CONTEXTUAL AND NEUROBIOLOGICAL FOUNDATIONS OF EXTERNALIZING BEHAVIOR IN EARLY ADOLESCENCE

by

LANDRY GOODGAME HUFFMAN

(Under the Direction of Assaf Oshri)

ABSTRACT

Early adolescence is characterized by significant neural, cognitive, and socioemotional development, which underlies vulnerability for psychopathology stemming from suboptimal rearing contexts. As a result, many adolescents develop externalizing problems such as aggression, delinquency, and antisocial behaviors, which confer increased risk for continued mental health problems and criminality into adulthood. Extant research informed by the Research Domain Criteria (RDoC) suggests that continual and bidirectional interactions between the individual and their environment contribute to disruptions in self-regulation and consequent externalizing behaviors. However, the specific mechanisms underlying longitudinal associations between the rearing environment, youth neurobiological function, and development of externalizing behaviors in early adolescence largely remain to be seen. Therefore, the aim of this dissertation was to explore interactions between the parenting context and youth neurobiological function across the five RDoC domains—social processes, positive valence, negative valence, arousal, and cognition—as predictors of externalizing psychopathology.

The data used in this dissertation come from two longitudinal studies: a diverse sample of low-income mothers and their children collected from the local community, and the ongoing Adolescent Brain Cognitive Development (ABCD) Study. We utilized observational, survey, physiological, and functional neuroimaging measures to assess parenting behaviors, youth neurobiological processes, and externalizing behavior between the ages of 8 and 12.

In the first and second studies, we investigated interactions between parenting and child neurobiological function as predictors of externalizing and antisocial behaviors across 18 to 24 months of early adolescence. In each study, we found that adverse parenting, characterized by high harshness or low support, exacerbated behavioral effects of neurobiological vulnerabilities for dysregulated behaviors. In the third study, we used a longitudinal, data-driven method to examine development of neural function underlying emotion regulation (ER) and working memory (WM) across 24 months. We found that early adolescents exhibit high between- and within-person variability in neural underpinnings of ER, but not WM. We also found that these patterns of neural function were not significantly associated with parental support or externalizing behaviors. The findings of this dissertation advance knowledge on the interactive contributions of parenting and child neurobiological function to development of externalizing psychopathology in early adolescence.

INDEX WORDS: Adolescence, Parenting, Physiology, Neural function, Neuroimaging, Psychopathology, Self-regulation, Externalizing

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DEDICATION

I'd like to dedicate this dissertation to all the middle schoolers: past, present, and future. Y'all are the best, weirdest, funniest humans out there. Keep up the good work.

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To my parents: Y'all instilled in me a curiosity for the world, a love for people, and a grossly inflated confidence in my own intellect. You made the perfect academic. I love you guys!

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CHAPTER 1

INTRODUCTION

Purpose of the Study

Fyodor Dostoyevsky wrote in *The Brothers Karamazov* that "the devil doesn't exist, but man has created him, he has created him in his own image and likeness" (Dostoyevsky, 1950). Many others have observed, albeit in more straightforward terms, the reality of destructive behaviors such as violence and crime that corrode the wellbeing of individuals, relationships, physical spaces, and institutions. Externalizing behaviors—those characterized by regulatory deficits that negatively impact the physical and social environment (Lake et al., 2017)—are one of the most common forms of maladjustment among young people (Campbell et al., 2000; Kessler et al., 2012; Merikangas et al., 2010). Recent studies estimate between 7 and 22% of American youths ages 13 to 17 exhibit externalizing behaviors such as aggression, delinquency, and antisociality (Kessler et al., 2012; Merikangas et al., 2010). These behaviors, especially when sustained throughout childhood and adolescence, confer risk for continued maladjustment into adulthood, including psychiatric disorders, violent offenses, substance use, and incarceration (Eisner & Malti, 2015; Kjeldsen et al., 2021; Reef et al., 2011). As such, a fundamental developmental question remains pressing as ever: what happens within and around an adolescent to compel them to behave in such a way? And what can be done to address it?

Developmental scientists and clinicians have worked to understand the causes and processes surrounding development of externalizing behavior since the inception of psychiatric and psychopathology research (Cicchetti & Lynch, 1993; Fiese et al., 2000; Wright et al., 1999). For many years, these lines of research were limited by medical model-based approaches that confined externalizing behavior into diagnostic categories, yielding limited and unsatisfactory insights into the etiology of externalizing behaviors and potential mechanisms for intervention (Insel et al., 2010). Moreover, mental health research was often compartmentalized within distinct and seemingly opposing traditions of

behaviorism and neurobiology; these disciplines and their relevant findings rarely intersected (Kozak & Cuthbert, 2016). In response to these methodological and theoretical limitations, mental health researchers created the Research Domain Criteria (RDoC) to "identify integrative models of neural circuitry and behavior rather than models that focus exclusively on one or the other" (Cuthbert & Insel, 2013a, 2013b). From the efforts of RDoC-informed research, a growing body of evidence confirms that youth externalizing behaviors are multi-faceted and multi-dimensional, and are themselves a product of complex interactions between the social environment, individual neurobiological contexts, and developmental timing (Beauchaine & McNulty, 2013; Hastings et al., 2011; Patrick et al., 2010; Turpyn et al., 2021). Despite the efforts of the RDoC initiative, much research into the etiology of youth externalizing problems and the early environment remains constrained by a focus on specific externalizing nosologies, inconsistent nomenclatures (e.g., *externalizing problems* versus *conduct problems* versus *problem behavior*), and a division between behavioral and neurobiological approaches that has stubbornly remained (Cuthbert, 2014). As such, the purpose of this dissertation is to examine, through the lens of the RDoC framework, the development of low self-regulation and consequent externalizing behaviors as a product of interactions between the five RDoC domains—*social processes, arousal, cognition, negative valence,* and *positive valence*—and two developmental contexts, the parent-child relationship and youth neurobiological function. As depicted in Figure 1.1, these domains may be considered as interlocking "gears" that rest on contextual "cogs," all of which crank together to produce self-regulation and externalizing psychopathology. I will explore these interactions using multiple levels of measurement, which correspond to several key RDoC domains and constructs (see Figure 1.2). Throughout, I will employ and discuss rigorous and novel quantitative methods, with which I hope to shed light on the complexity of externalizing symptom development without engaging in deterministic or reductionist views that have been so common in the past.

In the following introduction, I will lay the foundation for these aims by discussing: (1) the role of RDoC in research on externalizing behaviors, (2) parenting as a determinant of self-regulation and externalizing within the *social processes* domain, and (3) neurobiological processes underlying self-

regulation within the domains of *arousal, social processes, cognition, negative valence, and positive valence* that contribute to externalizing behavior.

Figure 1.1. Conceptual model of the present dissertation. Gears resemble interlocking RDoC domains (bold text), inside which are domain-specific measures included in the following studies. Grey concentric circles (cogs) represent major contexts of development that will also be discussed.

Externalizing Behaviors and Development

Externalizing behaviors impact between 7 and 22% of children and adolescents in the U.S. When present in early life, externalizing behaviors are linked to a wide range of suboptimal outcomes, including increased risk behavior (Reef et al., 2011), low psychosocial wellbeing (Kjeldsen et al., 2016; Korhonen et al., 2018), decreased social connectedness (Bongers et al., 2008; Loukas et al., 2016), internalizing problems (Nivard et al., 2017; Reef et al., 2011), violent crime (Beelmann & Raabe, 2009; Yu et al.,

2012), and substance use (Colder et al., 2018). Given the risk conferred by externalizing problems, as well as the present rise in mental health problems nationwide (*Youth Risk Behavior Survey Data Summary & Trends Report: 2009-2019*, n.d.), understanding their development and prevention is an issue central to public health. However, identifying the causes of externalizing behaviors is not easy, as their development is a result of complex person-environment interactions across time. Both individual factors, such as temperament and neurobiological functioning (Beauchaine, 2012; Bos et al., 2018; Leve et al., 2005; Shi et al., 2020), and contextual factors such as child maltreatment (Narayan et al., 2015; Oshri et al., 2011, 2013; Price et al., 2013), parenting (Galambos et al., 2003; Leve et al., 2005; Li et al., 2017; Rothenberg et al., 2020; Shi et al., 2020; Van Heel et al., 2019), neighborhood quality (Li et al., 2017), and socioeconomic status (McNeilly et al., 2021; Shi et al., 2020) are strongly linked to risk for externalizing problems. Therefore, an approach that considers the multi-level interplay of developmental contexts is paramount in order to better understand causes and treatment of externalizing behaviors.

RDoC and Research on Externalizing Behaviors

By the early 2000s, mental health research was having a crisis of confidence (Earp & Trafimow, 2015). Among other things, this crisis was caused by a persistent focus on medical-based psychiatric nosology—identifying and categorizing symptoms based on the Diagnostic & Statistical Manual (DSM), determining when these symptoms appeared or resolved, and defining their levels of chronicity (Clark et al., 1995; Van Praag, 2000). This system of diagnostic classification was limited by issues of within-disorder heterogeneity and between-disorder comorbidity (Clarkin & Huprich, 2011; Friedman, 2012; Hyman, 2010; Skodol, 2012; Wing et al., 2011). Moreover, the focus on nosology (e.g., establishing diagnoses based on symptom count) rather than etiology hindered the translational significance of psychopathology research and its ability to answer key questions about development timing, environmental contexts, and individual differences (S. E. Morris & Cuthbert, 2022).

In early 2009, researchers within the National Institutes of Mental Health who were dissatisfied with the DSM-based methodology met to establish novel and improved ways of studying psychopathologies. As a result of these efforts and others, many mental health researchers shifted their

focus from categorical diagnoses to underlying mechanisms of psychopathology. Founders of the developmental psychopathology theoretical framework such as Michael Rutter and Dante Cicchetti had already presented compelling arguments and early research identifying developmental mechanisms by which risk for psychopathology increased (Cicchetti & Lynch, 1993; Rutter & Sroufe, 2000). The developmental psychopathology perspective and research leveraged extant developmental concepts and methodologies, such as systems theory (Bertalanffy, 1968; Oyama, 2000), and merged them with new concepts, such as the graduated continuity between typical and atypical development, to elucidate multi-level origins of psychopathology. Central to this framework are the concepts of multifinality and equifinality, which might be best understood by observing the growth of oak trees (Cicchetti & Rogosch, 1996). In the case of multifinality, similar environmental contexts lead to distinct outcomes: two acorns can fall in the same forest, one reaching a height a 100 feet while another fails to reach the canopy. Equifinality, on the other hand, occurs when disparate contexts lead to similar outcomes: acorns can fall all over the east coast in different climates and soils and reach similar dimensions (C. Carvalho, personal communication, 2022). In human development, we may observe two siblings who both experience harsh discipline in childhood, and yet only one develops substance use problems in later life (multifinality). Conversely, a child from a low-income, resource-poor household might develop similar levels of anxiety as a child from a high-income household (equifinality).

These principles of developmental psychopathology became foundational to the RDoC framework. According to the RDoC framework, neural and behavioral vulnerabilities for psychopathology are transdiagnostic, conferring risk for multiple types, subtypes, and levels of adjustment problems (Beauchaine & Tackett, 2020; Cuthbert & Insel, 2013b). In the same vein, observed constructs that underlie psychopathology fall along a spectrum, rather than into predefined categories or diagnoses. Externalizing behavior, then, may be considered an “umbrella” rather than a “bucket,” encompassing a wide range of behavioral, psychosocial, and neurobiological underpinnings, which may manifest in similar or divergent ways depending on external factors (Kozak & Cuthbert, 2016; Patrick et al., 2010).

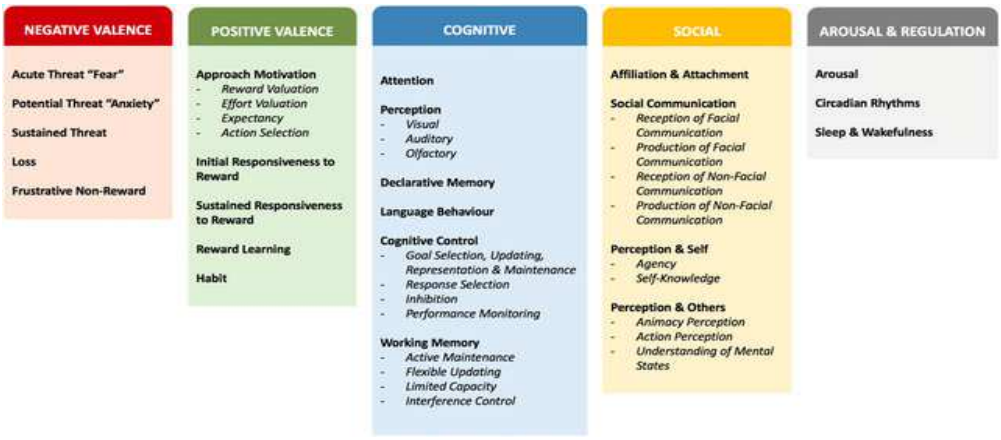

Figure 1.2. RDoC domains (white text) and constructs (black text).

The RDoC framework emphasizes the inextricability of psychopathology from both context and development (Beauchaine & McNulty, 2013; Zisner & Beauchaine, 2016). Adjustment problems progress throughout the lifespan as a dynamic function of both environmental exposures (e.g., family and community environments; Fiese et al., 2000; Gur et al., 2019; Turpyn et al., 2021) and time (e.g., developmental periods; Bongers et al., 2004; Kjeldsen et al., 2021). Although neurobiological risk factors for psychopathology exist, there is rarely, if ever, a 1:1 correspondence between a biological function and behavior, in part because "no man is an island" (Donne, 1624); every person is embedded in a socioemotional contexts that shape their development (Crowell et al., 2017; VanZomeren-Dohm et al., 2016). It is not a question of nature *or* nurture, but rather of how these elements intersect throughout a person's life to shape their behavioral adjustment and mental health (Beauchaine & McNulty, 2013; Hopwood et al., 2022).

Because development is embedded in time, focusing on specific developmental periods is paramount (Zisner & Beauchaine, 2016). The years of early and middle adolescence comprise a period of intense neurobiological, behavioral, and socioemotional development. At the neural level, synaptic growth and pruning and subsequent increases in white matter underlie changes in capacity for socioemotional and cognitive function (Fuhrmann et al., 2015; Shaw et al., 2008). According to the dual-systems and maturational imbalance hypotheses (Casey et al., 2008; Steinberg, 2017), early maturation of motivation and reward circuitry (e.g., ventral striatum, medial frontal and orbitofrontal cortices) paired

with more protracted maturation of cognitive control systems (e.g., lateral prefrontal, parietal, and anterior cingulate cortices) underlie increased propensity for emotional and behavioral dysregulation (although limitations of these theories are discussed in Pfeifer & Allen, 2012; Shulman et al., 2016). Moreover, changes in the social environment, such as increased autonomy and peer influence, interact with and, at times, exacerbate these neurobiological vulnerabilities (Guyer, 2020). As a result, adjustment problems often emerge during adolescence (Paus et al., 2008). Many scientists have thus identified adolescence as a sensitive period defined by increased vulnerability to environmental inputs and emergence of psychopathology (Fuhrmann et al., 2015).

RDoC provides an integrative theoretical and methodological framework from which to study this dynamic period and the patterns of behavioral adjustment that stem from it. A central aspect of this framework is its characterization of five major neurobehavioral domains (Figure 1.2) that underlie both normative and atypical variations in human functioning. In the following section, I identify and discuss how these domains (*social processes, arousal, cognition, negative valence,* and *positive valence*) and specific constructs within them (affiliation/attachment, physiological function, emotion processing, and working memory) are involved in the interplay between parenting, neurobiological underpinnings of self-regulation, and the consequent development of externalizing behaviors among adolescents.

Parenting as a Contributor to Externalizing Behaviors

The RDoC social processes domain encompasses all interpersonal social exchanges and their components, including interpretation and perception of others, as well as the socioemotional and behavioral sequelae of those exchanges (Clarkson et al., 2020; King et al., 2021). Within this domain are the constructs of affiliation, or positive social interaction with others, and attachment, or the socioemotional bond that occurs between two people as a result of affiliation (National Institute of Mental Health, 2022). Parenting is among the most proximal of early life developmental contexts, conferring arguably the greatest impact on child self-regulation and consequent socioemotional and behavioral adjustment (Bornstein, 2013; Fay-Stammbach et al., 2014; A. S. Morris et al., 2017). Extensive evidence suggests that both positive and negative inputs underlying affiliation and attachment from parents to their

children are strong predictors of child self-regulation and related externalizing behavior (Calkins et al., 2013; de Vries et al., 2016; Eisenberg et al., 2005; Guttmann-Steinmetz & Crowell, 2006; Pinquart, 2017; Rothenberg et al., 2020). Especially during the transition from childhood to adolescence, parenting behaviors that shape the parent-child affiliative process are crucial predictors of child externalizing behaviors (Donovan & Brassard, 2011; McCoby, 1983; Pinquart, 2017; Trentacosta & Shaw, 2008). In the context of accepting, nurturing, sensitive, and responsive parenting behaviors (hereby called *supportive parenting* or *parental support*), children are less likely to develop externalizing and antisocial behaviors and more likely to evince prosocial behaviors (Eisenberg et al., 2005; Rothenberg et al., 2020). As summarized by a meta-analysis of over 1 million children, greater parental support is consistently and longitudinally linked to less externalizing behaviors among adolescents (Pinquart, 2017). These findings are supported cross-culturally and at multiple developmental periods throughout childhood and adolescence (Lansford et al., 2018; Rothenberg et al., 2020). Conversely, when the parent-child emotional climate is characterized by harsh, rejecting, unresponsive, or aggressive parenting behaviors (hereby called *harsh parenting* or *parental harshness*), children are more prone to maladjustment, including externalizing behaviors (Burnette et al., 2012; Erath et al., 2011; D. S. Shaw et al., 2003; Wiggins et al., 2015). Several meta-analyses of children and adolescents confirm these associations and indicate that greater parental harshness predicts child externalizing behaviors both cross-sectionally and longitudinally throughout adolescence (Khaleque, 2017; Pinquart, 2017).

As made clear by this large body of research, there are critical processes of child socioemotional and cognitive development that are bolstered by parental support and undermined by parental harshness (Calkins et al., 2013; McCoby, 1983; Pinquart, 2017). Several prominent theories expound upon the mechanisms by which parental support and harshness lead to child externalizing behaviors (or lack thereof). According to attachment theory, insensitive and unresponsive parenting behaviors undermine affiliation between the parent and child and lead to insecure emotional bonds (Bowlby, 1979). As a result, children may learn to view others outside of the family as similarly unreliable and hostile, and treat them accordingly (Baer & Martinez, 2006; Contreras et al., 2000; Michiels et al., 2008; Oshri et al., 2015;

Pickreign Stronach et al., 2011). Kochanska (2001) builds upon this framework by arguing that children with healthy attachment to their parents experience less anxiety and negative emotion and thus have more "psychological resources" for development of higher-order cognitive processes such as self-regulation and empathy, which are pivotal to inhibiting and preventing externalizing behaviors (Laible & Thompson, 2002). Similarly, Cicchetti and Sroufe argue via the organizational perspective that children who evince insecure attachment with their caregiver (as a result of maltreatment) are less able to reorganize their behavior to match changing demands of each developmental period. These unresolved developmental milestones undermine social and emotional competence and precipitate psychopathology (Cicchetti & Beeghly, 1987; Cicchetti & Sroufe, 1978).

Social learning theory (Bandura, 1977) and the social-interactional perspective (Forehand & Scarboro, 1975; Patterson et al., 1989) also speak to parents' roles in child externalizing behaviors. According to these frameworks, parents model aggressive behavior when punishing their children or otherwise treating them in a harsh or coercive way (Snyder, 2016). Parental harshness often elicits similar negativity in children (C. Blair, 2002; Smith et al., 2015). When these feedback loops of negativity occur in the rearing context, children learn and internalize expressions of negative emotionality rather than self-regulatory behaviors, perpetuating existing behavioral problems (C. Blair, 2002; Patterson, 1986; Patterson et al., 1989). Conversely, parents can model for their children constructive ways of managing emotion, especially in stressful contexts (Power, 2004; Wiggins et al., 2015), thus facilitating their children's learning of self-regulation strategies.

Eisenberg et al. (2005) and Belsky et al. (2007) argue that self-regulation itself is the underlying mechanism linking parenting and externalizing behaviors. Lack of parental support and presence of harsh behaviors undermines development of youths' self-regulation, leading to increased behaviors characterized by low behavioral and emotional control (Belsky et al., 2007; Eisenberg et al., 2005). More specifically, parents' negative or hostile behaviors increase arousal in children, undermining children's learning and self-regulation efforts. In an over-aroused state, children are unlikely to shift or focus

attention as necessary, compromising their behavioral learning and subsequent self-regulation (Berger et al., 2007; Sarıtaş et al., 2013).

Parenting, Antisocial Behaviors, and Callous-Unemotional Traits

Callous-unemotional traits such as lack of guilt, shallow affect, and low empathy characterize a subgroup of youths at risk for chronic and severe antisocial behaviors in adulthood; see Frick & Viding, 2009; Frick & White, 2008). The parent-child emotional climate is equally formative in the development of callous-unemotional traits and antisocial behavior (e.g., conduct disorder and oppositional defiance disorder) as they are for other externalizing behaviors (Burnette et al., 2012; Trentacosta & Shaw, 2008; Waller et al., 2013). Parents may mitigate risk for antisocial behavior via positive and supportive interactions with their children, increasing their children's receptivity to socialization techniques and fostering greater prosociality (Kochanska, 2001; Waller et al., 2013). Although some studies suggest that youths exhibiting CU traits are less susceptible to parental socialization (Oxford et al., 2003), several others indicate that both parental harshness and warmth contribute to CU trait development as they do to antisocial behavior (Loney et al., 2003; Pardini et al., 2007; Waller et al., 2013). In line with social learning theory, children exposed to harsh rearing practices, including physical and emotional aggression, may learn to incorporate aggressive and callous behaviors in their social interactions (Bandura, 1973; Gershoff et al., 2018). Extant research supports this theory: increased CU traits such as low empathy, guilt, and concern for others' feelings have been documented among youths exposed to harsh parenting behaviors (Kochanska, 2001; Loney et al., 2003; Oshri et al., 2020; Trentacosta & Shaw, 2008).

Neurobiological Underpinnings of Self-regulation & Externalizing Behaviors

Development of self-regulation is key to successful navigation of life's day-to-day challenges. Self-regulatory capacity frequently predicts child and adolescent behavioral adjustment, especially in response to stressful or adverse experiences (C. Blair et al., 2016; Perry et al., 2018). Given that externalizing problems stem from regulatory deficits, a study of externalizing etiology necessitates (and is arguably synonymous with) a study of self-regulation (Eisenberg et al., 2017). Disruptions in self-regulation underlying externalizing behaviors stems not only from socioemotional inputs such as the

rearing context, but also from individual differences in neurobiological function (Castellanos-Ryan et al., 2014; Patrick & Bernat, 2009). Three measures of neurobiological function will be discussed here, due to their relevance both within the RDoC framework and as proxies of self-regulation and externalizing behavior risk: (i) physiological function, which belongs to the *arousal and regulation* domain; (ii) neural processing of emotional faces, which belongs to the *social processes* domain; (iii) and neural processing of working memory, which belongs to the *cognitive* domain (National Institute of Mental Health, 2022).

