Contextual Influences on Youth Socioemotional and Corticolimbic Development

by

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A dissertation submitted in partial fulfillment
of the requirement for the degree of
Doctor of Philosophy
(Psychology)
in the University of Michigan
2019

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DEDICATION

To my father, Robert Gard, and grandmother, Jean Scott. Thank you for raising me to be curious,

authentic, and tenacious.

ACKNOWLEDGMENTS

There are many people that helped me complete this dissertation. Most importantly, thank you to everyone in my social support network. Completing a dissertation and persisting through graduate school is a marathon, and I would not be at the finish line without your encouragement, wise words, and humor. This includes my 2013 Developmental Cohort – particularly Dr. Tyler Hein, Dr. Jasmine Manalel, and Amira Halawah. Thank you to all of my colleagues and friends in the Biodemography Lab – especially Dr. Erin Ware and Dr. Lauren Schmitz. I want to acknowledge all current and past members of the MiND Lab, particularly Dr. Rebecca Waller and Dr. Laura Murray, for helping me develop as a scientist and acting as a sounding board during times of stress. Thank you to my family – my godparents Jill Alfano and Tom Gray are my biggest cheerleaders and have guided me through intense personal and professional challenges. I would not be here without the support of mom, Stacy Scott, my sister Gianna Ciaccio, my uncle, Dean Scott, my best friend, Ashley Baskin, and my life partner who I adore and who inspires me to think deeply about the world, Ariel Binder.

I would like to thank my advisor, Dr. Luke Hyde, for his steadfast and sound mentorship. I cannot thank you enough for your support of my professional and personal growth over the last six years. I am continually inspired by the breadth of your knowledge, ambitious approach to research, and the seamless way in which you weave together developmental science and clinical research.

To the other members of my committee – Dr. Vonnie McLoyd, Dr. Christopher Monk, Dr. Colter Mitchell, and Dr. Chandra Sripada – thank you for your enthusiastic support. Dr.

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Mcloyd, thank you for helping me think about inequality and the heterogeneity in people's lived experiences. Your groundbreaking research over the last few decades is an inspiration, and I feel humbled to collaborate with you. Dr. Monk – thank you for teaching me to think critically about developmental neuroscience and for making my research possible through the use of the SAND dataset. There is no doubt in my mind that a successful career as a young scholar is made possible through access to such unparalleled data. Dr. Mitchell – thank you for your consistent support of my interdisciplinary training, financially and professionally, and for being honest about the challenges of this type of research career. Dr. Sripada – thank you for reading my [very long] dissertation and providing helpful comments and suggestions. I look forward to future collaborations.

Lastly, I would like to thank the University of Michigan Rackham Graduate School and the Department of Psychology for funding my graduate studies. I am grateful for the ability to think about science every day without worrying about how to survive financially.

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ABSTRACT

The formation of adaptive socioemotional skills is a key developmental competency in childhood and such behaviors are supported, in part, by neural function within the corticolimbic system. Multiple features of the social context (e.g., harsh parenting) and individual-level markers of maturation (e.g., pubertal development) are robust predictors of youth socioemotional outcomes, but several gaps in the literature remain. However, more research is needed to investigate the timing and specificity of contextual and maturation effects on youth socioemotional and corticolimbic development, using population-based studies that allow for generalization of the results to a broader population. This three study dissertation integrates research on socioeconomic disadvantage, neural correlates of emotion processing, and internalizing and externalizing behaviors in childhood in service of these goals. Study 1 tests a longitudinal Family Stress Model using prospectively-collected data from a population-based nationwide study of children followed from birth through age 9, with an oversample of disadvantaged families. Study 2 builds on the results of Study 1 by examining the influence of initial levels and changes in harsh parenting across childhood on corticolimbic function during adolescence. Finally, Study 3 evaluates the effects of age and puberty on amygdala-prefrontal connectivity during face processing, using a large cross-sectional population-based sample of twins from Southeast Michigan. The general discussion chapter of this dissertation highlights theoretical and empirical considerations for this research, as well as outlines several future directions.

CHAPTER I

Introduction

One marker of successful development through childhood is the formation of adaptive

socioemotional skills, including emotion regulation, impulse control, and interpersonal

competencies (Cole, Hall, & Hajal, 2008; Neuhaus & Beauchaine, 2013). By contrast,

psychopathology that emerges in childhood, including internalizing (e.g., anxiety, depression)

and externalizing (e.g., aggression, rule-breaking) behaviors, shapes the development of mental

health into adulthood through transactional processes between the individual and the

environment that unfold over time (Masten & Cicchetti, 2010; Sroufe & Rutter, 1984). Such

forms of psychopathology are associated with poorer health, wealth, and wellbeing across the

lifespan (Masten & Cicchetti, 2010; Moffitt, 2018; Scott et al., 2011; Steptoe, Deaton, & Stone,

2015). Multiple features of the social context, particularly correlates of socioeconomic

disadvantage, exert robust effects on psychopathology (Duncan, Ziol-Guest, & Kalil, 2010;

McLoyd, 1998). Alterations in brain function is one plausible mechanism of these associations

(Bruce S. McEwen, 2012; Shonkoff et al., 2012). To improve treatment and prevention efforts

for the development of youth psychopathology, more research is needed to understand how and

when the social context predicts the emergence of these behaviors. The current dissertation

integrates research on socioeconomic disadvantage, neural correlates of emotion processing, and

internalizing and externalizing behaviors in childhood in service of this goal.

Theoretical Frameworks

This dissertation uses several theoretical frameworks to explore how and when socioeconomic disadvantage impacts youth brain and behavioral development. Bronfenbrenner's Bioecological Model of Human Development (Bronfenbrenner & Morris, 2007) posits that proximal contexts within and across multiple nested systems are the primary predictors of child development, from the microsystem (e.g., families) to the macrosystem (e.g., cultural values). Developmental stage and person characteristics further moderate the effects of contexts on youth outcomes. For example, infants and young children (i.e., birth to 5 years) are physically and emotionally dependent on their caregivers for survival (M. D. S. Ainsworth, Bell, & Stayton, 1974; Bowlby, 2008). Caregivers can exert relatively more control over young children than adolescents, who possess greater physical and psychosocial maturity and spend more time in social networks outside the home (e.g., peer relationships, neighborhoods) (Bornstein, 2005; Edwards & Liu, 2005; Smetana, Campione-Barr, & Metzger, 2006). Similarly, person characteristics such as pubertal development may moderate youth socioemotional development at both the behavioral and biological level (Dahl, 2004). All three studies in this dissertation examine components of Bronfenbrenner's Bioecological Model of Human Development (Bronfenbrenner & Morris, 2007), including the influence of proximal processes (e.g., parenting), developmental stage, and person characteristics, on youth socioemotional and biological development.

A second theoretical framework that informs this dissertation is the Family Stress Model (FSM), which highlights the centrality of parenting and the parent-child relationship within the microsystem of the developing child (Bronfenbrenner & Morris, 2007; Conger, Conger, & Martin, 2010). Originally informed by research linking economic stress to marital functioning (Elder, Conger, Foster, & Ardelt, 1992), Conger and colleagues extended this model to study

how stress within the family influences socioemotional development in children (Conger et al., 1993, 2010). The basic form of the FSM posits that: (1) economic hardship (e.g., job loss, low income-to-needs) generates economic pressure felt by parents, defined as the psychological experience of material hardship and food insecurity; (2) such economic pressure translates into parent psychological distress, including maternal depression and substance use, which (3) impacts marital and parent-child relationship quality as measured by inter-parental conflict and parenting behaviors; finally, (4) greater marital instability and parenting characterized by high harshness and low warmth leads to maladaptive youth emotional and behavioral problems (Conger & Conger, 2002; Conger et al., 1993, 2010; Conger, Ge, Elder, Lorenz, & Simons, 1994; Masarik & Conger, 2017).

Parenting and the Parent-Child Relationship

The FSM places parents at the center of the associations between socioeconomic disadvantage and youth socioemotional outcomes. Several theories of parenting (Becker, Peterson, Luria, Shoemaker, & Hellmer, 1962; Bugental & Grusec, 2006; Darling & Steinberg, 1993; Maccoby & Martin, 1983) converge in their distinction between two primary dimensions of parenting behaviors: warmth and harshness (also characterized as responsiveness and demandingness; support and control). Meta-analyses indicate small to medium effects sizes of parental harshness and warmth on youth internalizing (e.g., anxiety, depression) and externalizing (e.g., conduct problems) behaviors that vary by the dimension of parenting behavior and child outcome examined. For example, larger effects of negative dimensions of parenting (e.g., control, rejection) have been reported for childhood depression symptoms (McLeod, Weisz, & Wood, 2007), than for childhood anxiety symptoms (McLeod, Wood, & Weisz, 2007). Hoeve et al., (2009) also reported larger effect sizes of parent psychological

control than parental support (e.g., affection, involvement) on youth antisocial behavior. Thus, multiple dimensions of parenting behaviors, including harshness and warmth, are relevant predictors of youth socioemotional development.

Development of parenting behaviors. Although parenting behaviors change over time as function of contextual demands (Belsky, 1984; McLoyd, 1998), the parent-child relationship is first formed during the early postnatal years. Attachment theorists highlight the importance of an attachment bond between the caregiver and the infant (M. D. S. Ainsworth et al., 1974; Bowlby, 1982). Physically dependent on their caregivers for survival, infants act to maintain proximity to their caregivers (e.g., crying to elicit caregiver attention) (Ainsworth, 1979). When such infant attachment behaviors are met with parenting behaviors that encourage (e.g., warmth) or discourage (e.g., hostility) affiliation, the child's expectations of the physical environment and attachment figures are represented into "working models" that shape interpersonal functioning across the lifespan (Bowlby, 1982, 2008; Bretherton & Munholland, 2008; De Wolff & Van IJzendoorn, 1997; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000). Social learning theorists also emphasize the importance of the parent-child relationship in the early childhood period, where youth antisocial behavior results from coercive parent-child interactions (Bugental & Grusec, 2006; Patterson, 1982; Shaw & Bell, 1993).

Parental warmth and harshness change across childhood in ways that mirror the developmental competencies of each developmental stage. Several investigations in community samples (Dallaire & Weinraub, 2005; Lipscomb et al., 2011) have reported increases in overreactive and harsh parenting from infancy (6 to 9 months) through toddlerhood and early childhood (2 to 5 years). These increases in parental control are thought to parallel developing child mobility and normative increases in emotionality starting around age 2 (Shaw & Bell,

1993). Warmth has also been shown to remain stable between ages 1 and 2 (Verhoeven, Junger, Aken, Dekovic, & Aken, 2007), but decrease overall from 6 months to 6 years (Dallaire & Weinraub, 2005). As children move into middle childhood (5 to 12 years), new developmental challenges associated with starting school (e.g., self-regulation, academic skills, social interaction with peers) further alter the parent-child relationship and requisite parenting behaviors (Collins, Madsen, & Susman-Stillman, 2005). Affective expression often decreases during middle childhood, where both children and parents both show less overt affection and negative behaviors (e.g., coercion, emotional outbursts) (Collins et al., 2005; Forehand & Jones, 2002; Shanahan, McHale, Crouter, & Osgood, 2007). In adolescence, when youth begin to spend more time with peers and less time with parents (Smetana et al., 2006), parental warmth has been shown to decrease (Barber, Maughan, & Olsen, 2005) or remain stable (Shanahan, McHale, Crouter, et al., 2007), while psychological control has been shown to increase as adolescents seek greater independence from parents (Barber et al., 2005).

Biological Mechanisms of Context Effects on Youth Socioemotional Outcomes

Efforts to understand how the social context, including the parent-child relationship, impact youth socioemotional outcomes have explored whether brain function is a biological mechanism of these pathways. Broadly, the brain processes stress (both positive and negative forms) by coordinating physiological processes that result in behavioral change (Bruce S. McEwen, 2009). Exposure to stressful contexts results in physiological stress responses (e.g., activation of the hypothalamic-pituitary-adrenocortical axis, which results in the release of cortisol in humans) that are adaptive in the short term (e.g., aid the individual in responding to a threat), but may be maladaptive in the long-term (e.g., hypervigilance to relatively innocuous

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stimuli, repeated exposure to circulating stress hormones) (Gunnar & Quevedo, 2007; Bruce S. McEwen, 2012).

The hypothesis that the brain mediates the effects contextual stress on behavioral outcomes sources from research in animal models. Early work by Meaney and colleagues showed that variations in maternal licking and grooming in rat pups was associated in concomitant changes in physiological stress responses that were mediated by transcription of genes encoding such stress hormones and their receptors within the brain (Kaffman & Meaney, 2007). Moreover, multiple studies in rats have shown that exposure to varying levels of maternal licking and grooming behavior during the early postnatal period are associated with changes in stress hormone signaling efficiency in the brain that persists into adulthood and is associated with anxiety-like behavior (e.g., Liu et al., 1997; Weaver, 2007).

Translation work in humans has begun to parallel these findings in animal models, with much of this research operationalizing contextual stress as childhood maltreatment. For example, McGowan et al., (2009) showed that relative to non-abused suicide victims, suicide victims with a history of childhood maltreatment showed lower transcriptional expression of the glucocorticoid gene (which is associate with greater physiological stress reactivity) within the hippocampus. In another example, childhood physical abuse was associated with greater physiological reactivity to a stressor and alterations in associated neural regions (Banihashemi, Sheu, Midei, & Gianaros, 2015).

The Corticolimbic System. The corticolimbic system is one candidate brain network through which stressful contexts may impact youth socioemotional development. Neural regions within this system broadly support emotion processing (Benes, 2010; Ahmad R. Hariri, 2015). Animal and human studies show that brain regions within this system, including the amygdala,

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medial prefrontal cortex (mPFC), and the anterior cingulate cortex (ACC), are structurally and functionally connected to one another (Di, Huang, & Biswal, 2017; Roy et al., 2009; Saygin et al., 2015; Whalen & Phelps, 2009) and are activated during tasks that present emotional facial expressions (Fusar-Poli, Placentino, Carletti, Landi, & Abbamonte, 2009).

Given its critical role in emotion processing, salience detection, and fear learning (LeDoux, 2000), the amygdala forms the "hub" of the corticolimbic region. Neurons within the amygdala, specifically the basolateral (BL) and superficial (SF) subregions, integrate information about the external environment from sensory cortices and the olfactory bulb with contextual information from the hippocampus (Hariri, 2015; Heimer & Van Hoesen, 2006; LeDoux, 2000; Price, 2003). This information is then relayed through intercalated cells to the centromedial amygdala subdivision (CM), which sends efferent projections to other subcortical (e.g., hypothalamus) and cortical (e.g., PFC) regions to stimulate behavioral responses (e.g., activation of the stress response, attention allocation) (LeDoux, 2000; Whalen & Phelps, 2009).

Amygdala reactivity has been studied extensively in response to emotional facial expressions that signal threat (i.e., anger) and distress (i.e., fear) (Ekman & Friesen, 1976; Oatley & Johnson-Laird, 1987; Whalen & Phelps, 2009). Greater amygdala reactivity to threatening facial expressions has been associated with dysregulated cortisol signaling (Henckens et al., 2016; Veer et al., 2011, 2012), and multiple forms of psychopathology including externalizing behaviors (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Hyde, Shaw, & Hariri, 2013; Marsh & Blair, 2008) and internalizing behaviors (Etkin & Wager, 2007; Kim et al., 2011; Monk, 2008). Moreover, childhood maltreatment has also been associated with greater amygdala reactivity to threatening facial expressions (for a meta-analysis see Hein & Monk, 2017), with

some investigations showing similar patterns in adults exposed to more normative variations in childhood stress (e.g., harsh parenting, family conflict) (Burghy et al., 2012; Gard et al., 2017a).

Regions of the PFC are also critical for function of the corticolimbic circuit (Benes, 2010; Ahmad R. Hariri, 2015). The medial PFC (mPFC), which includes the anterior cingulate cortex (ACC), is involved in attention and emotion (Fuster, 2001; Price, 1999). Dense structural and functional connections link the amygdala to the ventro-medial PFC (vmPFC) (Whalen & Phelps, 2009), and human patients with vmPFC lesions exhibit greater amygdala reactivity to threatening emotional facial expressions than healthy controls (Motzkin, Philippi, Wolf, Baskaya, & Koenigs, 2015). Thus, bi-directional connections between the amygdala and mPFC reflect bottom-up processing of salient features in the environment by the amygdala with top down attentional control by prefrontal cortices (Kim et al., 2011).

Though seminal work linking amygdala function to psychopathology has been critical to our understanding of biological risk factors for and correlates of psychopathology, complex socioemotional behaviors are thought to result from interactions between neural regions (Menon, 2011; Sporns, Chialvo, Kaiser, & Hilgetag, 2004; Woo, Chang, Lindquist, & Wager, 2017). Indeed, amygdala-PFC connectivity during the processing of angry, fearful, and ambiguous neutral faces has been associated with internalizing disorders (e.g., Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010; Gard et al., 2018; Kim et al., 2011). Yet despite increased interest in studying functional connectivity within the corticolimbic system as it relates to psychopathology, more research is needed to explore the associations between contextual stress and amygdala-PFC functional connectivity during emotion processing.

Individual-level contributors to corticolimbic function

Beyond highlighting how contextual stress and psychopathology are associated with corticolimbic function, it is important to understand how normative developmental processes impact amygdala and PFC reactivity and connectivity. Age and pubertal development are two maturational processes that have been studied independently in relation to emotion processing within the corticolimbic system. However, though correlated, chronological age and puberty capture different aspects of maturation (Dahl, 2004; Dorn, Dahl, Woodward, & Biro, 2006). Pubertal development, which is associated with social, biological, and physical changes, is specifically thought to impact affective outcomes – emotion, motivation, arousal, and reward processes (Dahl, 2004). Age impacts such affective processes but is also correlated with changes in environmental contexts that shape youth exposure to social stimuli (e.g., entering school in middle childhood) (Baltes & Smith, 2004). Yet few studies have tested whether chronological age and pubertal development exert unique effects on corticolimbic function during emotion processing. Moreover, though pubertal development is oftentimes studied within the adolescent period, pubertal development can start as young as age six with the onset of adrenarche, when adrenal androgens begin to rise but physical signs of puberty have not yet started (Dorn et al., 2006). Gonadarche, which overlaps with adrenarche, is characterized the maturation of primary and secondary sexual characteristics (e.g., in girls, ovaries, breast development); this phase of pubertal development is what is most often typified as "puberty" (Dorn et al., 2006). Therefore, identifying unique effects of age and puberty on corticolimbic function will require large samples from diverse populations where these two constructs are not perfectly correlated (e.g., same-age participants who are at different pubertal developmental stages) (Garcia & Scherf, 2015a).

Population-Based Studies in Neuroscience and Psychology

Population-based studies that are well-sampled to represent a population of interest and capture sociodemographic (e.g., race and ethnicity, income) diversity are critical for generalizing any research finding. Despite widespread agreement that external validity is a critical aspect of research design, the use of population-based sampling methods within neuroscience is a relatively new concept (Paus, 2010). Although the past several years have seen more attention given to sampling methodology in neuroimaging studies (e.g., Adolescent Brain Cognitive Development Study, Generation R Study; Garavan et al., 2018; White et al., 2013), much of the research linking contextual stress to brain development has relied upon racially- and economically-homogenous samples of predominantly White middle-to-upper class families (Falk et al., 2013). A recent paper quantified the influence of sample composition on neuroimaging results and found earlier maturation of cortical and subcortical structures when the study sample (skewed towards White middle-to-upper class families) was weighted to be representative of the U.S. population (LeWinn, Sheridan, Keyes, Hamilton, & McLaughlin, 2017).

One conclusion to draw from population neuroscience is the importance of defining the target population, which should be considered along with the research question (Falk et al., 2013; Paus, 2010). For investigations linking contextual stress to brain development, the target population (and study sample) should represent families living in disadvantaged contexts who are disproportionately at the greatest risk for exposure to stress and concomitant socioemotional outcomes (Kessler et al., 2010; McLaughlin, Costello, Leblanc, Sampson, & Kessler, 2012; McLoyd, 1998). By contrast, for investigations of normative brain maturation, the target population should represent participants from all sociodemographic backgrounds to better approximate a broader population (e.g., children living in the U.S.; LeWinn et al., 2017).

Specific Aims of this Dissertation

The purpose of this dissertation is the integrate research on contextual predictors of youth socioemotional outcomes with neurobiological research on emotion processing. The three studies that form this dissertation utilize studies that are well-sampled to represent a specific population of interest (i.e., children born in large U.S. cities with an oversampling of non-marital births; twins in Michigan with an oversample living in impoverished neighborhoods), making the results of this dissertation more generalizable than convenience samples that are common in developmental science research and neuroimaging (Davis-Kean & Jager, 2017; Falk et al., 2013; Henrich, Heine, & Norenzayan, 2010).

Study 1 (Chapter II): Evaluation of a Longitudinal Family Stress Model (FSM) in a Population-Based Cohort

> **Aim 1.** Evaluate a longitudinal FSM (where constructs are measured at sequential time points) and a longitudinal change FSM (where the lagged constructs at preceding time points are included to reflect change in each construct) in a population-representative sample of children.

Study 2 (Chapter III): Prospective Longitudinal Effects of Harsh Parenting on Corticolimbic Function during Adolescence

> **Aim 1.** Document patterns of maternal harshness across childhood in a population-based sample.

> **Aim 2.** Evaluate the effects of initial levels and changes in maternal harshness on corticolimbic function during adolescence

Study 3 (Chapter IV): Contributions of Age and Pubertal Development to Amygdala-Prefrontal Connectivity during Face Processing

Aim 1. Examine associations between chronological age and perceived pubertal development on amygdala-PFC connectivity during face processing

Aim 2. Report amygdala-PFC connectivity patterns that are unique and shared across amygdala subregions

Aim 3. Examine whether maturation effects on amygdala-PFC connectivity during face processing varies by the facial expression examined

CHAPTER II

Evaluation of a Longitudinal Family Stress Model in a Population-Based Cohort

Socioeconomic disadvantage is a potent predictor of mental and physical health problems across childhood (Bradley & Corwyn, 2002; McLoyd, 1998) and across the life course (Duncan et al., 2010). Much of the research on socioeconomic status (SES) and youth outcomes has been guided by the Family Stress Model (FSM) (Conger & Conger, 2002), which posits that socioeconomic disadvantage affects children by affecting parents. In the FSM, economic hardships (e.g., low family income-to-needs ratio, parental job loss) predict greater economic pressure on parents (e.g., material hardship, inability to pay bills or make ends meet). Economic pressure, in turn, gives rise to greater emotional distress in parents (e.g., parental depression, anxiety), who are challenged with the physical and psychological strains of economic pressure. Greater parental distress leads to family conflict, including parenting that is high in harshness and low in warmth, which can lead to youth internalizing (e.g., depression, anxiety) and externalizing (e.g., aggression, rule-breaking) behaviors (Masarik & Conger, 2017). Thus, a key feature of the FSM is the recognition that socioeconomic disadvantage taxes family processes, including parent-child relationships, that lead to the emergence of youth psychopathology (Conger et al., 2010; Edin & Kissane, 2010).

The Importance of the Family Stress Model in the First Decade of Life

The FSM is particularly relevant for understanding how family processes lead to youth internalizing and externalizing problems during the first decade of life. Theories of emotional development (Eisenberg, Cumberland, & Spinrad, 1998; Grusec, 2011) highlight the importance

of parents' emotion-related socialization behaviors from infancy through middle childhood, when children spend most of their time in the home (Shaw & Bell, 1993). Parents shape the development of youth socioemotional competence through their own emotional expressions (e.g., anxiety, personal distress) and their responses to child behaviors (e.g., harshness, emotional responsivity, warmth) (Eisenberg et al., 1998). As children age into early adolescence, peer and neighborhood influences become more salient and dampen the relative impact of family processes on youth outcomes (Smetana et al., 2006). For example, meta-analyses show stronger associations between maternal depression, parenting behaviors, and multiple dimensions of youth psychopathology among younger children than adolescents (Goodman et al., 2011; Hoeve, Dubas, Eichelsheim, van der Laan, et al., 2009). Additionally, exposure to poverty and poverty-related sequelae (e.g., economic pressure) during early childhood is particularly predictive of child outcomes (Walker et al., 2011), in part due to early sensitivity of the developing brain (Shonkoff et al., 2012; N. Tottenham, 2009). Thus, the FSM can be used to understand how socioeconomic disadvantage in early childhood leads to emergent youth psychopathology in middle childhood via family processes that shape emotion socialization.

Generalizability of the Family Stress Model

Empirical support for the FSM has been established across a wide range of contexts including diverse cultural backgrounds (i.e., African-American, European-American, and Mexican-American families), family structures (i.e., two-parent and single-parent families), urbanicity (i.e., urban and rural samples), and in populations within and outside of the U.S. (Conger et al., 2002; Jocson & McLoyd, 2015; Kinnunen & Feldt, 2004; Linver, Brooks-Gunn, & Kohen, 2002; Gutman, McLoyd, & Tokoyawa, 2005; Parke et al., 2004; Solantaus, Leinonen, & Punamäki, 2004). This research highlights the important role that parents play in mediating the

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effects of socioeconomic disadvantage on children's development and the validity of the FSM as a family process model. However, there are several quantitative and theoretical limitations of this work that warrant consideration (Conger et al., 2010).

Measurement of Socioeconomic Disadvantage

First, most tests of the FSM overwhelmingly rely on economic aspects of SES (i.e., family income) to indicate hardship (Conger et al., 2010), which may mask the complexities of the effects of socioeconomic disadvantage on family processes. Traditional components of SES include income, parental education, and occupational status (Bradley & Corwyn, 2002). Each component is differentially stable across time, captures different social and economic aspects of hardship, and has varying effects on child outcomes (Bradley & Corwyn, 2002; Conger et al., 2010; Duncan & Magnuson, 2003). For example, whereas income is sensitive to exogenous changes in the environment, education status is comparatively stable in adulthood (Krieger, Williams, & Moss, 1997). Few studies have examined parental age or marital status, which capture social capital or resources inherent to SES and may be important to the family processes proposed by the FSM (Bradley & Corwyn, 2002). Younger mothers may have fewer social resources than older parents (e.g., social support, relevant peer networks), and children of young parents are rated as being more aggressive and impulsive and score lower on cognitive tests than their peers with older mothers (Baldwin & Cain, 1980; Furstenberg Jr., Brooks-Gunn, & Chase-Lansdale, 1989). Family structure also predicts parent and child outcomes (McLanahan & Sandefur, 1994): married compared to single-parent households report lower levels of parent psychological distress and more parental warmth (S. L. Brown, 2004; McLanahan & Sandefur, 1994). Previous tests of the FSM have either not included parental education, maternal age, or marital status in their models (Gutman et al., 2005; Neppl, Senia, & Donnellan, 2016; Simons et

al., 2016), or have specified these variables as model covariates (Newland, Crnic, Cox, Mills-Koonce, & Family Life Project Key Investigators, 2013) or as predictors of family income (Gershoff, Aber, Raver, & Lennon, 2007). Although several examinations of the FSM have found consistency in the model across family structures (e.g., Conger et al., 2002; Gutman et al., 2005), family structure itself (i.e., two-parent versus single-parent households) may proxy SES (McLanahan & Sandefur, 1994). The present study addresses these limitations by examining the unique contributions of multiple indicators of SES as a broad construct (e.g., maternal age, marital status, education, income) to family processes as hypothesized by the FSM.

