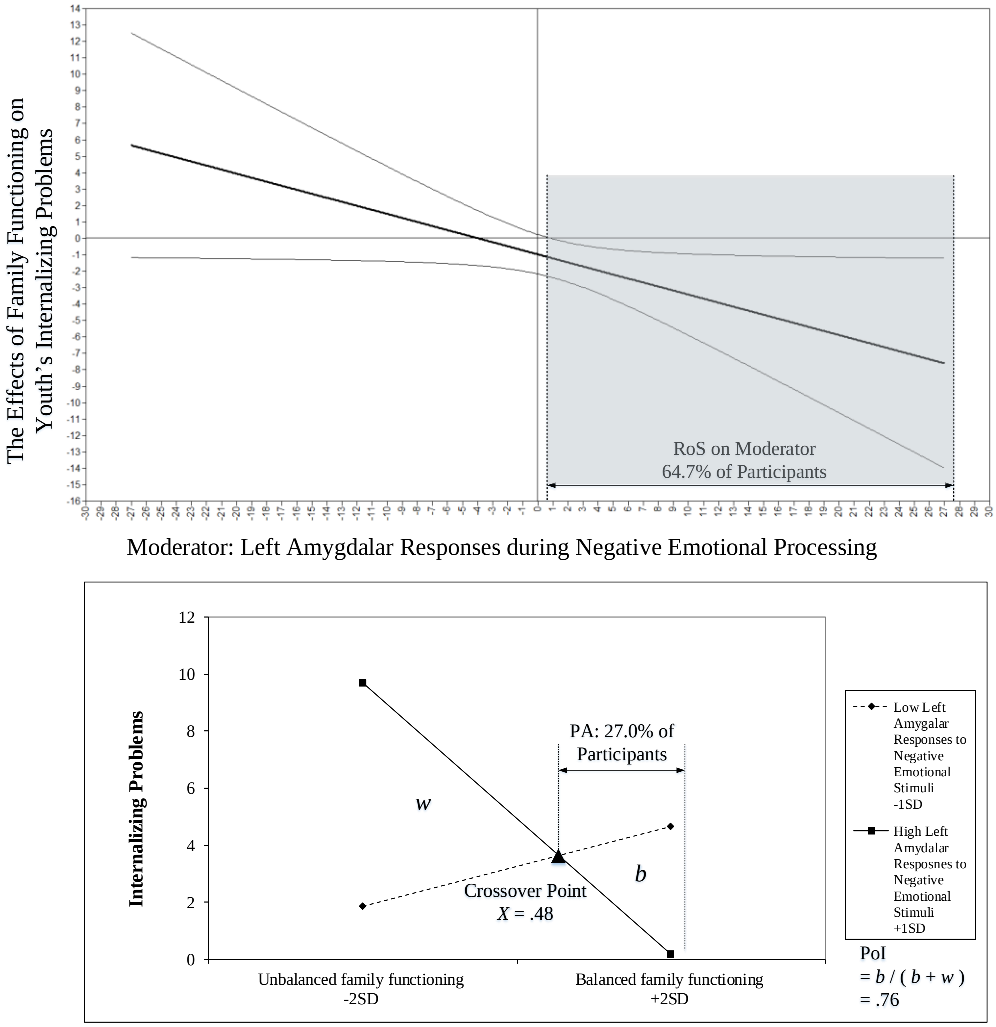

Figure 4.10. The Interpretation of the Significant Moderation Effect of Left Amygdalar Activations during Negative Emotional Stimuli on the Associations Between Family Functioning and Youth Internalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = “for better”; *w* = “for worse”. The dashed line indicates youth with low left amygdalar responses during negative emotional stimuli, while the solid line indicates youth with high left amygdalar responses during negative emotional stimuli.

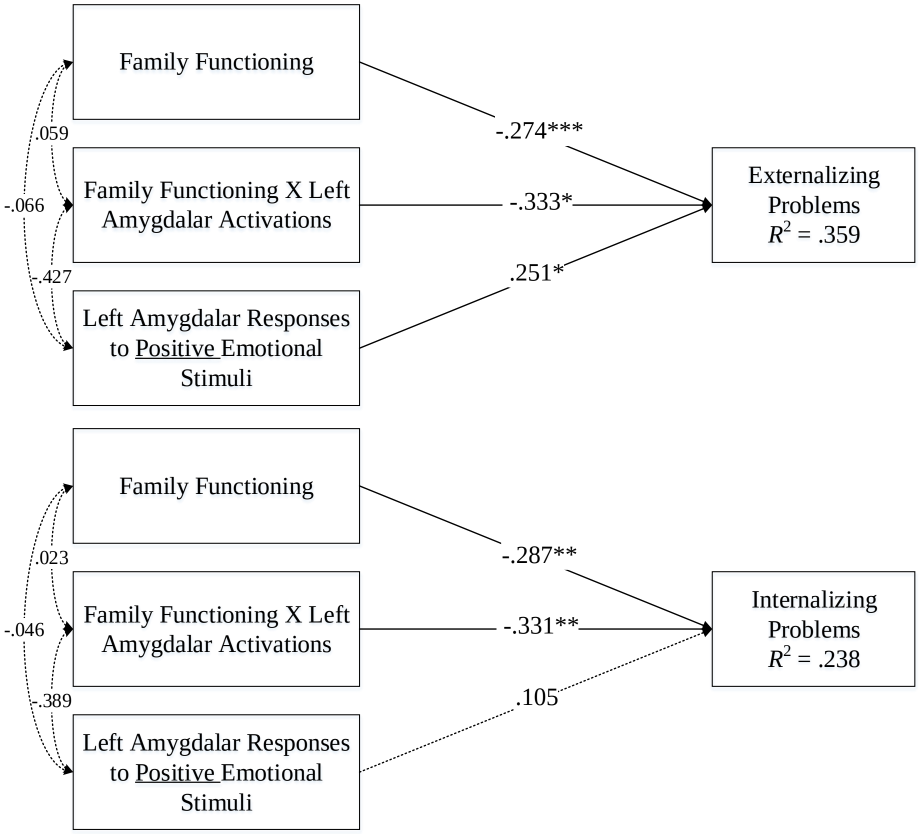

Figure 4.11. The Structural Equation Model of the Associations among Family Functioning, Left Amygdalar Responses to Positive Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Internalizing model: $\chi^2(4) = 3.597$ ($p = .983$); CFI = 1.000; TLI = 1.027; RMSEA = .000; SRMR = .036; Externalizing model: $\chi^2(4) = 3.739$ ($p = .91$); CFI = 1.000; TLI = 1.014; RMSEA = .000; SRMR = .036. *$p < .05$, **$p < .01$, *** $p < .001$.