Physiological Function

According to the neurovisceral integration model, individual differences in autonomic nervous system (ANS) function interact with the central nervous system to precipitate behavioral self-regulation (Thayer & Lane, 2000). As such, autonomic nervous system function is a central neurobiological factor in self-regulatory capacity and subsequent risk for externalizing behaviors (Beauchaine, 2012). The ANS plays a crucial role in the body's response to environmental inputs, interacting bidirectionally with the CNS to coordinate physical and behavioral responses (Beauchaine, 2001; Thayer & Lane, 2000). It comprises two branches: the parasympathetic and sympathetic nervous systems (PNS and SNS, respectively). The PNS is involved in coordinating "resting and digesting" functions, which allow the body to gather and conserve energy resources. Although there is no one objective index of PNS activity, it is often indirectly indexed by assessing vagal regulation of cardiac rate and resulting variations in high-frequency inter-beat intervals known as resting sinus arrhythmia (RSA; Dollar et al., 2020). As Thayer argues in the neurovisceral integration model (Thayer et al., 2009), RSA may proxy prefrontal cortex (PFC) function and related capacity for executive function, as evidenced by (1) inhibitory efferent pathways from the medial PFC to the PNS (Williams, 2010), (2) positive associations between RSA and PFC activity when measured simultaneously (Nguyen et al., 2022), and (3) a growing body of research linking basal RSA and RSA reactivity to self-regulatory ability and psychopathology (Beauchaine & Bell, 2020; Colzato et al., 2018). To the last point, both low basal RSA and high RSA reactivity (vagal withdrawal) to emotionally salient stimuli are linked to an array of externalizing psychopathology,

including conduct problems, aggression, ADHD, and CU traits (Beauchaine et al., 2019; Fortunato et al., 2013; Yan et al., 2021; W. Zhang & Gao, 2015).

The SNS underlies the body's response to threatening environmental demands by enabling the body to engage in fight, flight, or freeze behaviors. This sympathetic response is facilitated by increases in blood flow to the heart and consequent increases in respiration and sweat production. SNS influence over the heart can be measured indirectly by pre-ejection period (PEP), or the timing between the depolarization of the left ventricle and release of blood from the aorta (Newlin & Levenson, 1979). Shortened PEP, both at rest and in response to acute stress, is indicative of greater cardiac output and sympathetic influence on the heart. Lengthened PEP suggests sympathetic underarousal and attenuated sympathetic influence on the heart. Research on the associations between SNS function and psychopathology has produced mixed results. On the one hand, less SNS arousal may indicate lack of behavioral inhibition, which underlies fearlessness and sensation-seeking behaviors (Raine, 2002; Beauchaine et al., 2013). Conversely, excessive SNS arousal is associated with decreases in self-regulation, increased sensitivity to negative environments, and subsequent externalizing behaviors among youths (Hinnant et al., 2016; Tonacci et al., 2019).

Because self-regulation often influences individual responses to external pressures, it necessitates study wherein both self-regulatory capacities *and* external inputs are measured (C. Blair, 2010; Bush & Boyce, 2014). The biological sensitivity to context (BSC) theory (Ellis & Boyce, 2008) provides a helpful framework for studying externalizing behaviors as a product of interactions between environmental inputs and the physiological underpinnings of self-regulation. BSC theory posits that (1) physiological function is a biological proxy for individual sensitivity to environmental inputs, such as the family context or parenting behaviors, and (2) individuals with heightened biological sensitivity will evince developmental outcomes in closer conjunction with the quality of environmental inputs. As such, a child with a biologically sensitive profile may develop more optimally in the context of positive rearing contexts (e.g., supportive parenting) *and* more maladaptively in the context of negative contexts (e.g., harsh parenting). This theory has been bolstered by a growing body of research that links SNS and PNS activity (as

measured by PEP and RSA, respectively) to individual sensitivity to environmental inputs and risk for externalizing behaviors (Bubier et al., 2009; Doom & Gunnar, 2013; Duprey et al., 2021; Erath et al., 2011; Hinnant et al., 2016; Muhtadie et al., 2015; Obradović et al., 2011; Obradović, 2012; Oshri et al., 2020; Pickreign Stronach et al., 2011). Among youth reporting adverse parenting behaviors such as those characterized by harshness (Bubier et al., 2009), permissiveness (Hinnant et al., 2016), and conflict (Obradović et al., 2011), those exhibiting reactive or under-regulated physiological profiles, defined by shortened PEP, low resting RSA, and excessive vagal withdrawal (e.g., decreases in RSA from baseline) during stress are more at risk for development of externalizing behaviors when compared to those with less reactive physiological profiles.

Although these findings suggest biological sensitivity to parenting behaviors, most have been conducted among young children. Studies among older children and pre-adolescent samples have yielded mixed results; moreover, they are limited by cross-sectional designs and demographically homogenous samples (for review, see Sijtsema et al., 2013). Additionally, studies of BSC theory are often limited to one branch of the autonomic nervous system (parasympathetic versus sympathetic) and/or one type of autonomic function (basal function versus stress reactivity). Finally, extant research on BSC theory and its relevance to externalizing outcomes lacks specificity in the type of outcome (e.g., delinquency versus aggression), which is necessary in order to better understand the nuanced etiologies of problem behaviors.

Emotional Face Processing

Being able to recognize the emotions of others is essential to healthy socioemotional functioning and attendant self-regulatory capacities (Kaltwasser et al., 2017). Neural functions underlying emotion processing reflect a whole-brain convergence of multiple functional domains, including visual and somatosensory processing, executive function, and valuation (Haxby et al., 2000; Kanwisher et al., 1997; Richler & Gauthier, 2014). Among healthy individuals, observed facial emotion typically precipitates increases in activity of visual cortices, including the fusiform gyrus and inferior occipital gyrus (Fusar-Poli et al., 2009; Haxby et al., 2000; Vuilleumier & Pourtois, 2007). Recruitment of several regions, such as the primary and secondary somatosensory cortices, insula, supramarginal gyrus, and basal ganglia,

facilitates internal representation of emotional states (Kropf et al., 2018; Sel et al., 2014). At the same time, several prefrontal, temporal, and parietal regions, including the vmPFC (encompassing the medial orbitofrontal cortex, superior frontal gyrus, and anterior cingulate), posterior cingulate, precuneus, and superior temporal sulcus evaluate emotional stimuli, integrate whole-brain responses, and modulate subcortical structures to produce an adaptive affective and behavioral response (Frank et al., 2014; Ghashghaei et al., 2007; Kohn et al., 2014). Finally, several subcortical structures are involved in emotion processing, particularly the amygdala, hippocampus, and ventral striatum. The amygdala is particularly active during threat detection and fear conditioning (Adolphs, 2008; Fusar-Poli et al., 2009), although it is involved in processing a wide array of emotional salient stimuli, including anger, sadness, and happiness (Lindquist et al., 2012; Sergerie et al., 2008) (Sergerie et al., 2008; Lindquist et al., 2012). The amygdala also exhibits anatomical and functional connections with the vmPFC—a region central to emotional cognition, including emotion regulation, reward processing, valuation, and self-referential thinking (Hiser & Koenigs, 2018). During passive viewing of emotion, regions of the amygdala and vmPFC often exhibit concurrent positive activation (Yang et al., 2020). Conversely, during emotion regulation of negative emotional stimuli, the vmPFC often exhibits increased activation, while the amygdala decreases in activation. These patterns highlight the vmPFC's role as a down-regulator of amygdala activity and subsequent emotional experience in response to negative emotion (Motzkin et al., 2015; Yang et al., 2020). The hippocampus, which exhibits reciprocal connections to the amygdala, is central to memory processes, especially fear conditioning (Chaaya et al., 2018), and as such is most often recruited during processing of negative emotion (Yang et al., 2020). The ventral striatum, on the other hand, is active primarily during processing of positive emotion, given its role in the dopaminergic motivation circuit and reward processing (Frank et al., 2014; Yang et al., 2020).

Extensive evidence suggests that socioemotional processing disruptions are a foremost contributor to low self-regulation and related externalizing behaviors (Eisenberg et al., 2017; Hiser & Koenigs, 2018; Poon et al., 2022). The RDoC domains of *social processes, positive valence*, and *negative valence* correspond to socioemotional constructs of facial emotion processing, social reward, and

potential threat, respectively. These constructs in turn help elucidate unique associations between neural processing of emotion and externalizing behaviors (Hulvershorn et al., 2013; Kret & Ploeger, 2015). Within the *social processes* domain is the construct of perception of facial communication. Perception of facial communication underlies emotion knowledge, or the ability to recognize and understand positive, negative, and ambiguous emotional cues of others (Denham et al., 2003; Bassett 2012). Successful recognition of facial communication and resulting emotion knowledge are key to effective self-regulation (Di Maggio et al., 2016). Accordingly, disruptions in facial emotion processing and emotion knowledge are consistently linked to dysregulated psychopathology (Trentacosta et al., 2010) and externalizing behaviors (Aspan et al., 2013; Acland et al., 2021; Ip et al., 2019).

Processing of positive emotional stimuli such as happiness elicits neural circuitry underlying approach motivation, or the impulse to move toward potentially rewarding stimuli (Lang and Bradley, 2008). Approach motivation processes are often measured and observed via monetary reward tasks; however, processing of positive emotion and monetary reward often elicit similar neural pathways, such that positive emotion is often defined as a subjective state elicited by *social* reward (Rolls, 1999). Positive emotion processing, which falls within the *positive valence* domain, is a salient, albeit less studied, predictor of dysregulated behavior problems in adolescence. For example, Fortunato et al. (2013) found that decreased RSA withdrawal while viewing a happy video was linked to increased externalizing, but not internalizing, behaviors among youth. Similarly, Bunford et al. (2017) found that decreased cortical response to happy faces (measured by event related potential) was associated with increased rule-breaking and social problems among anxious adolescents.

Negative emotional stimuli, such angry or fearful faces, elicit neural circuitry underlying threat processing and is thus categorized within the RDoC domain of negative valence (McCrory, De Brito et al., 2011; Wiessman et al., 2020). Dysregulated neural response to negatively-valenced faces and images is a salient risk factor for development of externalizing behaviors, especially severe forms of antisocial behaviors. In fact, a growing body of research suggests that neural processing of negatively-valenced stimuli may differ based on the presence of callous-unemotional (CU) traits (e.g., lack of empathy and

remorse), which characterize a subgroup of youths with externalizing problems who are particularly at risk for chronic and severe behavior problems in adulthood (Frick et al., 2014). According to the foundational work of Blair (2008; 2014), neural underpinnings of emotion processing among youths with externalizing problems may be characterized by difficulties in two distinct processes within the *negative valence* domain: empathy and threat processing. Youths with CU traits often exhibit *attenuated* neural response to negative emotional stimuli within the amygdala, OFC, vmPFC, and visual cortex (e.g., inferior occipital gyrus, fusiform gyrus, superior temporal sulcus; Herpers et al., 2012; Jones et al., 2009; Rhoads et al., 2020; Viding et al., 2012; White, Brislin, et al., 2013). This pattern of diminished neural response underlies decreased avoidance when perceiving distress, impaired emotional learning, and consequent lack of empathy (R. J. R. Blair et al., 2014; Honk & Schutter, 2006; Northam & Dadds, 2020). In the absence of CU traits, however, disrupted neural processing of emotion can take on a different form, which underlies difficulties in threat processing rather than empathy (Blair 2014). Adolescents with externalizing problems but not CU traits often exhibit *elevated* amygdala response and decreased prefrontal response (e.g., dorsal ACC, dmPFC) to threatening or otherwise negative emotional stimuli (Alegria et al., 2016; Dotterer et al., 2020; Sebastian et al., 2014; Viding et al., 2012). Often, these types of externalizing behaviors develop concurrently with other symptoms of emotional and behavioral dysregulation, such as anxiety and impulsivity (Beauchaine et al., 2017; Knappe et al., 2022).

Executive Function, Working Memory, and Emotion Regulation

Emotion processing rarely occurs in isolation, but rather in the context of goals, tasks, and social interaction. Executive function (EF), an essential neurocognitive process underlying successful self-regulation, includes a number of conscious functions that mediate subordinate cognitive processes in order to accomplish goal-directed behaviors (Hum & Lewis, 2013). Across the lifespan, low EF is one of the most consistent risk factors for dysregulated psychopathology, including internalizing, externalizing, and comorbid disorders (Banich, 2009; Levens & Phelps, 2008; Mikels et al., 2008). During adolescence, normative increases in neural reactivity to affective and emotional stimuli, particularly within the striatum, medial PFC, and orbitofrontal cortex, are accompanied by protracted development of

frontoparietal regions underlying EF, such as the lateral PFC, lateral parietal cortices, and anterior cingulate (Luna et al., 2010). As a result, EF capacity during adolescence is increasing but inconsistent, making adolescents vulnerable to maladaptation stemming from low behavioral regulation (Fuhrmann et al., 2015; Luna et al., 2010; Carlson & Zelazo, 2011). This vulnerability to externalizing psychopathology in adolescence (Moffit, 1993; Kjeldsen et al., 2021; Fairchild et al., 2013; Stringaris et al., 2014; Ogilvie 2011; Paus et al., 2008) necessitates research that elucidates with greater specificity links between EF constructs and behavioral dysregulation.

Working memory (WM)—the cognitive process of maintaining and manipulating information for a short period of time in order to guide behavior—is a core component of EF that may be especially formative in the emergence of externalizing behaviors during adolescence. Among both adolescents and adults, WM is critical to an array of higher-order cognitive functions (Baddeley, 1998). In healthy adolescents, WM tasks involve frontoparietal areas such as the middle frontal gyrus, anterior cingulate, precuneus, inferior parietal lobule, superior frontal gyrus, and cerebellum (Andre et al., 2016; Zhang et al., 2021). Deficient WM underlies a number of psychiatric disorders, particularly those characterized by behavioral and emotional dysregulation (Huang-Pollock et al., 2017; Vuontela et al., 2013). Attenuated response of associated brain regions such as the vmPFC and ventral striatum underlies low WM capacity and is linked to decision-making deficits (Fellows & Farah, 2005; Oshri et al., 2019; White, Pope, et al., 2013). These deficits in decision-making can impair social functioning, leading to more impulsive and less adaptive responses in social situations—for example, responding aggressively, rather than prosocially, to a disagreement with a peer or family member (Flouri et al., 2016; McQuade et al., 2013). WM deficits and related decision-making problems are also linked to dysregulated motivational circuitry, which is another frequently observed risk factor for development of externalizing behaviors (Ernst et al., 2003).

Although WM on its own is a strong predictor of maladaptation, it has also long been considered in the context of emotional influence to elucidate emotion regulation, or executive processing that occurs in the context of affective stimuli (Banich, 2009; Mikels & Reuter-Lorenz, 2019). In many circumstances,

affective stimuli can modulate WM capacity and at times "overload" it, leading to decreases in WM capacity (Mikels & Reuter-Lorenz, 2019; Oshri et al., 2019; Schultz et al., 2004; Woltering et al., 2015; Hughes, 2011). The opposite of this "overloading" is emotion regulation (ER), or when WM and other EF processes modulate affect, both by influencing the emotional experience directly (Kellerman, 2012; Van Dillen et al., 2009) and by bolstering other ER strategies such as suppression and reappraisal (Schmeichel and Zell, 2007; Pe et al 2013 a and b). Among those exhibiting externalizing psychopathology, affective information disrupts executive function more frequently than healthy individuals (Oschner & Gross, 2005). These disruptions of cognition by emotion are evident at the neural level. For example, in two early studies, Gray et al. (2002) and Herrington et al. (2005) found that affective stimuli modulated activity in executive function regions; positive emotional face stimuli increased dlPFC activation, whereas negative emotional stimuli decreased dlPFC activation, during a WM task. In a meta-analysis of 33 fMRI studies, Schwiezer et al. (2019) found that vlPFC, amygdala, temporal, and occipital activation increased during a WM task when visual stimuli were emotionally salient, indicating the greater cognitive (and thus metabolic) demand on EF regions in the context of emotion. In day-to-day life, this overload of cognitive processing by emotional information disrupts the ability to maintain and regulate goal-directed behaviors in the context of emotion, leading to low behavioral regulation and an increased risk for externalizing psychopathology (Figueria et al., 2017).

Longitudinal Change in Neural Function

Significant changes in neural function underlying WM and emotion processing occur during adolescence (Simmonds et al., 2017; Geier et al., 2009; Scher et al., 2006; Klingberg et al., 2002; Kwon et al., 2002; Del Piero et al., 2016; Pfiefer et al., 2011; Passarotti et al., 2009; Pitski et al., 2011; Wiggins et al., 2016). According to Simmonds et al. (2017), improvements in WM performance, which persist through the early 20s, stem from linear increases in sensorimotor-related activity within the primary visual and visual association cortices and inferior temporal gyrus and decreases in frontal and subcortical executive areas (e.g., inferior parietal lobule, dorsolateral prefrontal cortex, anterior cingulate, insula, and basal ganglia) observed from late childhood to age 30. Longitudinal changes WM-related brain function

have also been observed in the dorsolateral prefrontal, parietal, and visual cortices, although findings are mixed. Geier (2009) and Scherf (2006) found that adolescents evinced greater activation in these regions during a WM task than children and adults; conversely, Klingberg (2002) and Kwon (2002) found that it was adults who exhibited greater activation when compared to children and adolescents. Findings from emotion processing and ER studies similarly indicate decreased response to emotion in frontal and subcortical areas. In a review of 24 neuroimaging studies by Del Piero et al. (2016), changes in adolescent neural reactivity to emotion were characterized by linear decreases in amygdala and lateral prefrontal response and increases medial prefrontal/anterior cingulate response. Several other studies have also found significant age-related increases in response to emotion within the basal ganglia (Pfiefer et al., 2011; Passarotti et al., 2009) and decreases within the insula (Pitskel et al., 2011) and fusiform gyrus (Passarotti et al., 2009; Wiggins et al., 2016).

There is still much we don't know about the age-related development of neural circuitry underlying emotion and executive function systems, which necessitates additional studies and methodologies that model development of brain-wide function at the individual level (Foulkes & Blakemore, 2018; Telzer et al., 2018). A growing body of evidence suggests high within- and between-person variability of both structural and functional brain development across adolescence. This variability necessitates person-centered over variable-centered analytic approaches. Whereas variable-centered methods focus on mean-level associations between variables, person-centered methods characterize intra- and inter-individaul variability and change by identifying subgroups of people based on their multivariate similarities. Preliminary evidence from person-centered studies using fMRI, EEG, and neurocognitive tasks indicate substantial within- and between-person variability in neural function as well (Kjelkenes et al., 2022; Ordaz et al., 2013; Tang et al., 2018). Ordaz et al. (2017) used mean growth curve modeling to characterize high within-person variability in neural function underlying EF in an accelerated longitudinal sample of 123 participants ages 9 to 26. Using latent class growth analysis within a sample of 43 12 to 16-year-old females, Tang et al. (2017) detected several unique trajectories of frontal alpha symmetry, suggesting both intra- and inter-individual variability of functional neural risk for psychopathology (Coan

& Allen, 2004). Finally, Kjelkenes et al. (2022) used a normative modeling framework to identify inter-individual deviations from the norm in neurocognitive ability among youths ages 12 to 16. However, no studies to date have used person-centered methods to investigate developments of brain-wide neural function underlying EF and emotion processing in adolescence.

The Emotional N-back

The emotional N-back (EN-back) has been widely used to study functional neural underpinnings of working memory in an emotionally salient context (Kirchner 1958; Owen 2005). Built upon the n-back task (Kirchner, 1958), participants view emotionally salient stimuli (typically human faces) interspersed with emotionally neutral stimuli, while reporting whether the current stimulus matches another stimulus "n" trials back. In order to complete the trials accurately, participants must hold information in short-term memory while implicitly processing emotional inputs (Rougier 2005; Diamond, 2015). A meta-analysis of 17 functional imaging EN-back studies among children and adolescents (n = 260) found common frontoparietal activation during the working memory condition, including the left superior frontal gyrus and dorsal cingulate gyrus, right inferior parietal gyrus, bilateral superior parietal lobule, and precentral gyrus, as well as the insula (Yaple & Arsalidou, 2018). When limiting brain activation to emotion-processing conditions (i.e., statistically removing effects of working memory stimuli to isolate effects of emotional stimuli), adolescents typically show brain activation in visual areas (fusiform face area, occipital lobe) and, less consistently, affective- and salience-processing areas such as the amygdala, insula, and anterior cingulate (Ahmed et al., 2015).

Recent cross-sectional studies using the EN-back paradigm suggest links between neural function underlying WM and externalizing problems. Among a large sample (n = 1,129) of adolescents, Shanmugen et al. (2016) found that decreased activation within the frontoparietal cortex, thalamus, and cerebellum during the WM condition was associated with increased externalizing and antisocial behaviors. Similarly, Lees et al. (2021) found among an even larger sample (n = 6,146) of preadolescents that decreased activation during WM in the rostral and caudal anterior cingulate, insula, nucleus accumbens, putamen, and pallidum was linked to increased externalizing behaviors. Notably, the authors

did not find any patterns of neural activity to be uniquely associated with internalizing or thought-based psychopathology.

Limitations of Current Brain-Behavior Research

Although informative, the translational implications of the imaging studies discussed here are constrained by several limitations. First, extant links between neural circuitry of emotion regulation and externalizing psychopathology have been largely established using cross-sectional methodologies. Only recently have studies employed longitudinal methodologies to investigate the implications of neural function for externalizing behavior development across adolescence (Poon et al., 2022; Ramphal et al., 2020; Thijssen et al., 2021). Although a certain pattern of brain function may be *correlated* with a behavior, it does not always follow that pattern of brain function *predicts* behavior longitudinally (e.g., behavioral development; Hyde et al., 2020; McCormick et al., 2021). Additionally, most neuroimaging studies investigating brain-behavior associations, both cross-sectionally and longitudinally, are constrained by small sample sizes ($n < 100$). Recent simulation and replication studies using neuroimaging and behavioral data suggest that replicability of neural activation patterns *increase* and effect sizes between brain-behavior associations *decrease* as sample size increases (Grady et al., 2021; Gratton et al., 2022; Marek et al., 2022). Finally, few longitudinal imaging studies have contextualized these brain-behavior associations within the parent-child emotional climate. Given the salient impact of parenting on youth neurobiological and cognitive-emotional development, this is a pressing limitation (Hyde et al., 2020). A better understanding of (1) the types of neural function that confer greatest risk for development of externalizing psychopathology and (2) how this neural function interacts with parenting behaviors to predict externalizing psychopathology will increase the specificity and efficacy of future interventions among adolescents at risk for externalizing psychopathology.

The Present Dissertation

Following extant findings and gaps in the current literature, this dissertation uses novel methods to explore interactions between parenting and neurobiological processes underlying self-regulation as predictors of externalizing psychopathology across late childhood and adolescence. Using two distinct

samples of youths and caregivers, I aim to answer the following questions: (1) How does parasympathetic and sympathetic ANS function interact with harsh parenting behaviors to predict externalizing behaviors? (2) How does neural processing of emotion interact with parental support to predict antisocial behaviors across high and low levels of CU traits? (3) How does neural function underlying emotion and WM change over time, and how are these changes linked to parental support and externalizing psychopathology?

In Chapter 2, I utilize a low-income sample of preadolescents (Mean age = 10.3) and their mothers to longitudinally examine physiological function as an indicator of biological sensitivity to the parenting context. I examine whether risk for externalizing psychopathology *and* biological sensitivity to harsh parenting is conferred uniquely by SNS and PNS function, both at rest and during acute stress. I also test whether interactions between harsh parenting and ANS function differentially predict aggressive versus delinquent behaviors 18 months later.

In Chapter 3, I continue to examine interactions between parenting and neurobiological function by assessing neural function underlying emotion face processing. Considering that biological risk for AB and CU traits can vary by family context, I test the longitudinal associations between neural response to positive and negative emotion (as elicited by the EN-back paradigm), parental support, and the interaction between the two as predictors of AB among adolescents with both low and high levels of CU traits.

In Chapter 4, I employ a preregistered, longitudinal, data-driven approach to examine neural circuitry underlying the intersection of emotion processing and working memory and its change over time. Using latent transition analysis, I aim to identify latent subgroups of neural function during the EN-back faces vs. places and 0-back versus 2-back conditions and subsequently evaluate continuity and discontinuity of these subgroups across 24 months. Moreover, I test whether parental support and externalizing psychopathology are linked to continuity or discontinuity of these functional subgroups.