Longitudinal Evidence

Second, most empirical tests of the FSM are cross-sectional, with constructs (e.g., parental distress, parenting behaviors, and child behavioral problems) measured at the same time point. This design limits our understanding of the direction of effects and the scale by which they cascade across development. By contrast, longitudinal designs provide greater confidence in directional associations, particularly when these models account for the same constructs measured at previous time points (MacCallum & Austin, 2000; Maxwell & Cole, 2007). For example, White, Liu, Nair, and Tein (2015) tested an adapted FSM in a longitudinal sample of 749 Mexican-origin adolescents, and included lagged FSM constructs at previous time points so that path estimates between two constructs reflected the effect of *change* in the predictor variable on the outcome variable. Results showed that mother's perception of economic pressure predicted *increases* in harsh parenting and subsequent *increases* in child externalizing behaviors across middle childhood to adolescence (White et al., 2015). Few other longitudinal tests of the FSM employ this "longitudinal change" design across *all* constructs in the FSM (e.g., Simons et al., 2016 accounted for the lagged measures of youth conduct problems, but not earlier nurturant

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parenting; see Kavanaugh, Neppl, & Melby, 2018; Landers-Potts et al., 2015; Neppl, Senia, & Donnellan, 2016, for similar examples). As the estimates of mediation effects from longitudinal auto-regressive models are less biased than those from cross-sectional mediation models (Maxwell & Cole, 2007), more rigorous longitudinal tests of the FSM are needed.

Population-Based Samples

A third limitation of FSM research is the lack of replication in large, population-based surveys that include large numbers of families living in disadvantaged contexts. Population-based studies that implement data collection and sampling strategies to recruit participants across diverse sociodemographic groups and geographies ensure sample diversity that reflects the population of interest (Groves et al., 2009). The issue of generalizability, stemming from the lack of representative samples (i.e., the demographic composition of the sample does not match the target population), plagues developmental science more broadly than tests of the FSM specifically (Davis-Kean & Jager, 2017; Henrich et al., 2010). Although studying family dynamics within subpopulations is critical to our understanding of diverse developmental trajectories (García Coll et al., 1996), large population-based samples can be used to validate psychological theories and broaden the impact of findings from community-based research (Davis-Kean & Jager, 2017).

Current Study

The current study tested the FSM longitudinally using the Fragile Families and Child Wellbeing Study (FFCWS), a large population-based sample of families in large U.S. cities followed from childbirth through age 9 and oversampled for nonmarital births. The FFCWS was appropriate for an examination of the FSM for several reasons. First, the study measured economic hardship and economic pressure at childbirth and age 1, which is a developmental

period during which infants are particularly sensitive to the neurobehavioral effects of environmental adversity (N. Tottenham, 2009). Second, maternal psychological distress and parenting behaviors, which are key features of emotion socialization theory (Eisenberg et al., 1998), were collected at sequential time points during early childhood when these constructs have been shown to exert the largest effects on youth psychopathology (Goodman et al., 2011; Hoeve, Dubas, Eichelsheim, van der Laan, et al., 2009). Lastly, the FFCWS collected youth internalizing and externalizing behaviors at age 9, when children are still largely under the influence of family processes. Moreover, psychopathology that emerges in middle childhood is often predictive of more serious socioemotional impairments that emerge in adolescence and continue into adulthood (Rutter, Kim-Cohen, & Maughan, 2006).

We tested both a longitudinal FSM (in which constructs were measured at sequential time points) and a longitudinal *change* FSM (in which lagged constructs at preceding time points were included to reflect change in each construct) to assess how estimates change after accounting for construct stability. We included multiple indicators of social and economic aspects SES (i.e., family income to needs ratio, family structure, maternal education, and maternal age) to measure economic hardship at childbirth (Bradley & Corwyn, 2002; Furstenberg Jr. et al., 1989; McLanahan & Sandefur, 1994). We hypothesized that low family income-to-needs ratio, low maternal education, young mothers' age, and mothers' unmarried status at childbirth would independently predict greater youth internalizing and externalizing behaviors at age 9 via greater economic pressure at age 1, greater maternal distress at age 3, and greater maternal harshness and less maternal warmth at age 5. In models that accounted for the lagged constructs at preceding time points, we hypothesized that these associations would be attenuated, but that high harsh parenting and low maternal warmth at age 5 would continue to be significant mediators of

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the effect of socioeconomic disadvantage at childbirth on youth internalizing and externalizing behaviors at age 9.

Method

Sample

Participants were part of the Fragile Families and Child Wellbeing Study (FFCWS), a longitudinal cohort of 4,898 (52.4% boys) children born in 20 large U.S. cities between 1998 and 2000 (Reichman, Teitler, Garfinkel, & McLanahan, 2001). The study oversampled non-marital births (~ 3:1) and when weighted, the sample is representative of families living in U.S. cities with populations of 200,000 or more between 1998 and 2000 (for detailed information about cohort retention across waves, see https://fragilefamilies.princeton.edu). At childbirth, mothers identified as White, Non-Hispanic ($N = 1,030$, 21.1%), Black, Non-Hispanic ($N = 2,326$, 47.5%), Hispanic ($N = 1,336$, 27.3%), or other (N = 194, 4.0%). Nearly 40% of the mothers reported less than a high school education at the childbirth interview, 25.3% with a high school degree or equivalent, 24.3% some college or technical training, and 10.7% who earned a college degree or higher. At childbirth, 1,088 (23.9%) biological mothers were married, 1,668 (36.7%) were cohabitating with a partner, and 1,791 (39.4%) were neither married nor cohabitating (which we denote as "single"). Thus, the FFCWS contains substantial racial, ethnic, and socioeconomic diversity, ensuring variation in the FSM constructs.

Biological mothers were interviewed at the time of the child's birth and again at 1, 3, 5, and 9 years. Telephone surveys were administered at each wave, with a subsample participating in-home visits with trained interviewers at ages three and five. The current study included 2,918 families. We excluded 1,980 families who did not have in-home observational data either at age three or five. Moreover, we marked data as missing at the ages 3, 5, or 9 interviews where the

biological mother was not the respondent to prevent artifacts introduced by changing informants across time. Note that we did not exclude these families from our analyses because in the majority of these cases, the target child still lived with the biological mother most or all of the time. Mothers included in present analyses identified as Non-Hispanic Black (n = 1544, 53.1%), White Non-Hispanic (n = 541, 18.6%), Hispanic (n = 738, 25.4%), or other (n = 87, 3.0%). Most mothers did not earn a high school degree (n = 1173, 40.3%), with roughly a quarter of the sample earning a high school degree or equivalent (n = 753, 25.8%) or some college or a technical degree (n = 725, 24.9%), and 9% (n = 263) with a college diploma or higher. Consistent with the original sampling frame of the study, 78.2% (n = 2269) of the mothers were unmarried at the birth of the target child. Compared to the full FFCWS cohort, the sample used in the present analyses were more likely to be unmarried ($\chi^2[1]$ = 25.37, $p < .001$) and have less education ($\chi^2[1]$ = 21.54, $p < .001$), and included a larger proportion of Black Non-Hispanic mothers and less White Non-Hispanic and Hispanic mothers ($\chi^2[1]$ = 93.88, $p < .001$).

Measures

      Socioeconomic Disadvantage. Four indicators of socioeconomic disadvantage were measured at childbirth: (a) family income to needs ratio was a ratio of total household income (total income before taxes) to official U.S. Census Bureau poverty thresholds based on family composition, where higher values indicate less poverty (M = 2.10, SD = 2.31, Min – Max = 0 – 14); (b) maternal education status, coded ordinally as 1=less than a high school degree (40.3%), 2=high school or equivalent (25.8%), 3=some college or technical degree (24.9%), or 4=college degree or more (9.0%); (c) maternal age in years (M = 24.92, SD = 5.89, Min – Max = 14 – 47); and (d) maternal marital status dichotomized as cohabitating or single (78.2%) versus married (21.8%). We collapsed cohabitating and single mothers into one group based on literature

showing that children of cohabitating and single mothers differ more from children of married

mothers than from each other with regards to socioemotional outcomes (S. L. Brown, 2004).

Economic Pressure. Economic pressure was measured at age 1 (but not at childbirth)

using 12 mother-reported dichotomous (Yes/No) items about experiences in the past 12 months

(e.g., evicted, received free food/meals, telephone service disconnected, borrowed money to pay

pills) taken from the Survey of Income and Program Participation (Bauman, 1998) and the Social

Indicators Survey (Social Indicators Survey Center, Columbia University School of Social Work,

1999). Total scores ranged from 0 to 11 ($M = 1.21$, $SD = 1.67$], $\alpha = .70$). Note that this construct

has also been termed 'material hardship', but we use the term 'economic pressure' to be

consistent with original conceptions of the FSM.

Maternal Distress. We created a latent factor of maternal distress at ages 1 and 3, using

diagnoses of current Major Depressive Episode (MDD), Generalized Anxiety Disorder (GAD),

and four items that measured self-reported stress from parenting from an abbreviated version of

the Parenting Stress Inventory (PSI) (Abidin, 1995). Diagnoses of MDD (Age 1: 12.9% or 358

cases; Age 3: 15.2% or 428 cases) and GAD (age 1: 3.3% or 92 cases; age 3: 5.2% or 146 cases)

were determined using criteria from the Composite International Diagnostic Interview – Short

Form (CIDI-SF) (Kessler, Andrews, Mroczek, Ustun, & Wittchen, 1998). A sum score of the

four reverse-coded items from the PSI (e.g., "Taking care of children is more work than

pleasure"), which were rated by mothers on a 4-point Likert scale (1 = *strongly agree* to 4 =

strongly disagree), was created at ages 1 ($M = 8.74$, $SD = 2.68$, Min – Max = 4 – 16, $\alpha = .61$) and

3 ($M = 9.06$, $SD = 2.66$, Min – Max = 4 – 16, $\alpha = .64$). By creating a latent factor of maternal

distress, we combined information from multiple overlapping but unique measures of maternal

psychological functioning, and addressed low reliability of the abbreviated scales implemented

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in the FFCWS (e.g., $\alpha < .6$ for the PSI at both ages one and three). Moreover, in several

structural models of psychopathology, anxiety and depression load onto a single internalizing

factor where the shared phenotype underlying both disorders is negative affect (Krueger &

Markon, 2006). The FSM predicts that when economic pressure is high, parents are at increased

risk for emotional distress, which Conger and colleagues (2010) broadly define to include

anxiety and depression as well as alienation. Consistent with prior cross-sectional tests of the

FSM (Gershoff et al., 2007), we included parenting stress as an additional indicator of negative

affect (Deater–Deckard, 2008). Results from the confirmatory factor analysis of latent maternal

distress are presented in Supplemental Figure 2.1. To decrease overall model complexity in tests

of the FSM, we extracted the resultant factor scores for each participant at each age for

subsequent analyses.

Parenting. Maternal warmth and harshness were measured using interviewer-reported

items from the Home Observation for Measurement of the Environment (HOME) scales and the

parent-report Conflict Tactics Scale (CTS) during the home visits at ages 3 and 5 (Caldwell &

Bradley, 1984; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). Maternal warmth (e.g.,

"Parent spontaneously vocalized to child twice") was captured as a sum of nine dichotomous

(Yes/No) items at age 3 ($M = 7.92$, $SD = 1.74$, Min – Max $= 0 – 9$, $\alpha = .79$) and a sum of eight

dichotomous (Yes/No) items at age 5 ($M = 6.15$, $SD = 2.07$, Min – Max $= 0 – 8$, $\alpha = .80$).

Maternal harsh parenting at both timepoints was captured using (a) a sum score of four

dichotomous (Yes/No) items from the HOME scales (e.g., "parent shouted at child", "parent

expressed annoyance or hostility towards child") (Age 3: $M = .35$, $SD = .83$, Min – Max $= 0 – 4$,

$\alpha = .73$; Age 5: $M = .40$, $SD = .87$, Min – Max $= 0 – 4$, $\alpha = .72$), and (b) a mean score of 10

parent-reported items from the CTS, measuring both physical and psychological aggression (Age

3: $M = 1.41$, $SD = .90$, Min – Max $= 0 – 5.30$, $\alpha = .76$; Age 5: $M = 1.46$, $SD = .90$, Min – Max $=$ 0 – 4.90, $\alpha = .77$). Total scores from the HOME and CTS at each age were z-scored and added together to create a multi-informant measure of maternal harsh parenting (Möller, Nikolić, Majdandžić, & Bögels, 2016).

       Child Behavior. Internalizing and externalizing behaviors at ages 5 and 9 were measured using parent-report of an abbreviated version of the Child Behavior Checklist/6-18 (CBCL) (Achenbach & Rescorla, 2001). Seventy-two (age 5) and 111 items (age 9) were administered to parents, who were asked to rate child behavior from zero (*Not true*) to two (*Very true*). To maintain consistency across both assessment periods, we only used the items that were collected at both ages (see Appendix A). Internalizing behaviors were constructed as mean scores of items from the anxious/depressed, withdrawn/depressed, and somatic problems subscales (Age 5 INT: $M = .20$, $SD = .18$, Min – Max $= 0 – 1.27$, $\alpha = .65$; Age 9 INT: $M = .22$, $SD = .24$], Min – Max $=$ 0 – 2, $\alpha = .87$). Externalizing behaviors were constructed as mean scores of items from the aggressive behavior and rule-breaking subscales (Age 5: $M = .27$, $SD = .22$, Min – Max $= 0 –$ 1.60, $\alpha = .85$; Age 9: $M = .19$, $SD = .22$, Min – Max $= 0 – 2$, $\alpha = .87$).

Analytic Strategy

       We employed structural equation modeling in Mplus version 7.2 (Muthén & Muthén, 2006) to evaluate (1) a longitudinal FSM, with the constructs of interest measured at consecutive ages from childbirth through age nine, and (2) a longitudinal *change* FSM model, which included the lagged constructs of interest at the preceding time points from childbirth through age nine (see Figure 2.1 for the conceptual model). We present the results of our path models using the unweighted survey data to maintain the sociodemographic diversity of the sample that is relevant for an examination of the FSM, and because our sample restrictions (e.g., families where

biological mother was the primary caregiver at all waves) would not be captured by the sampling weights.

To account for a missing data (Supplemental Table 2.1), all statistical analyses used full information maximum likelihood (FIML) estimation. FIML uses the covariance matrix of all available data to produce unbiased estimates and standard errors in the context of missing data (McCartney, Burchinal, & Bub, 2006), and has been shown to be a more powerful method of dealing with missing data than listwise deletion or imputation (J. W. Graham, 2009). Simulation studies indicate that FIML estimation provides unbiased estimates with greater power than listwise deletion even when up to 50% of data are missing (Enders & Bandalos, 2001; Schafer & Graham, 2002). Although covariance coverage was acceptable for FIML (>60% for all variables), we included auxiliary variables at ages 3 and 5 that were predictive of missingness as suggested by Graham (2009). At ages 3 and 5, families without observational data reported living in less poverty (Age 3: $t[4229] = 6.27$, $p < .001$; Age 5: $t[4137] = 5.96$, $p < .001$), living in households with less children (Age 3: $t[4209] = -6.31$, $p < .001$; Age 5: $t[4113] = -5.91$, $p < .001$) and more adults (Age 3 only: $t[4209] = 3.95$, $p < .01$), and were more likely to be married (Age 3: $\chi^2[1] = 38.91$, $p < .001$; Age 5: $\chi^2[1] = 35.47$, $p < .001$). Thus, the number of children and adults living in the household, family poverty ratio, and mother marital status (0 = unmarried, 1 = married) at ages 3 and 5 were included as auxiliary variables in all models. All models included child gender (0 = *girl* and 1 = *boy*) as a covariate.

Aim 1: Longitudinal FSM. Our strategy in testing path models was to include every possible path from predictors to outcomes (e.g., for internalizing and externalizing outcomes at age 9, we included direct paths from all mediators including SES at childbirth), followed by pruning of non-significant paths to improve model fit when necessary. We also modeled the

covariance between the four SES variables at childbirth so that significant paths from any of the SES variables to outcomes represented unique estimates over and above their shared covariance. We considered model fit acceptable if the Root Mean Square Error of Approximation (RMSEA) and Comparative Fit Index (CFI) met established guidelines (RMSEA < .06, and CFI > .95; Hu & Bentler, 1999). We were particularly interested in testing indirect effects from SES at childbirth to child internalizing and externalizing behaviors at age 9 via economic pressure at age 1, maternal distress at age 3, and maternal harsh parenting and low maternal warmth at age 5. We employed two complementary methods of testing indirect effects in Mplus v7.2 (Muthén & Muthén, 2006): (i) a product coefficient test (i.e., the 'Sobel method') to quantify the magnitude of the indirect effect (which can be less powerful and biased, but does provide an effect size), and (ii) unbiased confidence intervals using bootstrapping methods (i.e., 1000 draws of a Monte Carlo simulation), which do not assume normality of the indirect effects and thus represent more accurate and powerful tests of indirect, mediated pathways (Dearing & Hamilton, 2006).

Aim 2: Longitudinal change FSM. To test for longitudinal change (e.g., economic pressure at age 1 predicts increases in maternal distress from age 1 to 3), we included measures of maternal distress at age 1, parenting at age 3, and child internalizing and externalizing behaviors at age 5 as predictors of the same constructs at subsequent time periods (see Figure 2.1 for conceptual model). We allowed constructs that were measured at the same age (e.g., economic pressure and maternal distress at age 1) to be correlated. A fully recursive model, where we regressed youth socioemotional outcomes at age 9 on all lagged constructs (e.g., maternal distress at age 1) is not presented because model fit was worse than our hypothesized models (i.e., higher RMSEA, and lower CFI and TLI) and these paths were non-significant (results available upon request).

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Results

Zero-order correlations between all variables included in the FSM are displayed in Table 2.1. With a large sample size, most estimates will be significant at the $p < .05$ level; thus, we focus on effect sizes. As expected, SES indicators at childbirth (i.e., family income to needs ratio, high maternal education, maternal age, and maternal marital status [married]) were moderately correlated $(.33 < r < .51)$[1]. Lower SES at childbirth was weakly to moderately associated with greater economic pressure at age 1 and maternal distress at age 3 $(.05 < r < .18)$, and weakly to moderately associated with lower maternal warmth and higher maternal harsh parenting at age 5 $(.07 < r < .18)$. Lower SES at childbirth was consistently associated with externalizing behaviors $(.07 < r < .11)$ but not internalizing behaviors at age 9 (Table 2.1).

Longitudinal FSM

We specified a longitudinal FSM with constructs measured from childbirth through age 9. Figure 2.2 displays the results of our path analyses. Consistent with our hypotheses that multiple indicators of SES would be relevant for the FSM, we found that family income to needs ratio ($\beta = -.14$, $p < .001$) and biological mother single or cohabitating ($\beta = .10$, $p < .001$) at the target child's birth each uniquely predicted greater economic pressure at age 1. Greater economic pressure at age 1 predicted greater maternal distress at age 3 ($\beta = .28$, $p < .001$) which, in turn, predicted lower observed maternal warmth ($\beta = -.08$, $p < .001$) and greater maternal harshness ($\beta = .09$, $p < .01$) at age 5. Low maternal warmth and high maternal harshness at age 5 each uniquely predicted greater child externalizing but not internalizing behaviors at age 9, with the largest effect size between harsh parenting and child externalizing behaviors (harshness: $\beta = .14$,

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[1] One concern with including multiple indicators of socioeconomic disadvantage in the same model is high multicollinearity, where correlations between exogenous indicators larger than .50 increase the Type II error rate (Grewal, Cote, & Baumgartner, 2004). As the largest correlation among sociodemographic indicators in our sample was .51 (Table 2.1), we do not believe multicollinearity is a threat to our findings.

p < .001; warmth: β = -.08, *p* < .01). There were also direct effects from economic pressure at age 1 and maternal distress at age 3 to greater child behavioral problems at age 9 (.09 < β < .12; *p*s > .01); the effect sizes of these direct effects were similar to those linking parenting to child outcomes, suggesting that contextual factors earlier in development and parenting in later childhood may be equally important in predicting youth internalizing and externalizing behaviors at age 9 (Figure 2.2).

In contrast to zero-order correlations, there were no direct effects of SES at childbirth on youth externalizing behaviors at age 9; these effects operated indirectly via parenting and economic pressure (Table 2.1). Mother marital status (single or cohabitating with a partner) and low maternal education, but not low family income-to-needs ratio or mother age, at childbirth predicted greater child externalizing (but not internalizing) behaviors at age 9 via lower maternal warmth at age 5 (aβs = .01, *p*s < .05). Younger mother age at childbirth also predicted greater child externalizing behavior at age 9 via higher maternal harshness at age 5 (aβ = .01, *p* < .05). In addition to parenting, economic pressure mediated SES pathways to youth externalizing and internalizing behaviors: mother marital status and low family income-to-needs ratio at childbirth each predicted greater youth externalizing and internalizing behaviors at age 9 via greater economic pressure at age 1 (Table 2.1).

Longitudinal *Change* FSM

Next, we tested a longitudinal *change* FSM, where the lagged constructs of maternal distress, parenting, and child behaviors were included in the path analysis. Figure 2.3 shows that after accounting for maternal distress at age 1, economic pressure at age 1 did not predict maternal distress at age 3. However, increases in maternal distress from ages 1 to 3 predicted decreases in maternal warmth (but not increases in maternal harshness) from ages 3 to 5 (β = -

27

.08, $p < .01$). Moreover, changes in maternal warmth ($\beta = -.08$, $p < .01$) and maternal harshness ($\beta = .06$, $p < .01$) from ages 3 to 5 predicted changes in child externalizing behaviors from ages 5 to 9 (i.e., greater increases in maternal warmth and decreases in maternal harshness led to greater decreases in externalizing behaviors) (Figure 2.3).

After accounting for the lagged FSM constructs, maternal marital status (single or cohabitating versus married) and low maternal education at childbirth continued to predict greater increases in externalizing behaviors from ages 5 to 9 indirectly via greater decreases in maternal warmth from ages 3 to 5 (Table 2.1). Similarly, economic pressure at age 1 continued to mediate the effects of maternal marital status and low family income-to-needs ratio at childbirth on youth externalizing and internalizing behaviors at age 9 (Table 2.1). The only indirect effect that became nonsignificant after accounting for the lagged FSM constructs was younger mother age at childbirth to youth externalizing behaviors at age 9 via maternal harshness at age 5.

Post-hoc Analyses: Moderation by Race and Ethnicity

Income disparities by race and ethnicity (where Black Americans and Hispanic Americans disproportionately live below the poverty line) (Semega, Fontenot, & Kollar, 2017) and cultural differences (e.g., the role of extended family networks) suggest that some aspects of the FSM may vary across subpopulations (Cassells & Evans, 2017), though there have been no examinations of whether race and ethnicity moderates the FSM pathways within a longitudinal framework. Therefore, as a post-hoc analysis where we did not have any clear hypotheses, we took advantage of the racial and ethnic diversity in the FFCWS to examine whether any of the "core FSM" pathways in the longitudinal FSM were significantly different among Black Non-Hispanic families ($N = 1544$), White Non-Hispanic families ($N = 541$), and families of Hispanic

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origin (N = 738). Moderation was tested by comparing model fit ($\Delta\chi^2$) of a freely estimated

model (i.e., every path allowed to vary across groups) to a model where one of the core FSM

paths was fixed across groups. Figure 2.4 shows the results of the longitudinal FSM freely

estimated across White Non-Hispanics, Black Non-Hispanics, and participants of Hispanic

origin. With regards to the paths from economic hardship at birth to economic pressure at age 1,

only maternal marital status at birth to economic pressure at age one was moderated by

race/ethnicity ($\Delta\chi^2[2]$ = 13.53, p < .001). Single or cohabitating mothers at birth reported greater

economic pressure at age 1, and this relation was only significant for White Non-Hispanic

families (β = .30, p <.001). The paths from economic pressure at age 1 to maternal distress at age

3, as well as maternal distress at age 3 to parenting behaviors at age 5, were not moderated by

race/ethnicity. Lastly, the paths from harsh parenting ($\Delta\chi^2[2]$ = 9.56, p < .01) and maternal

warmth ($\Delta\chi^2[2]$ = 11.68, p < .001) at age 5 to child internalizing behaviors at age 9 were

significantly moderated by race and ethnicity. Greater maternal warmth at age 5 was associated

with lesser child internalizing among Black Non-Hispanic families (β = -.09, p < .05) and White

Non-Hispanic families (β = -.16, p < .01), with no effect among Hispanic families. Similarly,

harsher parenting at age 5 was significantly associated with greater child internalizing behaviors

at age 9 among White Non-Hispanic (β = .12, p < .05) and Hispanic families only (β = .18, p <

.01). There were no significant indirect pathways from economic hardship variables at childbirth

to youth behaviors at age 9 among any of the racial and ethnic groups.

Discussion

The current paper tested a longitudinal FSM in a large, population-based sample of urban

U.S. families from childbirth through age 9 and included measures of socioeconomic

disadvantage that extended beyond economic aspects of SES. We found evidence for direct

effects of economic pressure, maternal distress, and maternal parenting behaviors across childhood on children's socioemotional outcomes at age 9, as well as indirect effects of socioeconomic disadvantage at childbirth on youth outcomes via parenting and economic pressure. In our most rigorous model that accounted for stability in constructs over time, maternal marital status (single or cohabitating) and low maternal education at childbirth predicted increases in child externalizing behaviors from ages 5 to 9 via decreases in maternal warmth from ages 3 to 5, highlighting the importance of non-economic indicators for the FSM (Bradley & Corwyn, 2002; Duncan & Magnuson, 2003). Our results provide support for the centrality of parenting within the FSM, while also highlighting the relevance of economic pressure and maternal distress in pathways linking socioeconomic disadvantage to youth socioemotional outcomes across childhood.

Although prior tests of the FSM have predominately relied on economic aspects of SES (i.e., family income) to probe the effects of socioeconomic disadvantage on youth outcomes, our results suggest that maternal marital status and education at childbirth are unique contributors to the FSM. Mothers who are married oftentimes have more non-economic resources than single-parent or cohabitating parents, including social support and lower psychological distress (S. L. Brown, 2004; McLanahan & Sandefur, 1994). Similarly, higher maternal education confers additional cognitive resources that can be used to strategize in times of economic distress, as well as parent more effectively (Bradley & Corwyn, 2002). Although family income-to-needs ratio at childbirth was associated with greater economic pressure at age 1, maternal distress at age 3, and maternal warmth at age 5, it did not predict youth outcomes via parenting, which is in contrast to most previous tests of the FSM (Conger et al., 2010). In supplemental analyses that did not include maternal age or marital status at childbirth in the longitudinal FSM, the indirect effects

from low family income-to-needs ratio at childbirth to greater youth externalizing behaviors at age 9 via low maternal warmth and high harsh parenting at age 5 were significant ($\alpha\beta$s = .01, ps < .01; results available upon request). This suggests that after accounting for the covariance between family income-to-needs ratio and marital status, maternal marital status and education may be more proximal predictors of youth externalizing behaviors within the FSM in the FFCWS. Although the addition of non-economic indicators to the FSM may be considered a departure from the original FSM, our results suggest that future tests of this family process model should include indicators of socioeconomic disadvantage that extend beyond family income.

This study also contributed to the existing FSM literature by testing both a longitudinal model and a longitudinal change model (i.e., a cross-lagged model). When the lagged constructs of maternal distress, parenting, and child socioemotional outcomes were added to the longitudinal model, some path estimates became non-significant: economic pressure at age 1 was not associated with maternal distress at age 3, and maternal distress at age 3 only predicted maternal warmth, and not harshness, at age 5. Changes in the indirect effects were also observed: once the lagged FSM constructs were added to the longitudinal model, maternal age at childbirth no longer predicted youth externalizing behaviors via maternal harsh parenting. However, because all of the indirect effects were of such small magnitude (all aβs = .01), these changes may not be meaningful. Generally, across both modeling frameworks, socioeconomic disadvantage at childbirth appeared to have indirect effects on youth socioemotional outcomes at age 9 via maternal parenting behaviors at age 5 and economic pressure at age 1.

Although the FSM does not strictly hypothesize differential effects of harsh and warm parenting for youth internalizing and externalizing outcomes (Masarik & Conger, 2017), our

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results suggest otherwise. Indeed, several previous tests of the FSM measured socioemotional competence as a latent factor that included internalizing and externalizing behaviors (e.g., Raver et al., 2007), and parenting behaviors as a latent factor that included harsh and warm parenting (Landers-Potts et al., 2015). Yet among the existing longitudinal models that distinguish parenting dimensions, maternal distress has been shown to predict less sensitive but not more harsh parenting (Newland et al., 2013), a finding we replicated in both the longitudinal and longitudinal change FSMs. Only one previous longitudinal test of the FSM distinguished youth socioemotional competence along internalizing and externalizing domains: White et al. (2015) found that mothers' perceptions of economic pressure predicted youth externalizing behaviors, but not internalizing behaviors, via maternal harshness. The indirect effects via parenting in our models were also specific to youth externalizing outcomes. By contrast, economic pressure at age 1 mediated the effects of socioeconomic disadvantage at childbirth on youth externalizing *and* internalizing behaviors at age 9, consistent with previous research (Hobbs & King, 2018; Whitaker, Phillips, & Orzol, 2006; Zilanawala & Pilkauskas, 2012).