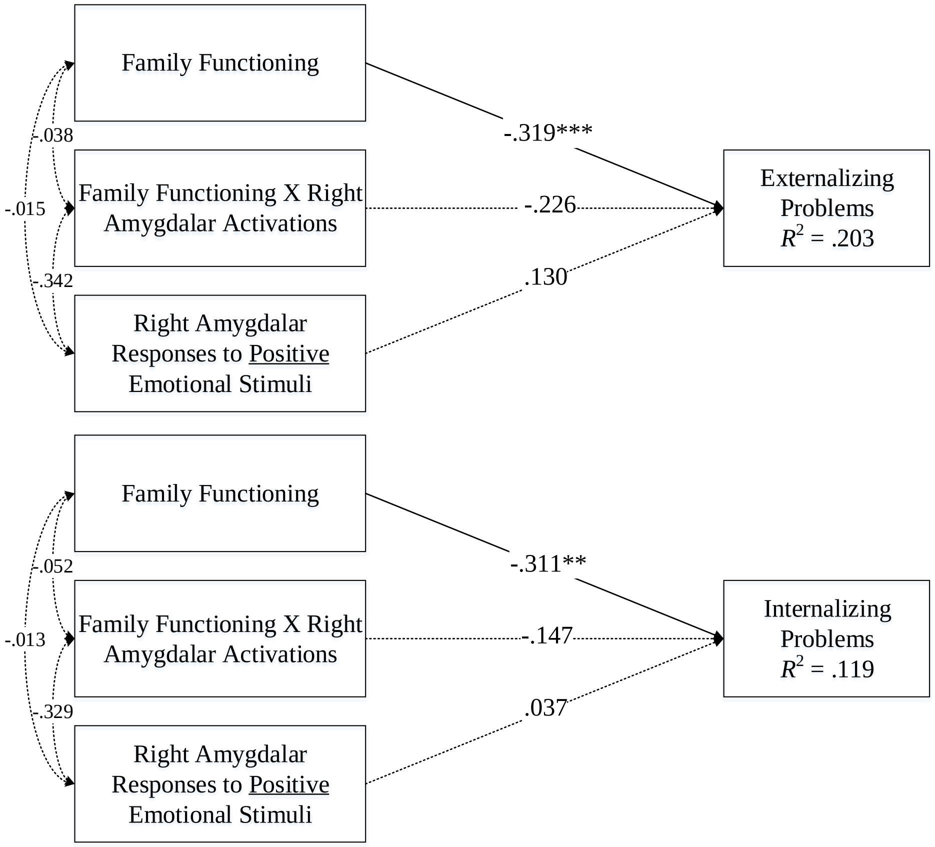

Figure 4.12. The Structural Equation Model of the Associations among Family Functioning, Right Amygdalar Responses to Positive Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Internalizing model: $\chi^2(0)$ = .000 (p = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000; Externalizing model: $\chi^2(0)$ = .000 (p = 1.000); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000. *p < .05, **p < .01, *** p < .001.

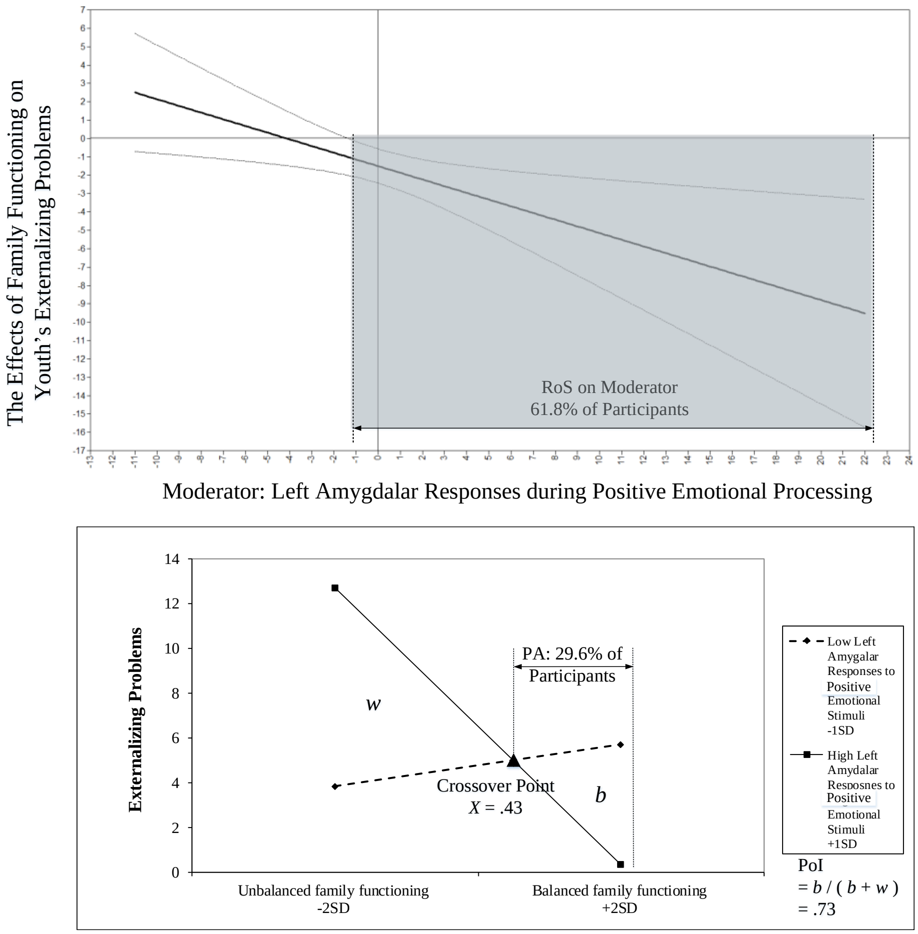

Figure 4.13. The Interpretation of the Significant Moderation Effect of Left Amygdalar Activations during Positive Emotional Stimuli on the Associations Between Family Functioning and Youth Externalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = “for better”; *w* = “for worse”. The dashed line indicates youth with low left amygdalar responses during positive emotional stimuli, while the solid line indicates youth with high left amygdalar responses during positive emotional stimuli.