CHAPTER 2

AN AUTONOMIC NERVOUS SYSTEM CONTEXT OF HARSH PARENTING AND YOUTH AGGRESSION VERSUS DELINQUENCY[1]

[1]Huffman, L. G., Oshri, A., & Caughy, M. (2020). An autonomic nervous system context of harsh parenting and youth aggression versus delinquency. Biological psychology, 156, 107966. Reprinted here with permission of the publisher.

Abstract

Harsh parenting is a significant predictor of youth aggression and delinquency. However, not every child exposed to adverse parenting develops such problem behaviors. Recent developmental evolutionary models suggest that variability in stress response reactivity to parenting, reflected by autonomic nervous system (ANS) functioning, may affect the impact of adverse parenting on youth behavioral adjustment. The aim of the present study was to investigate whether the parasympathetic and sympathetic branches of the ANS moderate the association between parenting and aggressive and delinquent behaviors. The study sample included low-income, ethnically diverse preadolescents (M = 10.28 years old; N = 101) and their caregivers. Direct effects were found from basal RSA to delinquent behaviors. In addition, harsh parenting predicted increased youths' aggressive and delinquent behaviors in the context of high RSA withdrawal and increased youths' delinquent behaviors in the context of shortened basal PEP. Implications for prevention and intervention are discussed.

Introduction

Harsh parenting behaviors are significantly associated with the development of a broad spectrum of externalizing behaviors in adolescence (Patterson, 2002). However, a counterintuitive but empirically documented fact is that similar family environments seem to differentially affect youth adjustment, a phenomenon referred to as multifinality (Cicchetti & Rogosch, 1996). Growing evidence indicates that these varied effects may stem from individual neurobiological differences (Dyer et al., 2016; El-Sheikh & Whitson, 2006; Erath et al., 2011). The biological sensitivity to context (BSC) hypothesis suggests that youth physiological response to stress (e.g., stress reactivity) can magnify or diminish the effect of parenting on youth behavior, such that elevated stress reactivity heightens the effect of parenting on children's behavior (Boyce & Ellis, 2005). Given the role of the autonomic nervous system (ANS) in stress reactivity, a growing body of research has been modeling its moderating role in the effect of parenting on youth adjustment (Skibo et al., 2020; Sturge-Apple et al., 2016). BSC theory has been extensively tested among children and adolescents and has yielded mixed results (Sijtsema et al., 2013), thus requiring further study. Moreover, scarce research has examined the role of the autonomic nervous

system in the connection between harsh parenting and youth externalizing behaviors (a) using both basal and reactivity data of the parasympathetic as well as the sympathetic branches and (b) considering these measures in relation to two distinct aspects of the externalizing spectrum, aggressive and delinquent behaviors (Beauchaine, 2015; Dennis et al., 2012). Thus, the main aim of the present study was to test the link between harsh parenting behaviors and two distinct domains of youth externalizing behavior, aggression and delinquency, in the context of varying levels of autonomic reactivity.

Harsh Parenting on Externalizing Behaviors in Adolescence

Harsh parenting encompasses a wide range of behaviors, such as negative affect toward the child, coercion, aggression, and punitive punishments (Erath et al., 2011; Wiggins et al., 2015). Extant research implicates harsh parenting as a salient factor in the development of psychopathology, especially externalizing symptoms such as aggression and delinquency (Patterson, 2002). Social learning theory posits that parents who exhibit harsh, negative, or aggressive behaviors socialize their children to exhibit similar behaviors during interpersonal interactions outside of the family (Bandura, 1978; Wiggins et al., 2015). Harsh or coercive parenting practices not only socialize aggression in children but also reduce parent attention toward their child's self-regulatory capacities (Sarıtaş et al., 2013). Moreover, child self-regulation abilities interact concurrently with parenting, such that poor self-regulation abilities elicits increased negativity from parents and confers greater risk for externalizing behaviors (Belsky et al., 2007, p. 200).

The study of externalizing behavior problems is particularly relevant during the transition from childhood to adolescence, a period characterized by increased psychological and neurobiological changes, and the attendant vulnerability to environmental adversity (Mendle, 2014). On the one hand, normative transitions within social and academic contexts, such as that from elementary to middle school, occur during the time in which youth consolidate autonomy and increasingly attribute more salience to peers (Chan & Chan, 2013; Daddis, 2011). On the other hand, these psychosocial changes coincide with adolescent hormonal and neurobiological developments that are associated with orientation towards high arousal, emotionally driven behaviors, and heightened sensitivity to reward (McLaughlin et al., 2011).

Together, preadolescence is a developmental phase of increased behavioral and biological plasticity as well as a heightened vulnerability for development of externalizing behaviors (Allwood et al., 2011). Because parent-child interactions are prolonged, frequent, and thus critically formative in youth socio-emotional development, parenting is one of the most potent shaping factors in the development of antisocial behaviors throughout the lifespan (Patterson et al., 2017).

Externalizing Problems Spectrum: Aggression versus Delinquency Symptoms

Externalizing problem behaviors are the most common form of maladjustment during childhood and adolescence and are a robust determinant of later psychopathology (Dishion & Patterson, 2015; Reef et al., 2010). The spectrum of externalizing problem behaviors includes two main subtypes: *overt aggressive behavior* such as fighting, teasing, hitting, and arguing, and *covert delinquent behavior* such as stealing, lying, and cheating (Achenbach & Ruffle, 2000; Prinzie et al., 2006; Reef et al., 2011). Research indicates that aggression and delinquent behaviors are developmentally distinct in both etiology and trajectory (Stanger et al., 1997). For example, coercive parenting strategies, such as intrusiveness and coercion, are more frequently used among parents of aggressive children as compared to parents of delinquent children (Dishion & Patterson, 2015). Moreover, the average trajectory of aggressive behaviors typically decreases linearly from childhood to adolescence, whereas delinquency often decreases throughout childhood but re-emerges during preadolescence, between 10 and 12 years (Bongers et al., 2004). Longitudinal research shows adolescents who exhibit primarily aggressive behaviors are more likely to commit crimes as adults than those who exhibit delinquent behaviors (Oshri, Liu, Duprey, & MacKillop, 2018; Patterson, 2002b). Similarly, childhood physical aggression more consistently predicts later adolescent health risk behaviors such as substance use and risky sexual activity than delinquent behaviors (Timmermans et al., 2008). The etiological and developmental distinction of these externalizing subtypes points to the need for empirical research that can concurrently yet separately model overt aggressive and covert delinquent behavioral outcomes.

Autonomic Nervous System Functioning

How children respond to adverse rearing environments is significantly influenced by their ability to self-regulate (Blair, 2010). The ANS plays a foundational role in the neurobiological mechanisms that underlie stress reactivity and self-regulation (Holzman & Bridgett, 2017). According to the neurovisceral integration model, the ANS and CNS influence one another bi-directionally, such that cognitive and affective neural structures within the CNS mediate the ANS to affect behavioral self-regulation (Thayer & Lane, 2000). Accordingly, a growing body of research suggests that patterns of stress reactivity—specifically, the individual's psychobiological capacity for self-regulation under acute stress—partially explain variability in parent influence on child adjustment (McQuade & Breaux, 2017; Obradović et al., 2010; Oshri et al., 2018).

Overview of Sympathetic and Parasympathetic Nervous Systems

The ANS is divided into two integrated but functionally unique parts: the sympathetic nervous system (SNS), responsible for energy expenditure, and the parasympathetic nervous system (PNS), responsible for energy conservation. The sympathetic nervous system coordinates behavioral processes that involve mobilization of bodily resources to meet environmental demands (Dennis et al., 2012). When a threat is perceived, the SNS increases cardiac ventricular contractility, and thus cardiac output, allowing the body to more efficiently carry out fight or flight behaviors. As such, the SNS has been shown to act as a proxy and predictor of individual variability in response to stress (Erath et al., 2009). Although no single measure can directly indicate SNS or PNS activity, study of autonomic functioning certainly reveals precise patterns that reflect the influence of the two branches (Jänig & Häbler, 2000). As such, the patterns in which the SNS influences cardiac contractility may be proxied by pre-ejection period (PEP).

The use of PEP, or the timing between the depolarization of the left ventricle and the release of blood from the aorta, is thought to be an effect proxy of sympathetic influence on cardiac contractility (Beauchaine, 2015; Cacioppo et al., 1994). PEP shortening indicates greater cardiac output and sympathetic influence on the heart. Variations in PEP occur during the stress response and have been shown to reflect individual variations in stress responsivity (Beauchaine et al., 2001). Shortened basal PEP, as well as increased PEP shortening during stress, indicate increased sympathetic activity. This

augmentation in SNS influence over cardiac contractility is highly associated with decreases in self-regulation: Beauchaine et al. (2013) found that children with shorter basal PEP exhibited increased conduct problems and aggression. In turn, attenuated self-regulation proxied by PEP shortening often exacerbates risks conferred by early childhood adversity, leading to maladaptive behavioral outcomes such as aggression and impulsivity (Bubier et al., 2009; Erath et al., 2009). Bubier et al. (2009) found that in the context of shorter basal PEP, harsh parenting predicted increased child externalizing behaviors. Similarly, among children with elevated PEP reactivity (i.e., PEP shortening) high permissive parenting was associated with increased affiliation with deviant peers, which is closely linked to risk behaviors (Hinnant et al., 2016). These findings indicate that development of youth maladjustment is signigicantly associated with patterns of sympathetic functioning, namely increases in sympathetic influence on cardiac contractility as proxied by PEP shortening. Although these findings are certainly informative, investigation of parasympathetic influence is also necessary to further elucidate implications of autonomic functioning on development.

The parasympathetic nervous system (PNS) is associated with "resting and digesting" behaviors used to gather and save energy resources. Similar to the SNS, there is no general index for PNS function; however, measuring vagal regulation of cardiac rate allows us to indirectly assess parasympathetic influence. The tenth cranial nerve, also known as the vagus nerve, is the mechanism by which parasympathetic influence is exerted on the heart. Vagal efferent pathways stemming from the nucleus ambiguus lead to cyclical variations in inter-heartbeat intervals known as heart rate variability. The high-frequency range of heart rate variability determines respiratory sinus arrhythmia, or RSA (Beauchaine, 2001; Porges, 2007). Vagal activity during resting states, also known as baseline RSA, is a proxy of individual ability to self-regulate and maintain homeostasis when reacting to environmental stimuli, including those emanating from family interactions (Cacioppo et al., 1994; Skibo et al., 2020; Wagner et al., 2017). Attenuated baseline RSA is linked to increased conduct problems, hostility, and internalizing behaviors (Balzarotti et al., 2017; Beauchaine et al., 2001; Dyer et al., 2016). Vagal activity during acute stress, as measured by RSA reactivity (RSA-R), includes both vagal withdrawal (decrease in RSA from

baseline) and augmentation (increase in RSA from baseline). Typically, vagal withdrawal occurs during emotionally negative or stressful stimuli and leads to increased heart rate and metabolic output, possibly indicating individual ability to adaptively respond to stress (Perry et al., 2012; Porges, 2007). Vagal augmentation slows heart rate and suggests propensity toward immobilization in response to environmental demands (Erath et al., 2011). However, extreme levels of vagal withdrawal during acute stress may indicate decreased capacity for self-regulation (Hastings & Miller, 2014; Kahle et al., 2018). As such, patterns of heightened vagal withdrawal has been associated with maladaptive stress response patterns and consequent externalizing problems (Dennis et al., 2012; Fortunato et al., 2013; Miller et al., 2013; Obradović et al., 2010; Tabachnick et al., 2021).

The parasympathetic and sympathetic systems are highly related during behavioral processes (Porges, 2007). Gray's motivational theory (Gray, 1982, 1987a, 1987b) identifies two interdependent dimensions of behavior: inhibition and activation. According to Gray, the SNS comprises the Behavioral Approach System (BAS), which is responsible for approach behaviors and active avoidance behaviors that respectively maximize reward and minimize aversive consequences during situations of threat. Conversely, the Behavioral Inhibition System (BIS) inhibits approach behaviors when aversive consequences are anticipated. In line with Gray's motivational theory is the association between maladaptive behavioral outcomes and imbalances in functioning of the SNS (behavioral activation) and PNS (behavioral inhibition). Considering the interdependence of both PNS and SNS as determinants of behavior, an investigation of the ANS as a contextual factor for psychopathology would benefit from increased methodological rigor in which activity of both branches is measured.

The ANS as a Determinant of Biological Sensitivity to Context

Higher stress response reactivity, as indicated by biomarkers of the SNS (PEP shortening) and PNS (vagal withdrawal), are associated with increased vulnerability to maladaptive outcomes (El-Sheikh et al., 2007; Hastings & Miller, 2014). According to BSC theory, physiological reactivity amplifies individual sensitivity to environmental influence such that some individuals physically experience the input of their context in a more acute way than others (Boyce & Ellis, 2005). Moreover, BSC theory

posits that sensitivity to contextual factors may not only be quantified (by measures such as RSA and PEP) but also used to predict developmental outcomes throughout the lifespan. Indeed, child physiological stress reactivity has been shown to interact concurrently with environmental factors to predict child outcomes such that highly reactive physiological profiles prove maladaptive in unsupportive or adverse contexts but adaptive in supportive contexts (El-Sheikh et al., 2007; Erath et al., 2009; Obradović et al., 2010). Although multiple studies have corroborated the BSC theory with young children, studies among pre-adolescent samples have yielded mixed results and lack longitudinal samples as well as economic and ethnically diverse samples (for review, see Sijtsema et al., 2013). Moreover, measurement of parenting behaviors has often been based on parent- or child-report, which has been shown to predict outcomes less consistently as compared to direct observation of parenting behavior (Faith et al., 2012; Pritchett et al., 2010). Furthermore, studies of BSC theory are often limited to one branch of the autonomic nervous system, investigating basal activity or stress reactivity, but not both. Finally, extant research on BSC theory and its relevance to externalizing outcomes lacks specificity in the type of outcome (i.e. delinquency versus aggression), which is necessary in order to better understand the nuanced etiologies of problem behaviors.

The Present Study

The main goal of the present study was to investigate the differential effects of harsh parenting on child aggressive and delinquent behaviors across varying levels of ANS functioning. Thus, we aimed to test the role of autonomic functioning at rest and during acute stress in moderating associations between harsh parenting and pre-adolescent aggressive and delinquent outcomes. Based on BSC theory, we hypothesized that high (i.e., shortened) basal PEP would exacerbate the effects of harsh parenting behaviors on aggressive and delinquent behaviors. We hypothesized that high PEP reactivity (i.e. PEP shortening during stress) would exacerbate the effects of harsh parenting on aggressive and delinquent behaviors. Additionally, we hypothesized that low baseline RSA and high RSA withdrawal during stress would exacerbate the impact of harsh parenting on aggression and delinquent behaviors.

The current study offers methodological advances that may assist in addressing methodological gaps in the BSC literature in several ways. First, we employed a longitudinal, community sample in order to assess developmental outcomes. Second, we assess ANS functioning via proxies of both SNS and PNS influence (PEP and RSA, respectively) during a modified Trier Social Tress task. We also quantified residual change (controlling for expected intra-individual variability) from baseline to stress to more comprehensively measure the child's autonomic functioning (Bubier et al., 2009). We measured parenting via an observed interaction task between parent and child, which builds on extant literature that primarily employs parent or child report. Moreover, we made the distinction between aggressive and delinquent behavioral outcomes to increase the specificity of our study and extend the literature regarding implications of autonomic functioning on problem behaviors. Finally, we used a low-income, ethnically diverse sample to test biological sensitivity in the context of the various stressors that are related to low SES and status as a racial or ethnic minority.

Methods

Sample

The sample was drawn from a small city in the southeastern U.S. and consisted of 101 pre-adolescent youths ages 9 to 12 years (M_{age} = 10.28 years; 43% male, 57% female) and their primary caregivers, who were mostly mothers (90.1%, M_{age} = 35.30 years, SD = 6.51). Other caregivers included fathers, grandparents, and other extended family members. All families came from economically impoverished backgrounds (at or below 200% of poverty line), and as such, 42.5% of caregivers had received food stamps and/or Women, Infants, and Children (WIC) benefits in the past year. The sample was diverse, with 72.5% of participants identifying as African American, 12.8% identifying as White, and 6.4% as Hispanic/Latino.

Procedures

Study and procedures were approved by the University of Georgia Institutional Review Board (Parenting and Children's Decision Making, approval number 00003946). In order to connect with community liaisons, the research team engaged the assistance of a local community organization that

works to promote well-being among youth and families and has many established relationships with community leaders. The community organization worked with researchers to identify individuals who resided in the community of interest and were socially well-connected who could serve as study liaisons to recruit study participants. Those who agreed to be liaisons were fully informed of the purpose and procedures of the study and were compensated $100 for each family that participated in the complete study.

Eligibility requirements for potential participants included availability of the primary caregiver, ability of caregiver and child to speak English, and family income level being below 200% of the poverty line. Enrolled families took part in data collection in a lab setting on the university campus. After consent and assent for caregiver and child were obtained, both were attached to an electrocardiogram via dermal electrodes for the duration of the survey. One electrode was placed on each side of the bottom rib cage, on each side of the clavicle, below the sternum, on the upper spine, and the lower spine. Biometric data were acquired using the BioLab software provided through MindWare Technologies. In order to stimulate participants' autonomic response to a simulated acute social-cognitive stressor, child participants were asked to perform a five-minute mental arithmetic stress task shown in prior research to produce a significant physiological reaction (Berntson et al., 1996).

Measures

PEP Reactivity. Sympathetic nervous system activity, proxied by basal PEP and PEP-R, was collected using a mobile impedance cardiograph (MindWare Technologies, Ltd., Gahanna, OH). To measure PEP, impedance cardiography analysis was conducted by isolating the time interval between the initial electrical stimulation of the heart (onset of the R peak) and the opening of the aortic valve (B point of the dZ/dT wave; Lozano et al., 2007). Using the MindWare IMP 3.1.4 Software module, impedance data were ensemble-averaged in 30-second epochs and combined with R waves that were obtained from the electrocardiogram. Research assistants were extensively trained to cross-inspect and correct abnormal R-R intervals such as severe fluctuations, inadvertent cardiac fluctuations, and ectopic beats due to

physical movement or breathing. Mean values of PEP across the 30-second epochs were calculated for the baseline and stress interaction task.

To account for within-person variability in PEP-R, a residualized change score was created using the mean level of PEP during the rest period and during the stress task (Cacioppo et al., 1994). A lower PEP residualized change score indicates a decrease from baseline to the stress task, and thus more sympathetic influence over the heart.

$$\Delta PEP = \frac{PEP_{\,Stress} - PEP_{\,Baseline}}{SD(PEP_{\,Baseline}) \times \sqrt{1 - r(PEP_{\,Stress},\ PEP_{\,Baseline})}}$$

Basal RSA and RSA Reactivity. RSA and RSA-R were utilized to assess caregiver and child parasympathetic physiological activity (Thayer & Lane, 2000). Baseline RSA was measured 3 minutes after watching a relaxation video, and RSA-R was measured during the acute stress task. Procedures were in accordance with current standards for measuring RSA in psychophysiological research (Berntson et al., 1996). RSA was measured using MindWare Technologies BioLab software (Version 3.0.6; MindWare Technologies, Ltd., Gahanna, OH). Data were digitized with the MindWare 3.1.0 software module at a sampling rate of 1,000 Hz. High-frequency components of RSA (0.12 to 0.42 Hz) were obtained via power spectrum analysis (Akselrod et al., 1981) to target parasympathetic neural activity. Past research has indicated that high-frequency components of RSA are indicative of more effective self-regulation (Akselrod et al., 1981; Appelhans & Luecken, 2006). Because accurate assessment of RSA depends on correct specification of respiration rates, and these rates decrease significantly from childhood to adulthood, two different respiratory bands were employed for parents and children (Zisner & Beauchaine, 2016). Therefore, the frequency threshold of RSA for adults was 0.15 Hz, while for youths it was .25 Hz. To estimate and correct for individual differences in respiration, spectral analysis of thoracic impedance was utilized (Ernst et al., 1999). Derived from the ECG signal, interbeat intervals (IBIs) were converted into 120-second segments using an interpolation algorithm, and physically improbable IBIs were detected using a MindWare Minimum Artifact Deviation and Maximum Expected Deviation (MAD/MED)

algorithm that places some bounds on the variability of the IBI from beat to beat, and flags beats which exceed these limits (Berntson et al., 1990). Data were also manually inspected by trained researchers to detect abnormal R-R intervals such as inadvertent cardiac fluctuations and ectopic beats in RSA due to participants' physical movement or breath. Abnormal R-R intervals were manually corrected by deleting extra beats and inserting mid-beats, according to MindWare Technologies instructions. A residualized change score (equation below) was calculated for RSA reactivity using participants' mean RSA during the baseline period and during the stress period (arithmetic task). The use of a residualized change score allows for the adjustment of within-person variability in baseline RSA (Berntson et al., 1996). A lower RSA residualized change score indicates a decrease from baseline to the stress task (i.e., more vagal withdrawal), and thus suggests a higher level of RSA reactivity.

$$\Delta HRV = \frac{HRV_{\,Stress} - HRV_{\,Baseline}}{SD(HRV_{\,Baseline}) \times \sqrt{1 - r(HRV_{\,Stress},\ HRV_{\,Baseline})}}$$

Child Stress Task. The task was constructed based on the Trier Social Stress Task (Allen et al., 2014, 2017; Kirschbaum et al., 1993). At the beginning of the task, children were asked to imagine that they were in a math competition taking place in front of their teachers, parents, and peers in which they would be completing a series of backward digit counting tasks. Though caregivers sat next to the child during the task, they were advised by the researchers not to participate or intervene during the task. It should be noted, however, that this instruction was not given during the initial stages of the study, and as such, a small number of caregivers (less than 5% of the sample) did intervene during the task. When reviewing task instructions, the research assistant led the child through an example trial by demonstrating how to start at 100 and count down by 1. Once the research assistant determined the child understood the task, the five-minute timer began. Children were asked to start at 100 and count down by 2 until they were told to stop. In order to maintain the level of stress for the child, the difficulty increased throughout the length of the task according to the competency displayed by the child (e.g., "Now start at 100 and count down by 6").

Conflict Discussion. During a six-minute conflict discussion task, caregivers and children were presented with a stack of notecards, each listing a different topic about which parents and children often disagree (e.g., chores, completing homework, fighting with siblings). The dyad was asked to work together to select the three topics they most frequently disagree about. Once they chose their three topics, dyads were told they would have six minutes to discuss the selected issues and try to achieve possible solutions. Dyads were instructed to try and talk about all three of the problems, and if they finished before the time was up, they were asked to go through the remaining notecards and discuss any additional problems. The research assistant then left the room and returned when the 6 min were over. Parenting behaviors were coded using a 7-point Likert scale (1 = *very low*, 7 = *very high*) based on an existing global coding system (Martin et al., 2008). Coded behaviors for harsh parenting included negativity toward child, intrusiveness, and coercion. Inter-rater reliability was estimated using an intra-class correlation (ICC) based on double coding 24.5% of the videos. Intra-class correlations were .84, .88, and .79 for negativity, intrusiveness, and coercion, respectively.

Child Psychopathology Symptoms. Caregivers completed the Child Behavior Checklist (CBCL; Achenbach & Ruffle, 2000) to report on their child's externalizing problems (Cronbach's alpha = .91). The CBCL contains 118 items rated on a 3-point scale ranging from 0 (*not very true*) to 2 (*very true or often true*) and yields a broadband externalizing measure as well as Delinquent and Aggressive subscales, which have been shown to be reliable and valid in similar samples (Dutra et al., 2004). Whereas T-scores between 65 and 70 represent borderline clinical levels, T-scores greater than 70 indicate clinical levels of symptoms (Achenbach & Ruffle, 2000). In the current sample, 5% of children met criteria for borderline clinical levels of aggressive symptoms and 5.9% met criteria for borderline delinquent symptoms at T1. Additionally, 2.8% of children demonstrated clinical levels of delinquent symptoms and 2.8% demonstrated clinical levels of aggressive symptoms at T1. T-scores for these domains were used in the analyses.