The effects of socioeconomic disadvantage at childbirth on youth socioemotional outcomes operated through parenting and economic pressure (i.e., there were no direct effects of family income-to-needs ratio, maternal education, marital status, or maternal age at childbirth to youth socioemotional behaviors at age 9). Though these findings support the mediation effects hypothesized by the FSM (i.e., via parenting), the indirect pathways via economic pressure and maternal warmth were of similar magnitude, suggesting that both constructs are important to the FSM. We also observed modest *direct* effects of economic pressure and maternal distress across childhood on youth socioemotional outcomes at age 9. That is, we found evidence that economic pressure and maternal distress may predict youth outcomes via mechanisms outside of parenting.

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This result is difficult to compare with previous tests of the FSM model as results have been

mixed – some studies have not tested for these direct effects (e.g., Conger et al., 2002), some

have found evidence for them (e.g., Kavanaugh et al., 2018), and others have not (e.g., Diggs &

Neppl, 2018). However, there is good reason to believe that economic pressure and maternal

distress may predict youth outcomes via multiple pathways. Mother-reported economic pressure

in the current study included items that asked parents about material hardship, eviction, and food

insecurity (Bauman, 1998; Social Indicators Survey Center, Columbia University School of

Social Work, 1999). Several reviews show that low SES increases risk for child socioemotional

problems through physical environmental exposures (e.g., housing quality, toxicant exposure) in

addition to interpersonal processes within the parent-child relationship (Evans, 2004; Evans &

Kantrowitz, 2002). Indeed, recent studies in the FFCWS have shown that adverse housing

conditions (Jackson, Newsome, & Lynch, 2017) and food insecurity (Hobbs & King, 2018),

significantly increase risk for youth socioemotional problems. Future research should integrate

physical environment exposures with interpersonal processes to better understand pathways from

economic hardship to youth socioemotional outcomes.

The indirect effects from maternal marital status and maternal education at childbirth to

youth externalizing behaviors at age 9 via parenting at age 5 did not fully explain the

associations between socioeconomic disadvantage and youth outcomes. In the longitudinal

change FSM, the indirect effects via parenting explained roughly 50% of the total effect from

SES at childbirth to youth outcomes, suggesting sizeable contributions from the direct effects

(e.g., economic pressure at age 1, maternal distress at age 3, parenting at age 5). Indeed, previous

longitudinal tests of the FSM that examined this model across timespans of more than a few

years report similarly small or non-significant indirect effects of parenting (Diggs & Neppl,

2018; Kavanaugh et al., 2018; Neppl et al., 2016; R. M. B. White et al., 2015). By contrast, Landers-Potts et al., (2015), whose longitudinal test of the FSM measured constructs only two years apart, reported much larger effect sizes ($a\beta$ = .16, 85% of the total effect) for the indirect effects of economic pressure on youth outcomes via parenting (see also Henninger & Luze, 2014). These studies thus suggest that the hypothesized indirect effects of SES on youth outcomes via parenting, as outlined in the FSM, may operate along short timeframes (e.g., on the order of weeks or months or a year or two). Alternatively, though our inclusion of both warm and harsh parenting behaviors was a strength of our study, other unmeasured aspects of parenting in early childhood (e.g., positive behavior support strategies) may be important in mediating the impact of socioeconomic disadvantage on youth socioemotional outcomes (Dishion et al., 2008). More research is needed to investigate whether the small indirect effects observed in the current analyses result from a discrepancy in the time scale of such family processes, or whether there are other mechanisms (e.g., access to quality childcare, variation in material hardship, parenting behaviors beyond harshness and warmth) linking socioeconomic disadvantage to youth socioemotional outcomes.

Small indirect effects via parenting do not discount the importance of parenting behaviors in the development of youth socioemotional outcomes. By contrast, in our models, both harsh and warm parenting at age 5 exerted modest effects on youth externalizing behaviors at age 9. Of course, one limitation of these findings is that they could reflect gene-environment correlation – that parents who are harsh also pass genetic risk for such aggression to their children, who display higher levels of externalizing behaviors (Manuck & McCaffery, 2014). However, several studies that have used adoption designs to disambiguate genetic and environmental influences on youth psychopathology, report effects of adoptive parents' behavior (e.g., warmth) on child

socioemotional outcomes (e.g., Hyde et al., 2016). Parents are critical regulators of context

effects on children (Conger et al., 2010; McLoyd, 1990, 1998), particularly during early

childhood when children spend much of their time in the home (Shaw & Bell, 1993).

Importantly, the relations between parenting behaviors and child socioemotional outcomes were

continuous; greater maternal warmth and lower harsh parenting predicted fewer child

internalizing and externalizing behaviors. Thus, framing our results within a resilience

framework highlights maternal warmth as promotive factor for families living in poverty (Ager,

2013; Kim-Cohen, Moffitt, Caspi, & Taylor, 2004).

Post-hoc moderation analyses of the FSM by race and ethnicity showed some of the core

FSM pathways differed across groups. This study is the first to test moderation by race and

ethnicity within a longitudinal FSM with several interesting results: First, maternal marital status

(single or cohabitating versus married) at childbirth was associated with economic pressure at

age 1 only among White non-Hispanic families than other groups. It may be that ethnic minority

mothers rely more on extended kinship networks, as well as having larger immediate families,

which functions as an adaptive strategy to pool resources (Garcia Coll, 1990; MacPhee, Fritz, &

Miller-Heyl, 1996). Second, although maternal warmth and harshness at age 5 were associated

with child internalizing behaviors at age 9 for White Non-Hispanic families, these associations

were not consistent for Non-Hispanic Black families and Hispanic families. In a cross-sectional

FSM, Raver and colleagues (2007) also found stronger effects of parental warmth on child

socioemotional competence among White families than among families of color. Parental

warmth may be expressed differently in African American and Latino families (Chao & Otsuki-

Clutter, 2011); for example, children in families of color may perceive acceptance and emotional

security through high behavioral expectations rather than expressions of affection and praise

(Hill, Bush, & Roosa, 2003). The differential associations between parenting behaviors and youth internalizing outcomes by race and ethnicity in our study suggest that more research is needed to evaluate the measurement and meaning of parenting behaviors across cultural groups.

Limitations

Several limitations of this study are noteworthy. First, as is common in survey research, the study implemented abbreviated measures of our constructs of interest, which resulted in lower than expected scale reliabilities (e.g., there were only 10 of the original 13 items from the internalizing subscales of the Child Behavior Checklist [Achenbach & Rescorla, 2001] available at both assessment periods; total sample $\alpha = .69$). Thus, the abbreviated measurement of constructs and low scale reliability may have contributed to some of the non-significant outcomes (e.g., child internalizing behaviors) and mediators (e.g., maternal distress) in our models. Moreover, although we only used items from the Child Behavior Checklist that were measured at both ages 5 and 9 to maintain consistency across assessment periods, age-specific items may have developmental significance (Widaman, Ferrer, & Conger, 2010). In follow-up analyses that utilized all available items at both ages, results from the longitudinal FSM were largely convergent, though the effect sizes of the paths from contextual variables to youth psychopathology were attenuated. The abbreviated version of the Child Behavior Checklist also prevented us from examining whether youth psychopathology was in the clinical range. Thus, future longitudinal examinations of the FSM should integrate additional measures of internalizing and externalizing behaviors, including clinical assessments where possible, to evaluate a range of youth psychopathology. Second, original conceptions of the FSM included interparental conflict within married dyads as an additional mediator linking socioeconomic disadvantage to youth outcomes, which we did not include in the present paper. As the FFCWS

purposely oversampled non-marital births as part of the original study design (Reichman et al., 2001) and nearly 80% of the mothers in our sample were unmarried at childbirth, we did not want to introduce uncertainty into our models with changing partner dyads over time. Although we believe the strengths of the FFCWS dataset outweigh this limitation, interparental conflict is an important predictor of youth socioemotional outcomes (Spjeldnes & Choi, 2008) and should be studied further within the FSM using large, population-based datasets. Third, though we used both observational and self-report measures of parenting in our models, many families did not participate in the in-home interview at age 3 and/or 5. To account for this missing data, we excluded families without at least one wave of observational data and in doing so, altered the composition of the sample from the original FFCWS. That data were systematically missing may undermine the representativeness of the sample (Groves et al., 2009). Finally, there is increasing concern that the regression parameters from cross-lagged panel models include both within-person and between-person effects (Berry & Willoughby, 2017; Curran & Bauer, 2011; E. L. Hamaker, Kuiper, & Grasman, 2015). As a result, it is unclear whether the patterns observed in such models reflect stable between-person differences and/or time-specific within-person processes. Though alternative models can be used to disambiguate between- from within-person variance (e.g., the random intercepts cross-lagged panel model, Hamaker et al. [2015]; the autoregressive latent trajectory model, Bollen & Curran [2004]), these models require at least three waves of data. In the current study, we had specific hypotheses about the relevance of the FSM during the first decade of a child's life (Eisenberg et al., 1998; Shaw & Bell, 1993), leaving us with fewer measurement time points. Moreover, alternative modeling strategies have relied on a small number of constructs; our examination of seven major constructs in the model may have made this complex modeling of change unwieldy. Future studies should integrate these

alternative longitudinal modeling techniques, but this will require dense observations of the FSM constructs during relevant developmental periods (i.e., as we argue, early and middle childhood). Thus, results from the current paper should be interpreted as population-level estimates averaged across individuals.

The current test of a longitudinal FSM in a relevant population-based sample of urban U.S. births contributes to our understanding of the longitudinal relations between socioeconomic disadvantage and youth socioemotional outcomes. The study leveraged the diversity and sample size of the Fragile Families and Child Wellbeing Study dataset to examine the unique influences of multiple indicators of SES to family processes, and to construct a rigorous longitudinal change FSM model that accounted for stability in constructs. Results highlight the importance of replicating and extending existing psychological theories in large, population-based studies. In the case of the FSM, more research is needed to understand the timescale within which these family processes operate. As our results show, however, Moreover, the current results emphasize that economic pressure, parent psychological functioning, and parenting behaviors exert durable effects on youth socioemotional outcomes across the first 9 years of life.

Table 2.1. Zero-order correlations between variables included in the Family Stress Model

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
Childbirth	1. Family poverty ratio	-									
	2. Maternal education	.51***	-								
	3. Maternal age (years)	.33***	.37***	-							
	4. Mother married	.40***	.35***	.37***	-						
Age 1	5. Economic pressure	-.17***	-.09***	-.05*	-.18***	-					
Age 3	6. Maternal distress	-.10***	-.09***	-.05**	-.06**	.29***	-				
Age 5	7. Maternal warmth	.17***	.18***	.12***	.15***	-.02	-.09***	-			
	8. Harsh parenting	-.11***	-.07**	-.11***	-.10***	.07**	.10***	-.26***	-		
Age 9	9. Child internalizing	.05*	-.03	-.01	.01	.11***	.13***	-.05*	.05	-	
	10. Child externalizing	-.11**	-.08***	-.09***	-.07**	.15***	.18***	-.14***	.20***	.60***	-

Note. $1433 < N < 2917$ Spearman rank correlations were used for associations with mother marital status (dichotomous).
*$p<.05$, **$p<.01$ ***$p<.001$

Table 2.2. Indirect effects linking socioeconomic disadvantage at childbirth to youth socioemotional outcomes at age 9

	Longitudinal Model	Longitudinal Change Model
	Estimate (SE), Bootstrapped [95% CI]	Estimate (SE), Bootstrapped [95% CI]
Outcome: Youth Externalizing Behaviors at Age 9		
Predictor: Low Family Income-to-Needs at Childbirth		
Indirect Effect via Economic Pressure at Age 1	.01 (.004)**, [.006, .022]	.01 (.003)**, [.004, .017]
Predictor: Mother Marital Status (Single or Cohabitating) at Childbirth		
Indirect Effect via Maternal Warmth at Age 5	.01 (.002)*, [.001, .010]	.01 (.002)*, [.001, .009]
Indirect Effect via Economic Pressure at Age 1	.01 (.004)**, [.003, .018]	.01 (.003)**, [.002, .015]
Predictor: Low Maternal Education at Childbirth		
Indirect Effect via Maternal Warmth at Age 5	.01 (.003)*, [.001, 014]	.01 (.003)*, [.001, .012]
Predictor: Young Mother Age at Childbirth		
Indirect Effect via Maternal Harsh Parenting at Age 5	.01 (.004)*, [.002, .020]	ns
Outcome: Youth Internalizing Behaviors at Age 9		
Predictor: Low Family Income-to-Needs at Childbirth		
Indirect Effect via Economic Pressure at Age 1	.01 (.004)**, [.004, .020]	.01 (.003)**, [.002, .015]
Predictor: Mother Marital Status (Single or Cohabitating) at Childbirth		
Indirect Effect via Economic Pressure at Age 1	.01 (.003)*, [.002, .016]	.01 (.003)*, [.002, .013]

Note. $N = 2,918$. Standardized estimates are shown. For indirect effects, we provide the unbiased bootstrapped CI of this effect ($p<.05$) as well as an estimate of the product coefficients ($\alpha\beta$) (i.e., the 'sobel test') as an index of gross effect size.
* p < .05; ** p< .01
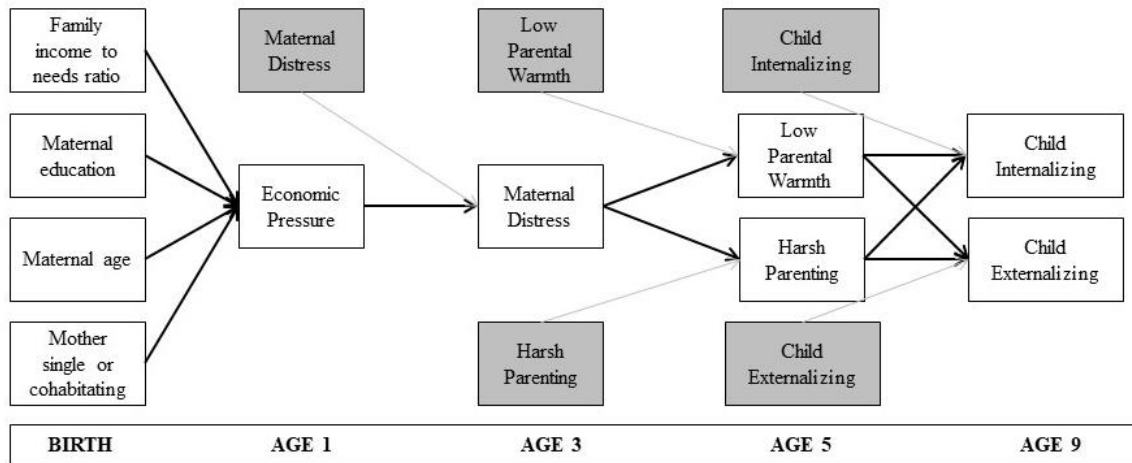
Figure 2.1. Conceptual Model
Aim 1 tested of the longitudinal family stress model (FSM) across childhood (from childbirth through age nine), as indicated by the constructs in white boxes. In Aim 2, we tested a longitudinal *change* FSM by adding the lagged constructs of maternal distress, harsh parenting, maternal warmth, and child internalizing and externalizing behaviors, as indicated by shaded gray boxes
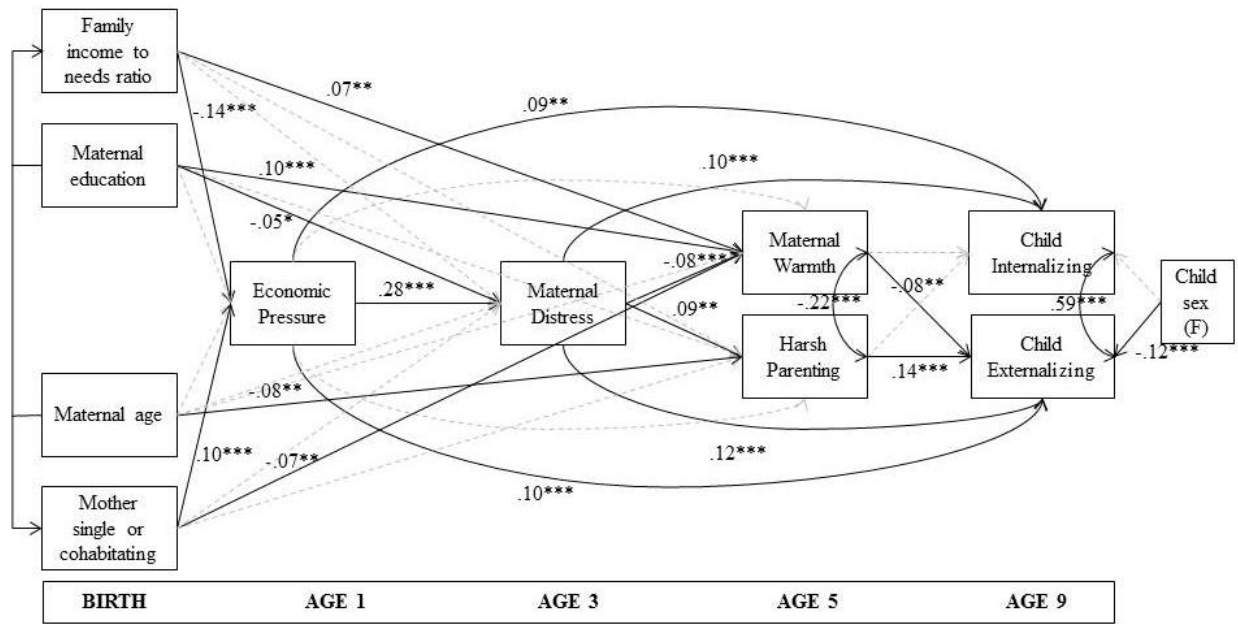
Figure 2.2. Path model testing the longitudinal family stress model
*p<.05, **p<.01, ***p<.001; Paths are marked with standardized estimates.
N=2,918. Model fit: X²(8) = 17.26, *p* = .03, RMSEA=.020, 90%CI [.01, .03], CFI=1.00, TLI=.97. Non-significant paths that were also modeled but not pictured: All measures of socioeconomic disadvantage at childbirth to child internalizing and externalizing at age 9.

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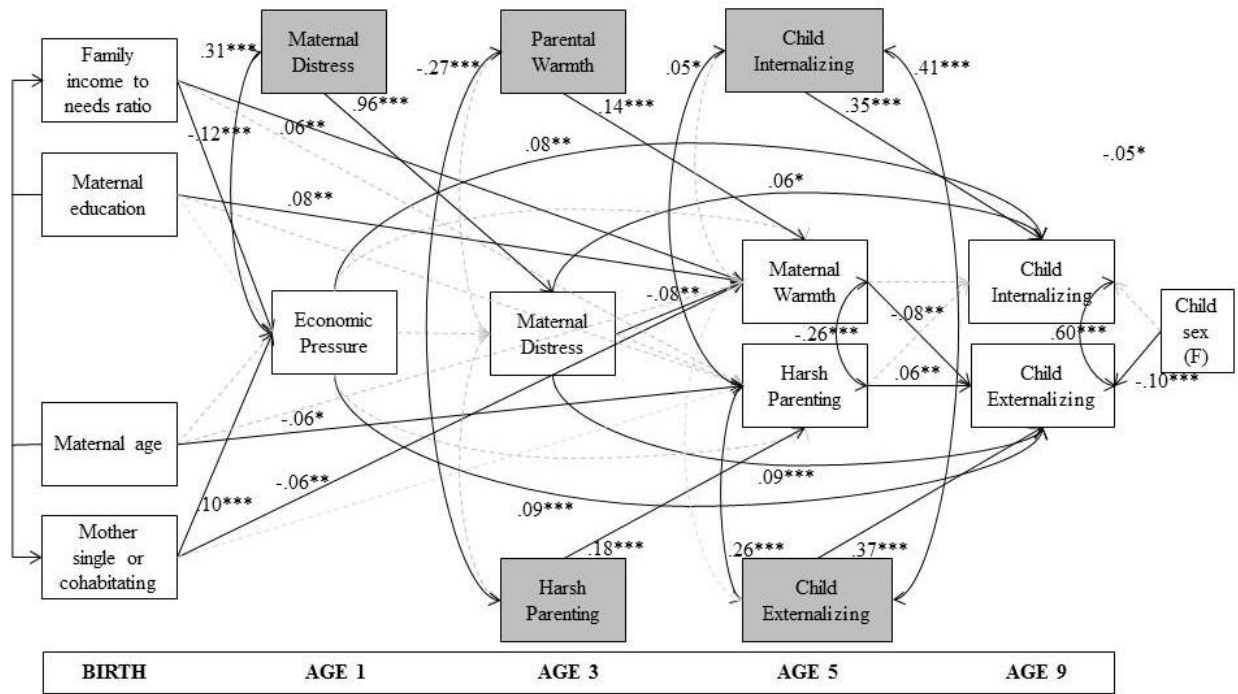
Figure 2.3. Path model testing the longitudinal change family stress model
*p<.05, **p<.01, ***p<.001; Paths are marked with standardized estimates.
N=2,918. Model fit: X²(60) = 628.46, *p* < .001, RMSEA=.06, 90%CI [.053, .061], CFI = .94, TLI = .92. Non-significant paths that were also modeled but not pictured: All measures of socioeconomic disadvantage at childbirth to maternal distress at age 3, and child internalizing and externalizing at age 9.

White: B(SE)= -.11(.03) β = -.21***
Black: B(SE)= -.06(.03) β= -.06
Hispanic: B(SE)=-.10(.04) β= -.11**

White: B(SE)= .06(.08) β= .04
Black: B(SE)= -.03(.06) β= -.02
Hispanic: B(SE)= .11(.08) β= .06

White: B(SE)= .12(.02) β= .31***
Black: B(SE)= .11(.01) β= .27***
Hispanic: B(SE)= .09(.01) β= .26***

White: B(SE)= -.003(.01) β= -.03
Black: B(SE)= -.01(.003) β= -.09**
Hispanic: B(SE)= -.01(.01) β= -.08

White: B(SE)= -.07(.14) β = -.03
Black: B(SE)= -.26(.11) β = -.08*
Hispanic: B(SE)= -.38(.15) β=-.11*

White: B(SE)= -.02(.01) β= -.16**
Black: B(SE)= -.01(.004) β= -.09*
Hispanic: B(SE)= -.004(.01) β= -.03

Family income-to-needs ratio			
Maternal education			
Economic Pressure	Maternal Distress	Maternal Warmth	Child Internalizing
		Harsh Parenting	Child Externalizing
Maternal age			
Mother single or cohabitating			

White: B(SE)= .002(.01) β= .01
Black: B(SE)= .02(.01) β= .05
Hispanic: B(SE)= .01(.01) β= .03

White: B(SE)=.08(.11) β= .04
Black: B(SE)= .23(.07) β= .11**
Hispanic: B(SE)= .11(.12) β= .05

White: B(SE)= .02(.01) β= .12*
Black: B(SE)= -.002(.01) β= -.01
Hispanic: B(SE)= .03(.01) β= .18**

White: B(SE)= 1.03(.18) β= .30***
Black: B(SE)= .23(.15) β= -.04
Hispanic: B(SE)= .27(.16) β= .07

White: B(SE)=.03(.01) β= .18**
Black: B(SE)= .02(.01) β= .15***
Hispanic: B(SE)= .03(.01) β= .18***

| BIRTH | AGE 1 | AGE 3 | AGE 5 | AGE 9 |

Figure 2.4. Moderation of the Family Stress Model by Race and Ethnicity
N=2,823 (White non-Hispanic=541; Black non-Hispanic=1544; Hispanic=738).
*p<.05, **p<.01, ***p<.001. Estimator=Full-information maximum likelihood (FIML). Paths are marked with standardized estimates from a freely estimated model (i.e., every path allowed to vary across groups). Moderation by race/ethnicity of the eight "core" family stress model (FSM) paths (indicated by solid black lines) was tested by comparing model fit ($\Delta\chi^2$) of the freely estimated model to a model where one of the core FSM was fixed across groups (this was repeated for each core FSM path). Model fit of the freely estimated model: $\chi^2(24)=33.60$, p=.09, RMSEA=.02 90%CI (.001,.04) CFI=1.00, TLI=.97. Pathways that were significantly moderated by race and ethnicity are outlined in black.

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Supplemental Table 2.1. Degree of missing data across all variables

	Available N (%)
Birth	
Child gender	2,918 (100%)
Mother age	2,917 (99%)
Family income-to-needs ratio	2,918 (100%)
Mother education	2,914 (99%)
Mother marital status	2,902 (99%)
Age 1	
Economic pressure	2,765 (95%)
Maternal distress	2,892 (99%)
Age 3	
Maternal distress	2,892 (99%)
Harsh parenting	1,939 (66%)
Maternal warmth	2,046 (70%)
Age 5	
Harsh parenting	1,722 (60%)
Maternal warmth	2,030 (70%)
Child internalizing behaviors	2,527 (87%)
Child externalizing behaviors	2,527 (90%)
Age 9	
Child internalizing behaviors	2,245 (77%)
Child externalizing behaviors	2,246 (77%)
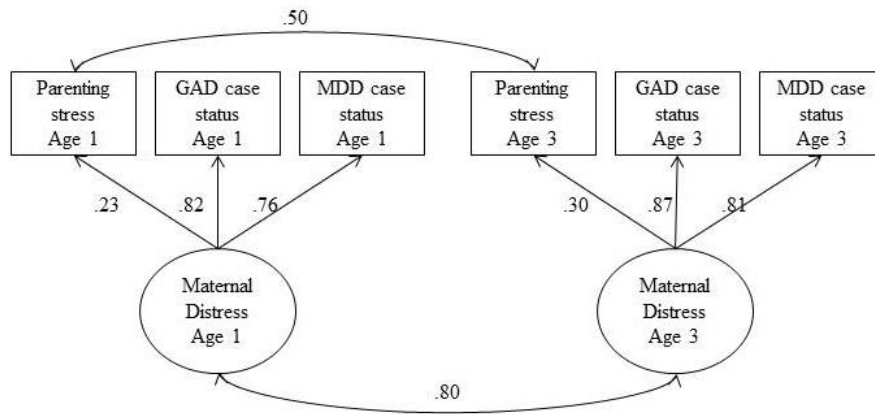
Supplemental Figure 2.1. Confirmatory factor analysis of maternal distress at ages 1 and 3 N=2,892. Standardized estimates are displayed, all significant at *p*<.001. Estimator = Weighted least squares mean- and variance-adjusted. Model fit was excellent: X²(7) = 11.79, *p* > .10, RMSEA = .02, 90%CI [.001, .03], CFI = 1.00, TLI = .99.

CHAPTER III

Prospective Longitudinal Effects of Harsh Parenting on Corticolimbic Function during Adolescence

Parental caregiving is a core determinant of child socioemotional development (Bornstein, 2001; Maccoby, 2000; Patterson, 1982) including children's attachment security (De Wolff & Van IJzendoorn, 1997) and self-regulation (Karreman, Tuijl, Aken, & Deković, 2006). Such socioemotional competencies are associated with lifelong health and socioeconomic outcomes (Kawakami et al., 2012; Moffitt, 2018; Sroufe, 2005), making our understanding of how parenting behaviors contribute to youth outcomes critical. Research is beginning to examine biological mechanisms that may mediate these associations, including stress-related neural function (Belsky & de Haan, 2011; McEwen, 2012; McEwen & McEwen, 2017). Yet much of the research linking parenting to neural function has focused on extreme forms of parenting (e.g., childhood maltreatment, early institutionalization). Even fewer studies have evaluated whether parenting behaviors during the early childhood period exert larger effects on neural function than parenting behaviors thereafter, or whether these associations differ by the neural region (e.g., subcortical, cortical) examined.