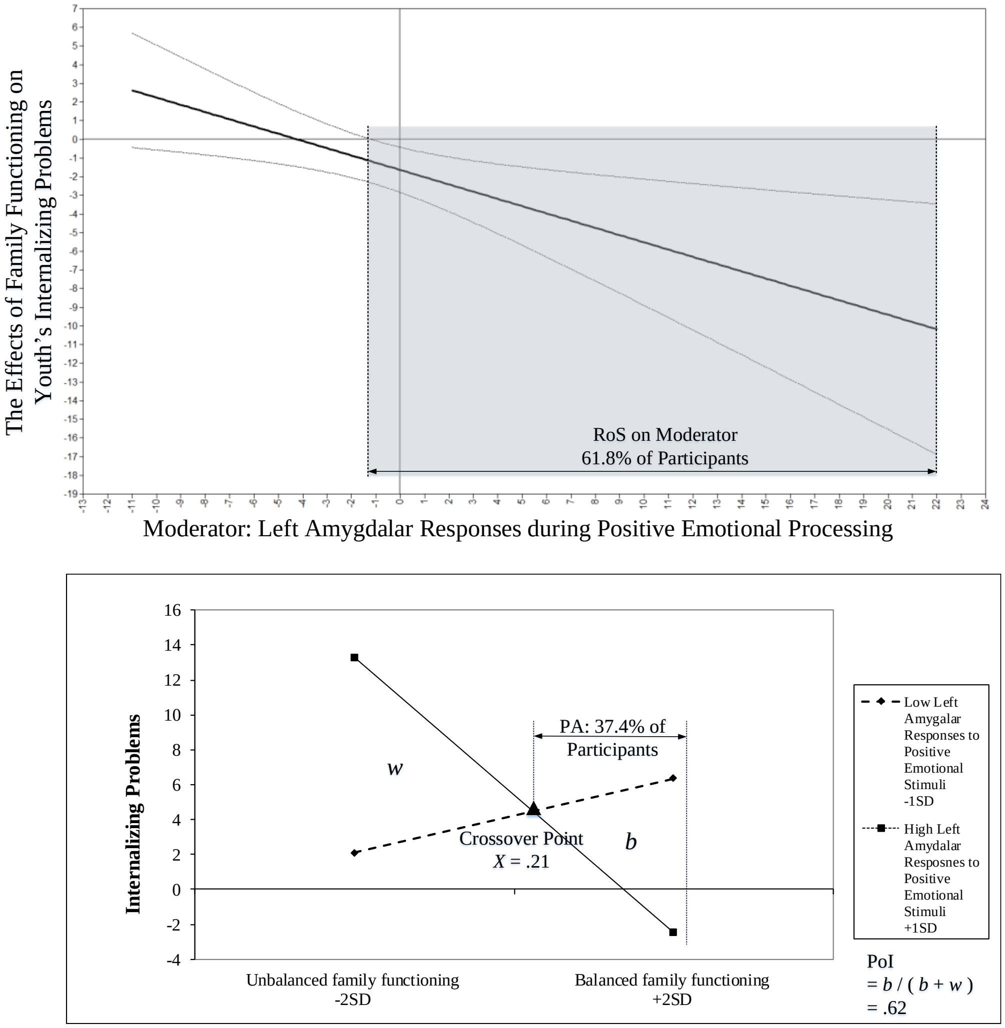

Figure 4.14. The Interpretation of the Significant Moderation Effect of Left Amygdalar Activations during Positive Emotional Stimuli on the Associations Between Family Functioning and Youth Internalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = “for better”; *w* = “for worse”. The dashed line indicates youth with low left amygdalar responses during positive emotional stimuli, while the solid line indicates youth with high left amygdalar responses during positive emotional stimuli.

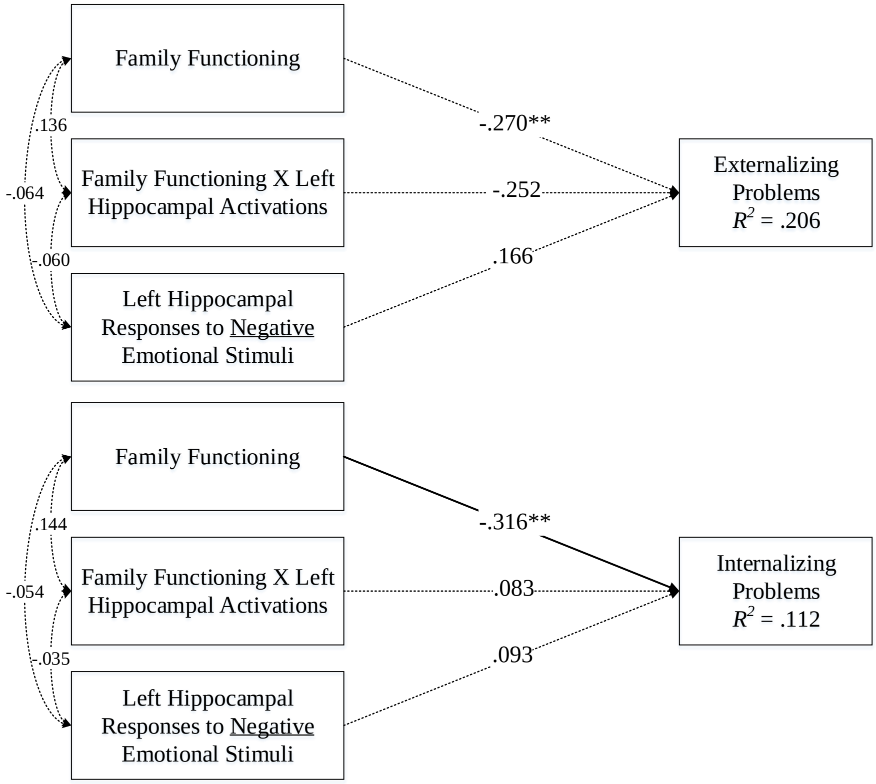

Figure 4.15. The Structural Equation Model of the Associations among Family Functioning, Left Hippocampal Amygdalar Responses to Negative Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Internalizing model: $\chi^2(0) = .000$ ($p = 1.000$); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000; Externalizing model: $\chi^2(4) = 4.695$ ($p = .829$); CFI = .942; TLI = .928; RMSEA = .038; SRMR = .037. $^*p < .05$, $^{**}p < .01$, $^{***}p < .001$.

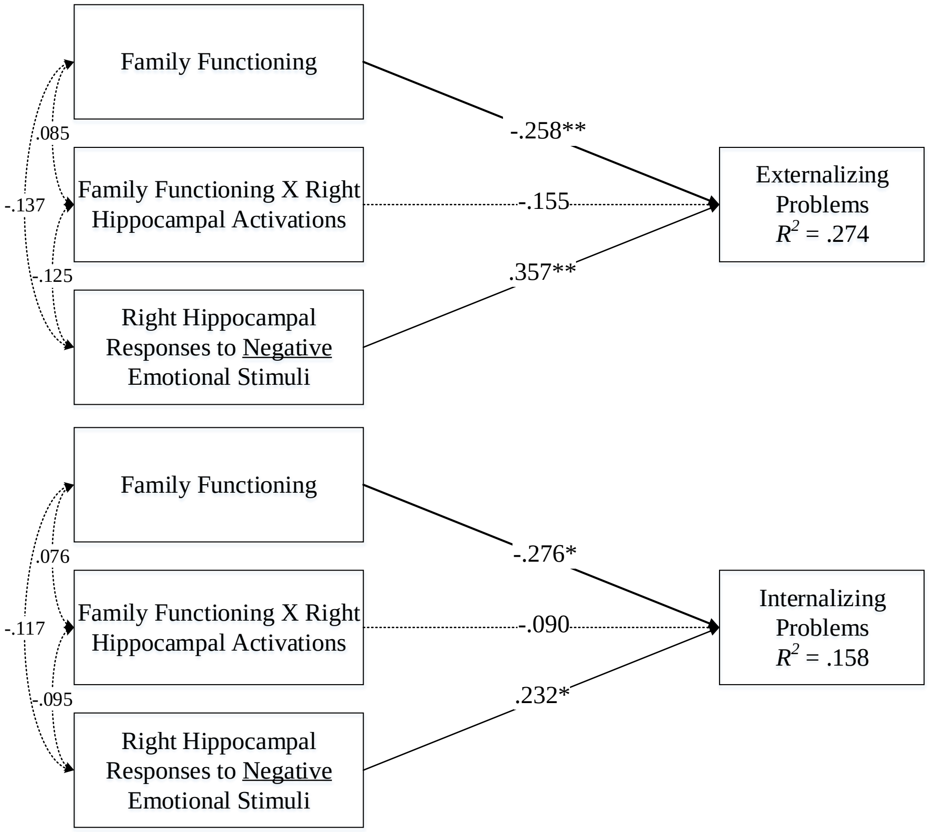

Figure 4.16. The Structural Equation Model of the Associations among Family Functioning, Right Hippocampal Amygdalar Responses to Negative Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Internalizing model: $\chi^2(0) = .000$ ($p = 1.000$); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000; Externalizing model: $\chi^2(0) = .000$ ($p = 1.000$); CFI = 1.000; TLI = 1.000; RMSEA = .000; SRMR = .000. *$p < .05$, **$p < .01$, *** $p < .001$.