Analytic Plan

Distributions of all variables were checked for normality, and skewed distributions of externalizing subtypes were logarithmically transformed before analysis. A structural equation model (SEM) in Mplus version 7.31 (Muthén & Muthén, 2014) was used to test study hypotheses. The SEM was estimated using maximum-likelihood estimation (Klein & Moosbrugger, 2000). Missing data at T1 ranged from 0 to 6.9%, as follows: 3.9 to 4.9% for physiological measures, 6.9% for parenting measures, and 0% for T1 child behavioral measures. Due to an attrition rate of 33% from T1 to T2, during which 10 to 13 months passed, missing data for T2 child behavioral measures was 33%. Little's Missing Completely at Random (MCAR) test suggested that missing data patterns met the MCAR assumption, χ^2 (172) = 162.120, p = .694. A CFA was then performed to test a latent construct of harsh parenting using three indicators: negative affect, intrusiveness, and coercion (see Figure 2.1). Next, SEM was used to model the interaction effect of the latent parenting construct and autonomic activity—basal RSA, RSA-R, basal PEP, and PEP-R—on child aggressive and delinquent outcomes. Finally, the Johnson and Neyman (1936) technique was employed to probe and interpret the regions of significance at differing values of the moderator.

Results

Descriptive Analysis

Descriptive statistics and bivariate correlations for all study variables are displayed in Table 2.1. Children with lower basal RSA exhibited higher levels of delinquent behaviors at T1 *(r* = -.254, $p < .05$). Children with lengthened PEP at baseline experienced increased parental intrusiveness ($r = .234$, $p < .05$). Children with elevated (shortened) PEP reactivity measured during the acute stress task also experienced increased parental negative affect *(r* = -.209, $p < .05$). Parental intrusiveness was also significantly associated with youths' increased delinquent behaviors at T1 ($r = .239$, $p < .05$). Older children exhibited more aggressive behaviors at T1 as compared to younger children ($r = .250$, $p < .01$). Boys were found to experience greater parent coercive behaviors as compared to girls ($r = -.266$, $p < .01$) and exhibited decreased basal RSA *(r* = -.341, $p < .01$).

Confirmatory Factor Analysis

A CFA of harsh parenting was conducted, with three indicators used: parent negative affect toward child, parent intrusiveness, and parent coercion (see Figure 1). All factor loadings were significant and above .50, as suggested by Brown (2014). Model fit was excellent (see Figure 1).

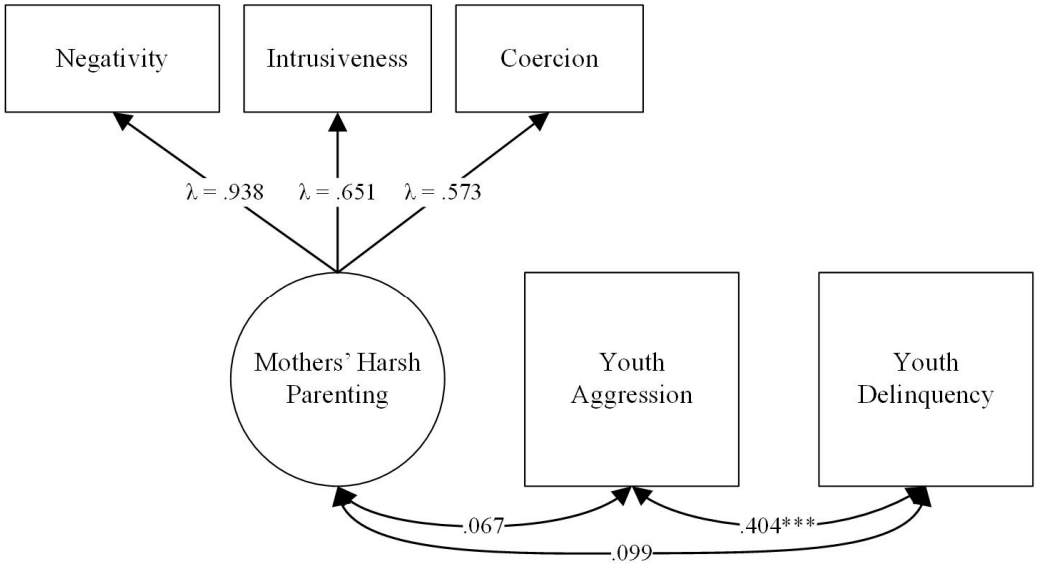

Figure 2.1. Measurement model for mothers' harsh parenting latent factor in relation to externalizing subtypes. *Note.* All values are standardized. All factor loadings are significant ($p < .000$). Model fit is excellent: χ^2 (4) = 0.377, p = 0.984; RMSEA = 0.000; CFI = 1.00; TLI = 1.151; SRMR = 0.012.

Table 2.1. Bivariate correlations, means, and standard deviations of study variables (N = 101)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. RSA_B	--													
2. ΔRSA	-.250*	--												
3. PEP_B	-.091	-.071	--											
4. ΔPEP	.090	-.031	-.577**	--										
5. RBK_T1	-.197	.143	.087	-.004	--									
6. RBK_T2	-.254*	.149	.020	.028	.391**	--								
7. AGG_T1	-.164	.048	.190	-.012	.771**	.274**	--							
8. AGG_T2	-.191	.179	-.004	-.056	.341**	.502**	.445**	--						
9. P_NEG	.001	.115	.165	-.209*	.164	.130	.085	.276*	--					
10. P_INT	-.043	-.026	.234*	-.166	.239*	.055	.109	.120	.611**	--				
11. P_CRS	.174	.051	-.041	.139	.048	-.005	.067	.110	.539**	-.368*	--			
12. SEX	-.341**	.102	.111	-.041	-.302	.211	.017	.133	-.05	-.077	-.266**	--		
13. AGE	-.195	.163	.079	-.013	.141	.077	.250**	.106	-.002	-.052	.011	.143	--	
14. INC	.025	-.036	-.007	-.001	-.098	.008	-.002	-.109	-.032	-.241	-.185	.199	0.24	--
Mean	6.95	-1.35	77.42	-.06	53.86	53.00	52.84	52.90	2.34	2.95	1.71	.52	10.28	21.74
SD	1.19	1.41	24.75	1.38	5.77	4.06	5.60	5.14	1.37	1.39	1.39	.502	1.19	12.8

Note. RSA_B = Basal resting sinus arrhythmia, ΔRSA = Residualized change RSA during acute stress task, ΔPEP = Residualized change PEP during acute stress task, RBK_T1 = Time 1 rule-breaking T score, RBK_T2 = Time 2 rule-breaking T score, AGG_T1 = Time 1 aggression T score, AGG_T2 = Time 2 aggression T score, P_NEG = Parent negative affect toward child, P_INT = Parent intrusiveness toward child, P_CRS = Parent coercion toward child, INC = Family median annual income (in thousands). *$p < .05$, **$p < .01$, ***$p < .001$.

Moderation Analyses

PEP Baseline & Reactivity. Moderation analyses were conducted with a harsh parenting latent factor to determine how basal PEP and PEP-R interacted with parenting to predict pre-adolescent aggression and delinquent behaviors in Time 2, after adjusting for variance in T1. No significant direct effect was found between harsh parenting and either aggression or delinquency. The interaction effect of harsh parenting and basal PEP was also not significantly associated with youths' aggression at T2. However, the interaction effect of harsh parenting and basal PEP was positively associated with delinquency at T2 after adjusting for variance at T1 (Figure 2.2; β = -.562; SE = .143; *p* = .000; 95% CI [-.797, -.327]). Post-hoc probing of the moderation, depicted in Figure 2.3, show that increased levels of harsh parenting predicted increased delinquent behavior among youths with shortened PEP (and thus increased SNS activity) at rest. The proportion of variance in youths' delinquent behaviors explained by this moderation was 68%. Moderation did not significantly differ based on sex, income, or age. Interaction terms involving PEP-R did not significantly predict youths' aggressive or delinquent behaviors.

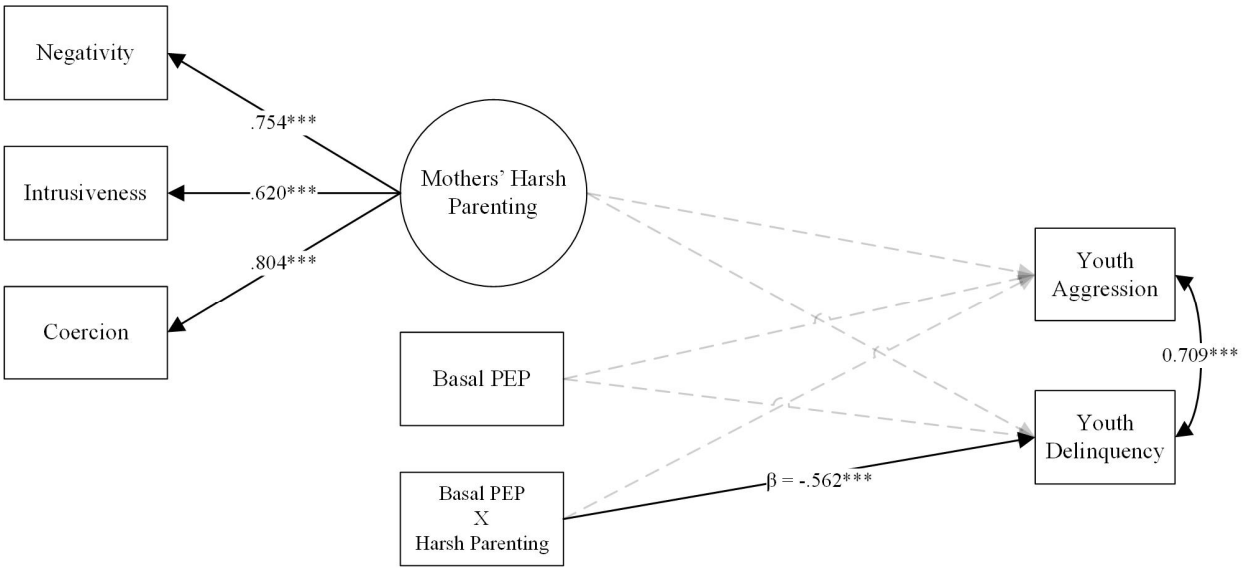

Figure 2.2. Moderation model with sympathetic activity at rest. All values are standardized. Dashed lines are insignificant. Delinquency and aggression at T1, household income, child sex, and child age were controlled for. * p < .05, ** p < .01, *** p < .001.

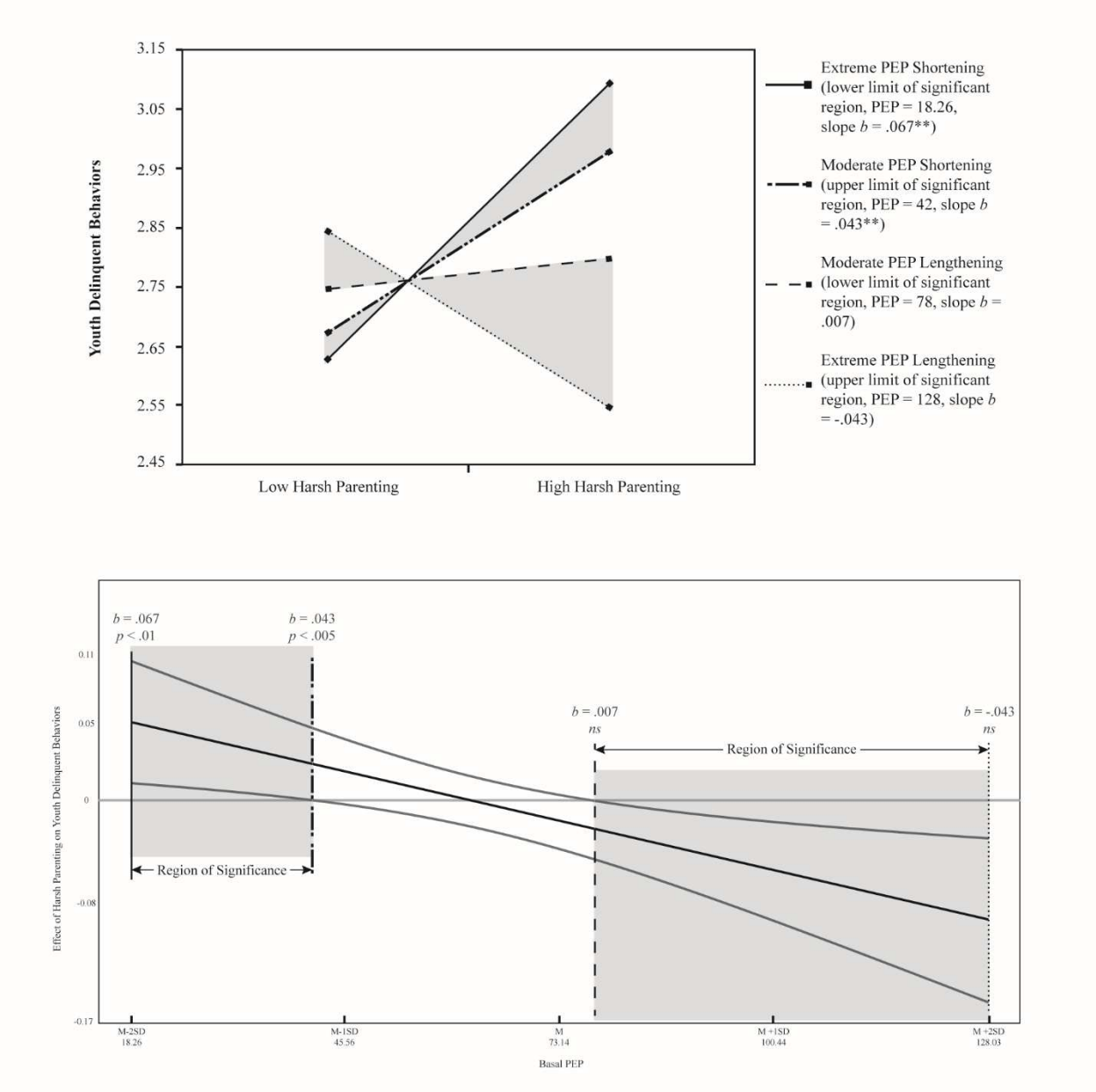

Figure 2.3. Interpretation of the moderating role of basal PEP on the link between harsh parenting and delinquent behaviors. The upper panel presents an adapted simple slope interpretation, and the lower panel presents the Johnson-Neyman plot. In both figures, the shadowed areas indicate regions of significance. The solid line represents an extremely shortened basal PEP level that is the lowermost boundary of the left significant region (PEP = 18.26, slope *b* = .067, p = .006). The dashed-dotted line represents the moderately shortened basal PEP level that is the uppermost boundary of the left significant region (PEP = 42, slope *b* = .043, p = .003). The dashed line represents the moderately lengthened basal

PEP level that is the lowermost boundary of the right significant region (PEP = 78, slope *b* = .007, *ns*). The dotted line represents the extremely lengthened basal PEP that is the uppermost limit of the significant region (PEP = 128, slope *b* = -.043, *ns*).

RSA Baseline & Reactivity. Basal RSA negatively predicted youths' delinquent behaviors at T2 after adjusting for variance in T1, such that attenuated RSA at rest predicted increased delinquency (β = -.426; SE = .145; *p* = .003; 95% CI [-.040, -.009]). No significant effect was found for the interaction between harsh parenting and basal RSA. The interaction effect of harsh parenting and RSA-R at T1 was significantly associated with youths' delinquent and aggressive behaviors at T2, indicating moderation (Figure 2.4; respectively: β = -.550; SE = .201; *p* = .006; 95% CI [-.881, -.218]; β = -.590; SE = .157; *p* = .000; 95% CI [-.848, -.332]). Post-hoc probing of the moderation, as seen in Figures 2.5 and 2.6, showed that among youths' who exhibited high RSA withdrawal during stress, increased levels of harsh parenting predicted increased aggressive and delinquent behaviors. The proportion of variance in youths' delinquent and aggressive behaviors explained by these pathways was 58.3% and 77.3%, respectively. Moderation did not significantly differ based on sex, income, or age.

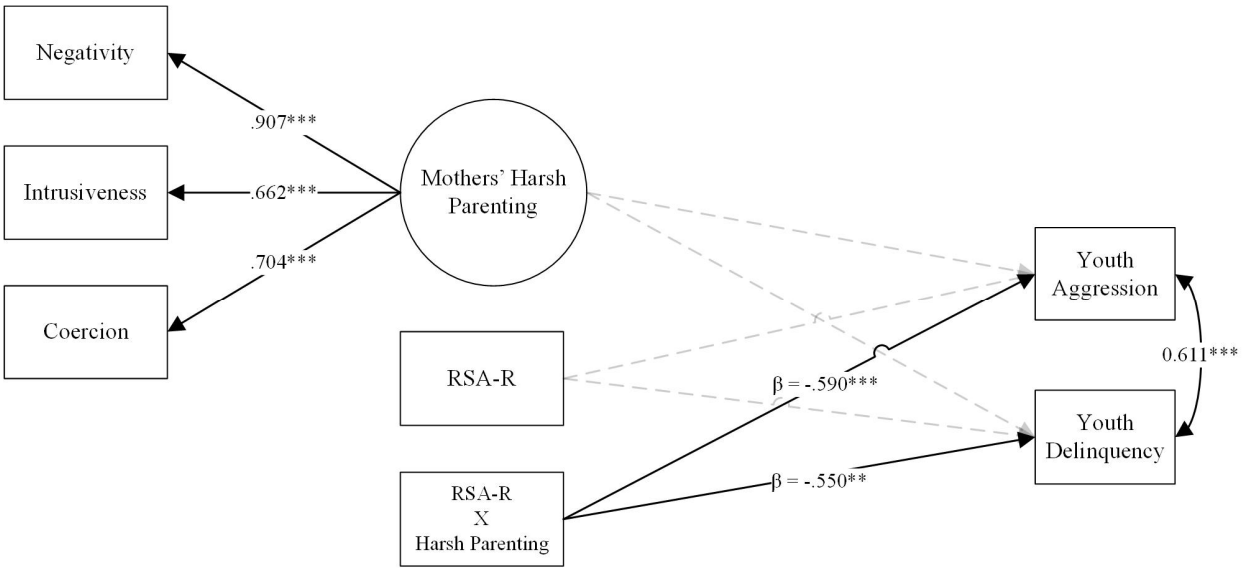

Figure 2.4. Moderation model with parasympathetic reactivity. RSA-R = Resting sinus arrhythmia reactivity. All values are standardized. Dashed lines are insignificant. Delinquency and aggression at T1, household income, child sex, and child age were controlled for. * p < .05, ** p < .01, *** p < .001.

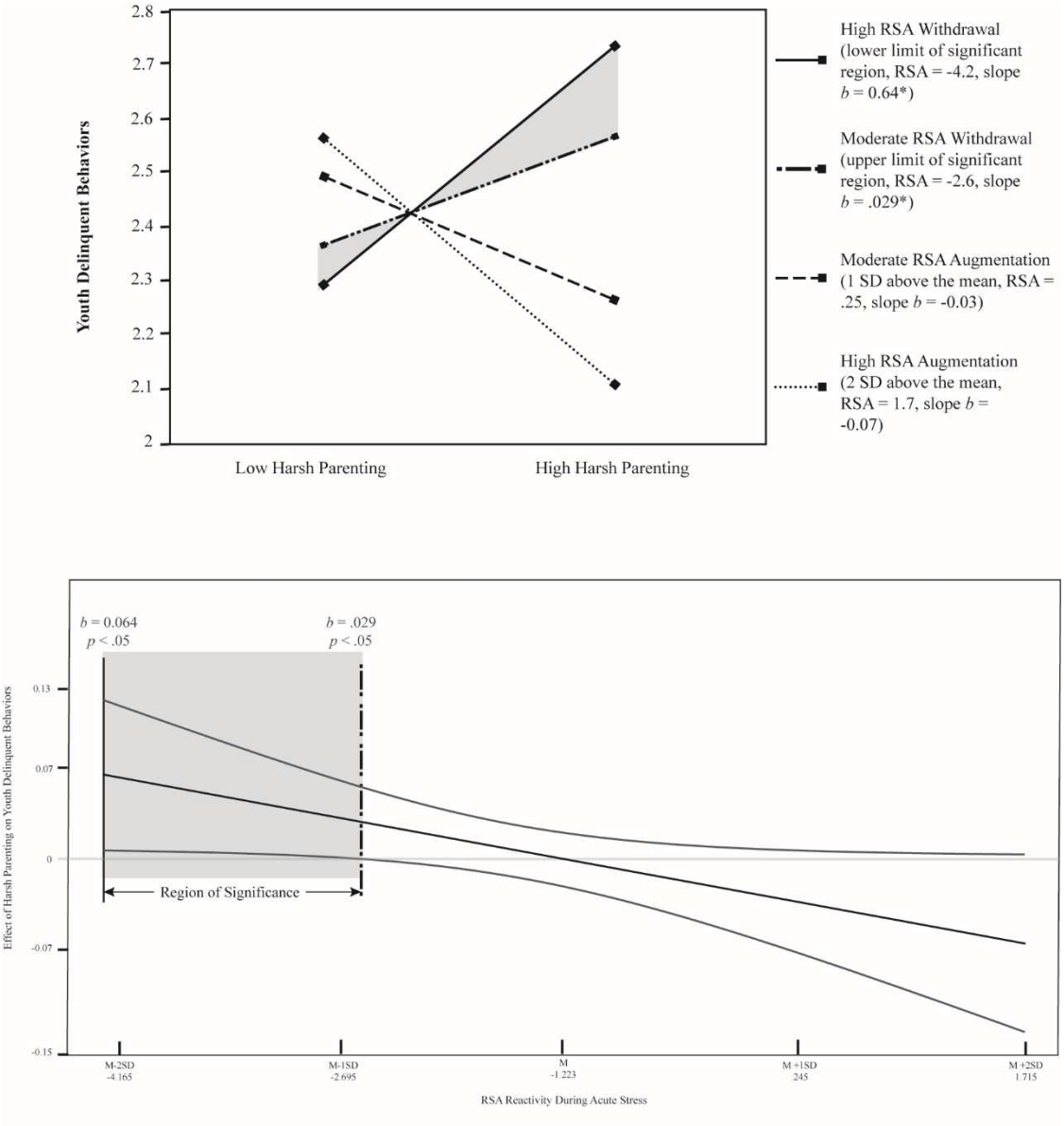

Figure 2.5. Interpretation of the moderating role of RSA reactivity on the link between harsh parenting and delinquent behaviors. The upper panel presents an adapted simple slope interpretation, and the lower panel presents the Johnson-Neyman plot. In both figures, the shadowed areas indicate regions of significance. The solid line represents a level of high RSA withdrawal that is the lowermost boundary of

the significant region (ΔRSA = -4.2, slope *b* = .064, p = .03). The dashed-dotted line represents a level of moderate RSA withdrawal that is the uppermost boundary of the significant region (ΔRSA = -2.6, slope *b* = .029, p = .047). The dashed line represents a level of moderate RSA augmentation that is one standard deviation above the mean (ΔRSA = .245, slope *b* = -.033, *ns*). The dotted line represents a level of high RSA augmentation that is two standard deviations above the mean (ΔRSA = 1.72, slope *b* = -.066, *ns*).