Harsh Parenting Across Childhood

One core dimension of parenting behaviors is harshness, characterized by high intrusion, coerciveness, and sometimes physical or verbal aggression (Bugental & Grusec, 2006; Darling & Steinberg, 1993). Overly harsh behaviors create an environment of inconsistency and

unpredictability that results in pronounced child behavioral problems and concomitant changes in biological stress responses (Bugental & Grusec, 2006; Loman & Gunnar, 2010). Several theories of parenting emphasize the importance of parenting behaviors during the early childhood period for the development of children's socioemotional competence (M. S. Ainsworth, 1979; Bowlby, 1982; Maccoby & Martin, 1983; Patterson, 1982; Shaw & Bell, 1993). For example, hostile and rejecting parenting behaviors during toddlerhood, when children become increasingly mobile and autonomous, facilitate coercive family processes that translate into later youth conduct problems (Patterson, 1982; Shaw & Bell, 1993; Shaw, Gilliom, Ingoldsby, & Nagin, 2003). Though harsh parenting behaviors tend to be elevated during toddlerhood and decrease thereafter (Dallaire & Weinraub, 2005; Kim, Pears, Fisher, Connelly, & Landsverk, 2010), few studies have reported how parental harshness changes from early to middle childhood. Most of this work has examined parenting within shorter developmental periods, such as infancy or adolescence only. As parenting behaviors are heavily influenced by child characteristics (e.g., difficult temperament), parent characteristics (e.g., maternal depression), and contextual stress (e.g., economic adversity) (Belsky, 1984), more work is needed to describe initial levels and changes in parental harshness in a large population-based study that is oversampled for families facing such risks (McLanahan & Sandefur, 1994). These patterns of parenting behaviors across childhood, in turn, could be used to probe the timing of parenting effects on brain development.

Neural Structures within the Corticolimbic System

Neural function within the corticolimbic circuit, which includes the amygdala and regions of the prefrontal cortex (PFC), is a plausible mechanism by which harsh parenting affects youth socioemotional outcomes (Callaghan & Tottenham, 2016; Gee, 2016; Perry et al., 2017). The

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amygdala contributes to salience detection and emotional learning through sensitivity to environmental stimuli that signal threat or uncertainty (LeDoux, 2000; Whalen & Phelps, 2009). In humans, the amygdala is robustly activated during functional magnetic resonance imaging (fMRI) tasks where participants either implicitly or explicitly view emotional faces, including fearful and angry facial expressions (Fusar-Poli et al., 2009; Shi, Wang, & Yao, 2013). Medial and lateral regions of the PFC, as well as the anterior cingulate cortex (ACC), support emotion processing by integrating affective valuations from the amygdala with inputs from other neural regions, including the brainstem and thalamus (Egner, Etkin, Gale, & Hirsch, 2007; Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Fuster, 2001; Ochsner et al., 2012). Functional parcellation of the medial frontal lobe suggests a rostral-ventral distinction in emotion processing where the dorsal ACC, dorsal-medial PFC (dmPFC), and dorsal-lateral PFC (dlPFC) support the cognitive components of emotion processing (e.g., appraisal), while the ventral ACC (including the rostral and subgenual components) and the ventro-medial PFC (vmPFC) and ventro-lateral PFC (vlPFC) support emotion regulation (Etkin, Egner, & Kalisch, 2011; Fuster, 2001). Though the amygdala and PFC uniquely contribute to emotion processing, connectivity between these two regions is more likely to explain complex socioemotional behaviors (e.g., psychopathology) that result from the coordinated function of multiple brain regions (Menon, 2011). Bottom-up signaling from the amygdala conveys salient information about emotional stimuli while top-down signaling from the PFC helps to regulate responses to emotional stimuli (Ochsner & Gross, 2005; Quirk, Garcia, & González-Lima, 2006; Stein et al., 2007). Indeed, weaker amygdala-PFC connectivity has been associated with multiple forms of psychopathology that are hallmarked by deficits in emotion processing (Hyde, Shaw, & Hariri, 2013; Kim et al., 2011; Price & Drevets, 2010).

Sensitive Periods for Environmental Effects on the Corticolimbic System

The developmental trajectories of the amygdala and the PFC suggest two developmental periods during which these regions may be more sensitive to the effects of environmental stress. Structurally, the rate of volumetric growth in the amygdala is largest during the early postnatal years (Payne, Machado, Bliwise, & Bachevalier, 2010), increasing in volume by more than 100% during the first year of life (Gilmore et al., 2012), with volumes reaching their peak between ages 10 to 12 in typically-developing humans (Uematsu et al., 2012). This rapid period of growth during the early postnatal years parallels functional patterns of greater amygdala reactivity to emotional facial expressions observed in children than among adolescents and adults (Monk, 2008). Greater sensitivity of the amygdala is thought to support normative childhood fears (e.g., separation anxiety) that peak during infancy and toddlerhood (Gee, 2016). In fact, Gee and colleagues (2013) reported a developmental shift in positive to negative amygdala-mPFC connectivity from childhood to adolescence that was mediated by a reduction in separation anxiety. In this analysis, positive amygdala-mPFC connectivity reflected more positively coupled amygdala and mPFC activation while participants were looking at fearful facial stimuli versus a baseline condition; in adolescents and adults, when amygdala reactivity decreases, the association between activity in these two regions became negative (i.e., thought to reflect lesser amygdala and greater mPFC activation) (Gee et al., 2013). In contrast to the amygdala, however, the PFC continues to develop throughout childhood into adolescence and adulthood (Gogtay et al., 2004; Sowell et al., 2003), with lateral PFC regions the last to develop. For example, gray matter density has been shown to peak during the pre-pubertal stage (i.e., 10 – 12 years), followed by synaptic pruning and dendritic arborization that is correlated with increasing cognitive control across the adolescent period (Andersen & Teicher, 2008; Casey,

Jones, & Hare, 2008; Lenroot & Giedd, 2006). During adolescence, projections from prefrontal regions to other brain regions become more well-defined, as children recruit more diffuse prefrontal regions when performing cognitive tasks than adolescents and adults (Casey, Jones, et al., 2008). For example, structural connectivity of the mPFC with the amygdala, defined by the uncinate fasiculus white matter tract, is stronger among adolescents than among children (Swartz, Carrasco, Wiggins, Thomason, & Monk, 2014) and multiple studies have shown relatively greater mPFC activation in adolescents than among adults (for a review, see Blakemore, 2008).

Though there is much research demonstrating that early life adversity impacts the development of corticolimbic system structure and function (Farah, 2018; Hein & Monk, 2017; Tottenham, 2009) and several recent reviews highlight the importance of timing of these effects (Callaghan & Tottenham, 2016; Gee, 2016; Perry et al., 2017), few studies have tested this hypothesis in humans (for reviews of the non-human literature, see Callaghan & Tottenham, 2016; Debiec & Sullivan, 2017). In one study, Gard et al., (2017) found that harsh parenting at age two predicted lesser amygdala reactivity to fearful facial expressions at age 20, even after accounting for harsh parenting at age 12; however, this paper did not examine PFC function or amygdala-PFC connectivity. Using a different paradigm to examine the timing of parenting effects on youth corticolimbic function, Gee et al., (2014) found that amygdala reactivity was suppressed among children (ages 4 – 10 years) but not adolescents (ages 11 – 17 years) when participants viewed their mother's face versus a stranger's face in the scanner. Moreover, amygdala-mPFC connectivity became more negative among children during the mother face condition, whereas stimulus condition (mother/stranger) did not modulate connectivity among

adolescents (Gee et al., 2014), suggesting that corticolimbic function may be most malleable to environmental experiences in early childhood.

Quantitative Considerations

A challenge in evaluating whether early parenting behaviors exert larger effects on corticolimbic function than parenting behaviors thereafter is determining how to quantitatively test this question. One method is to examine the unique impact of parenting behaviors at discrete time periods (i.e., after accounting for parenting behaviors at the other time periods) (Gard et al., 2017a). However, this strategy assumes that parenting can be parsed into discrete moments in time rather than the notion that parenting behaviors are a product of continuous reciprocal interactions within the changing context (Cicchetti & Toth, 2009). Alternatively, linear growth curve modeling (LGCM) can be used both as a form of data reduction and as a method to describe patterns of parenting behaviors across time (McCartney et al., 2006). An application of structural equation modeling, LGCM estimates of an intercept and a slope across repeated measures of a construct over time. Therefore, LGCM enables researchers to assess the unique impact of initial levels (i.e., intercept) and changes (i.e., slope) in a construct over time on an outcome. This approach may be especially useful for research questions in which we have specific hypotheses about the importance of initial development of a construct (e.g., parenting behaviors that form during the early childhood period) versus changes thereafter (Burchinal, Nelson, & Poe, 2006).

The Present Study

The current study sought to advance our understanding of how parental harshness across childhood impacts corticolimbic function in adolescence. First, in a large, nationally-representative sample of children born in large cities in 1999-2000 with a two-thirds oversample

for non-marital births (i.e., the Fragile Families and Child Wellbeing Study), we examined how parental harshness changes across childhood (i.e., from ages three to nine) using LGCM. Second, we evaluated the effects of initial levels and changes in harsh parenting across childhood on corticolimbic function during adolescence. Though as reviewed above, few studies have empirically tested the importance of timing for parenting or adversity effects on amygdala or PFC function or connectivity, our hypotheses were guided by growing research in this field linking early life stress or adversity during childhood on neural function within these structures. We hypothesized that initial levels of harsh parenting (operationalized by the intercept term from the LGCM estimated in Aim 1) would uniquely predict greater amygdala reactivity to threatening emotional facial expressions (i.e., anger and fear) during adolescence, after accounting for changes in parenting thereafter (i.e., operationalized by the slope term from the LGCM estimated in Aim 1). We also hypothesized that increases in harsh parenting across childhood, after accounting for initial levels, would predict lesser PFC activation (particularly during medial regions) during the processing of threatening emotional facial expressions. Lastly, as adolescents display the "mature" negative amygdala-mPFC connectivity pattern during face processing (Gee et al., 2013), we hypothesized that both initial levels and changes in parental harshness would predict less negative, shifting to positive amygdala-mPFC connectivity during fearful and angry face processing.

Methods

Sample

Participants were part of the Study of Adolescent Neurodevelopment (SAND), a cohort of 237 families drawn from the Detroit, Toledo, and Chicago subsamples of the Fragile Families and Child Wellbeing Study (FFCWS). The core FFCWS is a longitudinal cohort of 4,898 (52.4%

boys) children born in 20 U.S. cities between 1998 and 2000 (Reichman et al., 2001). In line

with the original aims of the core FFCWS, the study oversampled for non-marital births (~ 3:1),

which resulted in substantial sociodemographic diversity in the sample (Reichman et al., 2001).

At the birth of their child, mothers identified as Black Non-Hispanic (N = 2,326, 47.5%), White

Non-Hispanic (N = 1,030, 21.1%), Hispanic (N = 1,336, 27.3%), or other (N = 194, 4.0%).

Nearly 40% of the mothers reported less than a high school education at the birth interview,

25.3% with a high school degree or equivalent, 24.3% some college or technical training, and

10.7% who earned a college degree or higher. Additional details regarding sample characteristics

of the core FFCWS have been reported elsewhere (Reichman et al., 2001). Families in the

FFCWS were interviewed via phone and/or home visits at the birth of the target child, and again

at ages 1, 3, 5, 9, and 15 years old. Retention of the baseline sample was generally high at each

of the assessment periods (77% to 90% for mother or primary caregiver interviews, 62% to 72%

for home visits) (for detailed information about cohort retention across waves, see

https://fragilefamilies.princeton.edu).

At age 15, families from the Detroit, Toledo, and Chicago subsamples were asked to

participate in an additional follow-up study to investigate the role of the environment on youth

brain and behavioral development. Two-hundred and thirty-seven adolescents aged 15 to 17

(52.3% female) and their primary caregiver agreed to participate. Of the 237 families, teens self-

identified as Black Non-Hispanic (N = 179, 75.5%), Black Hispanic (N = 2, 0.8%) White Non-

Hispanic (N = 30, 12.7%), of Hispanic or Latino origin (N = 10, 4.2%), biracial (N = 13, 5.5%),

or other Non-Hispanic (N = 3, 1.3%). Primary caregivers were biological mothers (N = 216,

91.1%), biological fathers (N = 11, 4.6%), adoptive parents (N = 4, 1.7%), or other family

members (N = 6, 2.5%). Median annual family income was between $25,000 to $29,999, with

some primary caregivers reporting annual incomes below $4,999 (13%) and other reporting annual incomes above $90,000 (10.2%). Thus, the SAND sample is socioeconomically diverse and comprised of mostly Black American children and their biological mothers.

Procedure

The current paper uses data from both the core FFCWS and the SAND. Measures of maternal harsh parenting source from the core FFCWS telephone and in-person interviews at ages 3, 5, and 9. As the primary aim of this paper was to investigate whether initial levels and/or changes in parenting behaviors across childhood impacted youth corticolimbic function in adolescence, we limited our sample to families where the biological mother was the primary caregiver at the 3- , 5- , and 9-year assessments (i.e., to prevent artifacts introduced by changing informants across time). Only 216 families were excluded. Detailed descriptions of the study protocols for each of the core FFCWS assessment periods can be found on the study website (https://fragilefamilies.princeton.edu).

SAND subsample. At age 15, primary caregivers and adolescents in the SAND study participated in a one-day protocol that included collection of self-report, interviewer, observational, and biological data. Parents provided written consent and adolescents provided verbal assent for their participation in the SAND protocol. Families were reimbursed for their participation. All assessments and measures were approved by the Institutional Review Board of the University of Michigan (IRB protocol # XXX).

Measures

Maternal harshness. Maternal harshness was measured as a sum of five mother-reported items from the physical aggression subscale of the Parent-Child Conflict Tactics Scale (Straus et al., 1998) at the 3-year (Mean[SD] = 1.23[1.01], n = 3,284), 5-year (Mean[SD] = 1.10[.97], n =

2,935), and 9-year (Mean[SD] = .73 [.85], n = 3,083) assessments. Mothers were asked to rate how many times in the past year each disciplinary practice was used (e.g., "pinched him/her", "slapped him/her on the hand, arm, or leg"), from 0 (never happened) to 6 (more than 20 items). The reliability of the harsh parenting items was adequate (age 3: α = .61; age 5: α = .60; age 9: α = .70).

　　　　Covariates. Several covariates were included in the analyses linking parenting to corticolimbic function in adolescence, each of which have been shown to impact corticolimbic function (Alarcón, Cservenka, Rudolph, Fair, & Nagel, 2015a; Kubota, Banaji, & Phelps, 2012; Moore et al., 2012): (1) youth self-reported race and ethnicity at age 15, (2) youth gender (girl=1), and (3) youth self-reported pubertal development. Race/ethnicity was coded as one dummy code for the largest group in the SAND sample (Non-Hispanic Black [75.5%] = 1). Pubertal development was measured using youth report on the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988a), which includes two gender-specific items (e.g., for boys: voice changes; for girls: breast development), and three items for both genders (i.e., changes in height, skin, pubic hair). All items were rated on a 4-point Likert scale (1 = *process has not started*, 4 = *seems completed*), except for the menarche question for girls, which was dichotomous (1 = *not started*, 4 = *started*). Total pubertal development score was calculated as a mean of the five items for each gender (girls: Mean [SD] = 3.58 [.46]; boys: Mean [SD] = 2.86 [.50]).

Neuroimaging Data

　　　　fMRI task. Participants completed an implicit emotion face matching task during continuous fMRI acquisition (see Figure 3.1). In this task, participants were asked to identify the gender of the actor by pressing their thumb for male or index finger for female. Faces from the

NimStim set (Tottenham et al., 2009) were counterbalanced for gender and race (European American and African American). There were 100 pseudo-randomized trials, 20 trials each of the following emotions: fearful, happy, sad, neutral, and angry. Each trial consisted of a 500ms fixation cross followed by a face presented for 250ms. A black screen then appeared for 1500ms, during which participants responded to the stimulus presentation, followed by a jittered inter-trial interval (2000, 4000, or 6000ms). Total task time was 8.75 minutes. Accuracy and response times were collected using a non-metallic fiber optic transducer linked to a response box.

Data acquisition and pre-processing. MRI images were acquired using a GE Discovery MR750 3T scanner with an 8-channel head coil located at the UM Functional MRI Laboratory. High resolution T1-weighted gradient echo images were collected before the functional scans using the same field of view (FOV) and slices as the functional scans (TR = 9.0, TE = 1.8, TI = 400ms, flip angle = 15°, FOV = 22cm; slice thickness = 3mm; 256 x 256 matrix; 40 slices). Functional T2*-weighted BOLD images were acquired using a reverse spiral sequence (Glover & Law, 2001) of 40 contiguous axial 3mm slices (TR = 2000ms, TE = 30ms, flip angle = 80°, FOV = 22cm, voxel size = 3.44mm x 3.44mm x 3mm). Slices were prescribed parallel to the AC-PC line (same locations as structural scans). Images were reconstructed into a 64x64 matrix. Slices were acquired contiguously, which optimized the effectiveness of the movement post-processing algorithms. Images were reconstructed off-line using processing steps to remove distortions caused by magnetic field inhomogeneity and other sources of misalignment to the structural data, which yields excellent coverage of subcortical areas of interest.

Anatomical images were homogeneity-corrected using SPM, then skull-stripped using the Brain Extraction Tool in FSL (version 5.0.7) (Jenkinson, Beckmann, Behrens, Woolrich, & Smith, 2012; Smith, 2002). Functional data were pre-processed in the following steps: removal

of large temporal spikes in k-space data (> 2 std dev), field map correction and image reconstruction using custom code in MATLAB; and slice-timing correction using SPM12 (Wellcome Department of Cognitive Neurology, London, UK). To address head motion, functional images were realigned to the AC-PC plane in the mean image. Using SPM12, anatomical images were co-registered to the functional images. Functional images were normalized to the MNI Image space using parameters from the T1 images segmented into gray and white matter, cerebrospinal fluid, bone, soft tissue and air using a Tissue Probability Map created in SPM12. Images were then smoothed using an isotropic 8 mm full width at half maximum Gaussian kernel. Following preprocessing, the Artifact Detection Tools (ART) software package (http://www.nitrc.org/projects/artifact_detect) identified motion outliers (>2mm movement or 3.5° rotation); outlier volumes were individually regressed out of the participant's individual model. Additionally, because of the relatively extensive signal loss typically observed in the amygdala, single-subject BOLD fMRI data were only included in subsequent analyses if there was a minimum of 70% signal coverage in the left and right amygdala, defined using the Automated Anatomical Labeling (AAL) atlas in the WFU PickAtlas Tool, version 1.04 (Maldjian, Laurienti, Kraft, & Burdette, 2003). As the current paper additionally examined corticolimbic function within the PFC, participants with extensive coverage loss in prefrontal regions were removed by visual inspection. Lastly, participants were excluded if accuracy performance on the task was less than 70%. Of the 237 participants in the SAND neuroimaging study, usable fMRI data was available for 176 (74%) participants (Table 3.1). Participants without usable fMRI data did not differ from participants with usable fMRI data with respect to concurrent family monthly income, earlier measures of parental harshness, or youth gender or race and ethnicity (all ps > .10).

Activation analyses. The general linear model in SPM12 was used to estimate condition-specific BOLD activation for each individual and scan. Individual contrast images (i.e., weighted sum of the beta images) were then used in second-level random effects models to determine expression-specific reactivity using multiple regression. As the goal of this study was to examine corticolimbic reactivity during threat processing, we present results from the fearful facial expressions > neutral faces and angry facial expressions > neutral faces contrasts. We used two regions of interest (ROIs) to probe the effects of parenting of corticolimbic function: the amygdala and a large PFC mask. We defined the bilateral amygdala using the Automated Anatomical Labeling (AAL) atlas definition in the WFU PickAtlas Tool, version 1.04 (Maldjian et al., 2003). The PFC mask was defined by Broadman's areas 9 (dorsolateral), 10 (dorsomedial), 11 and 47 (orbitofrontal), 24 and 32 (dorsal anterior cingulate), and 25 (subgenual cingulate), using the WFU PickAtlas Tool, version 1.04 (Maldjian et al., 2003). We used this PFC mask because (1) larger ROIs require stricter correction for multiple comparisons (i.e., more voxels in the mask) and thus are less biased towards identifying effects, (2) by using a large mask composed of multiple PFC regions, we could compare our results to several studies that used different definitions of the mPFC (Gee et al., 2014; van Harmelen et al., 2014), and (3) the seven Brodmann's Areas we identified have each been shown in non-human primate neural tract-tracer studies to be structurally connected to the amygdala in previous studies (Amaral & Price, 1984; Ghashghaei, Hilgetag, & Barbas, 2007) and in the current sample (Goetschius et al., 2019). We corrected for multiple comparisons using 3dClustSim (Cox, Chen, Glen, Reynolds, & Taylor, 2017b) in AFNI (Cox, 1996). This procedure, which is dynamic to each group-level model based on changes in smoothing values, resulted in cluster size threshold of 25 voxels for amygdala activation analyses and 93 voxels for PFC activation analyses. These cluster sizes are ROI-

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corrected at a threshold of $p < .05$ and a voxel-wise threshold of $p < .01$. Our cluster thresholds were based on 2-sided tests and used the nearest neighbor definition of "face and edge" (i.e., 3dClustSim command: NN=2).

   Functional connectivity analysis. Psychological-physiological interaction (PPI) analyses in the generalized PPI toolbox (McLaren, Ries, Xu, & Johnson, 2012) in SPM12 were used to measure amygdala connectivity with regions of the PFC. In a PPI analysis, a design matrix is constructed at the level of the individual with the following columns of variables: (a) a physiological variable that represents the time course of the seed region (i.e., left or right amygdala) across the task, (b) a psychological variable indicating the experimental variable (e.g., onset times for fearful face stimuli), and (c) a product term of the interaction between the physiological and psychological variables. At the individual level, the regression coefficient for the interaction term represents the change in activity between the seed and identified "target region" across different conditions, such as emotional face types (i.e., the seed-target functional coupling is context-dependent). The gPPI toolbox developed by McLaren and colleagues (2012) allows for the simultaneous specification of all task conditions and interactions with the seed region time series in the same individual-level model (Friston et al., 1997). This is advantageous because it reduces the number of specified models and the overall type I error rate.

   As we were interested in reporting amygdala connectivity while participants viewed fearful and angry facial expressions versus neutral faces, we defined the left and right whole amygdala as seed regions using the AAL definition within the WFU PickAtlas Tool (Maldjian et al., 2003). Two general linear models at the individual level were constructed (i.e., one for each seed region). Using the gPPI toolbox, the time series of the left or right seed region was entered as the physiological variable in the design matrix, the explanatory variables for each of the five

conditions in our task (i.e., facial expressions of fear, anger, happy, sad, and neutral faces) were entered as psychological variables, and the five product terms between the amygdala seed and conditions were entered as the interaction terms. We specified two primary contrasts at the individual level: fearful facial expressions interaction term > neutral faces interaction term, and angry facial expressions interaction term > neutral faces interaction term. Practically, this can be interpreted as a difference in slopes: is slope A (i.e., the interaction between amygdala reactivity and the fear/angry condition) greater or lesser than slope B (i.e., the interaction between amygdala reactivity and the neutral condition). To determine the direction of connectivity between two regions (i.e., whether the slope is positive or negative), activation in the seed region (e.g., amygdala) can be plotted as a function of activation in the target region (e.g., PFC) during a given condition (e.g., presentation of angry facial expressions).Contrasts from the individual level models were then used in random effects, group-level models to evaluate the impact of initial levels and changes in harsh parenting on amygdala-PFC functional connectivity to fearful and angry facial expressions versus neutral faces. These models assess whether harsh parenting is associated with the difference in connectivity between conditions (or the difference in slopes). The contrasts of angry facial expressions > baseline, fearful facial expressions > baseline, and neutral faces > baseline were additionally used to confirm that our results were driven by connectivity during the emotion conditions (i.e., fear or anger) rather than the neutral face condition. Only ipsilateral connections between the amygdala and PFC were examined (e.g., left amygdala – left PFC), because neural tracer studies in non-human primates suggests that first order amygdala connections are primarily ipsilateral (Ghashghaei et al., 2007). Thus, we separated the same PFC mask from our activation analyses into left and right PFC masks for use as target regions in connectivity analyses. The same procedure using 3dClustSim (Cox et al.,

2017b) in AFNI (Cox, 1996) as in the activation analyses was used to correct for multiple comparisons in the functional connectivity analyses (cluster size threshold for left and right PFC masks = 77 voxels).

Analytic Plan

First, LCGM within Mplus version 7.2 (Muthén & Muthén, 2006) was used to estimate the intercept and slope of maternal harshness. Though our neuroimaging sample was composed of 167 participants, we used all available cases from the core FFCWS (N = 4,682 families, where mom was the primary caregiver at the 3-, 5-, and 9-year assessments) to estimate patterns of harsh parenting across childhood. Thus, the estimates of initial levels and changes in parenting behaviors across childhood are derived from a larger more representative sample as opposed to only SAND-recruited families from Detroit, Toledo, and Chicago. Cases with at least one data point were used in each analysis with the full maximum likelihood estimator with robust standard errors (FIML), resulting in a sample size of N=4,144 for the LCGM of maternal harshness. FIML estimation uses the covariance matrix of all available data to produce unbiased estimates and standard errors in the context of missing data (Enders & Bandalos, 2001; McCartney et al., 2006). Model fit was considered adequate if the Root Mean Square Error of Approximation (RMSEA) and Comparative Fit Index (CFI) values met established guidelines for good fit (i.e., RMSEA<0.06 and CFI>0.95) (Hu & Bentler, 1999).

To evaluate the effects of initial levels of and changes in parenting behaviors on corticolimbic function, estimates of the intercept and slope of maternal harshness were extracted for use in 2nd-level random effects models within SPM. First, the intercept or slope of maternal harshness was entered as the primary predictor in a linear regression model, with perceived pubertal status, gender, and race and ethnicity as covariates (16 models: 2 predictors [intercept or

slope] X [2 activation regions of interest + 2 functional connectivity models for the left and right amygdala seeds] X 2 contrasts). To evaluate the unique effects of the intercept/slope, a second set of models was estimated that additionally controlled for the slope/intercept of maternal harshness.

<div align="center">

Results

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Estimation of harsh parenting across childhood

The linear growth curve model of harsh parenting at ages 3, 5 and 9 in the nationwide FFCWS ($N = 4{,}144$; Figure 3.2) demonstrated good model fit ($X^2[1] = 5.62$, p = .02; RMSEA = .03, 90% CI [.01, .06]; CFI = 1.00, TLI = .99) and indicated that, on average, initial levels (i.e., the intercept) of harsh parenting were positive and significantly different from zero (estimated intercept mean [SD] = 1.23[.02], $p < .001$). On average, levels of harsh parenting decreased from ages 3 to 9 (estimated slope mean [SD] = -.09[.003], $p < .001$).

Harsh parenting effects on corticolimbic activation

We next used the estimated intercept and slope terms for each participant to evaluate whether initial levels of harsh parenting (i.e., the intercept) were most strongly associated with amygdala function and whether changes in harsh parenting across childhood (i.e., the slope) were most predictive of PFC function during emotional face processing at age 15. Across all models, the associations between harsh parenting and corticolimbic activation were specific to angry faces (i.e., anger versus neutral contrast) rather the fearful facial expressions (i.e., fear versus neutral contrast). First, greater initial levels of harsh parenting were associated with lesser left amygdala (but not PFC) reactivity to angry facial expressions versus neutral faces (see Table 3.2; Figure 3.3), controlling for changes in harsh parenting across childhood (i.e., the slope term) and harsh parenting at age 15 (i.e., the same age as the neuroimaging data collection). By contrast,

increases in harsh parenting from ages 3 to 9, controlling for initial levels of harsh parenting (i.e., the intercept) and harsh parenting at age 15, was associated with lesser bilateral dorsal ACC (but not amygdala) reactivity to angry facial expressions versus neutral faces (Table 3.2; Figure 3.3).