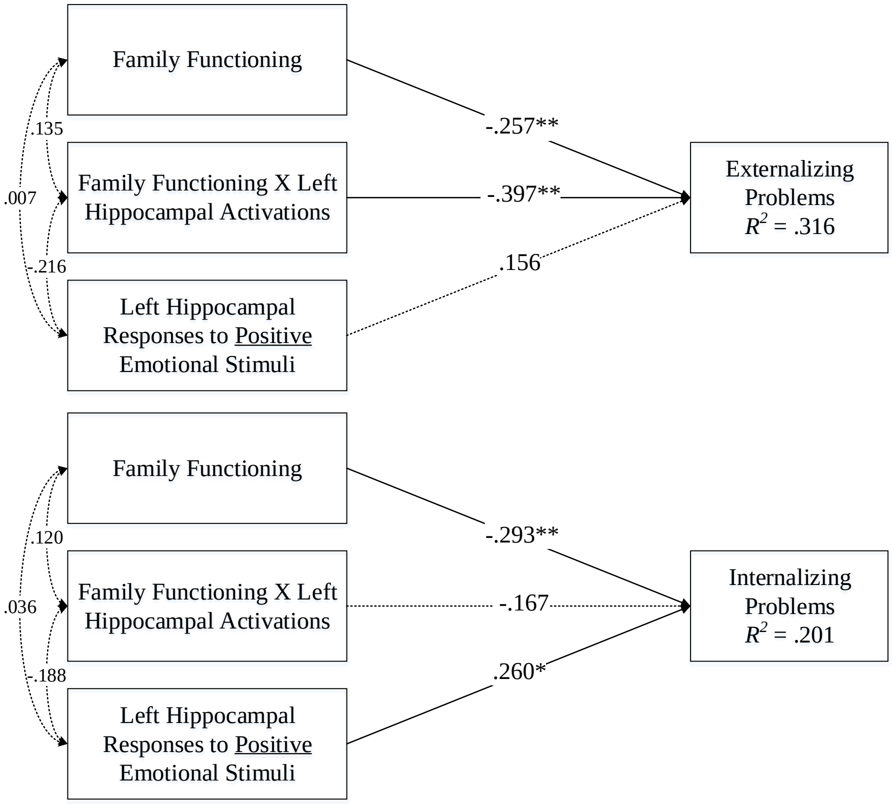

Figure 4.17. The Structural Equation Model of the Associations among Family Functioning, Left Hippocampal Amygdalar Responses to Positive Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Internalizing model: $\chi^2(4) = 4.236$ ($p = .888$); CFI = .983; TLI = .978; RMSEA = .022; SRMR = .037; Externalizing model: $\chi^2(4) = 4.439$ ($p = .881$); CFI = .979; TLI = .974; RMSEA = .030; SRMR = .038. *$p < .05$, **$p < .01$, *** $p < .001$.

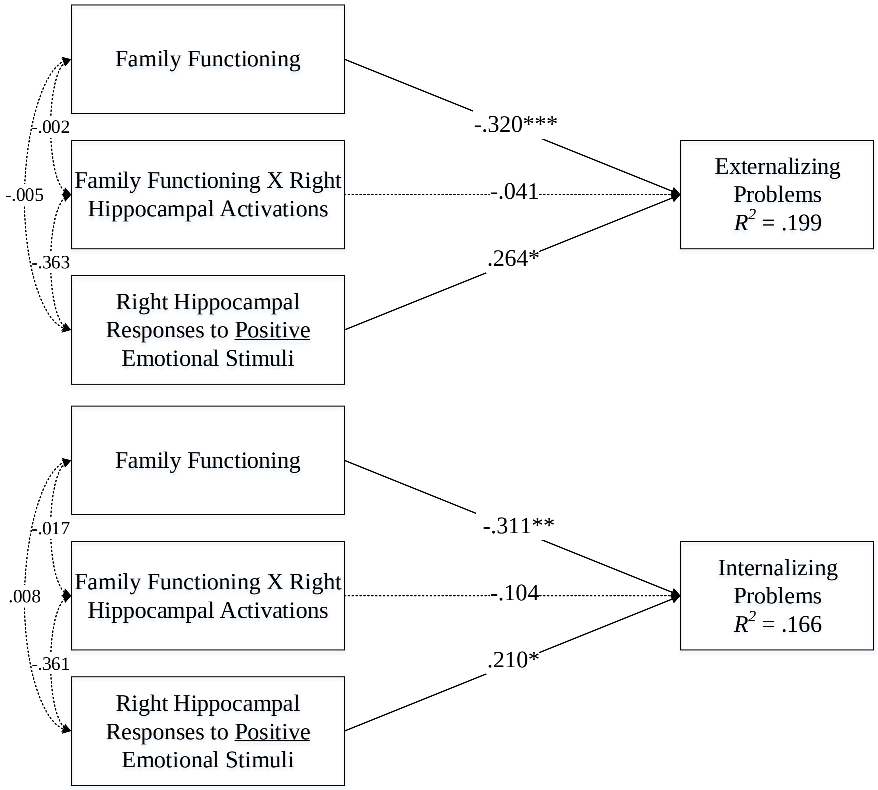

Figure 4.18. The Structural Equation Model of the Associations among Family Functioning, Right Hippocampal Amygdalar Responses to Positive Emotional Stimuli, and Youth Problem Behaviors

Note. The upper panel presents the model with externalizing problems as the dependent variable, and the lower panel presents the model with internalizing problems as the dependent variable. Solid lines indicate statistically significant associations while dotted lines represent insignificant associations. Standardized coefficients are presented in this figure. R^2 = Proportion of variance explained in this model. Models controlled for youth sex and household socioeconomic status. Covariates are omitted for figure clarity. Model fit: Internalizing model: $\chi^2(4) = 4.667$ ($p = .893$); CFI = .941; TLI = .926; RMSEA = .037; SRMR = .043; Externalizing model: $\chi^2(4) = .4.794$ ($p = .901$); CFI = .938; TLI = .922; RMSEA = .040; SRMR = .044. *$p < .05$, **$p < .01$, *** $p < .001$.