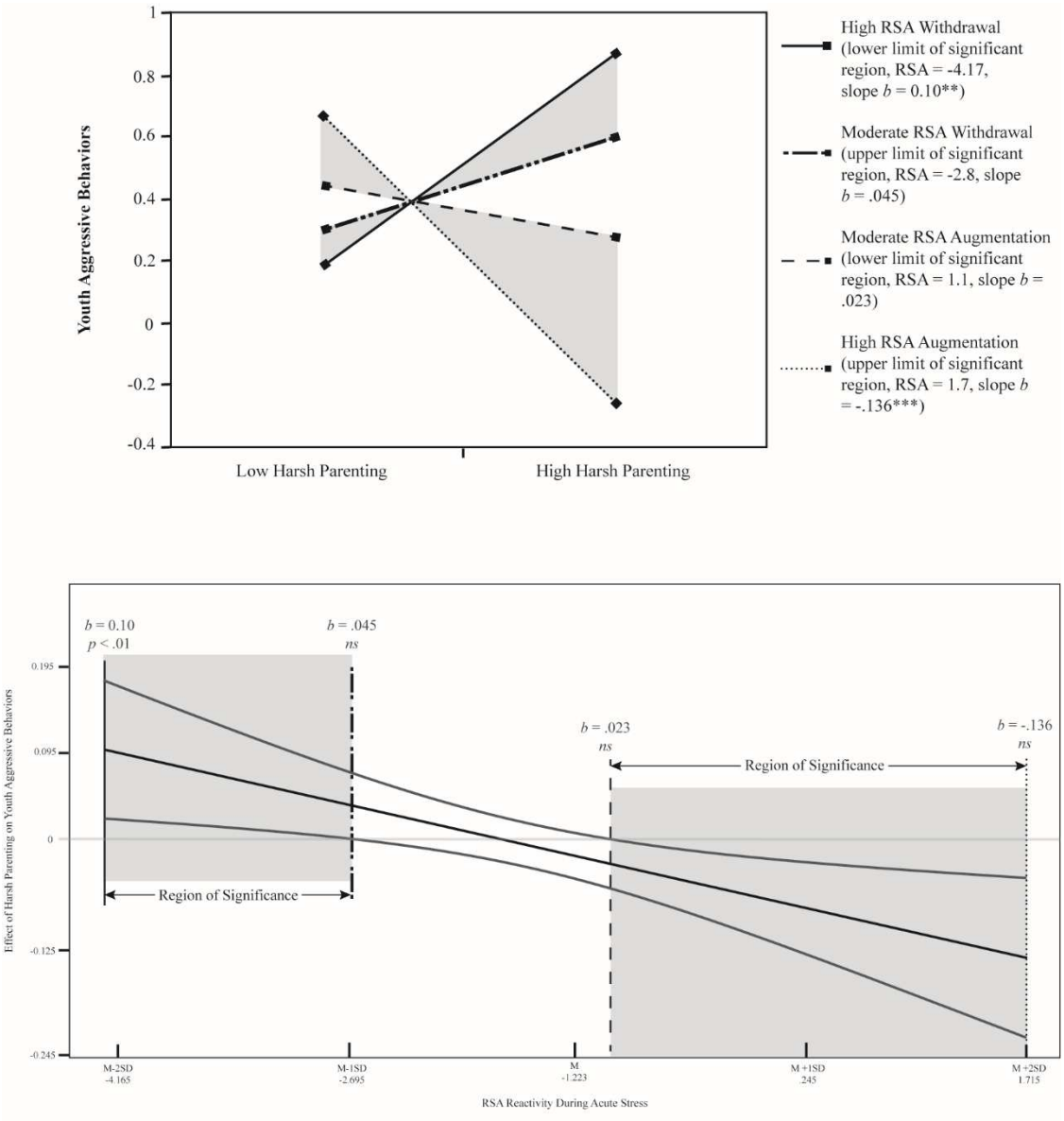

Figure 2.6. Interpretation of the moderating role of RSA reactivity on the link between harsh parenting and aggressive behaviors. The upper panel presents an adapted simple slope interpretation, and the lower

panel presents the Johnson-Neyman plot. In both figures, the shadowed areas indicate regions of significance. The solid line represents a level of high RSA withdrawal that is the lowermost boundary of the left significant region (ΔRSA = -4.2, slope *b* = .10, p = .004). The dashed-dotted line represents a level of moderate RSA withdrawal that is the uppermost boundary of the left significant region (ΔRSA = -2.8, slope *b* = .045, *ns*). The dashed line represents a level of moderate RSA augmentation that is the lowermost boundary of the right significant region (ΔRSA = -1.1, slope *b* = -.023, *ns*). The dotted line represents a level of high RSA augmentation that is the uppermost limit of the significant region (ΔRSA = 1.7, slope *b* = -.136, p = .000).

Discussion

Parenting behaviors are key in shaping youth socio-emotional adjustment and risk for psychopathology (Sarıtaş et al., 2013). Recent theory and research suggest the effect of parenting on youths' adjustment can vary significantly depending on individual psychobiological reactivity to the environment (Boyce & Ellis, 2005). Specifically, research suggests that youths' reactivity to parenting is modulated by autonomic functioning, which can increase or decrease risk for externalizing psychopathology. Therefore, in the present study we used using indirect indices of both sympathetic and parasympathetic functioning to investigate the moderating role of the ANS in the effects of harsh parenting on child aggressive and delinquent behaviors. Our findings corroborate and advance knowledge on the differential effects of harsh parenting and externalizing outcomes due to variations in autonomic functioning. This study provides insight into the roles of parasympathetic influence of cardiac rate and sympathetic influence on cardiac contractility, during baseline and stress, as a biological context for child externalizing outcomes. Specifically, the link between harsh parenting and youths' delinquent behaviors was exacerbated by shortened basal PEP, whereas the link between harsh parenting and both delinquent and aggressive behaviors was exacerbated by RSA withdrawal during stress. Because patterns of the autonomic nervous system, especially stress response reactivity, are subject to change via interventions such as mindful meditation, understanding the moderating role of the ANS as a protective or risk factor in

the association between harsh parenting and externalizing outcomes may lead to important prevention implications (Chen et al., 2017).

Our first hypothesis was partially supported. Among youths with shortened basal PEP, harsh parenting predicted increased delinquent behaviors at T2. Shortened basal PEP has been shown to be a risk factor for conduct problems in both children and adolescents, and is thought to be indicative of temperament-level emotionality (Beauchaine, 2001). Our findings partially replicate those by Bubier et al. (2009), who found that among children with shortened basal PEP, harsh parenting and low neighborhood cohesion predicted increased externalizing behaviors. Our findings are also in line with BSC theory, indicating that children with elevated baseline sympathetic functioning show increased sensitivity to environmental adversity and thus increased propensity for maladaptation in the context of harsh parenting.

Importantly, the moderating effect of basal PEP did not extend to aggressive behaviors, which may support extant research that distinguishes aggressive and delinquent behaviors as developmentally unique (Prinzie et al., 2006). Indeed, in their seminal study of child development from ages 4 to 18, Bongers et al. (2004) found a divergence in the trajectories of aggressive and delinquent behaviors. Although aggressive behaviors normatively declined in a linear fashion, delinquent behaviors took a curvilinear shape, decreasing linearly until ages 10 through 12, and increasing again thereafter. This trend is arguably at work within the current sample, which encompasses the critical period in which delinquent behaviors often re-emerge (rates of delinquent behavior are indeed marginally higher than rates of aggressive behaviors in the current sample). Although basal PEP may indeed be a salient and unique contributor to youth delinquent, and not aggressive, behaviors, further replication is necessary before this conclusion is drawn.

Our second hypothesis was not supported in that the effect of harsh parenting on child aggression was not influenced by PEP reactivity (shortening and/or lengthening) during acute stress. As such, we may conclude that sympathetic influence over cardiac contractility during acute stress may be less indicative of BSC, and less formative in the development of maladjustment, when compared to

sympathetic influence over cardiac contractility at rest. Once again, this finding is similar to that of Bubier et al. (2009), who found that PEP reactivity during stress did not moderate the link between harsh parenting and externalizing behaviors among children. The present finding diverges from those of Hinnant et al. (2016), which implicated PEP shortening during acute stress as a salient contributor to adolescent risk behaviors. This divergence may be due to assessment of PEP at differing periods of development, with Hinnant et al. employing a sample of middle adolescents rather than pre-adolescents. Although nascent findings have pointed to developmental increases of PEP during childhood, as well as stabilization of PEP during adolescence (Alkon et al., 2003; Matthews et al., 2002), a more recent study has yielded conflicting findings (J. Benjamin Hinnant, Elmore□Staton, & El□Sheikh, 2011). As such, additional study is necessary to further clarify the role of PEP reactivity in the link between harsh parenting and externalizing behaviors across differing stages of child and adolescent development.

Our third hypothesis was supported, in that the effect of harsh parenting on youths' aggressive and delinquent behaviors was exacerbated in the context of high RSA reactivity (vagal withdrawal). Moreover, the effect of harsh parenting on aggressive, but not delinquent, behaviors was attenuated in the context of low RSA reactivity (vagal augmentation). This finding partially supports a BSC framework, in which high and low autonomic reactivity act respectively as risk and protective factors in adverse contexts such as harsh parenting. Once again, replication of these results is necessary before further delving into the distinction between aggressive and delinquent behaviors and their association to vagal augmentation. Our results also highlight the necessity to distinguish between community and clinical samples when studying autonomic functioning. Blunted parasympathetic reactivity has been shown to increase risk for conduct problems among clinical samples (Brenner & Beauchaine, 2011). In contrast, the opposite trend is seen in community samples: low reactivity is shown to mitigate risk. This may be due to the less frequent incidence of maltreatment in community samples as compared to clinical samples (Dennis et al., 2012; Obradović et al., 2010).

In contrast to our last hypothesis, the effect of harsh parenting on externalizing behaviors did not significantly differ based on levels of basal RSA. However, we found that low basal RSA longitudinally

predicted youths' delinquent behaviors, thus corroborating similar findings on the associations between blunted parasympathetic functioning and behavioral dysregulation (Dyer et al., 2016; Eisenberg et al., 2012). Low basal RSA has been linked to increased negative emotionality and thus may indicate a lack of self-regulation, effects of which are seen in heightened risk behaviors (Beauchaine, 2001; Belsky & Pluess, 2009). This pathway may be further informed by extant links between emotion regulation and Theory of Mind (ToM; Frith & Frith, 1999), or the ability to attribute mental states to others in order to understand and predict their behavior. Deficits in ToM have been closely linked poor emotion regulation (Hudson & Jacques, 2014) and subsequent antisocial behaviors such as delinquency, aggression, and callous-unemotional traits due to an attenuated capacity for prosocial behaviors (Imuta et al., 2016). Therefore, children with low basal RSA are at a resting state that may increase their risk for development of maladaptive behaviors in adolescence, possibly via deficits in Theory of Mind (Hinnant & El-Sheikh, 2009; Quintana et al., 2012). Although nascent research has pointed to connections between physiological arousal and aspects of ToM (Gower & Crick, 2011; R. Zhang & Wang, 2020), more investigation is necessary to elucidate the nuances of these links.

In addition to elucidating nuances within autonomic functioning and its moderating context of the impact of parenting on youth adjustment, this study further highlights the theoretical and methodological importance of distinguishing externalizing subtypes as unique. As suggested by a developmental psychopathology perspective, environmental conditions can usher in dynamic developmental processes that eventuate in multiple developmental outcomes. This is expressed by the concept of multifinality and equifinality (Cicchetti & Rogosch, 1996). According to the principle of multifinality, individuals exposed to similar types of adversity may still exhibit varied outcomes due to the multi-level influences of the systems in which each individual operates. As such, harsh parenting has been shown to confer risk for aggression and delinquency for many, but certainly not all, children and adolescents. Variations in parasympathetic and sympathetic functioning, at rest and during acute stress, lead to differential outcomes despite the shared context of harsh parenting. Consequently, these findings serve as evidence for the framework of multifinality, in which shared context leads to an array of outcomes based on individual

differences. However, equifinality is also evidenced, in that varying patterns of autonomic functioning predicted similar outcomes of aggression and delinquency. Finally, our study supports the biological sensitivity to context (BSC) theory, in which patterns of high physiological activity and reactivity indicate greater sensitivity to environmental input, and thus greater vulnerability to the risks conferred by adversity.

Limitations

This study has several limitations that should be acknowledged. First, our limited sample size and power would be helped by replication among studies with increased sample sizes and similar design. Moreover, due to the circumscribed sample of low-income families and primarily mothers, our findings may not be generalizable across differing demographics and caregiver arrangements. However, it is necessary to study ethnic minorities and impoverished families to comprehensively study resilience, and thus this specificity of sample is also a strength. The study would also be better served to have an increased number of time points to study development of externalizing behaviors across adolescence. This limitation is mitigated by the study's employment of multiple reporters and data collection methods (parent report, physiological data, and coded video interaction) as well as two time-points that allow for longitudinal insights into development of externalizing behaviors.

This study extends literature on the functioning of the autonomic nervous system in the context of adolescent externalizing behavior. Pre-adolescence is a significant period in which both physiological and socioemotional development occurs, and these changes are salient contributors to externalizing behaviors in later adolescence and adulthood. Our findings corroborate and extend present study on the functioning of the autonomic nervous system in the development of problem behaviors. Moreover, our findings support current efforts to conceptually differentiate externalizing behaviors by implicating the parasympathetic and sympathetic branches as unique contributors to the long-term development of aggression and delinquency. Although our community sample limits a discussion of clinical implications, these findings may inform practitioners who directly treat children and adolescents with aggressive and delinquent behaviors.

CHAPTER 3

NEURAL SIGNATURES OF THE DEVELOPMENT OF ANTISOCIAL BEHAVIOURS AND CALLOUS-UNEMOTIONAL TRAITS AMONG YOUTH: THE MODERATING ROLE OF PARENTAL SUPPORT[1]

[1]Huffman, L. G., & Oshri, A. (2022). Neural signatures of the development of antisocial behaviours and callous□unemotional traits among youth: The moderating role of parental support. International Journal of Developmental Neuroscience, 82(3), 205-221. Reprinted here with permission of the publisher.

Abstract

Comorbidity of antisocial behaviors (AB) and callous-unemotional (CU) traits characterizes a subgroup of youth at risk for chronic and severe antisocial behavior in adulthood. Although aberrant neural response to facial emotion confers heightened risk for AB and CU traits, the behavioral effect of this neural response varies by family context. The present study examines the effects of neural response to emotional faces, parental support, and the interaction between the two as predictors of AB and CU traits in a longitudinal sample of preadolescents ($N_{baseline}$ = 11,883; M_{age} = 9.5; 47.8% female). Low CU youth who evinced attenuated response to fearful faces within the left superior temporal sulcus and fusiform gyrus showed smaller decreases of AB over time; these associations did not extend to high CU youth. Among high CU youth reporting low parental support, blunted response to fearful faces within the bilateral inferior parietal sulcus predicted smaller decreases of AB. Study findings highlight neurobehavioral differences between youth with high and low CU traits, as well as the interacting roles of negative face processing and parental support in the development of AB.

Introduction

Antisocial behaviors (AB) such as rule-breaking, aggression, conduct problems, and oppositional defiance confer significant financial costs to society and increased likelihood of adjustment problems in adulthood (Cohen & Piquero, 2009). Callous-unemotional (CU) traits characterized by lack of guilt, absence of empathy, and constricted emotions frequently co-occur with AB. This CU and AB comorbidity characterizes a severe subgroup of youth at elevated risk for even more chronic and severe antisocial behavior in adulthood (Frick et al., 2005; Frick & Marsee, 2018). Extant neuroimaging research has identified patterns of neural function in response to facial emotion that are distinctly correlated with AB and CU traits in youth (Ahmed et al., 2015; Gao et al., 2019; Johanson et al., 2020). However, these neural patterns seem to be significantly impacted by the rearing context of the child. For example, growing research in developmental neuroscience shows that family context moderates the risk of developing adjustment problems, particularly AB and CU traits during childhood and adolescence (Turpyn et al., 2021; Waller et al., 2013). Less is known, however, on the longitudinal interactions

between parenting and neural underpinnings of emotion processing that underlie development of AB/CU traits. Such a person-in-context (Deane et al., 2020; Whittle et al., 2016) examination is warranted, given that current knowledge of AB among youth is limited by a lack of examination of the parental context. Therefore, the present study aims to examine the moderating role of parental support in the connection between neural risk and behavioral indicators of AB at high and low levels of CU traits in a large, longitudinal sample of young adolescents.

Emotion Recognition, Antisocial Behavior, & Callous-Unemotional Traits

Youth with antisocial behavior (AB) problems often exhibit a diminished capacity for recognizing emotional cues in others (van Goozen, 2015; Schönenberg et al., 2016). These patterns of disrupted emotion processing are evident at the neural level, with a growing body of both neuroimaging and eye-tracking research among AB youth highlighting disrupted processing of emotion and emotional faces in key subcortical and cortical areas (Billeci et al., 2019; R. J. R. Blair, 2013; Dawel et al., 2012; Levantini et al., 2021). The amygdala, for example, is frequently implicated in the development of antisocial behaviors, given its role as a central hub of emotional processing and threat response. Disruptions in amygdalar processing through both *hypo*activation (Jones et al., 2009; Marsh et al., 2008; Rhoads et al., 2020; Viding et al., 2012; White, Marsh, et al., 2012) and *hyper*activation (Dotterer et al., 2017, 2020; Sebastian et al., 2014; Viding et al., 2012) are evident among youth with AB problems. Attenuated response to emotionally salient stimuli among AB youth has also been observed in other frontolimbic areas, such as the anterior cingulate (Stadler et al., 2007; Sterzer et al., 2005), orbitofrontal, ventromedial prefrontal, dorsolateral prefrontal, and medial prefrontal cortices, and insula (Fairchild et al., 2014; Fanti et al., 2018; O'Nions et al., 2014). Findings have also diverged based on presence of CU traits: diminished amygdala, prefrontal, and parietal responses to fearful and violent stimuli have been observed among those with high CU traits (Viding et al., 2012; White, Marsh, et al., 2012; White, Williams, et al., 2012) but not those with low CU traits (Dotterer et al., 2017, 2020; Sebastian et al., 2014; Viding et al., 2012). These patterns of blunted response among CU-presenting youth suggest that those

who present both AB and callous-unemotional (CU) traits may constitute a particularly severe subgroup characterized by attenuated neural response to emotion.

Despite this growing neuroimaging research on AB and CU traits, significant knowledge gaps remain, limiting generalizability of findings on the development of antisocial and CU traits in youth. Few neuroimaging investigations of emotion recognition and AB/CU traits have tested whether an altered neural response to emotion is specific to negative cues or extends as well to positive cues. This is a striking limitation, given not only the sensitivity of fronto-limbic regions to both negatively- and positively-valenced emotions, but also documented links between AB/CU traits and deficits across the emotion spectrum (Dawel et al., 2012; Dotterer et al., 2020). Moreover, only two relevant functional imaging studies have included community samples (see Table 3.1), despite community youth also evincing callous-unemotional traits and antisocial behaviors (Chabrol et al., 2012; Moran et al., 2009). Finally, despite ample evidence highlighting the family context as a critical developmental correlate of youth AB and CU traits, no fMRI studies to date have investigated the role of parenting processes in the prospective associations between neural substrates of emotion recognition and AB/CU trait development.

Table 3.1. Summary of fMRI studies of psychopathic and antisocial traits using emotion recognition paradigms, sorted by age ascending

Author(s), Date	ROI(s)	associated with behavioral type	Task condition	Behavioral type	Mean age	Male/Female	N	Clinical*
Jones et al., 2009	Right amygdala	Decreased	Fearful faces	Increased CP + CU	11	100% Male	17 CP/CU; 12 HC	Yes
Sterzer et al., 2005	Right dorsal ACC	Decreased	Negative pictures (IAPS)	Increased CD + Aggression	12.85	100% Male	13 CD; 14 HC	Yes
Stadler et al., 2007	Right ACC	Decreased	Negative pictures (IAPS)	Increased CD	12.85	100% Male	13 CD; 14 HC	Yes
Rhoads et al., 2020	Right amygdala	Decreased	Fearful faces (during "afraid for self" condition)	Increased CU	13.33	53.3% Male	30	Yes
Dotterer et al., 2017	Right amygdala	Increased	Angry faces	Increased AB (but not CU)	13.42	51.5% Male	220	No
Sebastian et al., 2013	Left amygdala	Increased	Fearful faces	Increased CP + Low CU	13.51	100% Male	17 High CU + CP, 17 Low CU +CP, 17 HC	Yes
Viding et al., 2012	Right amygdala	(1) Increased; (2) Decreased	Fearful faces	(1) Increased CP + Low CU; (2) Increased CP + High CU	13.72	100% Male	15 High CU + CP, 15 Low CU + CP, 16 HC	Yes
Marsh et al., 2008	Right amygdala	Decreased	Fearful faces	Increased CU	14.5	58.3% Male	12 CU + CD/ODD, 12 ADHD, 12 HC	Yes
Herpertz et al., 2008	Left amygdala	Increased	Negative pictures (IAPS)	Increased CD	14.7	100% Male	22 CD, 22 HC	Yes
Dotterer et al., 2020	Amygdala	Increased	All emotional faces	Increased AB + Low CU	15	65% Male	165	No
White et al., 2012	Superior parietal lobule, inferior parietal sulcus	Decreased	Fearful faces	Increased DBD + PT	15.51	76.5% Male	17 DBD + PT, 19 HC	Yes
White et al., 2012	Left amygdala	Decreased	Fearful faces (during decreased attentional load)	Increased DBD + PT	15.67	80% Male	15 DBD + PT, 17 HC	Yes
O'Nions et al., 2014	Bilateral OFC, right vmPFC, left insula, bilateral amygdala	Decreased	Angry faces	Increased CD	17.1-17.7	100% Male	27 Early-onset CD, 25 Adolescent-onset CD, 23 HC	Yes
Fairchild et al., 2008	Amygdala, superior temporal cortex, fusiform gyrus, dorsolateral PFC	Decreased	All emotional faces	Increased lifetime CD	17.62	100% Female	20 CD, 20 HC	Yes
Fanti et al., 2018	Medial PFC	Decreased	Violent stimuli	Increased CU + CP	19.92	50% Male	88	No
Decety et al., 2014	(1) Inferior occipital gyrus, fusiform gyrus, superior temporal sulcus, inferior frontal gyrus, orbitofrontal cortex; (2) dorsal insula	(1) Decreased; (2) Increased	(1) All emotional faces; (2) Negative emotional faces	Increased PT	18-50**	100% Male	80	Incarcerated

Note. CU = callous-unemotional; CP = conduct problems; CD = conduct disorder; AB = antisocial behavior; DBD = disruptive behavior disorder (e.g., CU + ADHD + oppositional defiance); PT = psychopathic traits; HC = healthy controls; PFC = prefrontal cortex. Mean age and % male columns are reported based on psychopathic/antisocial traits group for matched control studies.
*Indicates whether psychopathic traits of sample or portion of sample were above clinical levels. **No mean age provided.

Protective Effect of Positive Parenting on Risk for DBD & CU Traits

During childhood and the transition to adolescence, parenting behaviors are critical in moderating risk for maladaptive developmental trajectories among youth, above and beyond genetic influences (Burt et al., 2021). A large body of research has identified the salient contributions of negative parenting

behaviors, such as harshness, to the development of both AB and CU traits among youth (Burnette et al., 2012; Carlson et al., 2015; Duncombe et al., 2012; Oshri et al., 2020). Conversely, recent studies suggest that warm and accepting parenting behaviors (hereby known as parental support) may mitigate risk for development of AB and CU traits and even attenuate the effect of neurobiological vulnerabilities underlying AB/CU traits (Fontaine et al., 2011; Muratori et al., 2016; Pardini et al., 2007; Waller et al., 2013, 2014, 2018). However, no studies have examined the moderating role of supportive parenting in the link between neurocognitive underpinnings of AB/CU traits in youth (such as those evinced by neural function during emotion recognition) and AB/CU trait development. Consideration of the parenting context in neurocognitive risk for AB/CU trait development is key to understanding ways in which supportive parenting can promote resilience among youth exhibiting risk for AB and CU traits.