Harsh parenting effects on corticolimbic connectivity

Consistent with the corticolimbic activation results, all models linking harsh parenting to amygdala-PFC connectivity during emotional face processing were specific to the angry versus neutral face contrast. Consistent with our hypotheses, both greater initial levels and increases in harsh parenting from ages 3 to 9 were uniquely (i.e., accounting for their overlap) associated with greater amygdala-PFC connectivity during angry face processing than neutral face processing (Table 3.2). To determine the direction of amygdala-PFC connectivity (i.e., whether activation in the seed and target region was positively or negatively coupled), we extracted activation in the amygdala and the target region during the angry face versus baseline condition. As shown in Figure 3.4, greater initial levels of harsh parenting were associated with more positive left amygdala – left orbitofrontal cortex (OFC) connectivity during angry face processing; initial levels of harsh parenting did not predict connectivity during the neutral faces condition (Table 3.2). Increases in harsh parenting across childhood were also associated with more positive left amygdala – left OFC and left amygdala – left mPFC connectivity during angry face processing but not neutral face processing (Table 3.2; Figure 3.4). All associations remained significant after accounting for harsh parenting at age 15.

Post-hoc analyses

Though our results suggest that the timing of exposure to harsh parenting is important for corticolimbic function in adolescence, our results could also reflect cumulative risk effects (Sameroff, Seifer, Barocas, Zax, & Greenspan, 1987). That is, it may be that youth with the

highest levels of harsh parenting in early childhood were also exposed to the highest levels of harsh parenting at subsequent ages and, thus, our results could be accounted for by a cumulative effect of harsh parenting across childhood. Using methods traditional to cumulative risk research (Evans, Li, & Whipple, 2013), we calculated the number of waves (i.e., 3-, 5- , and 9-year waves; possible cumulative risk score $0 - 3$) during which an individual scored in the top quartile of harsh parenting. Of the 162 families with valid harsh parenting data at all three waves, most families (62.3%) were low risk across all three waves. Twenty-eight (17.3%) and 21 (13%) families were at risk in one or two waves, respectively, and 12 (7.4%) families scored in the top quartile of harsh parenting at all three waves. In three separate models that controlled for participant demographics at age 15, we examined whether the cumulative risk score was associated with (1) amygdala activation, (2) PFC activation, and (3) amygdala-PFC connectivity during angry versus neutral face processing at age 15. Consistent with the notion that timing of exposure to harsh parenting is important for corticolimbic development, the cumulative harsh parenting risk score was not associated with amygdala or PFC activation or connectivity (results available upon request).

Discussion

The current study examined how harsh parenting behaviors change across childhood in a large, population-based sample of sociodemographically-diverse families, and explored how initial levels and changes in harsh parenting across childhood impacted subsequent corticolimbic function during adolescence. One of the study's primary strengths was the integration of data from an existing nationwide study of nearly 5,000 families followed prospectively across childhood with neuroimaging data from a subsample recruited during adolescence. Consistent with prior research on trajectories of parenting behaviors during shorter developmental windows

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(Dallaire & Weinraub, 2005; Kim et al., 2010), harsh parenting was initially high during early childhood and decreased thereafter through age 9. Using neuroimaging data from an implicit emotional faces matching paradigm, we further demonstrated the importance of timing of harsh parenting effects on corticolimbic function: initial levels of harsh parenting were associated with subcortical amygdala function, increases in harsh parenting from ages 3 to 9 were associated with activation in the dorsal ACC, and both high initial levels and increases in harsh parenting across childhood were associated with more positive amygdala-PFC connectivity during angry face versus neutral face processing. Our results were consistent with our original hypotheses in terms of the *timing* of the hypothesized effects (i.e., initial levels versus changes in harsh parenting and amygdala versus PFC activation and connectivity), but not always the *direction* of hypothesized effects (i.e., initial levels of harsh parenting were associated with lesser, not greater, amygdala activation).

In a population-based sample of families followed prospectively across childhood (Reichman et al., 2001), parental harshness changed from ages 3 to 9 in ways that mirror the developmental competencies of each developmental stage. Maternal harshness was positive and significantly different from zero at age 3, when children are increasingly mobile and normatively evince greater emotionality (Shaw & Bell, 1993). Thereafter, from ages 3 to 9, maternal harshness decreased. During middle childhood (5 to 12 years), new developmental challenges associated with starting school (e.g., self-regulation, academic skills, social interaction with peers) alter the parent-child relationship and requisite parenting behaviors (Collins et al., 2005). Moreover, affective expression within the parent-child dyad also decreases during this period, where both children and parents show less overt negative behaviors (e.g., coercion, emotional outbursts) (Collins et al., 2005; Forehand & Jones, 2002; Shanahan, McHale, Osgood, &

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Crouter, 2007). Our results build upon previous work that tracked changes in parenting behaviors during shorter developmental windows, such as during infancy and early childhood (Dallaire & Weinraub, 2005; Lipscomb et al., 2011). Critically, reliance on a nationwide population-based sample suggests that these patterns of maternal harshness across childhood are reflective of the broader U.S. population, including families of lower socioeconomic position.

Although several recent reviews have posited that exposure to harsh contexts impacts corticolimbic function in a timing-specific manner (Callaghan & Tottenham, 2016; Gee, 2016; Perry et al., 2017), our study is the first to empirically test these hypotheses in humans using repeated measures of harsh parenting across childhood in a population-based sample of low-income adolescents. We found that initial levels of harsh parenting in early childhood were associated with lesser amygdala activation (consistent with our hypotheses in terms of timing, but not direction) and increases in harsh parenting thereafter were associated with lesser dorsal ACC activation (consistent with our hypotheses in terms of timing and direction) during angry versus neutral face processing. Much of the theoretical rationale for the notion of "sensitive periods" sources from foundational work in humans documenting the developmental trajectories of subcortical and cortical brain development (Gilmore et al., 2012), and from animal studies wherein environmental exposure can be manipulated. For example, rhesus monkeys separated from their mother at 1-week versus 1-month of age or control animals (no separation), showed a significant decrease in gene expression in amygdala nuclei (Sabatini et al., 2007). We also showed specificity of early exposure to adversity and amygdala function, though the direction of effects contrasted with some, but not all, previous research. A meta-analysis of largely cross-sectional studies or studies that used retrospective reports found that individuals exposed to childhood maltreatment exhibited *greater* amygdala activation to threatening emotional facial

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expressions (Hein & Monk, 2017). By contrast, two prospective longitudinal studies that recruited participants based on low family income or psychosocial adversity found that greater childhood adversity (operationalized as harsh parenting or a family adversity index) was associated with *lesser* amygdala reactivity to threatening facial expressions (Gard et al., 2017a; N. E. Holz et al., 2017), consistent with our results. Such a blunted pattern of amygdala activation following adversity may reflect an adaptive response to chronic stress exposure. Although amygdala sensitivity to environmental signals of threat or danger is adaptive in the short-term, particularly for youth living in adverse contexts, persistent hyperactivation of physiological response systems (e.g., the hypothalamic pituitary-adrenocortical axis) can lead to a wide array of diseases (McEwen & McEwen, 2017). Thus, blunted amygdala reactivity to threatening facial expressions following adversity may be an adaptive response that facilitates allostasis and minimizes exposure to neurotoxic physiological hormones (e.g., cortisol); our results suggest that earlier exposure to harsh parenting during a developmental period where the amygdala is undergoing substantial reorganization (Gilmore et al., 2012) may lead to this blunting effect.

Our results also showed specificity in the association between changes in harsh parenting from ages 3 to 9 and prefrontal (specifically dorsal ACC), but not amygdala, activation during angry face processing. In contrast to subcortical regions such as the amygdala, regions of the PFC continue to develop through adulthood (Lenroot & Giedd, 2006; Sowell et al., 2003). Structural MRI studies have shown that gray matter volume in the PFC increases during the pre-adolescent period, followed by post-adolescent decrease (Giedd et al., 1999); such volumetric changes correspond with increasing activation in the ACC and mPFC and parallel behavioral improvements in executive functioning and emotion regulation (Casey, Jones, et al., 2008).

Consistent with our results, van Harmelen and colleagues (2014) found that exposure to childhood maltreatment was associated with blunted mPFC activation during encoding and recognition of words. Several other studies have found negative associations between childhood adversity and activation in dorsal and lateral regions of the PFC (Fonzo et al., 2016; Liberzon et al., 2015), highlighting diffuse effects of adversity on PFC function. Identification of dorsal rather than ventral regions of the mPFC in the current study likely reflects the fact that our emotional faces matching task captured cognitive (i.e., perceptual processing) rather than regulatory components of emotion processing (Etkin, Egner, & Kalisch, 2011; Fuster, 2001).

In addition to region-of-interest analyses that examined the amygdala and regions of the PFC separately, we also explored the impact of initial levels and changes in harsh parenting across childhood on amygdala-PFC connectivity during emotion processing. Several studies using task-based or resting-state fMRI data have found that stronger negative amygdala-mPFC connectivity reflects regulation within the corticolimbic circuit (Gabard-Durnam et al., 2014; Gee et al., 2013). By contrast, positive amygdala-mPFC connectivity (or weaker negative connectivity) during threat processing is thought to reflect immature corticolimbic function and has been associated with anxiety in children (Demenescu et al., 2013; Gee et al., 2013), and internalizing and externalizing behaviors in adults (Gard et al., 2018; Waller, Gard, et al., 2018a). Although in contrast to our region of interest analyses, both high initial harsh parenting and increases in harsh parenting across childhood were associated with stronger positive amygdala-orbital/medial PFC connectivity during angry face compared to neutral face processing (consistent with our original hypotheses in terms of timing and direction), we do not believe these analyses conflict in reflecting the importance of timing of harsh parenting for later corticolimbic function. Functional connectivity as measured using PPI reveals how coupling

between two regions changes as a function of condition; this analyses does not reveal information about the absolute level of activation in either region (Friston et al., 1997). Thus, by pairing our PPI analyses with activation analyses, we were able to comment on the associations between harsh parenting and both activation and connectivity within two critical nodes of the corticolimbic system.

That the effects of harsh parenting on corticolimbic function were specific to angry face processing is consistent with previous reports showing that physically abused children process angry facial expressions differently than non-maltreated controls (Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003). Compared to non-maltreated children, physically abused children may require less perceptual information to correctly identify facial expressions of anger (Pollak & Sinha, 2002), and respond more quickly to targets cued by angry faces versus happy faces (Pollak & Tolley-Schell, 2003). Using the dot probe behavioral paradigm, Pine and colleagues (2005) showed that severity of childhood maltreatment (majority physical abuse) was negatively associated with attention bias towards angry/threatening faces; maltreated children were more likely to direct attention away from threatening stimuli, which was associated with having a diagnosis of Post-Traumatic Stress Disorder (Pine et al., 2005).

In drawing associations between the results presented here and previous research, it is important to highlight how we operationalized harsh parenting in the current study. Harsh parenting was measured as maternal report on the physical aggressions subscale of the Conflict Tactics Scale (Straus et al., 1998). As the items in this subscale captured corporeal punishment (i.e., "pinched him/her", "slapped him/her on the hand, arm, or leg", "spanked him/her on the bottom with your bare hand", "hit child on the bottom with some hard object", "shook child"), it could be argued that our measure identified dimensional child physical abuse. In supplemental

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analyses using the psychological aggressions subscale of the Conflict Tactics Scale (e.g., "yelled at the child", "called the child dumb or lazy"), we found strikingly similar results to those in the current study using items from the physical aggressions subscale; higher initial levels of maternal psychological aggression, indicated by an intercept term from a linear growth curve model across ages 3 to 9 years, were associated with *lesser* amygdala reactivity to angry facial expressions versus neutral faces, at a lower correction threshold (i.e., the direction of results was consistent, but the patterns were weaker for the psychological aggressions measure; results available upon request). Thus, we believe the harsh parenting measure included in the current study was reflective of broader harsh parenting practices across physical and psychological domains – leading to overall blunted corticolimbic function during threat processing.

Limitations and Future Directions

Despite the use of a large population-based sample of sociodemographically-diverse families followed from birth through adolescence, integration of repeated measures of harsh parenting across childhood with linear growth curve modeling, and examination of corticolimbic activation and connectivity within the same analyses, several limitations warrant consideration. First, although our results suggest that timing of exposure to harsh parenting is important for subsequent corticolimbic function, interpretations of our results as evidence for "sensitive periods" should be tempered. Such a claim would require repeated measures of neural function in addition to repeated measures of harsh parenting (Andersen & Teicher, 2008). Procuring measures of task-based corticolimbic function in early childhood is challenging; studies rarely collect such data in children younger than 5 years due to motion and attention constraints as well as the excessive noise of magnetic resonance data acquisition (A. M. Graham et al., 2015). In recent years, other imaging modalities such as resting-state fMRI, have been successfully

translated into younger populations including infants (A. M. Graham et al., 2015); such

approaches are promising for evaluating sensitive periods of environmental effects on brain

development. Nevertheless, our results should be interpreted within the context of robust animal

experiments documenting sensitive periods of adversity effects on corticolimbic development

(reviewed by Callaghan & Tottenham, 2016), structural MRI studies (reviewed by Teicher &

Samson, 2016), and the fact that our results using prospectively-collected measures of

psychosocial adversity is an improvement to existing cross-sectional and retrospective studies

(Hein & Monk, 2017).

Second, harsh parenting is only one type of environmental exposure thought to impact

functional brain development; neighborhood- and family-level socioeconomic disadvantage,

maternal psychopathology, and interparental conflict are just some examples of adversities that

often co-occur with harsh parenting (Green et al., 2010). Nevertheless, there ample evidence to

suggest that parenting behaviors are relevant targets for understanding how environmental stress

becomes biologically-embedded to predict maladaptive youth socioemotional outcomes. First,

the Family Stress Model posit that parents mediate the negative effects of socioeconomic

disadvantage on youth outcomes (Conger et al., 1994); this model has been supported across a

range of contexts – within urban and rural samples, cross-culturally, in racially- and ethnically-

diverse samples, in two-parent and single-parent families, and using cross-sectional and

longitudinal data (reviewed by Masarik & Conger, 2017). Second, some research has shown that

the impact of parenting behaviors on youth corticolimbic function is independent of other

correlated adversities (Gard et al., 2017a), though more research is needed to evaluate this claim.

The present study extends previous research by documenting how maternal harsh parenting

changes across childhood in a population-based sample of sociodemographically-diverse

families, and highlights that the effects harsh parenting on corticolimbic function depend on timing of exposure and the neural region examined. We present one pathway by which environmental adversity may become biologically-embedded to predict maladaptive youth socioemotional behaviors (McEwen & McEwen, 2017). Our results stimulate several future areas of research, including study designs that boast repeated measures of neural function, examinations of the specificity of harsh parenting effects on corticolimbic function, and the efficacy of parenting interventions on changes in brain development within emotion-processing-related regions.

Table 3.1. Faces task participant drop out

	Spiral Sequence		Echo-planar Sequence with Multiband Acquisition	
	Number Lost	Participants with data	Number Lost	Participants with data
Original sample		238		200
- Refused scan	16		12	
- Medical restriction*	25		8	
- Data collection/scanner error	2		1	
- Partial data	0		3	
Total lost	43			24
Sample with imaging data		195		176
- Whole brain coverage loss by visual inspection	2		1	
- Low amygdala coverage (< 90%)	7		58	
- Low prefrontal lobe coverage	3		19	
- Low task performance (< 70%)	11		3	
- Ghosting on functional scans	1		1	
- Exceeded movement thresholds	0		0	
Total lost	20			62
Sample with usable imaging data		171		94

*includes having a permanent retainer or braces, non-MRI safe implanted medical devices, having BBs/pellets or other non-removable metal inside of body, recent surgery, exceeds table weight limit, impaired vision not correctable with MRI-safe glasses. Note that an additional 21 families (42 twins) received an earlier version of the task that was not comparable to the current version; these participants were excluded from all analyses.

Table 3.2. Initial levels and changes in harsh parenting across childhood predict corticolimbic activation and connectivity during angry face processing

Activation Results

Predictor	Direction of relationship	Bilateral Amygdala (x,y,z), t and voxel extent (k)	Prefrontal Cortex (x,y,z), t and voxel extent (k)
Intercept of Harsh Parenting	Negative	Left: (-26,-4,-22), $t = 3.91$, $k = 41$	*no suprathreshold clusters*
Slope of Harsh Parenting	Negative	*no suprathreshold clusters*	Right dorsal ACC (BA32): (6,32,22), $t = 3.66$, $k = 151$ Left dorsal ACC (BA32): (-8,34,18), $t = 3.39$, $k = 119$

Connectivity Results

Predictor	Direction of relationship	Left Amygdala Seed Region – Left PFC	Right Amygdala Seed Region – Right PFC
Intercept of Harsh Parenting	Positive	Left OFC (BA10): (-16,68,8), $t = 3.44$, $k = 198$	*no suprathreshold clusters*
Slope of Harsh Parenting	Positive	Left OFC (BA10): (-8,46,16), $t = 3.75$, $k = 358$ Left mPFC (BA9): (-12,58,32), $t = 4.26$, $k = 110$	*no suprathreshold clusters*

Note. N = 159. Printed estimates are from the most stringent models that control for youth gender, race, pubertal status, the intercept/slope term, and harsh parenting at age 15. Harsh parenting at age 15 was measured by a mean of three parent-reported corporal punishment items from the Alabama Parenting Questionnaire (Frick, 1991). For activation, the results of the intercept and slope models of harsh parenting on corticolimbic reactivity were driven by less activation to angry facial expressions versus baseline (intercept model: $p_{voxel} < .05$, [-24,-2,-22], t=2.29, k=59; slope model: $p_{voxel} < .05$, [14,34,22], t=3.21, k=126), rather than greater activation to neutral facial expressions versus baseline (no clusters at $p_{voxel} < .05$). For connectivity, the results of the intercept and slope models of harsh parenting on corticolimbic connectivity were driven by greater amygdala-PFC connectivity during angry face versus baseline processing (left amygdala seed intercept model: $p_{voxel} < .05$, [-2,52,4], t=3.13, k=395; left amygdala seed slope model: $p_{voxel} < .05$, [-8,46,16], t=2.68, k=232), rather than lesser amygdala-PFC connectivity during neutral face processing versus baseline (no clusters at $p_{voxel} < .05$).
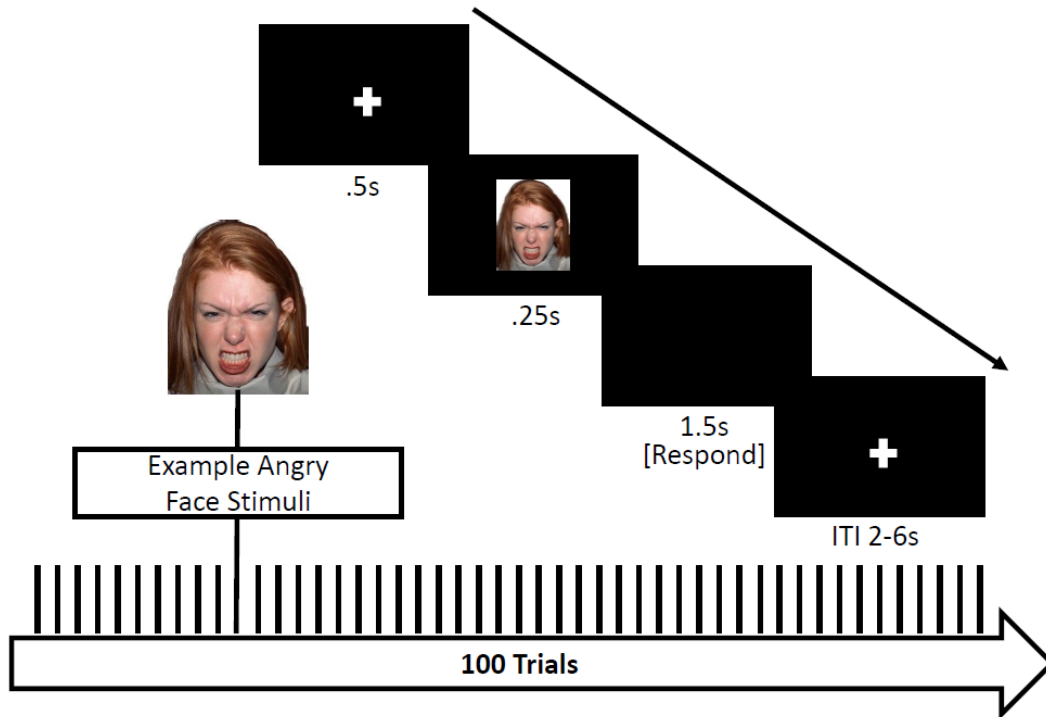
Figure 3.1. Implicit emotional faces matching paradigm

Note. This event-related task design included 100 trials, 20 each of the following facial expressions: angry, fearful, sad, neutral, and happy. Total task time was 8.75 minutes.
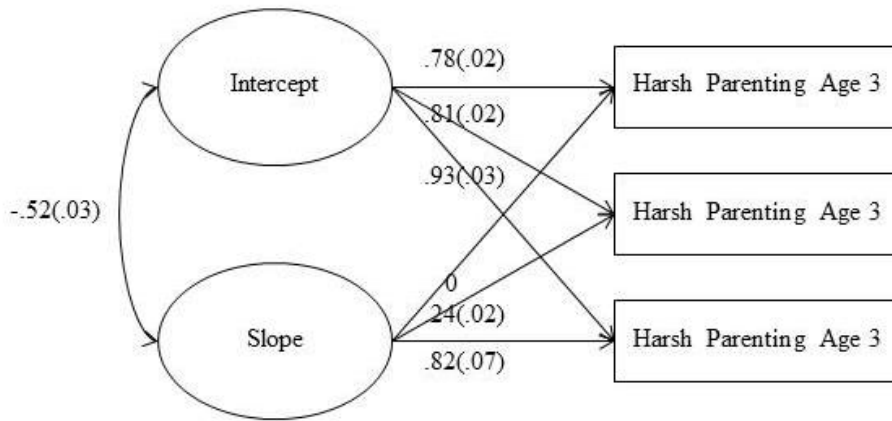
Figure 3.2. Latent growth curve model of harsh parenting across childhood
Note. N = 4,144. Standardized estimates are shown. All estimates were significant at *p* < .001.
Model fit: X^2 (1) = 5.62, *p* = .02; RMSEA = .03, 90% CI (.01, .06); CFI = 1.00, TLI = .99,
SRMR = .01. Loadings for the latent slope factor were specified as 0 (age 3), 2 (age 5), and 6
(age 9), and all loadings for the latent intercept factor were set equal to 1.
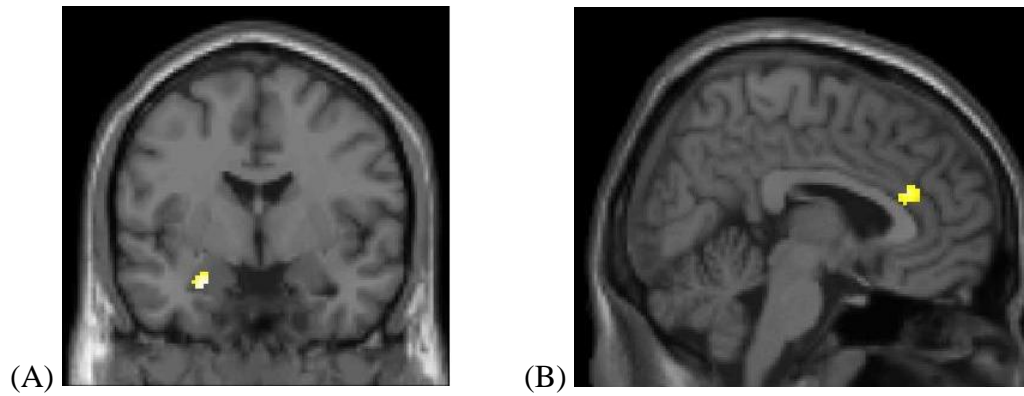
Figure 3.3. Initials levels and increases in harsh parenting across childhood are associated with lesser corticolimbic activation during angry face processing
Note. (A) Greater initial levels of harsh parenting (intercept) was associated with lesser left amygdala reactivity to angry versus neutral faces in adolescence. (B) Increases in harsh parenting from ages three to nine (indexed by a positive slope term) was associated with lesser dorsal anterior cingulate cortex reactivity to angry versus neutral faces in adolescence.

(A) *(B)*

(C)
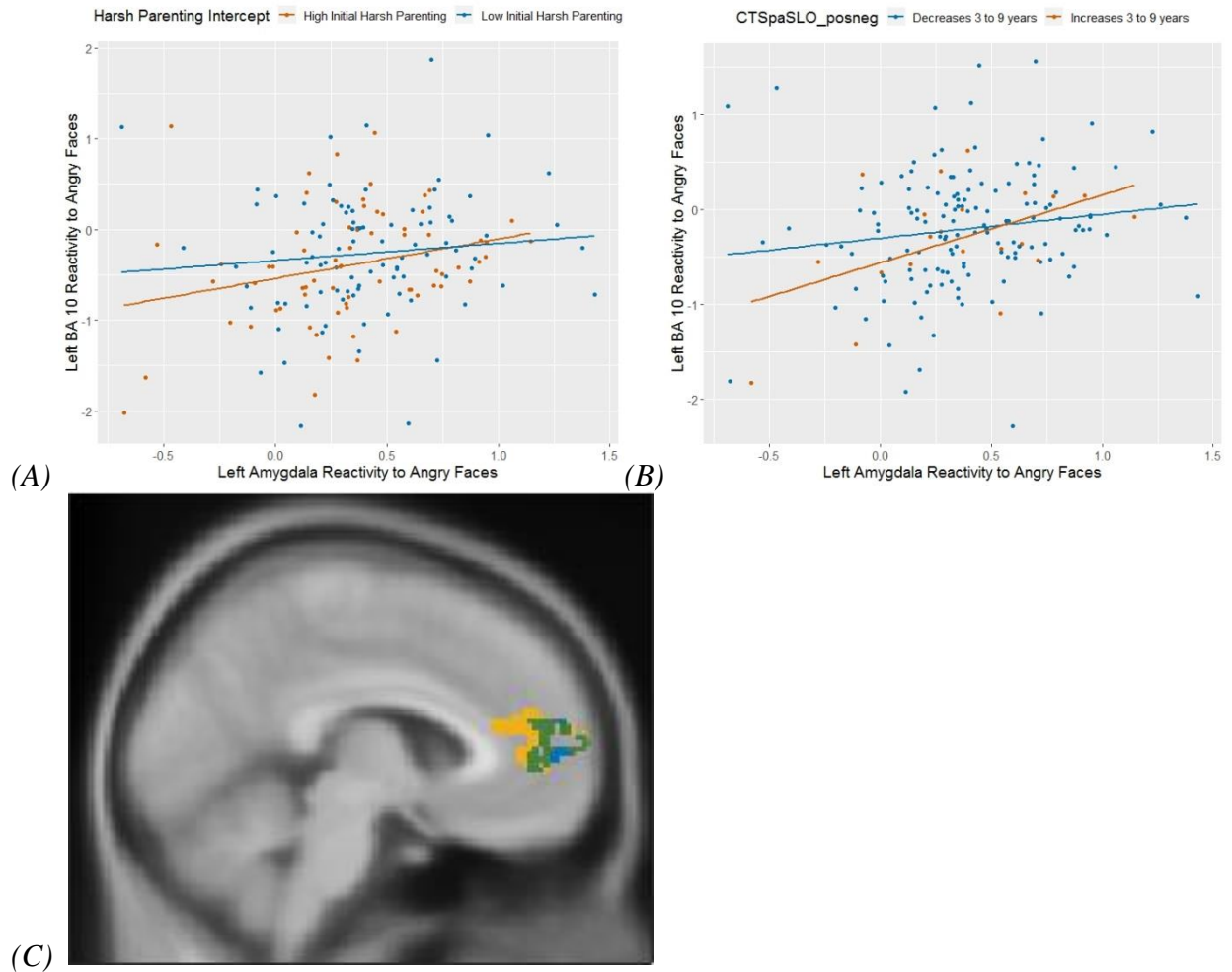
Figure 3.4. High initial levels and increases in harsh parenting across childhood are associated with more positive left amygdala-left prefrontal cortex connectivity during angry face processing
Note. (A) Greater initial levels of harsh parenting (intercept) predict more positive left amygdala-left orbitofrontal cortex (BA10) connectivity during angry face processing in adolescence. (B) Increases in harsh parenting from ages three to nine (indexed by a positive slope term) predict more positive amygdala-orbitofrontal cortex (BA10) connectivity during angry face processing in adolescence. (C) Image of identified clusters in (A) and (B) where yellow refers to the intercept results, blue refers to the slope result, and green areas show their overlap

CHAPTER IV

Age and Puberty Effects on Amygdala-Prefrontal Connectivity during Face Processing

Individual variability in processing emotional cues is linked to adaptive (e.g., social competence) and maladaptive (e.g., psychopathology) socioemotional outcomes (Cole, Hall, & Hajal, 2008; Feldman, Philipott, & Custrini, 1991). Much of this processing occurs at the level of the brain via connectivity between the amygdala and regions of the prefrontal cortex (PFC) (Cardinal, Parkinson, Hall, & Everitt, 2002; Somerville, Fani, & McClure-Tone, 2011). Investigations into the maturation of amygdala-PFC connectivity have informed our understanding of socioemotional development from childhood through young adulthood (Gabard-Durnam et al., 2014; Dylan G. Gee et al., 2013), but few studies have simultaneously examined the neural effects of age and pubertal development, which capture discrete features of maturation. Moreover, although amygdala subregions are structurally and functionally distinct (Bzdok, Laird, Zilles, Fox, & Eickhoff, 2013), no task-based designs have examined whether maturation effects on amygdala-PFC connectivity differ by amygdala subregion. Lastly, it is unclear whether these patterns occur during face processing broadly, or whether such patterns are specific to the most widely studied facial expression – fear. The current study sought to address these limitations by examining the effects of age and pubertal development on amygdala-PFC connectivity during emotional face processing, using a large population-based sample of children and adolescents.