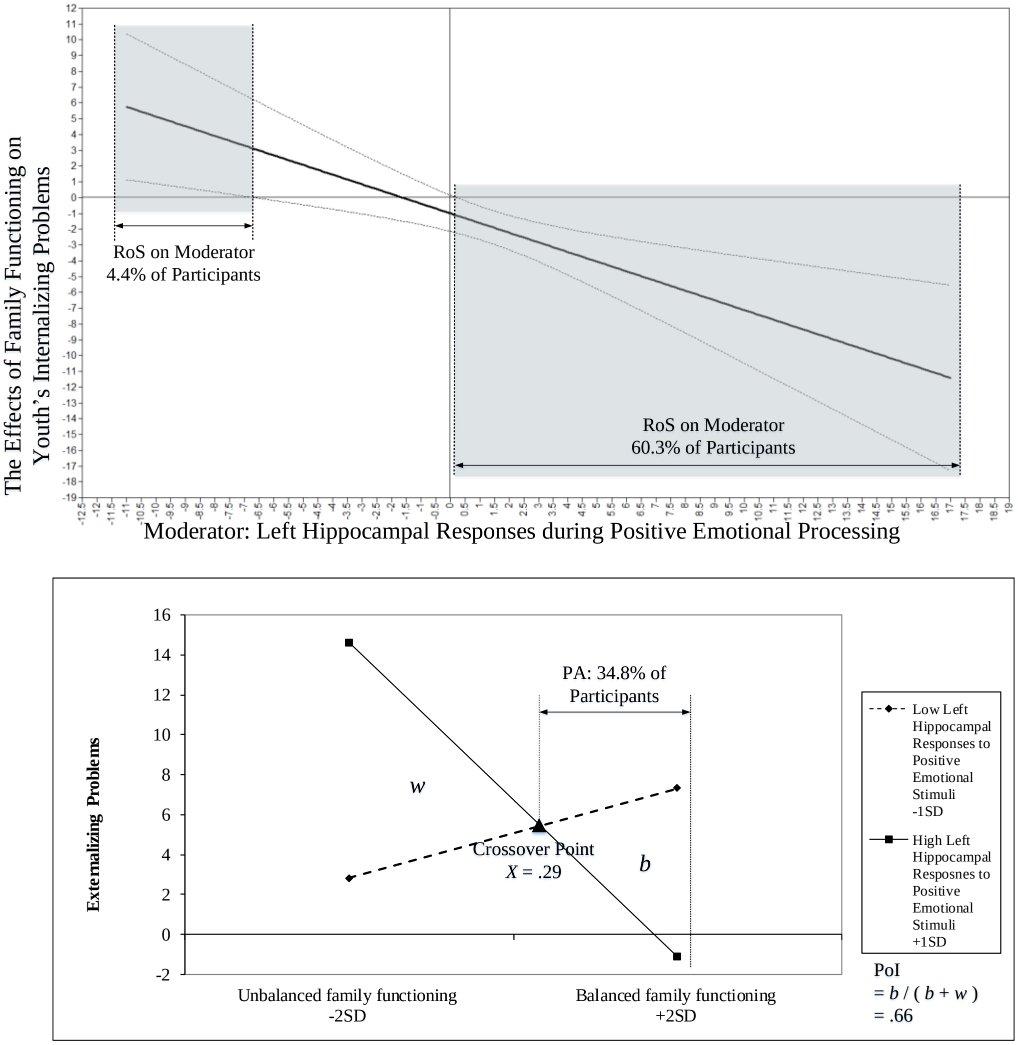

Figure 4.19. The Interpretation of the Significant Moderation Effect of Left Hippocampal Activations during Positive Emotional Stimuli on the Associations Between Family Functioning and Youth Internalizing Problems.

Note. The upper panel presents the Johnson-Neyman plot, and the lower panel presents the simple slope figure. RoI = Region of significance (on the moderator). *b* = “for better”; *w* = “for worse”. The dashed line indicates youth with low left hippocampal responses during positive emotional stimuli, while the solid line indicates youth with high left hippocampal responses during positive emotional stimuli.

CHAPTER 5

INTEGRATIVE DISCUSSION AND IMPLICATIONS

Review of Studies, Aims, and Findings

Family caregiving experiences are critical to youths' socioemotional and behavioral development (Luthar, 2006; A. S. Masten, 2001; Sturge-Apple et al., 2010). Healthy and supportive family environments contribute to positive youth adjustment (G. H. Brody, Yu, Chen, Kogan, et al., 2013; Rohner et al., 2005; Wickrama & Kaspar, 2007). In contrast, adverse rearing experiences are associated with elevated risks for developing problem behaviors and psychopathology among youth (Cummings et al., 2002; Kogan et al., 2019; Oshri et al., 2013; Wickrama et al., 2008).

Despite these robustly-documented main effects of rearing environments quality on youth adjustment, significant individual differences exist in how youth respond to positive and negative family influences (Luthar, 2006; A. S. Masten & Obradović, 2006). These differential responses to family environments are suggested to be embedded in neurobiological architecture that leads to differential sensitivity to environmental stimuli (Ellis et al., 2011). Specific neurobiological factors interact with positive and negative rearing environments and lead to youths' qualitatively different developmental outcomes (Ellis et al., 2011). For example, in interaction with adverse family environments, certain neurobiological factors that are associated with youths' elevated sensitivity to environmental influences can eventuate youth at elevated risk for developing psychopathology and risk behaviors (Monroe & Simons, 1991). In interaction with supportive rearing experiences, the same neurobiological factors can also potentiate youth with increased developmental competence and adaptive behavioral responses (Pluess & Belsky, 2013). In contrast, other youth who lack such neurobiological sensitivity factors are less responsive to positive and negative environmental influences, so their developmental outcomes are not significantly affected by rearing environments (Luthar, 2006; Obradović et al., 2010). Although these differential responses to family influences are consistently documented among children and adolescents (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Obradović et al., 2010; Skowron et al., 2014; Van IJzendoorn et al., 2012), little is known about the neural mechanisms that contribute to this differential sensitivity (Schriber & Guyer, 2016). This dissertation aimed to examine the neural architecture underlying youths' differential responses to the influences of similar family environments. Specifically, two studies were conducted to test the activations of two subcortical brain regions, the amygdala and hippocampus, as the neurobiological foci of youths' differential responses to family influences. Such neurobiological examination clarified why some youth were more responsive to the effects of positive and negative caregiving experiences than others (Schriber & Guyer, 2016).

Overview of Studies

The current dissertation included two studies. The first study employed the ABCD study dataset, a large nationally-representative longitudinal dataset with fMRI and multi-reporter (i.e., youth and their primary caregivers) assessments (Volkow et al., 2018). This study tested how the amygdala and hippocampus moderated the influences of family conflict and parental warmth on youth adjustment. This large dataset (*N* = 11,875) granted sufficient statistical power to test study hypotheses and yielded nationally-representative results (Casey et al., 2018). However, it also bore limitations in the measurement tools that were used to assess family rearing environments. Specifically, the trimmed or shortened scales used in large national datasets, such as the ABCD study, could not capture the full spectrum (from negative to positive) and dimensionality of caregiving experiences. This limited the ability to distinguish different sensitivity patterns (i.e., diathesis-stress, biological sensitivity to context, and vantage sensitivity) accurately (Boyce & Ellis, 2005).