The Present Study and Hypotheses

The present longitudinal study employs multilevel methods to investigate (1) associations between neural substrates of emotion recognition and development of antisocial behavior at high and low levels of CU traits, and (2) the moderating role of parental support in the link between neural emotion recognition and AB development at high and low levels of CU traits. The analyses are bolstered by a large prospective sample of children drawn from the Adolescent Brain Cognitive Development (ABCD) study and the use of both biological and behavioral data. Our hypotheses and choice of *a priori* ROIs were informed by imaging research among youth with antisocial behaviors (labeled as antisocial behavior, conduct problems, conduct disorder, disruptive behaviors, and psychopathic traits) and/or CU traits. We also considered the roles of 1) baseline household income and parent education, 2) child biological sex, 3) child age, and 4) co-occurring ADHD and anxiety disorders as potential covariates of AB development. These considerations were founded upon extant research suggesting that 1) youth from low socioeconomic status backgrounds show increased incidence and severity of AB (Piotrowska et al., 2015), 2) incidence and symptom severity of AB is greater in males than females (Odgers et al., 2008), 3) significant heterogeneity in AB is observed across late childhood and early adolescence (Fairchild et al., 2013), and 4) AB, ADHD, and anxiety disorders not only overlap clinically, but also are frequently co-

occurring among youth (Rubia et al., 2009; Storebø & Simonsen, 2016). We hypothesized that during both happy and fearful conditions of an implicit emotion processing task, heightened amygdala activation among the low CU group would predict greater AB from baseline to 24 months from baseline (T5). In contrast, we expected that diminished amygdala activation among the high CU group would predict increased AB. We also hypothesized that during both happy- and fearful-face emotion processing, diminished activation within frontal (medial and lateral orbitofrontal cortex, caudal and rostral middle frontal gyrus, caudal and rostral anterior cingulate cortex, inferior frontal gyrus, insula), visual (superior temporal sulcus, fusiform gyrus, lateral occipital gyrus, superior parietal sulcus), and attention-orienting regions (superior parietal lobule, inferior parietal sulcus) would predict increased AB traits from baseline to T5. Finally, we hypothesized that low parental support would exacerbate risk for increased AB, and high parental support would mitigate risk for increased AB, as conferred by aberrant ROI response to emotion, across both groups.

Methods

Participants

Study participants are enrolled in the national, longitudinal Adolescent Brain Cognitive Development (ABCD) study (https://abcdstudy.org) and included in the annual 4.0 data release (nda.nih.gov/study.html?id=901). The ABCD study recruited 11,883 healthy children (47.8% female) born between 2005 and 2008 (ages 8 to 10) to be studied into adulthood. Imaging, behavioral, and demographic data were collected at 21 sites, with sampling approaches intended to approximate national sociodemographic distributions. Institutional review boards at participating universities approved all study procedures, and youth participants and their legal guardians provided written assent and consent, respectively, for participation. Full information on recruitment and study design may be found in Garavan et al. (Garavan et al., 2018). Data for the current study included waves 1 (N = 11,876) and 5 (24 months after baseline, N = 10,414).

Measures

Antisocial behaviors. Antisocial behaviors were assessed by combining multiple indicators from two different parent-reported measures: (a) rule-breaking and (b) aggression from the Child Behavior Checklist (Achenbach, 1983), (c) combined symptom count (e.g., present symptoms) of the Diagnostic & Statistical Manual of Mental Disorders (DSM-5) conduct disorder and (d) oppositional defiant disorder, derived from computerized assessments using the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman & Schweder, 2004). Present symptoms included those that participants endorsed in the six months preceding the study visit. Using CFA, we created corresponding latent factors for both baseline and T5 using maximum likelihood estimation with robust standard errors.

CU traits. CU traits were quantified for baseline and T5 using a measure developed by Hawes et al. (2019) and validated using ABCD baseline data. Four items were measured via parent report: 1 item from the CBCL (Achenbach & Edelbrock, 1991; "doesn't seem to feel guilty after misbehaving") and 3 items from the Strengths and Difficulties Questionnaire (Goodman & Scott, 1999; "is considerate of others' feelings"; "is helpful if someone is hurt or upset"; "offers to help others"). Cronbach's alpha for both baseline and T5 indicators was acceptable (respectively: $\alpha = .746$; $\alpha = .766$). We also derived maximum a posteriori (MAP) scale scores, which provided person-specific CU trait factor scores accounting for variation in significant demographic covariates.

CU traits group classification. We divided the sample into two groups based on CU trait severity. Per methods employed previously in the ABCD sample (Waller et al., 2020), the High CU traits group had scores greater than or equal to 4 on the summed CU traits measures and CU MAP scores in the 90^{th} percentile ($n_{baseline} = 720$). In order to retain as much heterogeneity within the sample as possible, the Low CU traits group included all scores below the High CU traits group threshold ($n_{baseline} = 11{,}156$).

Parental support. Parental support at baseline was measured using the Children's Report of Parent Behavior Inventory support subscale (Schaefer, 1965). This subscale is the mean of 5 items, such as "makes me feel better after talking over my worries with him/her" and "believes in showing his/her love for me." Cronbach's alpha was acceptable ($\alpha = .71$).

Imaging Measures

EN-back task. The EN-back is an implicit facial emotion and memory processing task eliciting short-term memory processes while presenting equal numbers of happy, fearful, and neutral facial expressions, as well as places, in each run (Barch et al., 2013). This widely-used paradigm is known to elicit activity within frontal-amygdalar regions underlying emotion recognition and reactivity (Gee et al., 2013). When presented with a happy, fearful, or neutral face, or a place, participants were asked to respond as to whether the picture presented is a "Match" or "No Match." Conditions alternated between 0-back and 2-back conditions, for which youth were instructed to respond "Match" when the current stimulus corresponded to that presented at the beginning of the block or two trials back, respectively. Participants completed two runs of the task, with eight blocks per run and 10 trials (2.5 seconds each) per block. The current study employed two task conditions: *fearful faces vs. neutral faces* and *happy faces vs. neutral faces,* which were modeled to remove the effects of working memory and isolate effects of emotion processing. The contrast of happy with neutral faces and fearful with neutral faces allows for assessment of neural activation specific to the emotional stimulus (O'Hare et al., 2008).

Image preprocessing and calculation region-of-interest data. Preprocessing and analysis of MRI data was completed by the ABCD Data Analysis and Informatics Center and is outlined elsewhere (Casey et al., 2018; Hagler et al., 2019; see also Supplement). In short, parcellated cortical regions used in the study analyses were derived from Desikan atlas cortical surface reconstruction and subcortical segmentation performed in FreeSurfer 5.3.0 (Desikan et al., 2006). Estimates of task-related activation strength were computed for each individual using general linear modeling (GLM) in AFNI's 3dDeconvolve and released as contrast beta weights. The present study uses GLM beta coefficients averaged across both runs.

Covariates. Baseline measures of child age, biological sex, parent reported household income, and parent education were included as covariates in all analyses. All insignificant covariates were trimmed per each analysis. To account for potential confounding effects of co-occurring ADHD and

anxiety (Storebø & Simonsen, 2016), all analyses were repeated with CBCL DSM-5 ADHD and anxiety problem scores as a covariate.

Analysis Plan

Structural equation modeling in Mplus version 8.1 (Muthén & Muthén, 2014) was used to test all study hypotheses. Univariate latent change score (LCS) modeling with multiple indicators was used to model change in AB traits from baseline to T5 while taking into account within-person variability (McArdle & Hamagami, 2001). We then used the resulting LCS model to test direct effects of ROI activation and demographic covariates on latent change of AB (ΔAB). Second, we modeled interaction effects of ROI activation and parental support on ΔAB traits, adding demographic and psychiatric covariates in a stepwise manner (see Supplement for full results). Separate models were run for each ROI. Finally, we conducted exploratory analyses testing the direct effects of parental support, ROI activation, and demographic covariates on continuous CU traits within the full sample (results are reported in Supplement). All models were estimated using maximum likelihood with robust standard errors (MLR; Klein & Moosbrugger, 2000). To control for increased risk of type I error resulting from multiple statistical comparisons, all p-values were corrected using the Benjamini-Hochberg method (Benjamini & Hochberg, 1995). Multilevel modeling was used to account for clustering effects of participants within families and sites. Additionally, propensity weights were employed, calibrating ABCD distributions to nationally representative controls from the American Community Survey to mitigate potential selection bias in the ABCD sampling and recruitment process (Heeringa & Berglund, 2020).

Results

Descriptive Statistics

Descriptive statistics of demographic and behavioral variables are included in Tables 3.2 and 3.3. Separate equality tests (Table 3.3) indicated that low and high CU groups significantly differed based on AB behavioral indicators, parental support, and all demographic variables excepting age. Bivariate correlation analyses were conducted among all baseline environmental (e.g., parenting and demographic) and behavioral (e.g., CU and AB) variables (Figure 3.1). Parental support exhibited modest negative

correlations with total CU traits and all antisocial behaviors, as well as household income, youth biological sex, and parental education.

Table 3.2. Demographic information of full sample

Race/Ethnicity	Frequency	Percent
White	6182	52.04
Black/African American	1785	15.03
Hispanic	2411	20.30
Asian	245	2.14
Other	1247	10.50
Highest Education		
< HS Diploma	593	5.00
HS Diploma/GED	1132	9.54
Some college	3079	25.96
Bachelor	3015	25.42
Post-Graduate Degree	4043	34.08
Income		
< $5,000	417	3.84
$5,000 - 11,999	421	3.88
$12,000 - 15,999	273	2.51
$16,000 - 24,999	524	4.83
$25,000 - 34,999	654	6.02
$35,000 - 49,999	934	8.60
$50,000 - 74,999	1499	13.81
$75,000 - 99,999	1572	14.48
$100,000 - 199,999	3314	30.52
> $200,000	1250	11.51

Table 3.3. Descriptive statistics & equality tests between groups

	Low CU	High CU	Test Statistic	*p*
Age	M = 118.98	M = 119.05	t = -0.264	0.792
Aggression	M = 52.37	M = 59.99	t = -20.82	0.000
Rule Breaking	M = 52.37	M = 59.13	t = -23.04	0.000
ODD	M = 0.45	M = 2.15	t = -14.45	0.000
CD	M = 0.157	M = 0.84	t = -12.9	0.000
Parental support	M = 2.79	M = 2.68	t = 7.34	0.000
Household income	70.9% *	61.5% *	χ^2 = 59.84	0.000

Biological Sex	51.2% **	67% *	$\chi^2 = 67.66$	0.000
Parent education	59.89% ***	53.47% ***	$\chi^2 = 33.48$	0.000

Note. Low CU n = 11,156. High CU n = 720. *Percent earning above $50,000 annually. **Percent male. ***Percent with bachelor's or post-graduate degrees.

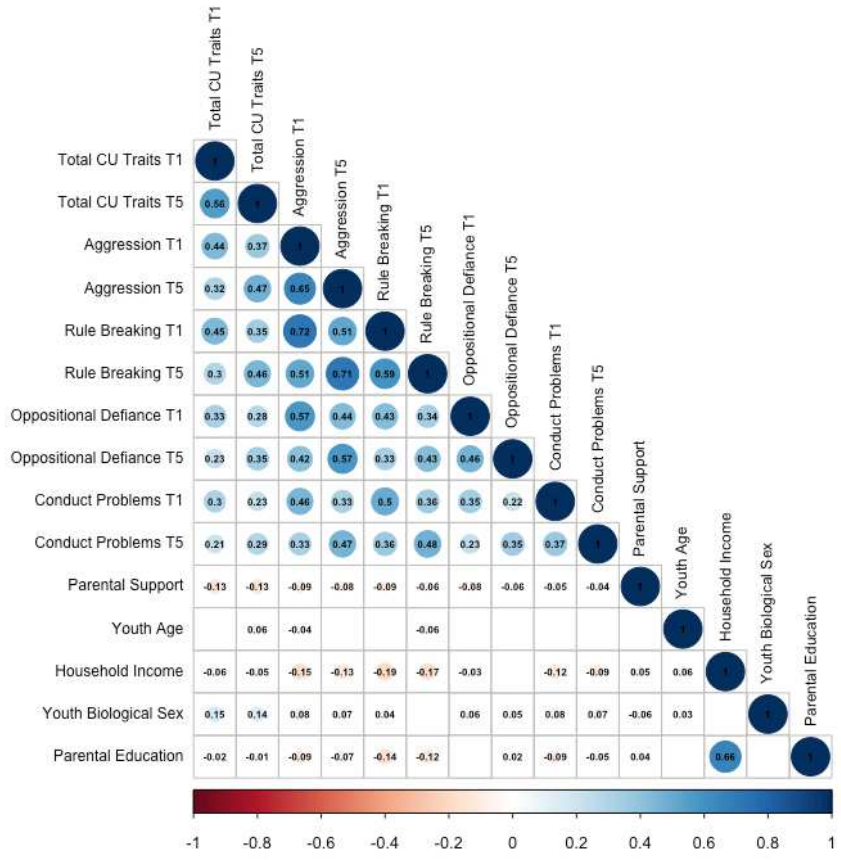

Figure 3.1. Bivariate correlations between demographic covariates, parental support, CU traits, and antisocial behaviors.

Measurement Model and LCS of AB Traits

Four separate but corresponding CFAs of AB were conducted: within the Low CU group at T1 and T5 and within the High CU group at T1 and T5 (model fit was acceptable; see Supplement). Subsequently, a latent change score of T1 and T5 AB factors was modeled within both the High CU and Low CU groups, yielding acceptable model fit (Figures 2 and 3). For both Low and High CU groups, the mean of the difference score was negative and significant at a .05 alpha, indicating an overall decrease in

AB trait severity from T1 to T5. The decrease within the High CU group was more pronounced than that of the Low CU group (-3.143 versus -0.241, respectively).

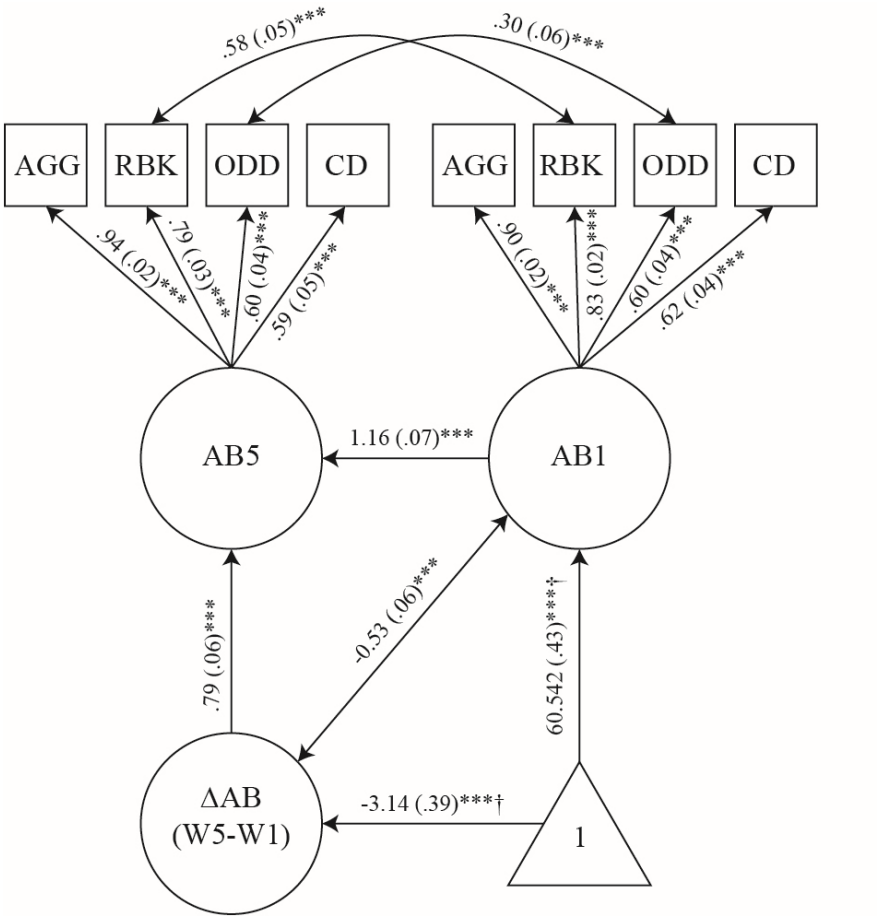

Figure 3.2. Latent change score model of AB within the High CU group. Model fit was acceptable: $\chi 2$ (17) = 67.99, p = .000; RMSEA = 0.07; CFI = 0.96; SRMR = 0.05. AB1 = antisocial behaviors at time 1, AB2 = antisocial behaviors at time 2, AGG = aggression, RBK = rule-breaking, ODD = oppositional defiance disorder, CD = conduct disorder. † For ease of interpretation, mean values of the latent difference score and AB1 are unstandardized. Standardized values are, respectively: -0.52 (.06)***, 6.75 (.27)***.

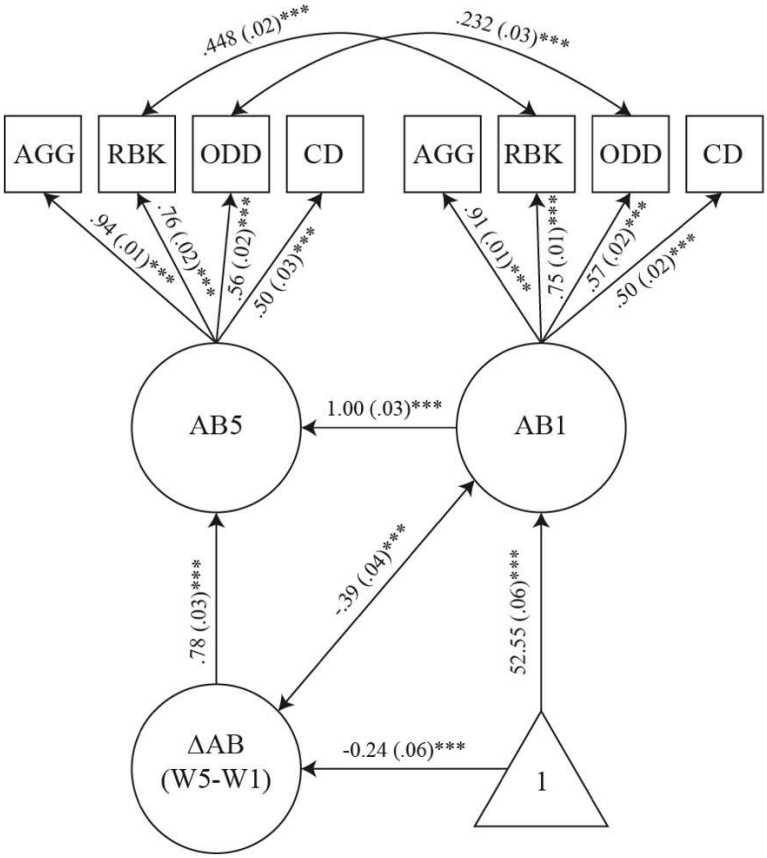

Figure 3.3. Latent change score model of AB within the Low CU group. Model fit was acceptable: $\chi 2$ (17) = 369.88, p < .000; RMSEA = 0.04; CFI = 0.94; SRMR = 0.042. AB1 = antisocial behaviors at time 1, AB2 = antisocial behaviors at time 2, AGG = aggression, RBK = rule-breaking, ODD = oppositional defiance disorder, CD = conduct disorder. † For ease of interpretation, mean values of the latent difference score and AB1 are unstandardized. Standardized values are, respectively: -0.07 (.02)***, 11.56 (.28)***.

Direct Effects

Results are indicated in Table 3.4. Within the Low CU group, activation of the right pars triangularis, left superior temporal sulcus, and left fusiform gyrus significantly predicted ΔAB. Specifically, attenuated response to fearful faces within the left STS and fusiform gyrus was associated with smaller decreases in AB from T1 to T5. These effects remained significant when controlling for child biological sex, age, and income, ADHD, and anxiety disorders (see Supplement). Within the High

CU group, no statistically significant associations were found between ROI response and ΔAB after correcting for multiple comparisons. Additionally, no significant direct effects were found between parental support and ΔAB within either group. Statistically significant results prior to correcting for multiple comparisons are reported in the Supplement.

Moderation Analyses

Moderation analyses were then conducted to determine how parental support interacted with ROI activation to both fearful and happy faces to predict ΔAB (Table 3.4). Within the Low CU group, no significant moderating effects of parental support were found after correcting for multiple comparisons. Within the High CU group, parental support significantly moderated the effect of response to fearful faces within the bilateral inferior parietal sulcus (IPS). These effects remained significant when controlling for effects of child age, biological sex, household income, anxiety disorders, and ADHD.

Post-hoc probing of the interaction terms were then conducted using both Johnson-Neyman and Dawson plots (Table 3.5, Figures 3.4 and 3.5). First, we employed the Johnson-Neyman technique to visualize the effect of IPS activation on change in AB at all values of the moderator (e.g., parental support) and thus determine the proportion of the sample for which the effect of predictors on change in AB was significant. Next, we used Dawson plots and simple slopes analysis to determine whether the slopes of each predictor were significantly different from one another at low, medium, and high values of the moderator. Given that the distribution of parental support within the sample skewed left (M = 2.7, minimum =1, maximum = 3), we identified low and medium parental support at -1 SD and -2 SD, respectively, and identified high parental support at the maximum value of 3, which was reported by approximately half of the sample (50.02%, n = 5,923). Results of these analyses converged to show that among high CU youth, low to moderately low parental support moderated the effects of the bilateral IPS response to fearful faces. Among those reporting low parental support, elevated IPS activation predicted greater decreases in AB, while attenuated ROI activation predicated smaller decreases in AB. Moderating effects were no longer statistically significant among those reporting high levels of parental support.

Table 3.4. Direct and moderation effects

	Direct effects								
	b	*SE*	*p*	*β*	*SE*	*p*	Adjusted *p*	*95% CI*	R^2
Support (Full sample)	-0.055	0.193	0.777	-0.005	0.017	0.777	*	[-.032, .023]	*
Support (High CU)	0.729	0.757	0.336	0.053	0.055	0.334	*	[-.037, .143]	*
Support (Low CU)	-0.065	0.206	0.754	-0.006	0.019	0.754	*	[-.038, .026]	*
Left superior temporal sulcus (Low CU)	-0.541	0.198	0.006	-0.05	0.018	0.006	0.048	[-.08, -.02]	0.088
Left fusiform gyrus (Low CU)	-0.481	0.195	0.014	-0.062	0.025	0.014	0.056	[-.103, -.021]	0.088
	Moderation Effects (High CU)								
Right inferior parietal sulcus	9.762	4.374	0.026	0.205	0.092	0.026	0.016	[.053, .357]	0.289
Left inferior parietal sulcus	13.636	4.563	0.003	0.262	0.091	0.004	0.052	[.112, .412]	0.309

Note. All models control for effects of ADHD and anxiety disorders. Non-significant demographic covariates were trimmed from the models.

Table 3.5. Simple slopes analysis

	Low (M-2SD = 1.90)			Moderate (M-1SD = 2.28)			Mean (2.66)			High (Max = 3)		
Fearful vs. Neutral, HCU	B	SE	p	B	SE	p	B	SE	p	B	SE	p
R inferior parietal sulcus	-8.925	3.583	0.013	-5.215	2.187	0.017	-1.506	1.5	0.315	1.814	2.161	0.401
L inferior parietal sulcus	-12.085	3.663	0.001	-6.903	2.231	0.002	-1.722	1.599	0.282	2.914	2.343	0.214

Note. Shaded areas indicate statistical significance at alpha of .05.