Amygdala-PFC Connectivity

The corticolimbic circuit, including the amygdala and medial regions of the PFC, supports socioemotional processing by attending to salient features of the environment (e.g., faces) and activating physiological processes and attentional networks (Hariri, 2015). As the

central node in this circuit, the amygdala is responsible for salience detection, emotion

processing, and contingency learning (Davis & Whalen, 2001; LeDoux, 2000). Medial regions of

the PFC (mPFC), extending into the anterior cingulate cortex (ACC), support socioemotional

processing by integrating affective valuations from the amygdala with inputs from other neural

regions, effectively modulating amygdala activation (Etkin et al., 2011; Fuster, 2001; Motzkin et

al., 2015; Ochsner & Gross, 2005; Quirk & Gehlert, 2003). Functional parcellation of the frontal

lobe suggests a rostral-ventral distinction in emotion processing where the dorsal ACC and

dorsal-medial PFC support the cognitive components of emotion processing (e.g., appraisal),

whereas the ventral ACC (including the rostral and subgenual components) and the ventro-

medial PFC support emotion regulation (Etkin, Egner, & Kalisch, 2011; Fuster, 2001). Lateral

regions of the PFC are also recruited during emotion processing to facilitate response inhibition

and selective attention (Fuster, 2001; Hampshire, Chamberlain, Monti, Duncan, & Owen, 2010).

A recent meta-analysis of 49 task-based connectivity studies (Di, Huang, & Biswal, 2017)

showed that the amygdala was more strongly functionally connected to both medial (Brodmann

Areas [BA] 24, 32, 10) and lateral (BA 47) regions of the PFC while participants viewed facial

expressions (particularly fearful) versus baseline.

Age Effects on Amygdala-PFC Connectivity

Efforts to track the development of amygdala-PFC connectivity have largely examined

the influence of chronological age, with some inconsistencies in the direction of associations and

regions of the PFC identified. Using a task-based design, Gee and colleagues (2013) found that

amygdala-mPFC (centered within the ACC) connectivity shifted from positive to negative across

development: younger children ages 4 – 9 years demonstrated positive connectivity between the

amygdala and mPFC while viewing fearful facial expressions versus baseline; adolescents and

young adults showed negative amygdala-mPFC connectivity. In this context, "negative" connectivity is thought to reflect greater prefrontal recruitment in adolescence and adulthood (Casey, Jones, et al., 2008), accompanied by decreasing amygdala activation (e.g., Guyer et al., 2008) with advancing age. Although Wu et al. (2016) replicated these results of greater negative amygdala-ACC connectivity among older participants, Vink, Derks, Hoogendam, Hillegers, & Kahn (2014) found that older adolescents and adults evinced greater *positive* amygdala-*orbitofrontal cortex* (OFC) connectivity while viewing emotional versus neutral scenes. As several resting-state fMRI studies have documented age-related changes in amygdala connectivity with multiple regions of the PFC (Gabard-Durnam et al., 2014), it may be that the region-of-interest (ROI) approaches employed in previous studies fail to capture the extent of age-related changes in amygdala connectivity with widespread regions of the PFC. Second, as one study found opposing patterns of age-related changes in amygdala-prefrontal connectivity in boys versus girls (Alarcón, Cservenka, Rudolph, Fair, & Nagel, 2015b), attending to gender differences may clarifying previous contradictory directional associations.

Puberty Effects on Amygdala-PFC connectivity

Although studies of age effects on amygdala-PFC connectivity are more common in the literature, pubertal development is another indicator of maturation thought to impact brain development through biological and social channels. Biologically, gonadal steroids (i.e., testosterone, estradiol) drive increases in cellular formation in both the amygdala and PFC (Ahmed et al., 2008; Koss, Lloyd, Sadowski, Wise, & Juraska, 2015). Socially, pubertal development during adolescence marks a period of increased motivation to form peer and romantic relationships (Crone & Dahl, 2012), which is facilitated by processing socially-relevant facial expressions (Garcia & Scherf, 2015b). In humans, increases in testosterone and more

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advanced pubertal development have been associated with both greater (Moore et al., 2012; Spielberg, Olino, Forbes, & Dahl, 2014; Tyborowska, Volman, Smeekens, Toni, & Roelofs, 2016) and lesser (Forbes, Phillips, Silk, Ryan, & Dahl, 2011) amygdala and PFC *activation* to affective facial expressions and during emotion regulation. Gender differences in pubertal timing and tempo (i.e., girls enter puberty 1-2 years before boys and typically progress through puberty at a faster rate), as well as the effects of testosterone and estradiol on biological processes (Ahmed et al., 2008; Berenbaum & Beltz, 2011a; Koss et al., 2015; Schulz & Sisk, 2016), may explain some of these inconsistencies, but very few studies have examined whether the effects of puberty on corticolimbic function vary by gender. In the only study to examine pubertal effects on amygdala-prefrontal *connectivity* during face processing, Spielberg and colleagues (2015) found that larger increases in testosterone levels were associated with larger decreases in amygdala-orbitofrontal cortex coupling during angry and fearful versus neutral face processing, with a steeper decline for girls than boys (Spielberg et al., 2015), reiterating that these associations may be different by gender.

Amygdala Subregion Connectivity

Though oftentimes examined as a single structure, the amygdala is composed of multiple nuclei that differ in their cytoarchitectonic properties and connectivity with prefrontal regions (Amunts et al., 2005; Roy et al., 2009; Whalen & Phelps, 2009). Moreover, as psychopathology is associated with differential patterns of amygdala subregion activation and connectivity (e.g., Hyde et al., 2015; Qin et al., 2014), more research is needed to identify how amygdala subregion connectivity matures across development. Using resting-state fMRI, Gabard-Durnam and colleagues (2014) found that centromedial (CM), basolateral (BL), and superficial (SF) amygdala connectivity with the mPFC (BA 10, 32) strengthened positively with age. The BL

also evinced stronger negative connectivity with widespread neural regions (e.g., dorsal ACC) in older participants, while both the CM and SF exhibited stronger positive connectivity with dorsolateral regions of the PFC in older participants (i.e., CM-dorsolateral superior frontal gyrus; SF-inferior frontal gyrus). Interestingly, Alarcón, Cservenka, Rudolph, Fair, & Nagel (2015) revealed several sex differences in the direction of resting state connectivity by amygdala subregion, reiterating the importance of examining gender differences. To date, no task-based fMRI studies have evaluated whether the associations between age, puberty and amygdala-prefrontal connectivity vary by amygdala subregion.

Consistency across Emotional Facial Expressions

Lastly, many studies of task-based amygdala connectivity have only focused on fearful face processing. However, the amygdala plays a broad role in processing threat, danger, and salience (Davis & Whalen, 2001; LeDoux, 2000), which have also been probed with angry facial expressions and ambiguous neutral faces (Marusak, Zundel, Brown, Rabinak, & Thomason, 2017; Neta & Whalen, 2010; Pollak & Sinha, 2002). Moreover, previous research has shown that amygdala-prefrontal connectivity during fearful, angry, and neutral face processing differentially predicts subtypes of psychopathology (Gard et al., 2018; Waller, Gard, et al., 2018b). Thus, more research is needed to evaluate how whether the maturation of amygdala-prefrontal connectivity during emotional face processing is consistent across facial expressions.

Current Study

The overall aim of the current study was to examine the effects of age and pubertal development on amygdala-PFC connectivity during emotional face processing. In line with some reports of opposing maturation effects on resting state amygdala-PFC connectivity by gender (Alarcón et al., 2015b), we tested for gender X age interactions, and analyzed pubertal effects

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separately by gender (Mendle, Beltz, Carter, & Dorn, 2019). To extend previous work in resting-state fMRI designs (Alarcón et al., 2015b; Gabard-Durnam et al., 2014), we examined patterns of amygdala-PFC connectivity that were shared or unique to different amygdala subregions (CM, SF, BL). Lastly, we evaluated whether maturation effects on amygdala-PFC connectivity varied by the facial expression presented (i.e., fear, anger, neutral).

Consistent with previous work (e.g., Gee et al., 2013), we predicted that amygdala-PFC connectivity during face processing would strengthen (and become more negative) with increasing age and pubertal development. As our emotion processing task was an implicit emotion processing task that did not involve explicit regulation, we expected that target regions within the PFC would be localized to dorsal and lateral regions (e.g., dACC, dmPFC, dlPFC) rather than ventral regions (e.g., vACC, vmPFC) (Etkin et al., 2011). Based on previous research using resting-state designs, we hypothesized that age- and puberty-related changes in amygdala-prefrontal connectivity would be more similar for the CM and SF subregions, which would show functional connections with the lateral PFC, than the more widespread prefrontal connectivity patterns of the BL subregion (Gabard-Durnam et al., 2014; Roy et al., 2009). Lastly, we hypothesized that both chronological age and pubertal development would be associated with stronger amygdala-PFC negative connectivity similarly across all face types, based on a previous study using a similar fMRI task (Wu et al., 2016).

Method

Participants

Participants in this study were part of the Michigan Twin Neurogenetics Study (MTwiNS), a sample of 240 families (i.e., 480 twins) living in south-central Michigan (recruitment ongoing). The sample was recruited from twin families who participated in the

Twin Study of Behavioral and Emotional Development in Children (TBED-C) within the

Michigan State University Twin Registry (Burt & Klump, 2013). Twins were originally recruited

at age 6 – 10 into one of two cohorts. The first cohort was sampled from birth records to

represent all families with twins living within 120 miles of Michigan State University. The

second cohort was recruited from the same area, but only included families living in U.S. Census

tracts with over 10% of families living below the poverty line (i.e., the median poverty threshold

for the state of Michigan at the time) (Burt & Klump, 2013).

The current study includes data from the first 219 families (438 twins) of the MTwiNS

sample who participated in the neuroimaging protocol. Note than an additional 21 families (42

twins) participated in an earlier version of the neuroimaging protocol that could not be combined

with the current data. Participants ranged in age from 7 to 18 years (Mean age = 14.24 years;

62% male). Based on the recruitment strategy, the sample represents families living in south-

central Michigan with oversampling for families living in impoverished neighborhoods. This

sampling approach yields advantages over many previous studies of neural correlates of emotion

processing, which typically rely on small, convenience samples of often higher income families

(Falk et al., 2013; Henrich et al., 2010). Annual family income in the current study ranged from

less than $4,999 to greater than $90,000, with over one-third of the sample "near poor": families

who reported annual incomes of less than 200% of the poverty line (Yoshikawa, Aber, &

Beardslee, 2012). Moreover, at the time of data collection, 68% of the families lived in

impoverished neighborhoods, defined as a neighborhood in which 10% or more of residents live

below the poverty line.

Procedure

Youth and their primary caregivers (95% biological mothers) participated in a day-long protocol that included questionnaires, parent-child interaction tasks, collection of biological samples, an MRI mock scanning session, and an MRI scan. Twin pairs were randomized to determine which twin participated in the protocol activities first (except for the questionnaires, which were completed throughout the day and split by twin so the caregiver would not confuse which youth was being rated). Parents and youth consented to participate in the study (minors provided informed consent), and the study protocol was approved by the Institutional Review Board at the University of Michigan.

Measures

Age and pubertal status. Maturation was measured using chronological age (years) and perceived pubertal development. Perceived pubertal development was captured using parent report on the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), a measure that has been validated in mixed gender samples and in both rural and urban community samples (Petersen et al., 1988b; Robertson et al., 1992). The PDS exhibits convergent validity with other self-reported measures of pubertal timing, physical exam, and hormonal measures (J. Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Shirtcliff, Dahl, & Pollak, 2009). The PDS includes five questions about physical development during puberty, scored from 1 (*has not started*) to 4 (*development seems complete*). Three questions are relevant for both genders (i.e., changes in height, skin, pubic hair), with two additional gender-specific questions (e.g., for boys: voice changes; for girls: breast development). A continuous measure of perceived pubertal development was constructed as a mean score of all five items (Mean [SD] = 2.80 [.96], Min-Max = 1 – 4), which maintains the original scale of the measure (Petersen et al., 1988b). Girls were rated as more pubertally advanced by their mothers than boys (t(235) = -12.16, *p* < .001), as

were older participants (r = .84, *p* < .001). Only one participant was missing data using the

continuous pubertal development scoring method. Chronological age and continuous pubertal

development were highly correlated in girls (r = .84, *p* < .001) and boys (r = .79, *p* < .001)

(Figure 4.1a). To visualize the overlap of age and pubertal status in our sample, we used a

method by Carskadon & Acebo (1993) to classify the boys and girls in our sample into pubertal

stages (Figure 4.1b). A chi-square difference test indicated that girls were more likely to be

categorized into later pubertal stages than boys (χ^2(4) = 95.94, *p* < .001): 93% of the girls in our

sample were classified as late-pubertal or post-pubertal, compared to 44% of boys (Figure 4.1b).

fMRI Face Processing Task

Participants performed a modified version of a classic implicit emotional face processing

task, which consists of four blocks of perceptual face processing interleaved with five blocks of

sensorimotor control (see also Gard et al., 2018; Hariri, 2002; Manuck, Brown, Forbes, & Hariri,

2007). Participants viewed a trio of faces and selected one of two faces (bottom) identical to a

target face (Figure 4.2). Each face processing block consisted of 18 images, balanced for sex and

race, all derived from the NimStim standard set of pictures of facial affect (Nim Tottenham et al.,

2009). Each of the four face processing blocks consisted of a different emotional facial

expression (i.e., anger, fear, happy, neutral), and participants were randomly assigned to one of

four different orders of block presentation. During the sensorimotor control blocks, participants

viewed 12 trios of simple geometric shapes (circles, squares, triangles) and selected one of two

shapes (bottom) identical to a target shape (top). In the face processing blocks, each of the 18

face trios was presented for 2s with a variable interstimulus interval (ISI) of 2 – 6s for a total

block length of 98s. A variable ISI was used to minimize expectancy effects and resulting

habituation, as well as to maximize amygdala reactivity throughout the paradigm. In the

sensorimotor control blocks, each of the 12 shape trios was presented for 2s followed by a fixation cross for .5s, for a total block length of 30s. An additional 4s of crosshair presentation followed each block. Total task time was 578s.

Imaging Data Acquisition

Each participant was scanned with one of two research-dedicated GE Discovery MR750 3T scanners. To take advantage of improvements in MRI data acquisition and harmonize our protocol with the Adolescent Brain Development Cognitive Development Study (Casey et al., 2018), we altered our acquisition protocol after the first 140 families (i.e., 280 twins). For the first 140 families, one run of 298 volumes was collected for each participant. Blood oxygenation level–dependent (BOLD) functional images were acquired with a reverse spiral sequence (TR/TE=2000/30 milliseconds, flip angle = 90°, FOV = 22cm), which covered 43 interleaved oblique slices of 3-mm thickness. High-resolution T1-weighted SPGR images (156, 1mm-thick slices) were aligned with the AC-PC plane, and later used during normalization of the functional images. For the remaining 100 families (i.e., 200 twins), one run of 730 volumes was collected for each participant. BOLD functional images were acquired with a gradient-echo sequence with multiband acquisition (TR/TE=800/30 milliseconds, flip angle = 52°, FOV = 21.6cm), which covered 742 interleaved axial slices of 2.4-mm thickness. High-resolution T1-weighted SPGR images (208, 1mm-thick slices) were aligned with the AC-PC plane, and later used during normalization of the functional images. For both acquisition sequences, BOLD functional images encompassed the entire cerebrum and most of the cerebellum to maximum coverage of limbic structures.

Imaging Data Processing

Preprocessing for both acquisition sequences was identical, unless otherwise specified.

Functional data were preprocessed and analyzed using Statistical Parametric Mapping version 12

(SPM12; Wellcome Trust Centre, London, United Kingdom). Raw k-space data from reverse-

spiral sequence acquisition were de-spiked before reconstruction to image space. For gradient-

echo sequence data with multiband acquisition, task-specific field maps are constructed from

volumes of both anterior-to-posterior and posterior-to-anterior phase encoding; field maps are

applied after image construction to reduce spatial distortions and minimize movement artifacts.

Slice timing correction was performed using the 23rd slide as the reference slice (reverse-spiral

data) or the 2nd slice of each 10-slice band (gradient-echo data with multiband acquisition). Data

from both acquisition sequences were then spatially realigned to the 10th slice of the volume.

These spatially realigned data were coregistered to the high-resolution T1-weighted image, and

segmented and spatially normalized into standard stereotactic space (MNI template). Finally,

functional data were smoothed to minimize noise and residual difference in gyral anatomy with a

Gaussian filter set at 6mm FWHM. After preprocessing, the Artifact detection Tools (ART)

software package (http://www.nitrc.org/projects/artifact_detect/) was used to detect global mean

intensity and translation or rotational motion outliers (> 4.5 SD from the mean global brain

activation, >2mm movement or 2° translation in any direction); for each participant, nuisance

covariates were created and included in the individual-level model for all volumes exhibiting one

of these criteria. Additionally, because of the relatively extensive signal loss typically observed

in the amygdala, single-subject BOLD fMRI data were only included in subsequent analyses if

there was a minimum of 90% signal coverage in the amygdala bilaterally, defined using the

Automated Anatomical Labeling (AAL) atlas in the WFU PickAtlas Tool, version 1.04

(Maldjian et al., 2003). As the current paper sought to characterize amygdala-PFC connectivity,

we also removed participants with < 85% coverage in the frontal lobe, using a broad, large

structural frontal lobe mask generated with the WFU PickAtlas Tool, version 1.04. Lastly,

participants were excluded if accuracy performance on the task was less than 70%. Of the 438

youth in the current sample, usable fMRI data was available for 265 (61%) participants (Table

4.1). Youth with valid imaging data did not differ (all ps > .10) from youth without valid

imaging data on youth characteristics (i.e., chronological age, continuous perceived pubertal,

gender, and self-reported race and ethnicity) or primary caregiver characteristics (i.e., education

or annual income).

Functional Connectivity Analyses

Psychological-physiological interaction (PPI) analyses in the generalized PPI toolbox

(McLaren et al., 2012) in SPM12 were used to measure whole brain functional connectivity of

the left and right amygdala. In a PPI analysis, a design matrix is constructed at the level of the

individual with the following columns of variables: (a) a physiological variable that represents

the time course of the seed region (e.g., left or right amygdala) across the task, (b) a

psychological variable indicating the experimental variable (e.g., onset times for an experimental

condition), and (c) a product term of the interaction between the physiological and psychological

variables. At the individual level, the regression coefficient for the interaction term represents the

change in activity between the seed and identified "target region" across different conditions,

such as emotional face types (i.e., the seed-target functional coupling is context-dependent). The

gPPI toolbox developed by McLaren and colleagues (2012) allows for the simultaneous

specification of all task conditions and interactions with the seed region time series in the same

individual-level model (Friston et al., 1997). This is advantageous because it reduces the number

of specified models and the overall type I error rate. Moreover, compared to standard correlation

methods (Rissman, Gazzaley, & D'esposito, 2004), gPPI methods have been found to be more powerful for detecting functional connectivity during block designs (Cisler, Bush, & Steele, 2014).

Seed regions were defined as the left and right whole amygdala, BL, CM, and SF (see Figure 4.3 for masks of these seed regions). The whole amygdala was defined using the AAL definition within the WFU PickAtlas Tool (Maldjian et al., 2003). The BL, CM, and SF subregions were defined using maximum probability maps of cytoarchitectonic boundaries developed by Amunts et al. (2005) and implemented in the SPM Anatomy Toolbox (Eickhoff et al., 2005). Eight general linear models at the individual level were constructed (i.e., one for each seed region). Using the gPPI toolbox, the time series of the left or right seed region was entered as the physiological variable in the design matrix, the explanatory variables for each of the five conditions in our task (i.e., facial expressions of fear, anger, happy, and neutral faces, and shapes) were entered as psychological variables, and the five product terms between the amygdala seed and conditions were entered as the interaction terms. We specified three primary contrasts at the individual level: fearful faces interaction term > shapes interaction term, angry faces interaction term > shapes interaction term, and neutral faces interaction term > shapes interaction term. Practically, this can be interpreted as a difference in slopes: is slope A (i.e., the interaction between amygdala reactivity and the fear/angry condition) greater or lesser than slope B (i.e., the interaction between amygdala reactivity and the shapes condition). Individual-level slopes (i.e., the betas corresponding to the interaction terms, e.g., fearful facial expressions, angry facial expressions) can then be extracted to determine the direction and strength of connectivity during the two conditions (e.g., fear, neutral). Contrasts from the individual level models were then used in random effects, group-level models to evaluate the impact of

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chronological age and pubertal development on amygdala-PFC functional connectivity during face processing. These models assess whether age and chronological age are associated with the difference in connectivity between conditions (or the difference in slopes).

Although the purpose of the current paper was to examine the influence of age and pubertal development on amygdala-PFC connectivity during face processing, it is important to note that we tested for whole brain group-level PPI effects. Generally, across the entire sample, the left and right amygdala exhibited greater positive connectivity between the left and right amygdala and visual processing regions (e.g., fusiform gyrus) while participants viewed fearful, angry, neutral, and happy facial stimuli versus shapes (Table 4.2). By contrast, the left and right amygdala exhibited greater negative connectivity with prefrontal regions (e.g., cingulate gyrus, middle frontal gyrus) during angry, happy, and neutral face processing versus shapes (Table 4.3). The absence of group-level negative PPI effects during fear versus shapes processing stands in contrast to previous reports (Di et al., 2017; Gard et al., 2018). However, as shown below, we believe this is due to robust maturational effects on amygdala-PFC connectivity during fearful face processing specifically.

Group-level analyses. To identify target regions in the PFC that were functionally coupled with the amygdala and associated with maturation, we used a created a large mask defined by Brodmann's areas 9, 10, 11, 47, 24, 32, and 25, using the WFU PickAtlas Tool, version 1.04 (Maldjian et al., 2003). We used this PFC mask because (1) larger masks require stricter correction for multiple comparisons (i.e., more voxels in the mask) and thus are less biased towards identifying effects, (2) by using a large mask composed of multiple PFC regions, we could compare our results to several studies that used different definitions of the PFC (Dylan G. Gee et al., 2013; Wu et al., 2016), and (3) the seven Brodmann's areas we identified have

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each been shown in non-human primate (Amaral & Price, 1984; Ghashghaei et al., 2007) and human studies (Goetschius et al., 2019) to be structurally connected to the amygdala. Only ipsilateral connections between the amygdala and PFC were examined, because neural tracer studies in non-human primates suggests that first order amygdala connections are primarily ipsilateral (Ghashghaei et al., 2007). To identify the precise location of target regions within the PFC, we used the WFU PickAtlas to report regional labels (i.e., AAL definitions) and Brodmann areas. To address confounding by scanner sequence (i.e., gradient-echo reverse spiral sequence or gradient-echo-planar sequence with multiband acquisition), all group-level models included a dummy code for sequence (0 = spiral; 1 = multiband).

We corrected for multiple comparisons using the updated version of 3dClustSim (Cox, Chen, Glen, Reynolds, & Taylor, 2017a) in AFNI (Cox, 1996). This procedure, which is dynamic to each group-level model based on changes in smoothing values, resulted in a minimum cluster size of 16 voxels that reached a mask-corrected threshold of $p < .05$ and a voxel-wise threshold of $p < .001$. Note that our cluster thresholds were based on 2-sided tests and used the nearest neighbor definition of "face and edge" (i.e., 3dClustSim command: NN=2).

To reduce the number of statistical tests, we implemented a stepwise approach to evaluate maturational effects on amygdala-PFC connectivity. For each predictor, we first examined its' effect on left or right amygdala-PFC connectivity; if there was a statistically significant association, we examined additional associations using the BL, CM, and SF seed regions. We used a masking approach method (Gabard-Durnam et al., 2014; Roy et al., 2009) to evaluate patterns of amygdala-PFC connectivity that were shared (i.e., "inclusive masking") to each amygdala subregion (i.e., BL, CM, SF). Note that this masking method did not add additional

statistical tests; this approach was merely used to identify the shared and unique patterns of amygdala-PFC during face processing by amygdala subregion.

Continuous age and pubertal development. Multiple linear regression models were used to evaluate the effects of age and pubertal development on amygdala-PFC connectivity. In the total sample, chronological age was specified as primary predictor, with participant race (0 = White [77%]; 1 = Non-White), and scanner sequence as covariates. In a separate set of models, we examined whether gender moderated the effect of age on amygdala-PFC connectivity by including gender and an interaction term between centered age in years and gender as covariates. Pubertal development was analyzed separately in boys ($N = 141$) and girls ($N = 106$), with the continuous puberty score as the primary predictor and participant race and scanner sequence as covariates. For each significant association between age or pubertal development and amygdala-PFC connectivity, we examined whether the associations remained significant after additional correction for the other measure of maturation (i.e., pubertal development/age).

Age- and puberty-restricted models. Given the high correlation between age and continuous pubertal development in our sample (Figure 4.1) that may preclude identifying independent effects of age and pubertal development, we specified two additional sets of models in restricted samples that held constant one of the two constructs. First, we selected late-pubertal youth ($N = 110$) and re-examined the effects of age in years on amygdala-PFC connectivity during face processing, controlling for participant race and scanner sequence. Second, we selected 15-year-olds (boys: $N = 32$; girls: $N = 28$) and re-examined the effects of continuous pubertal development on amygdala-PFC connectivity during face processing, controlling for participant race and scanner sequence.