To address the methodological issues in the ABCD study dataset, a second study was conducted using more rigorous measurement methods. The data of the second data were drawn from the Development of Risk and Resilience among Rural Youth (DORRY) project that included a sample of rural adolescents and their primary caregivers recruited from Georgia (*N* = 123). The DORRY project utilized an extensive and comprehensive measurement tool to assess the full continuum of the multidimensional family functioning (i.e., the quality of interpersonal relationships in family environments; Olson et al., 2004). This measurement tool allowed for an accurate examination of neurobiological sensitivity models (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, this second study investigated how amygdalar and hippocampal activations during positive and negative emotional processing moderated the influences of family functioning on youths' internalizing and externalizing problems. These two studies methodologically complemented each other and provided a thorough examination of study aims and hypotheses.

Overview of Aims and Findings

The first overarching aim of this dissertation was to test how the quality of rearing environments, including family conflict, parental warmth, and family functioning, directly affected youths' socioemotional adjustment. The findings from the first study suggested that higher levels of family conflict were associated with youths' increased externalizing problems and decreased prosocial behaviors. In addition, higher levels of parental warmth were associated with youths' decreased internalizing problems and increased prosocial behaviors. Similarly, the second study found that balanced family functioning, characterized by balanced cohesion and flexibility and effective communication skills, was connected to youths' reduced internalizing and externalizing problems. Overall, the first overarching hypothesis was supported in which adverse family rearing environments were associated with youths' socioemotional maladjustment (Cummings et al., 2002; Kogan et al., 2019; Oshri et al., 2013; Wickrama et al., 2008). Similarly, supportive and nurturing caregiving experiences were linked to positive developmental outcomes (G. H. Brody, Yu, Chen, Kogan, et al., 2013; Rohner et al., 2005; Wickrama & Kaspar, 2007).

The second overarching aim was to examine the moderating effects of amygdalar activations during emotional processing on the associations between family rearing environments and youth adjustment. Similarly, the third aim was to test how hippocampal responses to emotional stimuli interacted with family environments and affected youths' developmental outcomes. Additionally, the differential response patterns in significant moderating effects were investigated by distinguishing the diathesis-stress, biological sensitivity to context, and vantage sensitivity models (Del Giudice, 2017; Jolicoeur-Martineau et al., 2017; Roisman et al., 2012). Results of the first study suggested that left amygdalar responses to *negative* emotional stimuli significantly exacerbated the impact of family conflict on youths' externalizing problems. Left hippocampal activations to *negative* and *positive* emotional stimuli were also found to intensify the effects of family conflict and parental warmth on youths' internalizing problems. These results corroborated the biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005). Specifically, with heightened left amygdalar and hippocampal activations, youth who reported to grow under stressful family rearing environments developed elevated externalizing and internalizing problems. In contrast, the same elevated left amygdalar and hippocampal activations also intensified youths' developmental competence among those who reported low levels of family conflict and high levels of parental warmth.

In the second study, left amygdalar responses to negative and positive emotional stimuli were found to amplify the influences of family functioning on youths' internalizing and externalizing problems. Left hippocampal activations during positive emotional processing were found to significantly intensify the effects of unbalanced family functioning on youths' externalizing problems. Overall, youth with heightened left amygdalar or hippocampal activations in response to emotional stimuli displayed a sensitivity pattern consistent with the biological sensitivity to context theory (Boyce & Ellis, 2005; Ellis et al., 2005). Particularly, with heightened left amygdalar or hippocampal activations during emotional processing, youth who reported dysfunctional family rearing environments showed elevated problem behaviors. In contrast, the elevated left amygdalar or hippocampal activations also enabled youth to evince reduced internalizing and externalizing problems in response to balanced and healthy rearing environments. Overall, these findings provided empirical evidence that left amygdalar and hippocampal activations in response to emotional stimuli were associated with youths' sensitivity to the influences of positive and negative caregiving experiences.

Furthermore, both studies found that high levels of hippocampal *deactivations* (i.e., negative levels of hippocampal activations in response to emotional stimuli) promoted a "steeling" effect for youth who reported adverse caregiving experiences (R. T. Liu, 2015; Repetti

& Robles, 2016; Rutter, 2006). The “steeling” effect suggests that experiences of moderate levels of stress can strengthen youths’ resilience and thus promote the development of positive outcomes under adverse rearing environments (Finch & Obradović, 2017; Rutter, 2006). The findings of this dissertation suggested that, for youth with high levels of hippocampal deactivations in response to positive emotional stimuli, lower levels of parental warmth and unbalanced family functioning were linked to reduced problem behaviors. Therefore, hippocampal deactivations were shown to promote the development of resilience among youth who reported to be reared in adverse rearing environments.

The fourth aim of the current dissertation was to test the lateralization in amygdalar and hippocampal activations in response to emotional stimuli (Baas et al., 2004; Frings et al., 2006; Schriber & Guyer, 2016; Sergerie et al., 2008; Wager et al., 2003). The first study found that the right amygdala exhibited higher levels of activations in response to positive and negative emotional stimuli compared to the left amygdala. Despite being statistically significant, the effect sizes of these brain hemisphere differences in amygdalar activations were small. No statistically significant difference was found in left vs. right hippocampal activations when youth were responding to emotional stimuli. The second study also suggested no statistically significant brain hemisphere differences in either amygdalar or hippocampal activations. Overall, these findings converge to indicate that the amygdala and hippocampus may exhibit similar activation patterns across the left and right brain hemispheres during youths’ emotional processing (Baas et al., 2004; Sergerie et al., 2008). Additionally, both studies provided consistent evidence that only the left amygdala and hippocampus, rather than the right, significantly exacerbated the effects of positive and negative family rearing environments on youths’ socio-emotional adjustment.

Therefore, the left brain hemisphere is more engaged in regulating youths' differential sensitivity to family environments, compared to the right brain hemisphere.

Lastly, the first study also examined sex differences in amygdalar and hippocampal activations during emotional processing, as well as the moderating effects of biological sex on the neurobiological sensitivity models. Results revealed statistically significant, but weak sex differences in left amygdalar and hippocampal activations in response to positive emotional cues. For example, compared to females, males evinced higher levels of activations on the left amygdala and hippocampus during positive emotional processing. However, no statistically significant sex difference was found for right amygdalar and hippocampal responses to positive stimuli, as well as the neural responses to negative emotional stimuli. Further, when testing the moderating effect of biological sex on neurobiological sensitivity models, no significant sex difference was identified in amygdalar and hippocampal roles as neurobiological sensitivity indicators. These findings suggest that the roles of the amygdala and hippocampus as indicators of neurobiological sensitivity to family rearing environments may be universal across female and male groups.

Scientific Contributions of the Current Findings and Future Research Directions

The current dissertation is among the first empirical studies that examine amygdalar and hippocampal functions as neural mechanisms underpinning youths' differential sensitivity to the influences of family rearing environments. This project provides empirical support to the neurobiological sensitivity theories by showing that youths' heightened amygdalar and hippocampal activations are associated with their elevated sensitivity to the influences of family environments (Boyce & Ellis, 2005; Ellis et al., 2005). Further, the findings of this dissertation fill the gap on the neural architecture underlying youths' differential responses to the impacts of rearing environments (Schriber & Guyer, 2016). These findings reveal the neurobiological processes underpinning youths' adaptive and maladaptive development under the influences of positive and negative family environments. Therefore, this dissertation can advance knowledge of children's and adolescents' development of risk and resilience (G. H. Brody, Yu, Chen, Miller, et al., 2013; Cicchetti & Curtis, 2007; Ellis, Bianchi, et al., 2017; A. S. Masten & Obradović, 2006; Oshri et al., 2015b; Rutter, 2006).