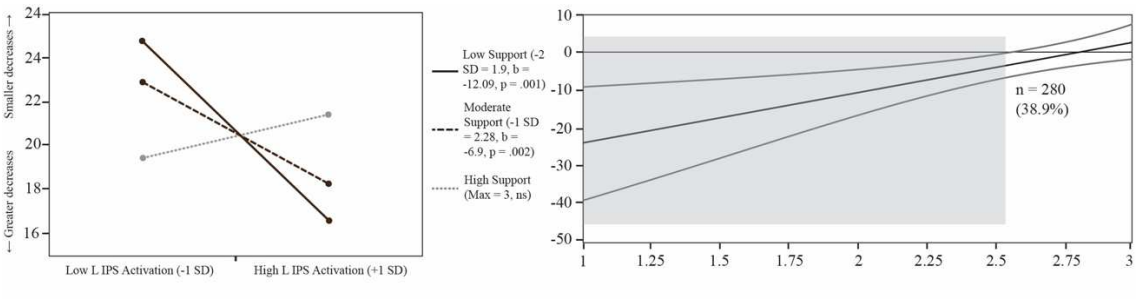

Figure 3.4. Probing of moderation of left IPS response on AB by parenting among youth with high CU traits. The x-axis of the Johnson-Neyman plot (right) represents parental support, and the y-axis represents effect size of the IPS*parenting interaction term on change in AB. Black and grey slope lines within Dawson plots indicate significant and non-significant slopes, respectively. Each black line within Johnson-Neyman plot represents the main interaction effect, and the grey outer lines represent the confidence interval of the effect.

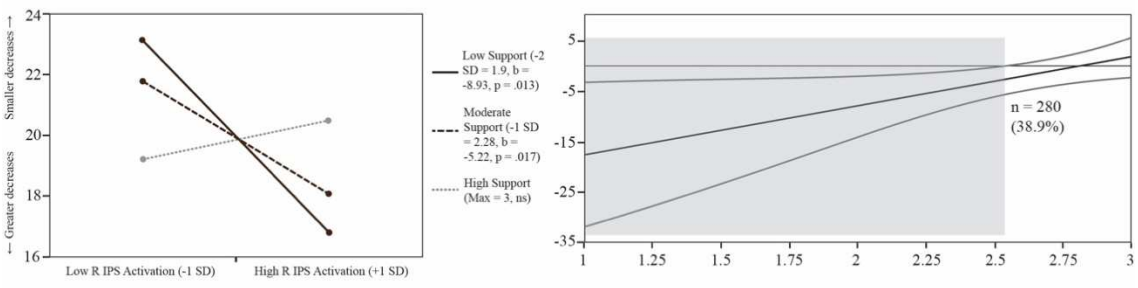

Figure 3.5. Probing of moderation of right IPS response on AB by parenting among youth with high CU traits. The x-axis of the Johnson-Neyman plot (right) represents parental support, and the y-axis represents effect size of the IPS*parenting interaction term on change in AB. Black and grey slope lines within Dawson plots indicate significant and non-significant slopes, respectively. Each black line within Johnson-Neyman plot represents the main interaction effect, and the grey outer lines represent the confidence interval of the effect.

Discussion

The development of AB in children and youth is a potent risk factor for problem behaviors across the life span, especially among those with elevated CU traits (Frick et al., 2005; McMahon et al., 2010; Viding & Kimonis, 2018). Although functional imaging studies have documented various neurocognitive

contributors to AB and CU traits, behavioral data suggest that the family environment may be a salient context for AB/CU development in youth. From an ecological neuroscience perspective (van Dijk & Myin, 2019), neural risk for maladjustment is embedded in the family context. Informed by this perspective, findings from the current study suggest that AB differences in youth with high CU traits may stem not only from neurocognitive response to emotion and the presence of CU traits, but also the parenting environment.

Our findings revealed divergent longitudinal associations between neural function and behavior among low and high CU trait youth. Low CU youth who exhibited attenuated left superior temporal sulcus (STS) and left fusiform gyrus response to fearful faces showed smaller decreases in AB. These findings correspond to those of Decety et al. (2014), who found that diminished response of the fusiform gyrus, STS, and inferior frontal gyrus (IFG, a region encompassing the pars triangularis) to emotional faces predicted increased psychopathic traits among incarcerated males ages 18 to 50. Fairchild et al. (2014) also found that decreased STC and fusiform gyrus response to emotional faces was linked to increased lifetime conduct problems among 20 CD-diagnosed females. The fusiform gyrus and STS are members of the core face processing network, processing (respectively) unchangeable elements of the face and more variable aspects such as gaze and expression (Kanwisher & Moscovitch, 2000). These regions are known to be 1) consistently activated by emotional faces in normatively-developing youth and adults, and 2) less active among those evincing antisocial and psychopathic traits. Importantly, STS and fusiform gyrus responses to positive faces were not predictive of AB, indicating a particular salience of negative emotion recognition deficits in the development of AB. Moroever, these findings did not extend to youth evincing high CU traits. Whereas lack of attendance to negative facial stimuli may confer greater risk for AB among youth evincing low to moderate CU traits, our findings suggest that neural response to emotional faces is not predictive of risk for AB among high CU youth. Taken together, these direct effects reveal evidence of contrasting neurocognitive vulnerabilities for AB between low and high CU youth.

Research suggests that neural risk for antisocial behavior among youth is moderated by the family context (Waller et al., 2013). The present study builds upon this literature, suggesting that among youth with high CU traits, parenting interacts with child neurocognitive characteristics to influence development of AB. High CU youth response to fearful faces within the bilateral IPS predicted greater decreases in AB over time, but only among those reporting low to moderately low parental support. Conversely, attenuated response of these regions predicted smaller decreases in AB over time and thus greater longitudinal risk for AB. The IPS is crucial in attending to and registering the salience of facial expressions (Corbetta & Shulman, 2002; Decety et al., 2014; Dotterer et al., 2020), which in turn is a central component of interpreting others' emotional states and developing empathy and prosociality. Our findings indicate that although IPS response to fearful faces alone is not predictive of behavioral adjustment, it is a salient risk factor for antisocial traits in the context of a challenging parental environment. Similar associations were found by Levantini et al. (Levantini et al., 2021) using eye-tracking technology: among boys (ages 7-12) with Disruptive Behavior Disorder, gaze pattern impairments to negative emotional faces were associated with greater CU traits only among those reporting negative parenting. Youth with high CU traits who develop in the context of negative or harsh parenting are already at increased risk for maladaptation (Waller et al., 2013, 2018; Whittle et al., 2016). Our findings indicate that this risk is further exacerbated if they are less able to attend to negative emotional cues. It may be that this lack of neurocognitive attendance to and salience of negative emotional stimuli increases parent-child conflict, which leads to further parental harshness and youth AB (D. J. Hawes et al., 2011; Trentacosta et al., 2019; Waller et al., 2014). Conversely, however, high-CU youth who evince greater ability to attend to negative emotional cues may be more resilient to the negative parental context. As discussed by Levantini et al. (2021), youth who experience harsh parenting but may still attend to emotional cues by seeking out supportive social networks outside of their caregivers, such as those among peers, teachers, and other relatives.

Contrary to our hypothesis and extant studies among non-clinical samples (Dotterer et al., 2017, 2020), amygdala response to emotional faces did not predict change in AB among youth with low or high CU traits, neither as a direct predictor nor when modeled in conjunction with parental support. Although a

number of studies have identified aberrant amygdala function as a salient predictor of AB, findings are mixed, with many studies failing to reproduce these results (Decety et al., 2014; Fanti et al., 2018; Stadler et al., 2007; Sterzer et al., 2005; White, Williams, et al., 2012). This result may be due to the strong recruitment of the amygdala during emotional face processing within the sample and consequent low variability across participants. It also speaks to the role of the amygdala as an "intersection" of emotion processing, rather than a "dead end." Although the amygdala certainly plays a significant role in emotion processing, its function moves far beyond simply representing the level of emotion expressed. Amygdala activity reflects the up-regulation required to evaluate a stimulus' motivational relevance and direct cognitive resources in accordance with that evaluation (Adolphs, 2010; Cunningham & Brosch, 2012; Dixon & Dweck, 2021). Our findings support this evaluative view of the amygdala. Whereas high and low CU youth exhibited significantly different levels of AB, it was the function of regions underlying attendance to and recognition of faces that perpetuated AB risk.

The present study has several limitations. Given that all analyses were conducted within a community sample, results may not generalize to other populations, especially those with clinical levels of antisocial or psychopathic traits. Accordingly, the study did not include a diagnostic measure of CU traits; however, the CU trait measure showed sufficient reliability and validity within the current sample, as indicated by prior studies (S. W. Hawes et al., 2019; Waller et al., 2020). Although the CU measure was comprised only of parent report, previous studies suggest that children may be less accurate as informants for disruptive behavior problems (Loeber et al., 1991), and parent reports of CU traits outperform youth and teacher reports (Docherty et al., 2017; Gao & Zhang, 2016). Additionally, only one dimension of parenting was included; follow-up studies are necessary to determine whether results hold for other dimensions of parenting, such as harsh parenting. The study was also limited by use of only happy and fearful face stimuli. Further investigation is necessary to determine whether current findings extend to sad and angry faces, which may also elicit disrupted processing among youth with CU traits as well (Billeci et al., 2019; R. J. R. Blair, 2001). Finally, the current study is based on two waves of neuroimaging data collection. Although this allowed us to explore change in CU traits over the course of

2 years, additional waves of data collection are necessary to probe the development of CU traits throughout adolescence using several time points. Despite these limitations, the present study highlights specific brain-by-environment mechanisms that may underlie differences in AB/CU trait severity, suggesting that variations in supportive parenting confer both risk and resilience among youth exhibiting neurocognitive risk for AB and CU traits. These findings hold significant translational significance for parenting-based behavioral interventions among highly callous and unemotional youth at risk for antisocial behaviors. Whereas previous studies have suggested lack of malleability to parental inputs among high-CU youth, our findings indicate that high CU youth may be particularly susceptible to the absence of parental warmth. As such, efficacy of prevention and intervention programs for youth with CU traits and AB problems may be improved if they included assessments of both CU trait severity and parental support as well as subsequent training for caregivers aimed at increasing supportive and warm behaviors.

CHAPTER 4

CONTINUITY VERSUS CHANGE IN NEURAL PROFILES OF EMOTION REGULATION AND WORKING MEMORY DURING ADOLESCENCE[1]

[1]Huffman, L. G., & Oshri, A. Continuity versus change in neural profiles of emotion processing and working memory during adolescence. Under revision at *Developmental Cognitive Neuroscience, 10/17/22*.

Abstract

Significant structural and functional brain development occurs during late childhood and adolescence. These changes underlie developments in central neurocognitive processes such as working memory (WM) and emotion regulation (ER). The preponderance of studies modeling trajectories of adolescent brain development use variable-centered approaches that focus on mean level change, omitting attention to individual differences. This is a striking limitation, given that between- and within-person variability may undergird neurobiological embedding of early life stress and attendant psychopathology. This preregistered, data-driven study used latent transition analysis (LTA) to identify 1) latent profiles of neural function during a WM and implicit ER task, 2) changes in profile membership (e.g., latent statuses) across 24 months, and 3) associations between latent statuses, parental support, and subsequent psychopathology. Using two waves of data from the ABCD Study (Mage T1 = 10; Mage T2 = 12), we found three unique profiles of neural function within *a priori* defined ROIs at both T1 and T2, which were differentiated primarily based on neural function underlying ER. The Typical, Emotion Hypo-response, and Emotion-Hyper response profiles were characterized by, respectively: moderate amygdala activation and fusiform deactivation; high ACC, fusiform, and insula deactivation; and high amygdala, ACC, and insula activation during the *faces vs. places* condition. Most individuals (69.5%) remained in the Typical profile from T1 to T2; however, nearly 10% transitioned from the Typical to the Hypo-response profile, and 7.3% transitioned from the Hyper-response to the Typical profile. No significant associations were found between parental support and likelihood of class membership. Although youth within the Typical profile evinced better working memory performance than those in the atypical profiles, they did not differ in rates of internalizing and externalizing symptoms.

Introduction

Throughout late childhood and adolescence, emotion regulation (ER) and working memory (WM) undergo significant changes, matched by underlying structural and functional developments in the brain. Although developmental science has documented normative adolescent brain development trajectories, the focus on average change may obscure inter-individual differences and intra-individual

change in brain development. Indeed, adolescents' brain development varies (Foulkes & Blakemore, 2018), particularly in response to risky and promotive environmental inputs. For example, positive and supportive parenting behaviors are critical in shaping children's neurocognitive development and resulting WM and ER capacity (Borelli et al., 2021; J. E. Clark & Frick, 2018; Deane et al., 2020; Oshri et al., 2021; Schroeder & Kelley, 2010; Whittle et al., 2016). Moreover, variability of brain development underlies neurobiological vulnerabilities and attendant risk for the development of psychopathology (Beauchaine & McNulty, 2013). This preregistered, data-driven study aimed to: 1) derive latent profiles of neural function during working memory and implicit emotion processing task in a priori ROIs, 2) identify latent statuses of neural function across 24 months (M_{age}, baseline = 11, M_{age}, T2 = 13), and 3) explore parental support and demographic covariates as predictors of statuses, and 4) evaluate between-status differences in the development of psychopathology.

Working Memory and Emotion Regulation during Adolescence

Adolescence comprises a period of significant neurocognitive growth. Early maturation of motivation and reward circuitry (e.g., ventral striatum, medial frontal and orbitofrontal cortices) paired with more protracted maturation of cognitive control systems (e.g., lateral prefrontal, parietal, and anterior cingulate cortices) precipitates greater attendance to and salience of emotional information (Casey et al., 2008; Pfeifer & Allen, 2012; Shulman et al., 2016; Steinberg, 2017). However, these neurocognitive developments also underlie gradual increases in adolescents' capacity for EF, or goal-directed control of thought and behavior, and ER, or modulation of emotional reactions in order to accomplish goals. Despite these increases in EF and ER, a maturational imbalance can occur between quickly-developing motivational systems and slowly-developing EF/ER systems in adolescence. This imbalance, in turn, can predispose adolescents to inconsistent behavioral regulation and heightened vulnerability for psychopathology (Carlson & Zelazo, 2011; Fuhrmann et al., 2015; Luna et al., 2010).

Growing evidence suggests increasing differentiation of EF components by late childhood and early adolescence, necessitating research among adolescents that focuses on specific EF measures. Above and beyond other EF components, working memory (WM)—the process of maintaining and manipulating

information for a short period of time in order to guide behavior (Baddeley, 1998)—is a central mechanism underlying self-regulation and consequent adaptation throughout adolescence (Huang-Pollock et al., 2017; Vuontela et al., 2013). However, day-to-day task demands rarely require "cold" WM, or that which occurs in isolation from emotion processing (Banich, 2009; Blair et al., 2007; Pessoa, 2008; Pessoa & Ungerleider, 2004). Given that emotion receives priority in neural processing of stimuli (Pessoa & Ungerleider, 2004), ER is critical to successful WM and overall EF (Banich et al., 2009; Levens & Phelps, 2008; Mikels et al., 2008). Although ER takes many forms, it can be generally categorized into implicit ER, which involves passive, automatic, and often unconscious processing of emotional information, and explicit ER, which involves conscious cognitive effort aimed at modifying the emotional response (Gyurak et al., 2011).

Paradigms such as the emotional N-back (EN-back) are designed to elicit brain function at the intersection of implicit ER and WM. The EN-Back task presents emotionally-salient stimuli (typically emotional faces) while prompting the subject to hold information in an active cognitive state for use in a working memory task (Rougier et al., 2005). Given that the EN-back requires management of automatic emotional responses at varying degrees of working memory load, it is also considered an implicit ER task. The confluence of WM and ER processes elicits increases in activity across multiple functional domains. EN-back studies in adolescence and adulthood reveal working memory-related activations within frontoparietal regions (e.g., middle and superior frontal gyrus, inferior parietal cortex) and deactivations in motivation-oriented regions such as the cingulate cortex and insula (Chaarani et al., 2021; Liu et al., 2021; Rosenberg et al., 2020; Vetter et al., 2017). Implicit emotion processing tasks such as the EN-back recruit areas underlying visual and somatosensory processing (primary and secondary somatosensory cortices, insula, supramarginal gyrus, and basal ganglia), executive control (medial orbitofrontal cortex, superior frontal gyrus, anterior cingulate, posterior cingulate, precuneus, and superior temporal sulcus), valuation and motivation (vmPFC, striatum), and memory (hippocampus, amygdala; Chaaya et al., 2018; Frank et al., 2014; Fusar-Poli et al., 2009; Ghashghaei et al., 2007; Haxby et al., 2000; Hiser & Koenigs, 2018; Kanwisher et al., 1997; Kohn et al., 2014; Kropf et al., 2018; Lindquist et

al., 2012; Richler & Gauthier, 2014; Sel et al., 2014; Sergerie et al., 2008; Vuilleumier & Pourtois, 2007; Yang et al., 2020).

Several studies have also examined the developmental trajectories of neural function underlying WM and ER throughout adolescence. According to a meta-analysis of 10 WM imaging studies among adolescents and young adults (ages 10-30, n = 382), WM-related function increased with age within the rostral middle frontal, precuneus, inferior parietal, and premotor cortices and decreased with age within the superior frontal, postcentral, and posterior cingulate cortices (Andre et al., 2016). However, in an accelerated longitudinal study of 8-30 year-olds, Simmonds et al. (2017) found decreases in the middle frontal cortex, anterior cingulate, insula, and basal ganglia, as well as increases in the primary visual, visual association, and inferior temporal cortices. These group-level changes were associated with improved WM performance over time. Meta-analyses and reviews of emotion regulation studies among adolescents also reveal somewhat mixed findings. For example, in a review of 24 neuroimaging studies by Del Piero et al. (2016), changes in neural reactivity to emotion from childhood to adulthood were characterized by linear decreases in amygdala, insula, and fusiform gyrus response and increases in medial prefrontal/anterior cingulate response from childhood to early adulthood. Taken together, these findings suggest that neural specialization, and subsequent efficiency, during WM processing increases during adolescence, as reflected by decreased recruitment of the medial PFC (e.g., the middle frontal gyrus and anterior cingulate) and increased activation within visual cortices. On the other hand, neural function underlying ER is characterized by increasing prefrontal influence, as the medial PFC (particularly the anterior cingulate) increases in activity and regions underlying threat, motivation, and face processing (the amygdala, insula, and fusiform gyrus, respectively) decrease in activity.

Although these average group-level trends are significant, they are limited by their variable-centered methodology, which focuses on mean-level associations between variables (for example, average magnitude of brain function predicting average level of behavior). Person-centered methods, on the other hand, characterize heterogeneity between and within individuals by identifying subgroups of people based on their multivariate similarities (Howard & Hoffman, 2018; Muthén & Muthén, 2000).

These approaches are warranted, as a growing body of evidence suggests high variability of structural brain development (e.g., cortical thickness and grey matter volume) across adolescence (Lebel & Beaulieu, 2011; Mills et al., 2021; Paus et al., 2008; Tamnes et al., 2013; Wierenga et al., 2014). Preliminary evidence from person-centered studies using fMRI, EEG, and neurocognitive tasks indicate substantial within- and between-person variability in neural function as well (Kjelkenes et al., 2022; Ordaz et al., 2013; Tang et al., 2018). Ordaz et al. (2017) used mean growth curve modeling to characterize high within-person variability in neural function underlying EF in an accelerated longitudinal sample of 123 participants ages 9 to 26. Using latent class growth analysis within a sample of 43 12 to 16-year-old females, Tang et al. (2017) detected several unique trajectories of frontal alpha symmetry, suggesting both intra- and inter-individual variability of functional neural risk for psychopathology (Coan & Allen, 2004). Finally, Kjelkenes et al. (2022) used a normative modeling framework to identify inter-individual deviations from the norm in neurocognitive ability among youths ages 12 to 16.

Latent profile analysis (LPA) and its longitudinal extension latent transition analysis (LTA) form another branch of person-centered methods that may be especially advantageous for examination of neural function over time (Bray et al., 2010; Collins & Lanza, 2009; Lanza et al., 2013). LPA is a dimension reduction technique that characterizes heterogeneity across multiple variables into unobserved homogenous subgroups at a single time point. LTA extends LPA across time by 1) characterizing latent profiles at multiple time points, and 2) estimating the probability of individual movement from one profile to another across time points. Unlike growth mixture modeling (GMM) and latent class growth analysis (LCGA), in which subgroups of individuals are characterized by their level and shape of change over time, LTA examines both between-person differences at static points *and* within-person continuity or discontinuity across time. As such, LTA has the potential to create a more comprehensive picture of brain function by characterizing inter-individual variability first at static points and then modeling development of intra-individual change (Bray et al., 2010).

LTA differs from other person-centered approaches in its capacity for multivariate modeling. Whereas LCGA/GMM models often encounter issues of convergence and under-identification when

modeling trajectories of more than 4 variables at a time, LPA and LTA models allow for inclusion of multiple indicators (extant studies have included between 4 and 24; Scotto Rosato & Baer, 2012; Wurpts & Geiser, 2014) and typically improve in performance with increases in number of high-quality indicators (Wurpts & Geiser, 2014). This multivariate capacity means that several brain regions may be included in an LTA model simultaneously, which allows for better characterization of brain-wide function and interaction between multiple regions during tasks. Given that WM and ER processes recruit numerous brain regions simultaneously (Ahmed et al., 2015; Andre et al., 2016), a method such as LTA may be ideal for modeling their neural underpinnings across time.

Parenting and Neurocognitive Development

Warm and supportive parenting behaviors are potent predictors of positive youth development, whereas a lack of parental support is tied to development of psychopathology in adolescence (Huffman & Oshri, 2022; Meeus, 2016; Oshri et al., 2021; Waller et al., 2013; Weitkamp & Seiffge-Krenke, 2019). This link between the parenting context and youth psychopathology is mediated by the development of central neurocognitive processes, namely ER and EF, that underlie behavioral adaptation (Butterfield et al., 2021; Reuben et al., 2016). Growing research suggests that parental support is central to the formation of effective emotion regulation (Kerr et al., 2019; Morris et al., 2017) and working memory (Hughes & Devine, 2019) at both the behavioral and neural levels. The parent-child relationship fosters development of emotion regulation abilities primarily through modeling, socialization, and family emotional climate. Parents can model for their children effective emotion processing and regulation; they can also teach their children how to manage their emotions via discussion, transmission of ER strategies, and encouragement (Meyer et al., 2014). Use of these strategies is linked to heightened ER abilities during late childhood and adolescence (Morelen et al., 2016; Morris et al., 2017). Similarly, the emotional climate of the family lays the foundation for attachment security between the parent and child, which is closely linked to development of emotion reactivity and regulation throughout the lifespan (Morris et al., 2017).

Above and beyond genetic influence, parental support and sensitivity to children's affective states is also a central predictor of child executive functioning, including working memory (Hughes & Devine,

2019; Lucassen et al., 2015; Towe-Goodman et al., 2014). Caregivers act as "external regulators" of their child's affect, especially during infancy and early childhood, which facilitates development of the child's self-regulation and executive function (Gunnar & Donzella, 2002). Moreover, consistent caregiver sensitivity and support allow for the child to interact with their immediate environment in a way that elicits positive, encouraging, and/or effective responses from the caregiver (Bernier et al., 2010), further promoting the internalizing of constructive self-regulatory strategies (Bernier et al., 2012). To this point, a recent meta-analysis spanning 2000 to 2016 confirmed consistent associations between positive parenting (characterized by warmth, responsiveness, and sensitivity) and overall executive function among children ages 0 to 8 (Valcan et al., 2018). Similarly, Sosic-Vasic et al. (Sosic-Vasic et al., 2017) found that greater parental involvement was associated with improved executive functioning, including working memory, response inhibition, and cognitive flexibility, among both children and adolescents.

ER and WM as Predictors of Psychopathology

Disruptions in both ER and WM are strong predictors of psychopathology in adolescence and adulthood. Low WM capacity underlies lack of self-regulation (Huang-Pollock et al., 2017; Vuontela et al., 2013), and is often implicated in externalizing, internalizing, ADHD, and poor academic achievement (Ahmed et al., 2015; Beck et al., 2010; Cassidy et al., 2016; Matthews et al., 2008). ER is a similarly powerful risk factor for psychopathology. A child with low ER may be less able to modify their emotional response in the face of daily challenges and as a result is more likely to develop internalizing and externalizing behaviors throughout their lifespan (Aldao et al., 2016; Halligan et al., 2013; Kim Spoon et al., 2013; Shapero et al., 2016; Sheppes et al., 2015).