Results

Effects of age on condition-specific amygdala-PFC connectivity

Chronological age was associated with amygdala-PFC connectivity while participants viewed fearful (but not angry or neutral) facial expressions versus shapes (Table 4.4). Older adolescents evinced greater positive right amygdala – right inferior frontal gyrus (IFG; BA 11) connectivity to fear versus shapes (Figure 4.4). An examination of the right amygdala subregions revealed that advancing age was also associated with greater positive right CM – right insula (BA 10) and right SF – right IFG (BA 47) connectivity during fear versus shapes. There was substantial overlap in the IFG target region between the right whole amygdala and the right SF (100%), with less overlap between the right whole amygdala and right CM (17%). None of the age associations remained significant when controlling for gender and the interaction of age and gender did not predict condition-specific amygdala-PFC connectivity (Table 4.4). However, subgroup analysis by gender revealed that for boys only, advancing age was associated with stronger positive right amygdala – right IFG connectivity during fear versus shapes and, as in the total sample, this effect was also seen using the right SF amygdala as the seed region (Table 4.4), though we caution that these results are tentative given the non-significant interaction between age and gender. When the continuous pubertal development score was included as a covariate in the model, chronological age was no longer predictive of condition-specific amygdala-IFG connectivity. In models that restricted the sample to late-pubertal adolescents ($N = 110$), designed to implicitly control for pubertal development, age was also not uniquely associated with condition-specific amygdala-PFC connectivity (Table 4.4).

Effects of pubertal development on condition-specific amygdala-PFC connectivity

For both boys and girls, pubertal development was also associated with amygdala-PFC connectivity while participants viewed fearful (but not angry or neutral) facial expressions versus

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shapes (Table 5), but in different directions. For boys, more advanced pubertal development was associated with greater positive right amygdala – right IFG (BA 11) connectivity to fear versus shapes, which was the same direction as the effects of chronological age (Table 4.5; Figure 4.5a). The identified cluster in the right IFG overlapped 100% with the cluster identified in models with age as the predictor. Moreover, advanced pubertal development was also associated with greater right SF – right IFG (BA 47) connectivity during fear versus shapes (Table 4.5), with 90% overlap in the IFG with right amygdala seed models. When chronological age was added as a covariate, pubertal development was no longer predictive of condition-specific amygdala-IFG connectivity. For girls, by contrast, more advanced pubertal development was associated with weaker positive left amygdala – left superior medial frontal gyrus (BA 9) connectivity during fear versus shapes (Figure 4.5b), and this association remained significant after accounting for chronological age (Table 4.5). Moreover, advanced pubertal development was also associated with weaker positive left BL – left superior medial frontal gyrus (BA32) connectivity during fear versus shapes, with 87% overlap in the superior medial frontal gyrus as in the left amygdala seed model (Table 4.5). In secondary analyses that restricted the samples to 15-year-old boys ($N = 32$) or girls ($N = 28$), pubertal development was not uniquely associated with condition-specific amygdala-PFC connectivity for either gender (Table 4.5), although these models were likely underpowered.

Discussion

In a population-based sample of twins aged 9 to 18 years, we examined the effects of chronological age and pubertal development on amygdala-PFC connectivity during face processing. Results suggested differential patterns by gender, such that age and puberty were associated with stronger positive amygdala-inferior frontal gyrus (IFG) connectivity for boys

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whereas puberty was associated weaker positive amygdala-superior medial frontal gyrus connectivity for girls. In results that largely paralleled existing research using resting-state fMRI, we found more similarities in prefrontal connectivity with centromedial (CM) and superficial (SF) amygdala subregions than the basolateral (BL) subregion. Lastly, the effects of maturation on condition-specific amygdala-PFC connectivity were not widespread across emotional facial stimuli: associations were localized to fear versus shapes processing.

Contrary to our hypothesis, older youth evinced stronger *positive* right amygdala – right IFG connectivity while viewing fearful facial expressions versus shapes. Thus, our findings were both in the opposite direction and localized to a different region in the PFC than Gee et al. (2013) and Wu et al. (2016), who reported stronger *negative* amygdala – mPFC connectivity during emotion processing in older participants. In supplemental analyses where we created masks of the mPFC by creating 10mm spheres around the peak coordinates from Gee et al. (2013) and Wu et al. (2013), age was not associated with condition-specific amygdala-mPFC connectivity (results not shown) suggesting that our discrepant results are not due to different masking procedures. Although Wu et al. (2016) implemented a similar emotional faces matching task and examined multiple facial expressions, Gee et al. (2013) utilized a paradigm that only presented female faces and exclusively examined fearful facial expressions; these study design differences may contribute to our contradictory findings. The IFG target region we identified overlapped with Brodmann's areas 11 and 47, which encompass dlPFC and lateral-OFC regions (Fuster, 2001). Both the lateral OFC and dlPFC are involved in the cognitive components of emotion processing (Cardinal et al., 2002; Fuster, 2001; Ochsner & Gross, 2005), and are engaged during cognitive-emotion interference tasks (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Cardinal et al., 2002). For example, greater activation of the lateral PFC has been associated with successful

inhibition of negative affect (Phan et al., 2005), engagement of mindfulness strategies (Opialla et al., 2015), and less cognitive interference by emotional distracters (Dolcos, Kragel, Wang, & McCarthy, 2006) during cognitive reappraisal tasks. Thus, our finding of stronger positive amygdala-IFG connectivity in older youth may capture a different aspect of emotion processing (i.e., cognitive inhibition) than negative amygdala-mPFC connectivity, thought to reflect affective inhibition (Dylan G. Gee et al., 2013; Wu et al., 2016).

The effects of pubertal development on condition-specific amygdala-PFC connectivity during fear versus shapes processing were different for boys and girls. More pubertally-advanced boys exhibited stronger positive right amygdala-right IFG (BA 11/47) connectivity, whereas more pubertally-advanced girls had weaker positive left amygdala – left superior medial frontal gyrus (SMFG; BA32) connectivity during fear versus shapes processing. That our results revealed opposite patterns of maturation effects on amygdala-PFC connectivity by gender is consistent with Alarcón and colleagues (2015), who found that age was associated with greater *positive* right BL-right superior frontal gyrus resting-state connectivity in boys but stronger *negative* connectivity in girls (see also Ernst et al., 2019). Gender differences in puberty effects on corticolimbic connectivity might reflect higher rates of internalizing disorders in girls (Kessler et al., 2012) that emerge during puberty and have been linked to gender differences in pubertal timing (Angold et al., 1998; Berenbaum & Beltz, 2011; Hyde, Mezulis, & Abramson, 2008; Negriff & Susman, 2011; Thapar, Collishaw, Pine, & Thapar, 2012).

For boys, the effects of age and pubertal development were indistinguishable: neither age nor pubertal development remained significant predictors of condition-specific right amygdala-right IFG connectivity when controlling for their overlap. Moreover, there was 100% overlap in the IFG target region across both analyses. Our attempts to disambiguate the neural effects of age

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and puberty (i.e., by restricting the sample to 15-year-old boys or late-developing youth) were unsuccessful, although these analyses were vastly underpowered. One explanation for confounded maturation effects on condition-specific amygdala-PFC connectivity in boys is that our sample only included four (2.8%) post-pubertal boys, compared to 47 (44.3%) post-pubertal girls. As previous research suggests that group differences in corticolimbic function may arise when comparing late- and post-pubertal youth to pre- and early-pubertal youth (Forbes et al., 2011), our results might reflect sample composition in boys' pubertal development. It could also be that case that pubertal tempo (i.e., how quickly an individual matures through puberty compared to their peers; Mendle et al. [2019]) rather than pubertal stage or timing is more relevant for brain development in boys. Differential effects of pubertal timing and tempo have been reported with respect to several socioemotional behaviors including internalizing and externalizing behaviors (Baams, Dubas, Overbeek, & van Aken, 2015; Castellanos-Ryan, Parent, Vitaro, Tremblay, & Séguin, 2013; Marceau, Ram, Houts, Grimm, & Susman, 2011), but few neuroimaging studies have integrated measures of puberty beyond timing (Goddings, Beltz, Peper, Crone, & Braams, 2019; Vijayakumar, Op de Macks, Shirtcliff, & Pfeifer, 2018).

In addition to identifying maturational effects on condition-specific amygdala-PFC connectivity, we also found patterns of amygdala-PFC connectivity by amygdala subregion. Age effects on stronger positive right amygdala – right IFG connectivity during fear versus shapes processing were also observed in models using the right CM and right SF as seed regions, with no overlap in BL models. Gabard-Durnam et al. (2014) similarly reported age-related increases in positive connectivity between the CM- and SF- and dorsolateral regions of the PFC during resting state fMRI ; our results extend this literature to the context of fear processing. A meta-analysis of 6,500 fMRI and PET experiments (Bzdok et al., 2013) found that both the CM and

the SF subregions, but not the BL, coactivated with the bilateral IFG (see also Bach et al. [2011] and Roy et al. [2009]), further highlighting that functional connectivity patterns may be shared across the CM and SF, particularly in connection to the IFG. By contrast, puberty effects on weaker positive left amygdala – left superior medial frontal gyrus connectivity (localized to the rostral ACC within BA32; Fuster [2001]) were observed in models using the BL as the seed region, but not the CM or SF. Although Gabard-Durnam et al. (2014) found that older youth showed stronger negative BL-rostral ACC connectivity during resting-state fMRI, discrepancies in the direction of effects may be due to our examination of puberty instead of age, implementation of task-based fMRI (i.e., as opposed to resting-state), and delineation of associations by gender. As previous research has found differential effects of amygdala subregion function with several socioemotional outcomes including externalizing (Aghajani et al., 2017; Hyde et al., 2015) and internalizing (Jalbrzikowski et al., 2017) disorders, post-traumatic stress disorder (V. M. Brown et al., 2014), and temperament (Roy et al., 2014), more research is needed to understand the maturation of the CM, SF, and BL amygdala subregions, particularly using task-based designs that index emotion processing.

Lastly, neither chronological age nor pubertal development were associated with changes in amygdala-PFC connectivity during angry or neutral face processing versus shapes, suggesting that the effects of maturation on corticolimbic connectivity are particularly relevant for fear processing. Group-level PPI models that did not consider maturation foreshadowed these effects: across the entire sample, the left and right amygdalae were more strongly negatively coupled with several prefrontal regions during angry and neutral but not fearful face versus shapes processing. That is, including youth of all ages and pubertal stages washed out the group-level PPI effect for fear versus shapes, but not for angry or neutral faces versus shapes. Behaviorally,

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there is some evidence to suggest that accuracy in matching emotional facial expressions increases with age more so for fearful facial expressions than for angry expressions or neutral faces (Herba, Landau, Russell, Ecker, & Phillips, 2006), although other behavioral paradigms report conflicting results (e.g., Montirosso, Peverelli, Frigerio, Crespi, & Borgatti, 2010). The specificity of maturational effects on corticolimbic connectivity during fear processing may also reflect the relative sparsity of fearful facial expressions compared to neutral or angry faces (Calvo, Gutiérrez-García, Fernández-Martín, & Nummenmaa, 2014; Somerville & Whalen, 2006). As Calvo and colleagues (2014) also found that recognition accuracy increased with the frequency of exposure, it may be that the relative sparsity of fearful facial expressions environmentally necessitates a longer developmental window for the corticolimbic system to support fear processing. To evaluate this tentative hypothesis, more research is needed to link subjective assessments of emotional facial expressions with corticolimbic connectivity during face processing, across multiple developmental windows.

Limitations

Despite the use of a large population-based sample of children and adolescents with variability in age and pubertal development, attention to condition-specific changes in amygdala-PFC connectivity that were shared and unique across amygdala subregions, and evaluation of whether maturational effects on corticolimbic connectivity varied by the facial expression examined, several limitations warrant consideration. First, our study relied on a cross-sectional design, which limits our interpretation to inter-individual differences in brain function (e.g., comparing younger and older youth) rather than intra-individual change, which would provide more robust evidence for maturational effects on condition-specific amygdala-PFC connectivity. As there are no existing longitudinal studies that have examined changes in pubertal status or age

on changes in task-based amygdala-PFC connectivity during face processing, this is an important future direction (see Jalbrzikowski et al., 2017, for an example using resting-state fMRI).

The current study was also limited by measurement of pubertal development. Although the Pubertal Development Scale (Petersen et al., 1988b) is widely used in the neuroimaging literature to index puberty (Goddings et al., 2019; Vijayakumar et al., 2018), it includes aspects of both adrenarche and gonadarche. Research from structural MRI studies suggests that these two phases of pubertal development may impact brain development differently (Byrne et al., 2017). Gonadarche may be more relevant than adrenarche for corticolimbic function during face processing for both biological and social reasons. Biologically, gonadal hormones (i.e., testosterone, estradiol) have been shown in animal models and human studies to target corticolimbic regions including the amygdala and mPFC (Ahmed et al., 2008; Goddings et al., 2019; Koss et al., 2015; Schulz & Sisk, 2016). Socially, the maturation of observable secondary sexual characteristics that occur during gonadarche (e.g., breast development in girls, voice changes in boys) influence how adolescents perceive themselves and how they are perceived and treated by others (Blakemore & Mills, 2014). Sociocultural influences during gonadarche could potentially impact interpersonal functioning and how youth process socially-relevant cues like emotional facial expressions (Scherf, Smyth, & Delgado, 2013).

There are also several limitations of our neuroimaging analyses. First, as is common in many emotional faces matching paradigms (Carré, Fisher, Manuck, & Hariri, 2012; Fakra et al., 2009; Gard et al., 2018; Hariri et al., 2002; Swartz, Knodt, Radtke, & Hariri, 2015), the baseline condition in our task was a non-face shapes stimulus. Thus, our maturation effects on amygdala-PFC connectivity during fearful face versus shapes processing could be capturing amygdala-PFC connectivity during general face processing rather than fear processing specifically. However,

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we would then expect to find maturation effects on condition-specific amygdala-PFC connectivity during other face conditions (i.e., angry, neutral), which we did not. Second, PPI analyses do not establish directionality; no information is provided about the temporal ordering of activation in the seed or target region (Friston et al., 1997). Network mapping approaches (e.g., GIMME; Beltz & Gates [2017]) can be applied to tasks with blocked designs to determine how temporal patterns of connectivity between several a-priori identified nodes (e.g., amygdala subregions) are different across conditions (e.g., fearful or angry faces). Lastly, although our sample was larger than the existing literature linking age or pubertal development to amygdala-PFC maturation, our sample was also composed of twins. By not accounting for the nested structure of our data (i.e., twins are nested within family), our results may be biased (Henry, Tolan, & Gorman-Smith, 2005). Therefore, the current results need to be replicated in a genetically independent sample (i.e., one random twin from each pair) or using multilevel modeling.

Despite these limitations, the current study builds upon previous work by charting the effects of age and pubertal development on amygdala-PFC connectivity during face processing. Our results stress the importance of examining gender differences in these associations, as boys and girls may show different patterns of maturation depending on the measure used (i.e., age or puberty). For girls, it may be that early-starting puberty increases risk for internalizing disorders via weaker amygdala-PFC connectivity during fear processing, a maturation pattern we identified in the current study.

Table 4.1. Faces task participant drop out

	Spiral Sequence		Echo-planar Sequence with Multiband Acquisition	
	Number Lost	Participants with data	Number Lost	Participants with data
Original sample		238		200
- Refused scan	16		12	
- Medical restriction*	25		8	
- Data collection/scanner error	2		1	
- Partial data	0		3	
Total lost	43		24	
Sample with imaging data		195		176
- Whole brain coverage loss by visual inspection	2		1	
- Low amygdala coverage (< 90%)	7		58	
- Low prefrontal lobe coverage	3		19	
- Low task performance (< 70%)	11		3	
- Ghosting on functional scans	1		1	
- Exceeded movement thresholds	0		0	
Total lost	20		62	
Sample with usable imaging data		171		94

*includes having a permanent retainer or braces, non-MRI safe implanted medical devices, having BBs/pellets or other non-removable metal inside of body, recent surgery, exceeds table weight limit, impaired vision not correctable with MRI-safe glasses. Note that an additional 21 families (42 twins) received an earlier version of the task that was not comparable to the current version; these participants were excluded from all analyses.

Table 4.2. Target neural regions exhibiting greater positive connectivity during face versus shapes stimuli processing

Contrast	Seed Region	Target Region	(x,y,z), t extent threshold, k cluster size
Fear > Shapes	left amygdala	left fusiform gyrus	(-36,-46,-20), $t = 4.36$, $k = 165$
	right amygdala	right fusiform gyrus	(36,-46,-18), $t = 5.25$, $k = 100$
Anger > Shapes	left amygdala	left fusiform gyrus	(-36,-52,-20), $t = 5.24$, $k = 276$
	left amygdala	left inferior parietal lobule	(-26,-56,42), $t = 4.55$, $k = 176$
	left amygdala	left lingual gyrus	(-8,-68,4), $t = 4.47$, $k = 361$
	right amygdala	right fusiform	(38,-52,-12), $t = 4.79$, $k = 90$
Neutral > Shapes	left amygdala	left hippocampus	(-20,-30,-4), $t = 5.22$, $k = 78$
	left amygdala	left postcentral gyrus	(-50,-20,40), $t = 4.14$, $k = 114$
	left amygdala	left middle occipital gyrus	(-26,-56,36), $t = 4.03$, $k = 106$
	left amygdala	left calcarine	(-6,-74,8), $t = 3.99$, $k = 221$
	right amygdala	right calcarine	(-20,-64,6), $t = 4.57$, $k = 131$
Happy > Shapes	left amygdala	none	NA
	right amygdala	right inferior frontal gyrus	(44,28,20), $t = 4.29$, $k = 95$
	right amygdala	right fusiform gyrus	(26,-74,-14), $t = 3.90$, $k = 66$

Note: N=265. Coordinates are in Montreal Neurological Institute (MNI) space.
Clusters are corrected for multiple comparisons across left or right whole brain at p < .05, using the 3dClustSim correction procedure and a voxel-wise threshold of $p < .001$ (minimum cluster = 62 voxels).

Table 4.3. Target neural regions exhibiting greater negative connectivity during face versus shapes stimuli processing

Contrast	Seed Region	Target Region	(x,y,z), t extent threshold, k cluster size
Fear > Shapes	left amygdala	none	NA
	right amygdala	none	NA
Anger > Shapes	left amygdala	left anterior cingulate – BA32	(-4,48,-4), t=5.42, k=264
	left amygdala	left middle cingulate – BA32	(-2,-18,36), t=4.81, k=217
	left amygdala	left middle temporal gyrus	(-52,-14,-14), t=3.91, k=141
	left amygdala	left precuneus	(-8,-58,14), t=3.78, k=79
	right amygdala	right superior medial frontal gyrus – BA 32	(2,48,0), t=4.90, k=66
	right amygdala	right middle temporal gyrus	(58,-12,-16), t=4.39, k=137
	right amygdala	right middle cingulate – BA31	(4,-28,40), t=4.09, k=83
Neutral > Shapes	left amygdala	left middle temporal gyrus	(-58,-6,-18), t=4.40, k=137
	left amygdala	left middle cingulate – BA31	(-10,-46,32), t=3.98, k=113
	right amygdala	right angular gyrus	(58,-52,36), t=3.91, k=190
Happy > Shapes	left amygdala	left middle cingulate – BA24	(-4,-20,38) t=4.71, k=142
	left amygdala	left cuneus	(-10,-64,22) t=4.38, k=444
	right amygdala	none	NA

Note: N=265. Coordinates are in Montreal Neurological Institute (MNI) space.
Clusters are corrected for multiple comparisons across the left or right whole brain at p < .05, using the 3dClustSim correction procedure and a voxel-wise threshold of $p < .001$ (minimum cluster = 62 voxels).

Table 4.4. Age effects on condition-specific amygdala-PFC connectivity during face processing in the total sample

Predictor and Contrast	Whole Amygdala	Centromedial Amygdala	Basolateral Amygdala	Superficial Amygdala
Total Sample				
Fear > Shapes	*Left*: none *Right*: (+) (30,36,-12) inferior frontal gyrus (BA11), $t = 4.15, k = 30$	*Left*: NA *Right:* (+) (28,22,-10) insula/inferior frontal gyrus (BA47) $t = 4.08, k = 29$	*Left*: NA *Right*: none	*Left*: NA *Right* (+): (26,32,-12) inferior frontal gyrus (BA47) $t = 3.78 , k = 28$
Anger > Shapes	*Left*: none *Right*: none	NA	NA	NA
Neutral > Shapes	*Left*: none *Right*: none	NA	NA	NA

Note. $N = 265$. Models controlled for participant race and scanner sequence. All estimates were corrected for multiple comparisons at a mask-corrected threshold of $p < .05$ and a voxelwise threshold of $p < .001$. The interaction of age and gender did not predict condition-specific amygdala connectivity with any region in the prefrontal cortex mask, but subgroup analysis revealed that the effect of age on condition-specific amygdala-PFC connectivity was only present in boys: right amygdala – right IFG (x, y, z = 30, 36, -12; t = 4.46; k = 58); right centromedial amygdala – right IFG/insula (x, y, z = 40, 20, -12; t = 3.89; k = 22); right superficial amygdala – right IFG (x, y, z = 28, 32, -10; t = 4.29; k = 35). In models that restricted the sample to late-pubertal adolescents ($N = 110$), age did not predict condition-specific left or right amygdala-PFC connectivity during fearful, angry, or neutral face processing versus shapes.

Table 4.5. Pubertal development effects on condition-specific amygdala-PFC connectivity during face processing, for boys and girls separately

Predictor and Contrast	Whole Amygdala	Centromedial Amygdala	Basolateral Amygdala	Superficial Amygdala
Girls				
Fear > Shapes	*Left*: (-) (-2,40,32) superior medial frontal gyrus (BA9)† t = 3.72, k = 79 *Right*: none	*Left*: none *Right*: NA	*Left*: (-) (-4,38,28) superior medial frontal gyrus (BA32) t = 3.59, k = 46 *Right*: none	*Left*: none *Right*: none
Anger > Shapes	*Left*: none *Right*: none	NA	NA	NA
Neutral > Shapes	*Left*: none *Right*: none	NA	NA	NA
Boys				
Fear > Shapes	*Left*: none *Right*: (+) (30,36,-12) inferior frontal gyrus (BA11) t = 4.51, k = 40	*Left*: NA *Right*: none	*Left*: NA *Right*: none	*Left*: NA *Right*: (+) (26,34,-10) inferior frontal gyrus (BA47) t = 4.28, k = 24
Anger > Shapes	*Left*: none *Right*: none	NA	NA	NA
Neutral > Shapes	*Left*: none *Right*: none	NA	NA	NA

Note. $N = 265$.

†significant when controlling for perceived pubertal development

All models controlled for participant race and scanner sequence. All estimates were corrected for multiple comparisons at a mask-corrected threshold of $p < .05$ and a voxelwise threshold of $p < .001$. In models that restricted the sample to 15-year-old boys ($N = 32$) or girls ($N = 28$), puberty did not predict condition-specific left or right amygdala-PFC connectivity during fearful, angry, or neutral face processing versus shapes.
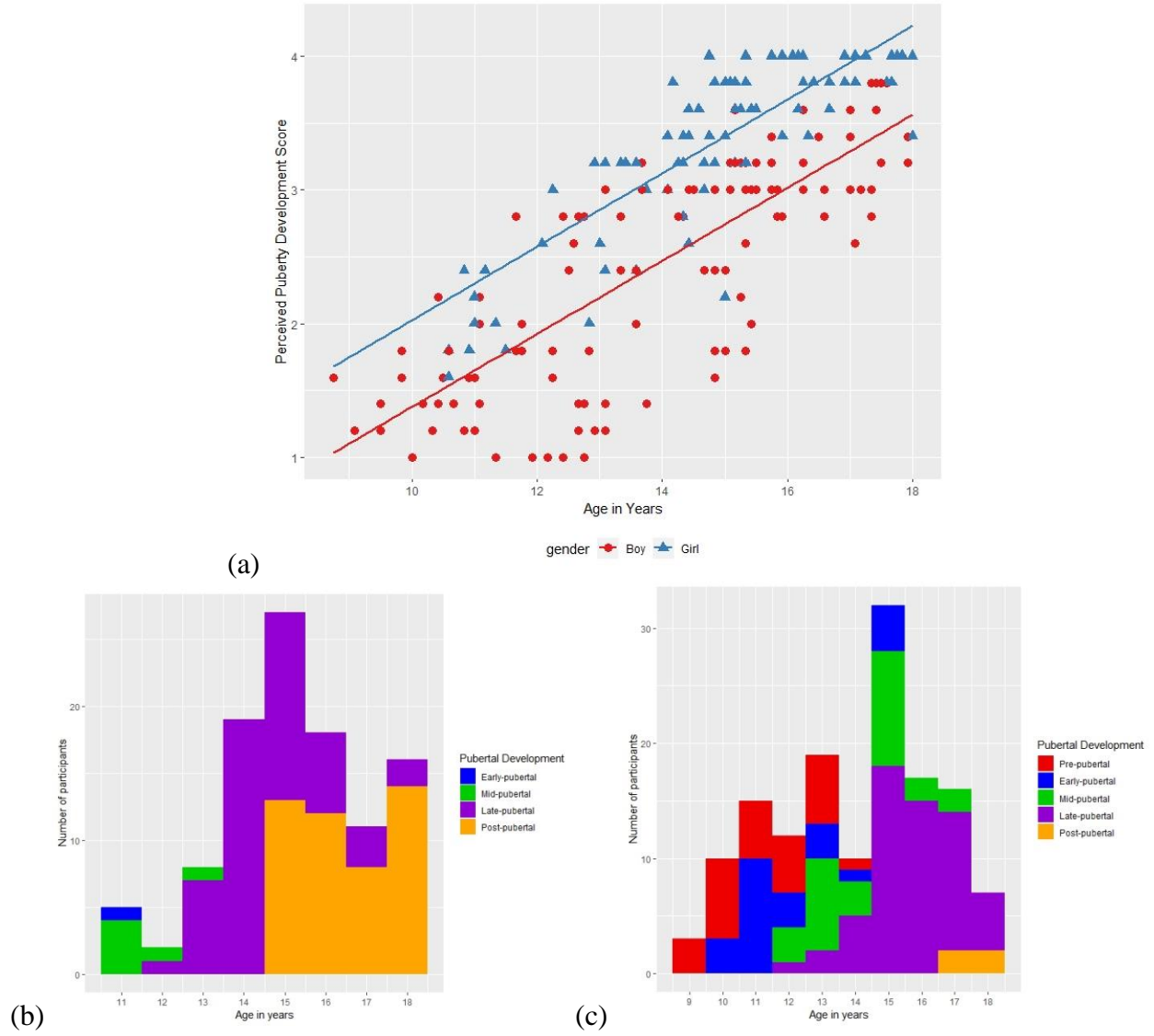
(a)

(b)

(c)

Figure 4.1. Associations between age and pubertal development in the current sample by gender
Note. (a) Zero-order correlation between chronological age and continuous perceived pubertal development for boys and girls separately. (b) Distribution of pubertal development categories across age for girls ($N = 106$); (c) Distribution of pubertal development categories across age for boys ($N = 141$).
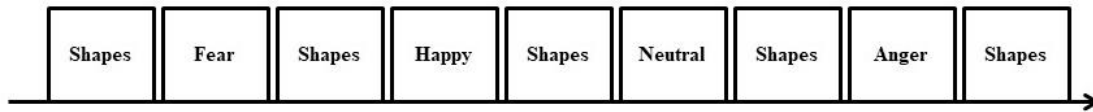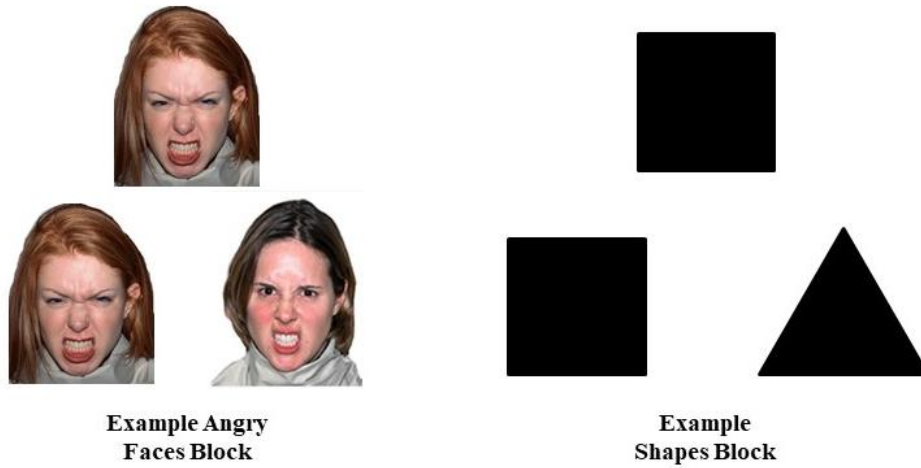
Figure 4.2. Implicit emotional faces matching fMRI task
Note. This blocked task design included four face blocks and five shapes blocks. Participants were randomly assigned to one of four different presentation orders. Total task time was 9.63 minutes.
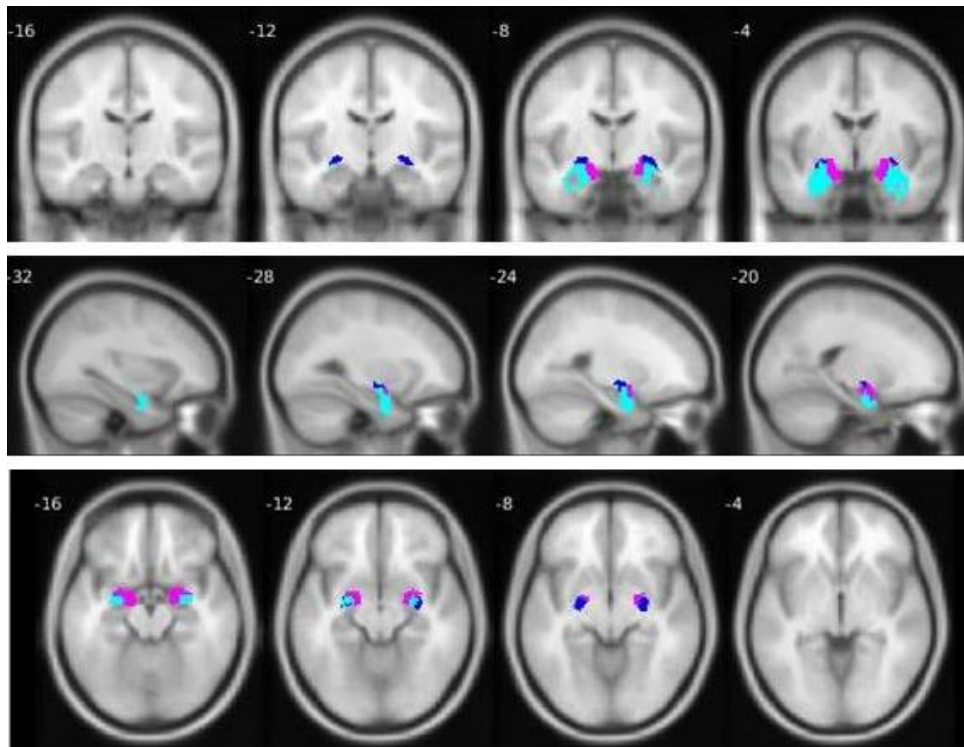
Figure 4.3. Basolateral, centromedial, and superficial amygdala subregions
Note. Light blue indicates the basolateral amygdala (BL) subregion, dark blue indicates the
centromedial amygdala (CM) subregion, and pink indicates the superficial amygdala (SF)
subregion. Group-level coverage of each amygdala subregion is as follows: BLA = 337 voxels;
CMA = 112 voxels; SFA = 209 voxels. All subregion masks were defined by Amunts et al.
(2005) and implemented in the SPM Anatomy Toolbox (Eickhoff et al., 2005).