The Direct Effects of Family rearing environments on Youth Adjustment: Risk and Resilience

This current dissertation provides empirical support to the theoretical frameworks of developmental psychopathology (Cicchetti, 2016), family science (Davies & Cicchetti, 2004), resilience (Luthar, 2006; A. S. Masten, 2001; A. S. Masten & Obradović, 2006; Rutter, 2006). The findings of this project first suggest that the quality of interpersonal relationships in the family rearing environments, such as family conflict, parental warmth, and family functioning, shapes youths' socioemotional adjustment outcomes. Results on the associations between family rearing environments and youth adjustment provide further support to the theoretical frameworks of developmental psychopathology (Cicchetti, 2016; Sroufe, 2009) and family science (Davies & Cicchetti, 2004). The integration of these theoretical frameworks emphasizes that within a family system, the dynamic and reciprocal interactions among family members form a base for youths' normal and abnormal development (Davies & Cicchetti, 2004). Furthermore, both studies suggest that there is a large variance in developmental outcomes for youth who experience similar family environments. Specifically, for youth who report adverse family rearing environments, a large proportion exhibit resilience and show developmental competence, as evinced by low levels of internalizing and externalizing problems. As suggested by the multifinality concept of developmental psychopathology framework, similar rearing environments can result in different developmental outcomes (Cicchetti & Rogosch, 1996). The findings of individual heterogeneity in developmental outcomes for youth who are reared in similar family rearing environments are aligned with this multifinality concept. These findings also provide empirical support to resilience theories (Luthar, 2006; A. S. Masten, 2001; A. S. Masten & Obradović, 2006; Rutter, 2006). Consistent with multilevel systems perspective on child development of risk and resilience (Cicchetti & Curtis, 2007), the results of this dissertation further indicate that youths' resilience is not only exhibited in their socioemotional and behavioral adjustment but also grounded in their neurobiological processes such as brain functions.

Neurobiological Sensitivity Indicators

The findings of this dissertation on the roles of the amygdala and hippocampus as neurobiological sensitivity indicators expand knowledge on why some youth are hypersensitive, while other youth are not responsive to the effects of rearing environment (Ellis et al., 2011). This dissertation is aligned with the perspective of developmental cognitive neuroscience (Baron-Cohen et al., 2000; M. H. Johnson, 2013; M. H. Johnson & De Haan, 2015; Munakata et al., 2004; Tager-Flusberg, 2013). The developmental cognitive neuroscience perspective bears the promises of revealing the connections of developmental changes between neural functions and youths' socioemotional adjustment.

The findings of this dissertation suggest that the social-affective brain circuitry is one critical neural system that mediates youths' differential sensitivity to similar environmental inputs (Crone & Dahl, 2012; Guyer et al., 2011). Despite the focus on the amygdala and hippocampus in the current dissertation, neurobiological sensitivity is a complicated process grounded in the inter-relations and reciprocal coordination among multilevel neural and physiological systems (Boyce & Ellis, 2005; Ellis et al., 2005). The functions of the amygdala and hippocampus may only account for a part of this differential sensitivity to family rearing environments (Schriber et al., 2017). Future research that examines the associations between neurobiological sensitivity and other brain structures, functions, and processes is needed to form a more comprehensive picture of the neural architecture underpinning youths' differential sensitivity to environmental influences (Schriber & Guyer, 2016).

Testing other brain regions of the social-affective brain circuitry as sensitivity indicators. Except for the amygdala and hippocampus, the social-affective brain circuitry includes other brain regions that can serve as potential sensitivity indicators (Schriber & Guyer, 2016). One critical brain region in youths' socioemotional development is the ventral striatum (VS), which includes the nucleus accumbens and is a subcortical brain region that belongs to the limbic system (Brodal, 2016; David et al., 2007; Herman et al., 2005). The VS is a center of reward processing. The functions of the VS in reward processing is particularly relevant to adolescents because they show peaks in reward-related behaviors, including rewards in social interactions (especially peers) and novelty-seeking (Spear, 2011; Telzer, 2016). Therefore, the VS has significant implications in regulating individuals' decision-making strategies and reward-related behaviors (Daniel & Pollmann, 2014; Windle et al., 2018). Emerging research indicates that the activations of the VS when youth are anticipating social rewards or avoiding punishments significantly moderate the effects of peer norms and on adolescents' risk-taking behaviors significantly (Telzer, Jorgensen, Prinstein, & Lindquist, in press). Specifically, with high VS activations, youth develop increased risk behaviors under the influence of negative peer norms (i.e., peers who encourage deviant and risky behaviors). However, youth with low VS activities are resilient to the impacts of negative peer norms (Telzer et al., in press). Therefore, heightened VS reward-sensitivity is also associated with youths' elevated neurobiological sensitivity to environmental influences.

Another example is the anterior cingulate cortex (ACC), which is located in the frontal lobe (R. A. Cohen et al., 2006; Stevens, Hurley, & Taber, 2011). Anatomically, the ACC can be divided into dorsal and ventral components. The dorsal ACC is more connected with the prefrontal cortex, which regulates the cognitive abilities. The ventral ACC, in contrast, is more linked to the limbic system that mediates emotional processing (Bush, Luu, & Posner, 2000). Functionally, due to the connections with both the limbic system and prefrontal cortex, the ACC serves as an integrative hub that mediates the cognitive control of emotions (Stevens et al., 2011). The ACC is a candidate neurobiological sensitivity indicator (C. L. Masten et al., 2009; Whittle et al., 2008). For example, C. L. Masten et al. (2009) suggest that activations of the dorsal ACC are linked to adolescents' individual differences in rejection sensitivity (i.e., a maladaptive risk factor) and interpersonal competence (i.e., a protective factor). Therefore, the reactivity of ACC is connected with youths' differential responses to positive and negative environmental influences (Schriber & Guyer, 2016).

Research provides preliminary evidence that supports the potential connections between the VS and ACC activations and youths' neurobiological sensitivity to family influences (C. L. Masten et al., 2009; Telzer, 2016; Whittle et al., 2008). However, empirical studies that directly examine the moderating effects of the VS and ACC on the associations between family rearing environments and youth adjustment is lacking (Schriber & Guyer, 2016). Future research is needed to test how the functions of other regions of the social-affective circuitry, such as VS and

ACC, interact with family rearing environments and affect youths' socioemotional and behavioral adjustment.

Revealing the interconnections between the social-affective brain circuitry and other sensitivity indicators. The social-affective brain circuitry primarily regulates youths' behavioral reactions in response to emotional and stress stimuli (Crone & Dahl, 2012; Guyer et al., 2011; Schriber & Guyer, 2016). Therefore, findings of the current dissertation on the role of the social-affective brain circuitry as sensitivity indicators reveal a connection between youths' emotional and stress regulation abilities and neurobiological sensitivity. Indeed, this connection has been implied by previous empirical research that investigates behavioral, genetic, and physiological sensitivity indicators (Ellis et al., 2011). These sensitivity indicators include difficult temperament (i.e., behavioral phenotype; Chen et al., 2019), dopamine- and serotonin-related genes (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Caspi et al., 2010; Van IJzendoorn et al., 2012), and physiological stress response indicators (Boyce & Ellis, 2005; Ellis et al., 2005; Obradović et al., 2010). Further, research in neuroscience and psychobiology documents that the functions of the social-affective brain circuitry are highly interconnected with these behavioral, genetic, and physiological sensitivity indicators (Buijs & Van Eden, 2000; DiCara, 2012; Herman et al., 2005; Sakai et al., 2005; Schwartz et al., 2003).

Future research can investigate how the interconnections of different sensitivity indicators (i.e., behavioral phenotype, genetic, physiological stress responses, and neural processes) account for the youths' differential sensitivity to family rearing environments (Schriber & Guyer, 2016). For example, recent studies that employ both neuroimaging and psychobiology methods propose the importance of the brain-autonomic coupling in youth development. Brain-autonomic coupling is defined as coordinated and matched activities between the brain and the autonomic nervous system (Thayer & Lane, 2009). Notably, research suggests that the brain-autonomic coupling is affected by youths' early experiences and bears significant influences on youths' development of problem behaviors (Weissman, Guyer, Ferrer, Robins, & Hastings, 2018, 2019). Future research can further examine how the brain-autonomic coupling moderates the associations between family rearing environments and youth adjustment. This body of research will further reveal how the coordinated activities of complicated neurobiological systems affect youths' development of risk and resilience (Weissman et al., 2019).

Testing the cognitive control brain circuitry as sensitivity indicators. Apart from the social-affective brain circuitry, the adolescent neurodevelopmental models (i.e., the "imbalanced models") indicate that the cognitive control system is also critical for youths' neurobiological sensitivity (M. Ernst & Fudge, 2009; Nelson et al., 2005; Steinberg et al., 2008). Indeed, the slower and gradual maturation of the cognitive control system during adolescence, compared to the early-developed social-affective circuitry, contributes to youths' sensitivity to rearing environments (Casey et al., 2008; M. Ernst, 2014; Nelson et al., 2005; Schriber & Guyer, 2016; Steinberg et al., 2008). Therefore, the *deactivations* of the cognitive control system during emotional or stress processing, such as the prefrontal cortex deactivations, may serve as candidate sensitivity indicators for adolescents.

Furthermore, these "imbalanced models" emphasize the role of the *disjoint* between the cognitive-control and social-affective circuits in affecting adolescents' impulsivity and risk-taking behaviors (Romer et al., 2017). This divide can be reflected by the functional dysconnectivity between the cognitive-control and social-affective brain circuits (Steinberg, 2010). Functional dysconnectivity, in opposite to functional connectivity, is defined as the temporal *incoherence* (i.e., negative correlations) of activations in different neural systems (Van Den Heuvel & Pol, 2010; Zhou et al., 2007). Therefore, future research on neurobiological sensitivity can also test the functional dysconnectivity between the cognitive-control and social-affective brain circuits as a potential sensitivity indicator for adolescents.

Implications for Translational Neuroscience

Theories developed using the framework of developmental psychopathology (Cicchetti, 2016) and family science (Davies & Cicchetti, 2004) have been widely applied to developing family-based intervention practices. These practices include programs designed to improve family relationships and youth competencies (Beach et al., 2014). Adopting similar translational strategies, the current dissertation can contribute to the field of *translational neuroscience*. Translational neuroscience is an interdisciplinary field that applies neuroscience research to the development of clinical practices, therapies, and preventive intervention programs (Fisher, 2016; Garcia-Rill, 2012). By specifying the underlying neural mechanisms, translational neuroscience can be used to develop precise and efficient preventive intervention programs that aim for mitigating the harmful impacts of early life stress and improving disadvantaged youths' well-being (Fisher, 2016). Aligned with the translational neuroscience perspective, findings of the current dissertation on the neural underpinnings of youths' differential sensitivity to family influences have significant implications for the design of prevention and intervention programs.

Implications for Prevention and Intervention Practices

During adolescence, rapid growth and changes occur in youths' brains (Casey et al., 2008; Romer et al., 2017). Therefore, adolescence is an essential developmental window with significant neuroplasticity needed for intervention programs to take effect in re-orienting youths' behaviors, especially for disadvantaged adolescents (Schriber & Guyer, 2016). Examining neurobiological sensitivity indicators has significant implications for identifying youth who can benefit the most from preventive intervention programs (Ellis, Bianchi, et al., 2017; Ellis et al., 2011). Through examining the activations of specific brain regions in response to socio-emotional stimuli (such as the amygdala and hippocampus), researchers can identify youth who have sensitive neural functions that can be amenable for intervention (Schriber & Guyer, 2016). Further, the inclusion of neurobiological sensitivity assessments in intervention and prevention practices could facilitate individualized program designs that are based on the knowledge of youths' previous early experiences, behaviors, and biological characteristics (Schriber & Guyer, 2016).

Significant brain changes occur during adolescence, including synaptic pruning, extensive myelination, volumetric changes, and rebalancing of excitatory and inhibitory inputs (Bredy, Zhang, Grant, Diorio, & Meaney, 2004). These brain changes enable youth with the potential to reprogram the impact of early life experiences at the neural level to be consistent with current experiences (Bredy et al., 2004; Schriber & Guyer, 2016). For example, if youth experience adverse rearing environments during childhood but are later put in a supportive context during adolescence, the neural plasticity renders them the ability to reprogram their neural functions to fit in the current positive environment. Preventive intervention programs may use empirical findings in the neurobiological sensitivity research to guide youths' neural reprogramming and improve their well-being (Schriber & Guyer, 2016).

Lastly, by assessing the neural mechanisms underlying youths' differential sensitivity to family rearing environments, researchers can parse the neurobiological sensitivity into different brain functional domains. These functional domains may involve affective reactivity

(McLaughlin et al., 2014), reward processing (Telzer et al., in press), and conflict monitoring (Carter & Van Veen, 2007). These functional domains are often less evident through survey-based, observational, or physiological assessments. However, neuroimaging research can use the evaluation of specific brain regions to identify which function domain is more relevant to youths' certain behaviors (Schriber & Guyer, 2016). For example, given that the amygdala is critical for emotional processing (LeDoux, 2000), the findings on amygdalar activations as sensitivity indicators emphasize the importance of emotional reactivity in youths' neurobiological sensitivity. This finding suggests that preventive intervention programs that target disadvantaged youth can incorporate contents that improve youths' emotional regulation abilities. Overall, through parsing neurobiological sensitivity into functional elements, the contributions of different classes of emotion, cognitive, and motivation to this sensitivity can be revealed. This will further inform the content designs and targeting components in preventive intervention programs that aim at improving at-risk youths' well-being.

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