(A)   (B)
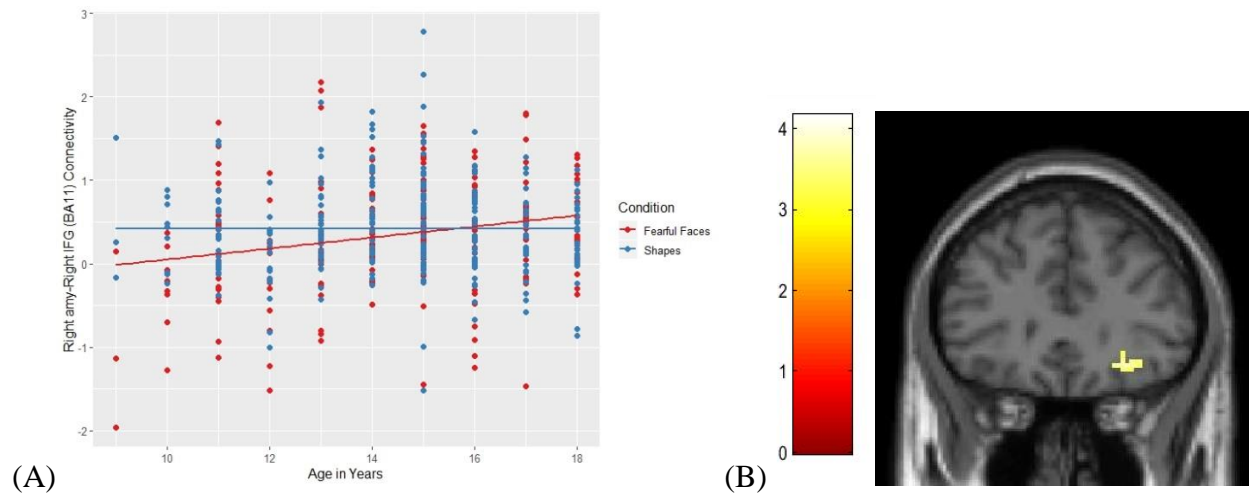
Figure 4.4. Age effects on amygdala-PFC connectivity during fear versus shapes processing in the total sample

Note. N = 265. (A) Chronological age is positively associated with right amygdala – right inferior frontal gyrus (IFG) connectivity during fear but not shapes processing. The data points are extracted mean connectivity estimates of right amygdala – right inferior frontal gyrus during the fear or shapes condition (i.e., from the betas of each individual's psycho-physiological interaction model). There were no non-linear effects of age on right amygdala-right IFG connectivity during fear versus shapes processing, which was tested by including age^2 (and centered age in years) in the model.
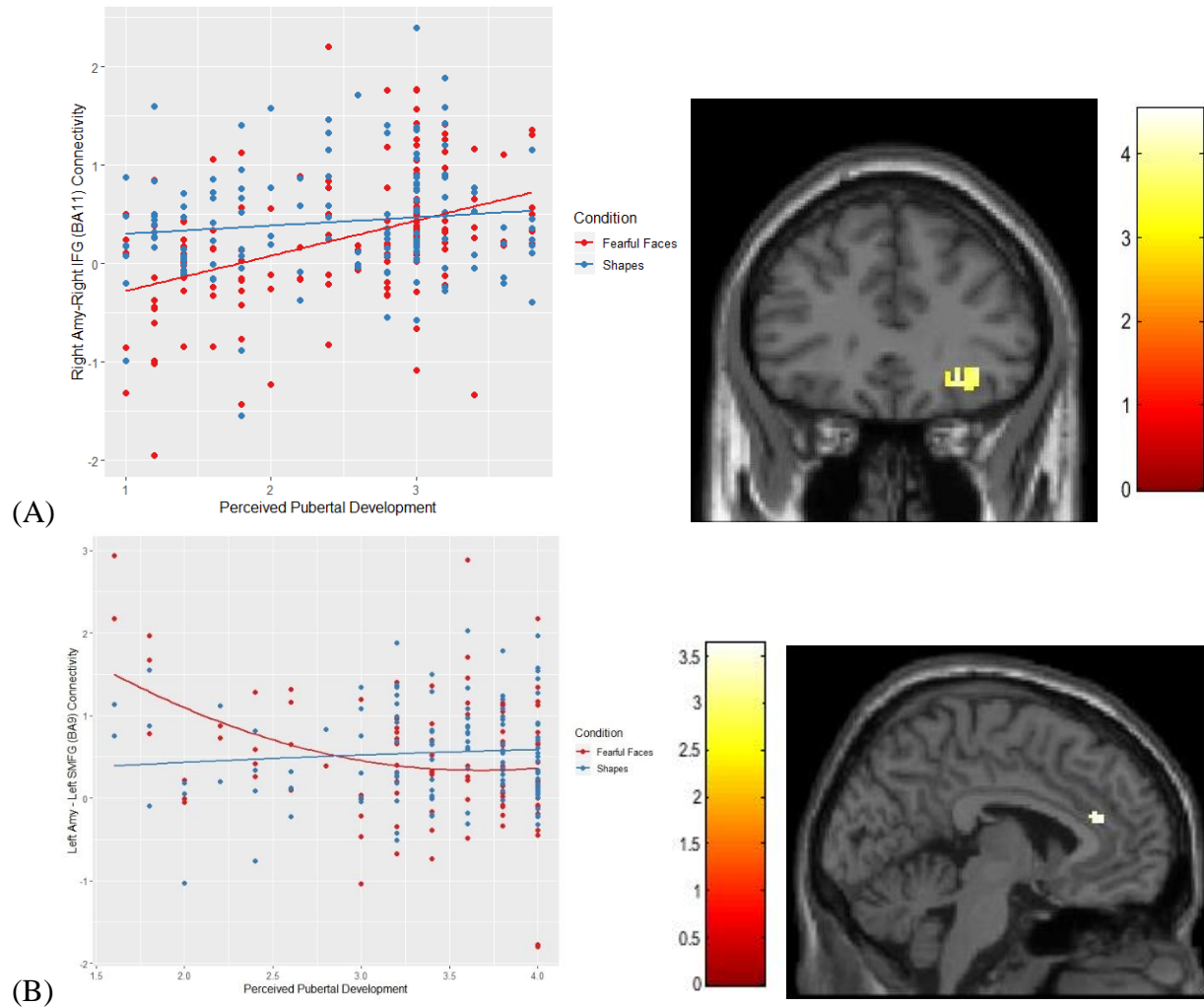
(A)

(B)

Figure 4.5. Pubertal development effects on amygdala-PFC connectivity during fear versus shapes processing, in girls and boys separately

Note. N = 265. (A) Girls: more advanced pubertal developmental was negatively associated with left amygdala – left superior medial frontal gyrus connectivity during fear but not shapes processing. There was a significant quadratic effect of puberty for girls, which was quantified by extracting the mean connectivity estimate (i.e., the betas) of left amygdala – left superior medial frontal gyrus during the fear condition (versus baseline). A multiple linear regression in R (R Core Team, 2018) revealed a significant positive effect of the quadratic term (B[SE] = .35[.14], *p* = .02). Visually, the effect of pubertal development on condition-specific changes in left amygdala-left superior medial frontal gyrus connectivity tapered off for more pubertally-advanced girls (B) Boys: more advanced pubertal developmental was positively associated with right amygdala – right inferior frontal gyrus connectivity during fear but not shapes processing; the data points are extracted mean connectivity estimates during the fear or shapes condition (i.e., from the betas of each individual's psycho-physiological interaction model). For boys, there were no non-linear effects of puberty on right amygdala-right IFG connectivity during fear versus shapes processing, which was tested by including puberty^2 (and the centered pubertal development score) in the model.

CHAPTER V
General Discussion

Socioemotional development in childhood includes the maturation of emotion regulation, impulse control, and interpersonal skills (Cole et al., 2008; Neuhaus & Beauchaine, 2013). Competency in these domains shapes the development of mental health into adulthood, which has implications for educational and economic success, as well as personal wellbeing (Masten & Cicchetti, 2010; Moffitt, 2018; Scott et al., 2016; Steptoe, Deaton, & Stone, 2015). Several features of the social context interact with maturational processes (e.g., age, pubertal development) to challenge adaptive socioemotional development (Masten & Cicchetti, 2010; Sroufe & Rutter, 1984), and one plausible mechanism of these associations is via functional alterations in emotion-related brain circuitry. The goal of this dissertation was to evaluate contextual effects, including parenting behaviors and individual-level markers of maturation, on youth socioemotional and corticolimbic development.

Summary

Study 1. In an extension of the Family Stress Model (FSM; Conger, Conger, & Martin, 2010), Chapter 2 evaluated whether maternal harshness and warmth mediated the effects of socioeconomic disadvantage on youth internalizing and externalizing behaviors. Using a population-based nationwide study of children and their families followed since birth, the effects of socioeconomic disadvantage at birth on youth externalizing behaviors at age 9 were mediated by both economic pressure at age 1 and low maternal warmth at age 5. Consistent with previous research (Kim-Cohen et al., 2004; Rutter, 2013), these results provide an opportunity to promote resilience in disadvantaged families; high maternal warmth can buffer children from the

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psychological effects of socioeconomic disadvantage. Second, the presence of indirect effects via economic pressure reiterates the potent effects of material hardship and food insecurity on youth socioemotional development. Thus, although the Family Stress Model focuses on psychosocial aspects of the child's microsystem (Bronfenbrenner & Morris, 2007), Study 1 suggests that future research should integrate aspects of the built environment (e.g., pollutants, residential crowding, food availability) into models of developmental psychopathology (Evans, 2004; Evans, Wells, & Moch, 2003).

  Study 2. Using a subsample of the Fragile Families and Child Wellbeing Study (i.e., the same sample used in Study 1), Study 2 examined the mechanisms and timing of harsh parenting effects on corticolimbic function. Whereas high initial levels of harsh parenting in early childhood were associated with amygdala function in adolescence, increases in harsh parenting from ages 3 to 9 were associated with prefrontal function during threat processing. Moreover, both high harsh parenting in early childhood and increases in harsh parenting across childhood were associated with weaker amygdala-prefrontal connectivity, a neural marker previously linked to multiple forms of psychopathology (L. W. Hyde et al., 2013; M. J. Kim et al., 2011). These results largely parallel animal models and some human studies indicating heightened sensitivity of subcortical brain regions in the first few postnatal years, and a second window of vulnerability for prefrontal regions during adolescence (Casey, Getz, & Galvan, 2008; Giedd et al., 1999; Lenroot & Giedd, 2006; Payne et al., 2010; Sabatini et al., 2007; N. Tottenham et al., 2011). Moreover, Study 2 showed that the effects of harsh parenting on youth brain development were durable and specific; the associations remained even when accounting for concurrent harsh parenting.

Study 3. One aspect of understanding how harsh environmental contexts impact brain function is clarifying how the corticolimbic system develops across childhood. Study 3 examined the effects of two markers of maturation – chronological age and pubertal development – on amygdala-prefrontal connectivity during face processing. Using a large cross-sectional sample of twins aged 7 to 18 years, Study 3 revealed gender-specific associations: pubertally-advanced girls showed weaker amygdala-prefrontal connectivity during fear versus shapes processing, while pubertally-advanced boys evinced stronger amygdala-prefrontal connectivity during fear versus shapes processing. Study 3 also documented that maturation effects on amygdala-prefrontal connectivity during face processing differed by the amygdala subregion examined, with greater convergence in prefrontal connectivity with centromedial and superficial than basolateral subregions. Overall, the results of Study 3 parallel some previous studies using resting-state fMRI (Alarcón et al., 2015a; Gabard-Durnam et al., 2014; Roy et al., 2009), while also highlighting the importance of attending to gender and puberty in investigations of corticolimbic development.

Considerations for Studies of Parenting Effects on Youth Development

Studies 1 and 2 highlighted the central role of parents in the development of youth socioemotional outcomes and corticolimbic function, while also identifying several considerations for future research.

Timescale. Studies 1 and 2 utilized secondary data from a nationwide study of families followed from birth through adolescence. Although generalizability was a strength of both studies, the use of a large population-based sample resulted in several data limitations. One of the resounding conclusions from Study 1 was that more research is needed to determine the timescale over which family processes operate. The effect sizes of the indirect pathways in Study

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1 were smaller than previous longitudinal studies that measured constructs within shorter

developmental windows (e.g., Landers-Potts et al., 2015), suggesting that the Family Stress

Model may occur on the order of months, rather than years (Hollenstein, Lichtwarck-Aschoff, &

Potworowski, 2013). With constructs measured every two years or so in the Fragile Families and

Child Wellbeing Study, this analysis is not possible using this dataset. Similarly, although Study

2 examined the effects of changes in harsh parenting from ages 3 to 9 on corticolimbic function,

changes in harsh parenting during toddlerhood (i.e., 2 to 4 years) may be more important for the

development of corticolimbic function associated with some forms of psychopathology (e.g.,

antisocial behaviors); observational data of harsh parenting were not available in the study.

Coercion theory posits that reciprocal parent-child interactions during the toddler years, as

children become increasingly autonomous and mobile, sets the stage for the development of

aggression and rule-breaking across childhood (Patterson, 1982; Patterson, DeBaryshe, &

Ramsey, 1990; Shaw & Bell, 1993). Thus, although population-based surveys, such as the

Fragile Families and Child Wellbeing Study, are important data resources for social scientists

studying macrolevel processes, these studies may not be well-suited to investigations of

microlevel processes (Jeanne Brooks-Gunn, Phelps, & Elder, 1991). Relatively short-term

repeated-measures studies of family processes should be implemented during developmental

periods of plasticity (Baltes & Smith, 2004), when both the social context and biological

development are rapidly changing (e.g., toddlerhood). Advances in longitudinal data analysis

support repeated-measures study designs. For example, daily diary data has been used to study

daily fluctuations in harsh parenting (Peterson, Tremblay, Ewigman, & Popkey, 2002), work-to-

family conflict (Butler, Grzywacz, Bass, & Linney, 2005), and psychopathology (Wright, Beltz,

Gates, Molenaar, & Simms, 2015). Repeated measures analyses would also enable researchers to

estimate between- and within-person effects (Cranford et al., 2006; Ellen L. Hamaker, Kuiper, & Grasman, 2015; Voelkle, Brose, Schmiedek, & Lindenberger, 2014), which was also a limitation of Study 1. Thus, incorporating studies of microlevel processes is an important avenue for future investigations of the effects of parenting and family processes on youth outcomes.

Genetic confounding. Neither Study 1 nor Study 2 accounted for genetic confounding. As parents and children are genetically related, several forms of gene-environment correlation (rGE), particularly evocative and passive rGE, might explain the associations between parenting behaviors and youth socioemotional outcomes and corticolimbic function (Manuck & McCaffery, 2014). Evocative rGE occurs when individuals, by nature of their heritable dispositions, elicit behaviors in others (e.g., children with aggression evoke harsh parenting behaviors). Passive rGE occurs when environments (e.g., harsh parenting) and phenotypes (e.g., child aggression) are correlated due to shared genetic liability among related individuals (i.e., parents transmit genetic risk for aggression). Multivariate genetic analyses suggest that although passive and evocative rGE are implicated in harsh parenting effects on youth externalizing behaviors, a true environmental effect remains (Burt, McGue, Iacono, & Krueger, 2006; Deater-Deckard, 2000; Hyde et al., 2016; Lynch et al., 2006; Waller, Hyde, Klump, & Burt, 2018). Further, some studies have shown that harsh parenting may exacerbate genetic effects on youth psychopathology (Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007; Li, Chen, Li, & Deater-Deckard, 2015), suggesting that parenting effects on youth outcomes operate along multiple pathways. Although no studies to date have leveraged behavioral genetic designs to examine the extent to which parenting effects on brain development are genetic or environmental in origin, several recent studies using resting-state fMRI indicate that some neural networks are largely explained by environmental influences (Achterberg et al., 2018; Yang et al., 2016). Thus,

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an important avenue for future research, which will be possible using the Michigan Twins Neurogenetics Study (PIs: Hyde, Burt), is to leverage genetically-informed designs to study environmental effects on brain development (Iacono et al., 2018).

In addition to genetically-informed designs, interventions and quasi-experiment designs can further our confidence in causal, environmental effects of parenting behaviors on youth outcomes (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Thapar & Rutter, 2019). The English and Romanian Adoption Study and the Bucharest Early Intervention Project (Sonuga-Barke et al., 2017; Zeanah et al., 2003) radically changed the caregiving environment by moving infants from deprived institutional-rearing environments into relatively advantaged homes. These studies have largely reported persistent effects of high quality foster care on several dimensions of psychopathology (Humphreys et al., 2015; Sonuga-Barke et al., 2017) and some measures of neural function using electroencephalogram methods (Wade, Fox, Zeanah, & Nelson, 2019). However, given that institutionalization is a relatively rare event, it is critical to extend this research to less severe and more common features of the caregiving context (Thapar & Rutter, 2019).

Longitudinal observational data, genetically-informed designs, and causal inference methods should be viewed as complementary methodologies in the service of study parenting effects on youth outcomes; genetically-informed designs reveal the complex interplay between heritable dispositions and environmental effects, causal inference methods can be used to estimate precise effects of the environment of youth outcomes, and longitudinal data often uncover the mechanisms by which environmental variation leads to behavioral change.

Integrative Themes

The three studies that form this dissertation evaluated contextual effects on youth socioemotional and corticolimbic development. From the examination of contexts within the microsystem (i.e., parent-child relations) to person characteristics that shape developmental processes (i.e., age), this dissertation was heavily influenced by Bronfenbrenner's Bioecological Model of Human Development (Bronfenbrenner & Morris, 2007). This dissertation took a broad view of "contextual effects" to include individual-level components of maturation (i.e., age, pubertal development) in addition to parenting behaviors. Study 3 revealed that the effects of puberty on corticolimbic connectivity varied by gender, where more advanced girls showed weaker (not stronger, as in boys) amygdala-prefrontal connectivity. In addition to biological differences in pubertal maturation by gender (Berenbaum & Beltz, 2011b), one might consider the possibility that pubertal development also reflects a contextual effect. One of the mechanisms by which pubertal timing is thought to increase risk for psychopathology is through evocative effects: girls who mature early and boys who mature late show observable signs of maturation (e.g., breast development in girls, facial hair in boys) that result in differential treatment by peers, teachers, and parents (Mendle & Ferrero, 2012; Negriff & Susman, 2011). In their model, Bronfenbrenner and Morris (2006) also view person characteristics as demand characteristics, where such qualities invite or discourage reactions from the social environment. Thus, this dissertation highlights an important future avenue of research to clarify how pubertal development is nestled within the Bioecological Model of Human Development.

All three studies in this dissertation were also informed by a developmental psychopathology approach. Arising from the notion that adult psychopathology traces its roots to childhood disorders, developmental psychopathology as a discipline seeks to elucidate the biological, psychosocial, and contextual contributions to normal and abnormal development

(Cicchetti & Toth, 2009). Multiple levels of analysis were integrated into this dissertation, including behavioral (Study 1) and neural (Study 2). Longitudinal and multi-method measures of parenting were featured in the first two studies. Study 3 charted maturational patterns of corticolimbic connectivity in a cross-sectional sample of healthy youth, an important prerequisite for future research linking childhood disorders to corticolimbic function. Despite these strengths, Study 2 and Study 3 drew literature from psychiatric neuroimaging to interpret results yet neither study determined whether the observed patterns of corticolimbic function were associated with psychopathology or, conversely, positive socioemotional functioning. A defining feature of the developing brain is its plasticity (Huttenlocher, 2009; Nelson, 1999). Although most of our knowledge of neural reorganization comes from studies of sensory deprivation (e.g., congenital blindness), there is increasing evidence that neurobiological systems underlying emotion processing also adapt to chronic stress (Feder, Nestler, & Charney, 2009; Herman, 2013; McEwen, 2013; McEwen, 2010). Thus, the patterns of corticolimbic function we observed in Study 2 and Study 3 could reflect neurobiological adaption to environmental stress (i.e., harsh parenting, pubertal development). Future studies examining contextual effects on corticolimbic function should incorporate measures of psychopathology and positive indicators of socioemotional development, to determine the phenotypic impact of corticolimbic adaptation to stress.

A final feature of all three studies in this dissertation is the reliance on population-based community samples, thereby increasing the generalizability of the results. Study 1 and Study 2 utilized data from the Fragile Families and Child Wellbeing Study (FFCWS), a population-based study of children recruited at birth from large U.S. urban cities between 1998 and 2000 (Reichman et al., 2001). Data in Study 3 came from the Michigan Twin Neurogenetics Study, a

population-based sample of twins recruited from Southeast Michigan (Burt & Klump, 2013). A

major limitation of existing research in the fields of developmental psychology and neuroscience

concerns the representativeness of study populations (Davis-Kean & Jager, 2017; Falk et al.,

2013). Convenience samples are typically skewed towards White middle-to-upper class families

or undergraduate students who do not represent the broader U.S. population (Henrich et al.,

2010). Population-based studies, by contrast, enhance generalizability due to systemic sampling

(e.g., defining the target population and tracking non-participation patterns) and data collection

methods (e.g., vary the number and timing of contacts and lessen interviewer workload) (Groves

et al., 2009).

Conclusions and Future Directions

Results from this dissertation reiterate the importance of the social context for youth

socioemotional and corticolimbic development, while also highlighting several future directions

to advance this research. Although examinations of the associations between context and

behavior and context and brain function are important first steps in identifying mechanistic

pathways underlying youth psychopathology, more research is needed to integrate data across

multiple levels of analysis (i.e., contextual, genetic, neural, and behavioral). Models of 'Imaging

Gene-x-Environment Interactions' (IGxE; Bogdan, Hyde, & Hariri, 2013; Hyde, 2015; Hyde,

Bogdan, & Hariri, 2011) can guide this research. IGxE models posit that brain function and

structure mediate the paths from gene-by-environment interactions to psychopathology. Phrased

differently, the social context is thought to increase risk for psychopathology via alterations in

brain structure or function, but only for some individuals (i.e., moderation by genetic variation).

Several examples of such models exist in the literature in the prediction of antisocial behavior

(Gard et al., 2017b; N. Holz et al., 2016), substance use (Carey et al., 2015), and depression

(Little et al., 2015). Relatively more research has examined the pathways linking genetic variation and psychopathology via brain structure or function (i.e., 'Imaging Genetics' models), without consideration of the environment (for a recent review, see Bogdan et al., 2017). As large sample sizes are needed to evaluate moderated mediation IGxE models (L. W. Hyde et al., 2011), recent multi-site collaborations such as the Adolescent Cognitive Brain Development study (Volkow et al., 2017) and the Philadelphia Neurodevelopmental Cohort (Satterthwaite et al., 2016) will undoubtedly foster this type of research.

Beyond identifying future avenues for research, it is imperative that scientists remind each other, policy-makers, and the public why research is important, who it is intended to benefit, and the results of our scientific inquires. This dissertation and my research program are informed by four facts: (1) 18% or 13 *million* children in the U.S. live at or below the federal poverty line which, at $25,750 for a family of four, is below the living wage for most U.S. counties (Glasmeier, 2018; Semega et al., 2017); (2) the lifetime prevalence of having any mental disorder as an adolescent in the U.S. is 49.5% (Merikangas et al., 2010); (3) through several mechanisms and decades of research from multiple disciplines, children who grow up in poverty are at dramatically greater risk for developing psychopathology (Bradley & Corwyn, 2002; Jeanne Brooks-Gunn & Duncan, 1997; McLoyd, 1998; Yoshikawa et al., 2012); and (4) at every stage of development, psychopathology is associated with poorer economic, educational, and health outcomes (Duncan et al., 2010; Miller, Chen, & Cole, 2009; Steptoe et al., 2015). Thus, even though children are not responsible for household incomes or economic recessions, they bear an alarming health burden simply by circumstance. Beyond the ethical obligations of caring for our children, childhood poverty in the U.S. is estimated to cost nearly $500 billion per year, which is roughly 4% of the U.S. gross domestic product (Holzer, Schanzenbach, Duncan,

& Ludwig, 2008). For these reasons, reducing child poverty should be a priority for scientists,

policy-makers, and society-at-large.

APPENDIX

Study 1: Supplemental Methods

Measures

The Fragile Families and Child Wellbeing Study (FFCWS) used abbreviated scales of the Child Behavior Checklist/6-18 (CBCL) (Achenbach & Rescorla, 2001) at the age five and nine assessments. Seventy-two items were administered to parents at age five, and 111 items were administered to parents at age nine. To maintain consistency across assessment periods, we only used the items that were available at both ages. Internalizing behaviors were constructed as mean scores of items from the anxious/depressed (10 available items out the 13 items from the original measure) and withdrawn/depressed (7 available items out of the 8 items from the original measure). Externalizing behaviors were constructed as mean scores of items from the aggressive behavior (all 18 items from the original measure) and rule-breaking (10 available items out of the 17 items from the original measure) subscales.

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