A number of studies have examined interactions between EF/WM process and ER at the neural level. In two early studies, Gray & Braver (Gray & Braver, 2002) (2002) and Herrington et al. (Herrington et al., 2005)2005) found that affective stimuli modulated activity in EF regions during a WM task: positive emotional face stimuli increased dlPFC activation, whereas negative emotional stimuli decreased dlPFC activation. In a meta-analysis of 33 fMRI studies, Schwiezer et al. (Schweizer et al., 2019) found that vlPFC, amygdala, temporal, and occipital activation increased during a WM task when

visual stimuli were emotionally salient, indicating the greater cognitive (and thus metabolic) demand on EF regions in the context of emotion. When interference in EF by emotional information is excessive, problems of self-regulation often ensue (Mueller, 2011). Indeed, among those exhibiting psychopathology, affective information disrupts executive function more frequently than healthy individuals (Ochsner & Gross, 2005). However, no studies have examined the development of WM-ER interactions over time, nor during adolescence—a period in which developing cognition-emotion interactions exert a particularly salient influence on behavior and psychopathology (Luna et al., 2010; Paus et al., 2008).

The Current Study

The current study aimed to 1) derive latent profiles of neural function during working memory and implicit emotion processing task in a priori ROIs, 2) identify latent statuses of neural function across 24 months (M_{age}, baseline = 10, M_{age}, T2 = 12), and 3) explore parental support and demographic covariates as predictors of latent statuses, and 4) evaluate the differences in youth internalizing and externalizing symptoms across latent statuses. Due to the complexity of study aims, we summarize all research questions, hypotheses, and analytic plans in Table 4.1.

1a) We hypothesized that neural activation during working memory and emotion regulation (e.g., the 2 back vs. 0 back and Faces vs. Places conditions of the EN-back, respectively) would form homogenous subgroups, or latent profiles, characterized by distinct patterns of task-activated regional function within regions delineated in Table 4.2. We anticipated that each profile would diverge in levels of activation, especially within the amygdala and medial prefrontal regions. We also hypothesized at least one "low regulation" profile at baseline that evinces particularly high amygdala and/or low ACC activation during the ER and low rostral middle frontal activation during the WM condition. Additionally, we hypothesized a "high regulation" profile at baseline that shows high anterior cingulate activation during ER and visual cortex activation during WM. Although we originally proposed to include biological sex and age as indicators alongside relevant ROIs during the model building process (given their relevance to organization and function of neural circuits underlying WM and ER [Hill et al., 2014; Stevens &

Hamann, 2012; Ullsperger & Nikolas, 2017; Zahn-Waxler et al., 2015]), inclusion in this way caused convergence issues (more details in Results section). As such, we included them as covariates (see number 3 below).

1b) Given the relatively short time frame between the two waves, we hypothesized the number of latent profiles yielded by the LPA to remain consistent from baseline to T2 (24 months after baseline).

2) We anticipated that most individuals in the sample would remain in or transition into the "high regulation" profile between baseline and T2. Conversely, we also hypothesized that very few youths would transition from a high to low regulation profile; rather, some youths who began in a low regulation profile would remain at T2.

3) Low parental emotional support and low family income would significantly affect LTA parameters by changing probability of latent statuses and/or decreasing probability for transition into in a high regulation class. We hypothesized that family history of mental illness would similarly impact LTA parameters, given the documented effect of parental symptoms on adolescent psychopathology (Schulz et al., 2021). Although we initially proposed to account for potential effects of scanner type on imaging data by also including it as a covariate (McCormick et al., 2021; McNeish & Kelley, 2019), this caused convergence issues (more information in Results section). As such, we included it as a clustering variable alongside family ID, which has also been used frequently within the ABCD sample (Saragosa-Harris et al., 2022; Lees et al., 2020; Bernanke et al., 2022; Pagliaccio et al., 2020; Lees et al., 2020).

4) We anticipated that latent status and transition probabilities would significantly affect rates of parent- and youth- reported internalizing and externalizing symptoms at T2, such that latent statuses and/or transitions evincing low neural regulation will show increased mean values of parent- and youth-reported internalizing and externalizing symptoms.

Table 4.1. Hypotheses and analytic plan

Research Question	Hypothesis	Analysis Plan	Effect of Interest	Threshold for Determining Support of Hypothesis
Are there qualitatively distinct subgroups of adolescents who evince unique patterns of neural response underlying ER and WM?	Inter-individual (between-person) differences in neural response during an ER and WM task will be evident at both T1 and T2[1] within relevant ROIs[2]. Optimal class solution will yield between 3 and 5 profiles. We hypothesize at least 1 "high regulation" profile (high ACC [ER] and rMFG/visual cortex [WM]) and 1 "low regulation" profile (high amygdala [ER], low ACC [ER], low rMFG [WM]).	We will use 2 separate LPAs to characterize homogenous subgroups of varying regional activation during the EN-back task 0- vs. 2-back (WM) and Faces vs. Places (ER) conditions. Indicators will include relevant ROIs (Table 2) as well as youth biological sex and age.	To determine optimal number of classes: BIC, A-BIC, class size; To identify characteristics of and differences between groups: probabilities of profile membership , item response probabilities[3], size of each profile	Hypotheses will be confirmed if we identify an optimal profile solution of 3-5 profiles that show differences in mean ROI activation within each task condition.
Is there change between latent profiles across time? If so, what is the probability that an adolescent's distinct pattern of neural function underlying WM and ER will change between ages 10 and 12?	1) The LPA at T1 and T2 will show measurement invariance. 2) Probability of transitioning from one latent profile at T1 to another profile at T2 will range from low to moderate (τ = .2-.5), with highest probabilities for transition observed among those moving into a "high regulation" profile.	We will conduct 1) repeated-measures LPA to determine measurement invariance, and 2) LTA to determine individual probability of transitioning from one latent profile at T1 to another 24 months later.	Difference in model fit of nested models using likelihood ratio difference test, BIC, a-BIC; Probability of latent statuses[4] (δ); Probability of transitioning from *k* profile at T1 to *k* profile at T2 (τ); item response probabilities	Hypotheses will be confirmed if we detect 1) a significant LRDT of nested models and lower BIC and a-BIC of constrained model, which indicate measurement invariance, and 2) more than one unique latent status (δ) and probability for transition (τ).
How do parental emotional support, family history[5], and family income impact latent statuses and transitions of neural function underlying WM and ER?	Low parental emotional support, low family income, and family history of mental illness will significantly affect profile transition probabilities, by changing probability of latent statuses and/or increasing probability for transition into in low-regulation class(es).	To test whether the slope of these covariates impacts latent statuses from T1 to T2, we will conduct two subsequent models: 1) parental support and family income will be included as covariates, and 2) family history of mental health problems will be added subsequently.	Changes in model fit parameters (significant LRDT of nested models); Probability of latent statuses (δ); Probability of transitioning from k profile at T1 to k profile at T2 (τ); item response probabilities	Hypotheses will be confirmed if we detect changes in model fit, model parameters (probability of latent statuses (δ), probability of transitioning from k profile at T1 to k profile at T2 (τ), and/or item response probabilities) after adding parent emotional support, family income, and family history of mental illness as covariates.
How do youth internalizing and externalizing symptoms at T2 differ across latent statuses?	Profile transition probabilities will significantly affect rates of internalizing and/or externalizing symptoms at T2, such that latent statuses evincing low neural regulation will show increased mean values of youth internalizing and externalizing symptoms.	We will estimate the mean values of parent- and youth-reported internalizing and externalizing symptoms (derived by latent factor scores) and conduct separate Wald tests to determine if symptoms differ significantly across statuses.	Mean values of internalizing and externalizing symptom scores per each latent status; Significance ($p < .05$) of each Wald test	Hypotheses will be confirmed if any Wald test shows significant differences in internalizing and/or externalizing symptoms across latent statuses ($p < .05$) after correcting for multiple comparisons using Benjami-Hochberg method (number of tests depends on number of profiles[6]).

Notes. [1]T1 = Time 1, T2 = Time 2, measured 24 months after T1; [2]ROIs = Region(s) of interest. See Table 2 for specific ROIs and directional hypotheses; [3]Item response probabilities are mean ROI activation per profile; [4]Latent statuses are latent profiles that remain consistent over time; Family history = sum score of parental history of alcohol use, drug use, depression, mania, hallucinations, trouble with the law, nervous disorders, hospitalization for mental health problems, and suicidality; [6]In 3-profile estimation: 2 outcomes*3 comparisons per outcome = 6 tests; 4-profile: 2*6 = 12 tests; 5-profile: 2*8 = 16 tests

Table 4.2. Hypotheses regarding function and change of ROIs

ROI	Function	Activity at T1	Change
Emotion regulation (*faces vs. places* condition)			
Amygdala	Emotion & threat detection	Increase	Decrease
Insula	Somatosensory, salience	Increase	Decrease
Fusiform gyrus	Visual face processing	Decrease	Decrease
Anterior cingulate	Integration, regulation	Increase	Increase
Working memory (*0-back vs. 2-back* condition)			
Rostral middle frontal gyrus	Executive function, memory	Increase	Decrease
Anterior cingulate	Integration, regulation	Decrease	Decrease
Lateral occipital	Visual processing	Increase	Increase
Inferior parietal	Visual processing	Increase	Increase

Note. Hypotheses are sourced primarily from systematic reviews and meta-analyses: Del Piero (2016), Andre et al. (2016). Simmonds et al. (2017) was also used to inform WM hypotheses change over time. Chaarani et al. (2021) was used to inform task-based activation specific to the ABCD sample.

Methods

To address our questions, we used data from the Adolescent Brain Cognitive Development (ABCD) Study. Launched in 2015, the ABCD Study recruited over 11,500 adolescents aged 9 to 10 at baseline. Participants were sampled in such a way to be representative of the population of the United States. The ABCD Study will continue to follow these youths every 6 months for a total of 10 years. Questions and tasks are similar across waves but may be adjusted as is age appropriate. Questions include demographic information, socioeconomic background, family history of physical and mental disorders, physical and mental health, and subjective experiences. We used a subset of participants with complete behavioral and functional neuroimaging (Emotional N-Back) data at baseline and 24 months after baseline. All subjects included passed the ABCD Study's quality control measures for functional imaging to ensure evaluation of interpretable data.

Measures

EN-back task. The EN-back is an implicit facial emotion and memory processing task eliciting short-term WM while presenting equal numbers of happy, fearful, and neutral facial expressions, as well as places, in each run (Barch et al., 2013). When presented with a happy, fearful, or neutral face, or a

place, participants were asked to respond as to whether the picture presented is a "Match" or "No Match." Conditions alternated between 0-back and 2-back conditions, for which youth were instructed to respond "Match" when the current stimulus corresponded to that presented at the beginning of the block or two trials back, respectively. Participants completed two runs of the task, with eight blocks per run and 10 trials (2.5 seconds each) per block. The current study employed two task conditions: *faces vs. places*, which was modeled to remove the effects of working memory and isolate effects of ER, and *0-back vs. 2-back*, which was modeled to remove the effects of ER and isolate effects of WM.

Image preprocessing and calculation region-of-interest data. Preprocessing and analysis of MRI data was completed by the ABCD Data Analysis and Informatics Center and is outlined elsewhere (Casey et al., 2018; Hagler et al., 2019). In short, parcellated cortical regions used in the study analyses were derived from Desikan atlas cortical surface reconstruction and subcortical segmentation performed in FreeSurfer 5.3.0 (Desikan et al., 2006). Estimates of task-related activation strength were computed for each individual using general linear modeling (GLM) in AFNI's 3dDeconvolve and released as contrast beta weights. The present study uses GLM beta coefficients averaged across both runs.

Working Memory (Indicators of LPA; IV & DV). WM ROIs were measured at T1 and T2 (24 months after T1) and include the rostral middle frontal gyrus (rMFG), rostral and caudal anterior cingulate (rACC, cACC), lateral occipital cortex (LOC), and inferior parietal cortex (IPC). To decrease computational burden and risk of convergence issues, mean values of bilateral activation were computed for each ROI.

Emotion Regulation (Indicators of LPA; IV & DV). ER ROIs were measured at T1 and T2 (24 months after T1) and included the amygdala, insula, fusiform gyrus, rACC, and cACC. To decrease computational burden and risk of convergence issues, mean values of bilateral activation were computed for each ROI.

Parenting Behaviors (IV). Parental support was measured at T1 using the CRPBI-Short Parental Acceptance subscale (Schaefer, 1965). Youths reported on the emotional support of their caregiver who participated in the study with them at baseline. This subscale is the mean of 5 items, such as "makes me

feel better after talking over my worries with him/her" and "believes in showing his/her love for me," which youths evaluated using a Likert scale (1 = *Not like them;* 3 = *A lot like them*).

Childhood Psychopathology (DV). Child psychopathology was measured at T2 using symptom subscales of the parent-reported Child Behavior Checklist (CBCL; 119 items) and youth-reported CBCL Brief Problem Monitor (BPM; 19 items; (Achenbach et al., 2017). The CBCL is 119 items and the BPM is 19 items. In the CBCL, parents report on the presence of youths' behaviors over the last 6 months using a Likert scale (0 = *Not True*, 1 = *Somewhat or Sometimes True*, 2 = *Very True or Often True*), such as rule-breaking ("Breaks rules at home, school, or elsewhere"), aggression ("Cruelty, bullying, or meanness to others"), anxious-depressed symptoms ("Too fearful or anxious"), and withdrawn-depressed symptoms ("Withdrawn, doesn't get involved with others"), and somatic complaints ("Headaches, nausea"). Internalizing problems are a sum of the anxious-depressed, withdrawn-depressed, and somatic complaints subscales, and externalizing problems are a sum of the rule-breaking and aggressive behavior subscales. The BPM has been designed as a brief counterpart to the CBCL. It is structured with the same Likert responses and produces youth-reported item ratings and scale scores for internalizing, externalizing, and attention problems that can be directly compared to the CBCL. Both the CBCL and BPM have been studied and validated in many different cultures among youths ages 6 to 18. The current study will employ raw scores of youths' anxious-depressed, withdrawn-depressed, rule-breaking, and aggressive symptoms (parent report), as well as youth-reported internalizing and externalizing symptoms.

Family History of Mental Health. Family history of parental mental health problems were assessed at T1 via the participating caregiver. Inclusion of family history data in the ABCD study is dependent on the participating caregiver verifying they had knowledge about the child's biological parents. Participating caregivers reported on whether the child's biological parents had ever (in their lifetime) evinced drug problems, alcohol problems, depression, mania, hallucinations, problem behavior (e.g., fighting, not holding a job, trouble with the law), nerve problems or nervous breakdowns, suicidality, or hospitalization due to these problems. Each answer is coded dichotomously, such that 1 =

Yes and 0 = *No*. A sum score of parent mental health problems was computed (minimum = 0, maximum = 9) prior to inclusion in the LTA.

Covariates. In addition to parental support and family history of mental health problems, we tested whether baseline covariates—annual family income, child age in months, and child biological sex (coded dichotomously as 0 = Female, 1 = Male) —were significant predictors of class membership, latent statuses, and likelihood for transition.

Analysis Plan

Missing data. All imaging data were filtered using quality control measures as outlined by Hagler et al., 2019 and in ABCD Release Notes 4.0, MRI Quality Control Recommended Inclusion (http://dx.doi.org/10.15154/1523041). Briefly, quality control metrics include imaging protocol compliance, mean head motion, framewise displacement, presence of artifacts, irregularities, or incidental findings, and behavioral task performance. Per ABCD data release 4.0, a single quality control index (abcd_imgincl01) has been added to indicate those who pass all quality control filtering measures. The current sample was filtered using this quality control index (0 = passing QC, 1 = not passing QC).

Inclusion/exclusion criteria. T1 (baseline) data included all imaging data present at baseline that passed quality control (n = 7,930) and all parental support and demographic data of those who have complete imaging data. T2 data include dall imaging data available at T2 that passes quality control (estimated n = 6,184) and all CBCL and YSR data of those who have complete imaging data.

Statistical outliers. Outliers can bias results of multivariate analyses such as latent profile and latent transition analysis (Fidell, 2001). During pre-processing of tabulated ABCD imaging data, beta values with greater than 5% signal change are censored (replaced with empty cells), accounting for less than 0.5% of the sample (Hagler et al., 2019). For imaging variables with skewness greater than 2, we will Winsorize the top and bottom .25%, which has also been recommended by Hagler et al. (2019). Both LPA and LTA will be conducted with Winsorized and non-Winsorized imaging variables to test robustness of the models to normality.

Sampling weights. The ABCD Study includes propensity weights, which are weighted estimates derived from the American Community Survey used to calibrate ABCD distributions to nationally representative controls (Heeringa & Berglund, 2020).

Statistical models

Structural equation modeling in Mplus version 8.1 will be used to test all study hypotheses (Muthén & Muthén, 2014). All models were estimated using maximum likelihood with robust standard errors (Klein & Moosbrugger, 2000). Multilevel modeling was used to account for clustering effects of participants within families and scanner type (Saragosa-Harris et al., 2022). Although scanner was originally proposed as a covariate, Mplus software is currently unable to include categorical covariates with more than 10 categories. Given this, we instead used scanner as a nesting variable alongside family ID. Propensity weights were also be used to calibrate distributions of the current sample to nationally representative controls from the American Community survey, thus mitigating potential selection bias in the ABCD sampling process (Heeringa & Berglund, 2020; Saragosa-Harris et al., 2022). Prior to conducting the LTA, we create two separate measurement models of child internalizing and externalizing symptoms. Using confirmatory factor analysis (CFA), we created a latent factor of internalizing symptoms using parent-reported subscales of youth anxious-depressed and withdrawn-depressed symptoms, as well as youth-reported internalizing symptoms. We also created a latent factor of externalizing symptoms using parent-reported subscales of youth rule-breaking and aggressive behavior and youth-reported externalizing behaviors. We saved the resulting factor scores to a separate data file and include them as distal outcomes in Step 4 of the LTA.

Latent Transition Analyses. *LTA Step 0.* We built separate LPAs with T1 and T2 imaging data using the aforementioned ROIs within Mplus version 8.1. For both baseline and T2 indicators, we fit separate LPAs starting with a null 1-class model and increasing the number of profiles by one until stopping criteria were reached. In a simulation by Whittaker & Miller (Whittaker & Miller, 2021), BIC and adjusted BIC (a-BIC) were found to predict number of correct classes with significantly greater accuracy than any fit indices, including AIC, entropy, VLMRT, and BLRT. As such, we used BIC and a-

BIC to determine whether the *k* class solution was better than the *k*-1 class solution, as indicated by decreases in both criteria. Scree plots were also used to visualize where BIC values began to display diminishing value for each additional class. The class number at the "elbow" of the plot where BIC values level out provides an indicator of best fitting number of classes (Nylund-Gibson & Choi, 2018). We also included as a stopping criterion a class size of less than 5% of the sample. This criterion was included to prevent fitting a model with so many classes that making comparisons between them becomes unwieldy and their qualitative differences (in both item response probabilities, probabilities of latent status, and probabilities of transitions) become meaningless. Finally, we planned not to estimate the *k*+1 model if the best loglikelihood did not replicate or the model did not converge. As such, the following 4 questions comprised our selection criteria; if the answer to any one of these questions was no, we planned to stop and select the *k*-1 class solution.

- Did the model converge?
- Was the best loglikelihood replicated?
- Are the BIC and the adj-BIC lower than the k-1 model?
- Are all class sizes greater than approximately 5% of the sample?

LTA Step 1. If finding the number of classes derived at each time point to be the same, we tested longitudinal measurement invariance by comparing model fit of a constrained and unconstrained model. In the constrained model, all item response probabilities are constrained to be equal across T1 and T2 (see Appendix for Mplus syntax). We then compared model fit between the constrained and unconstrained LTA models by comparing BIC, a-BIC, and conducting a log likelihood ratio difference test (LRDT). Measurement invariance may be concluded if the BIC and a-BIC are lower in the constrained model, and if the LRDT is significant. We planned to fit a repeated measures-LTA if the number of classes between T1 and 5 were not consistent or if the LTA did not evince longitudinal measurement invariance, based on the recommendation of Bray et al. (2010). After failing to establish measurement invariance, however, we conducted exploratory LTA of the non-invariant model, based on the recommendation of Nylund et al. (2022). *LTA Step 2.* We used the results of the LTA to characterize probability of latent statuses (e.g.,

latent profiles that remain consistent from T1 to T2), probability of transition (e.g., moving from *k* profile at T1 to *k* profile at T2), and item-response probabilities (e.g., mean ROI activation by profile). *LTA Step 3.* We tested whether parental support, family history of mental health problems, family income, child biological sex, and child age impacted LTA parameters (see Appendix for Mplus syntax). To do so, we ran the same LTA model repeatedly in a stepwise manner, including one additional covariate each time. In this way, we aimed to disentangle the unique effects of each covariate on the model. For each covariate model, we conducted an LRDT to determine whether inclusion of these covariates significantly changed the model fit compared to the previous model (Ryoo et al., 2018). Using odds ratios, we also determined whether inclusion of these covariates conferred a greater likelihood of belonging to one latent status over another. *LTA Step 4.* We then tested whether psychopathology symptoms differed across latent statuses by estimating the mean values of internalizing and externalizing factors (see Statistical Models, above) within each T5 profile. We conducted Wald tests to determine whether these differences were statistically meaningful (Nylund, Muthén, et al., 2007). Across all instances of multiple comparisons (e.g., comparing odds ratios of covariates and mean values of outcome variables), we corrected for family-wise error using the Benjamini-Hochberg method (Thissen et al., 2002).

Reliability and Robustness testing. Although a number of LPA, LCA, and LTA analyses have been conducted using cross-validation techniques, an LCA simulation study by Whittaker & Miller (2020) indicated that the most accurate cross-validation methods perform less accurately than BIC and a-BIC in a large single sample (n = 800). Considering our use of BIC and a-BIC to fit the proposed models, and also the large sample size and number of indicators of the current study (both of which increase estimation accuracy), we conducted our analyses using the full sample without cross-validation. We also tested if our findings were robust without controlling for clustering effects of family and scanner and no longer weighting estimates by propensity scores.

Power analysis. A previous simulation study by Nylund et al. (Nylund, Asparouhov, et al., 2007) indicated that for an 10-item complex LPA model with 4 unequal classes, a sample size of 1000 provided excellent coverage values for all parameters, including for the smallest class of 5%. This simulation also

showed that for both LMR and BLR tests, a sample size of 1000 provided sufficient power (greater than .80) to detect the *k* class model for a 10-item, 4 class complex LPA model. Given that the proposed sample is significantly larger than 1000, we anticipated adequate power to detect 3 and 4 class models. We confirmed this by conducting a Monte Carlo simulation study for a complex LPA model with 13 indicators, 5 covariates, and 4 outcomes. For both 3 and 4 class solutions, LMR and BLR tests were significant ($p < 0.01$) and the proportion of replications at the 5% level for the BLRT, indicating that the proposed sample size of roughly 7,000 provided adequate power to correctly identify the *k* class model. The simulation also indicated that the proposed sample size provided enough power to reject the null for each of the 4 outcome variables 100% of the time.

Results

CFA of T2 Child- and Parent-Reported Psychopathology

A two-factor model of child internalizing and externalizing behavior exhibited an excellent fit to the data (see Figure 4.1). Child-reported internalizing and externalizing were covaried due to high correlation and corresponding recommendation of modification indices. Resulting factor scores (e.g., individual Z-scores indicating factor-level standard deviations above and below the sample mean) were saved in a separate data file and used in subsequent analyses (see Step 4).

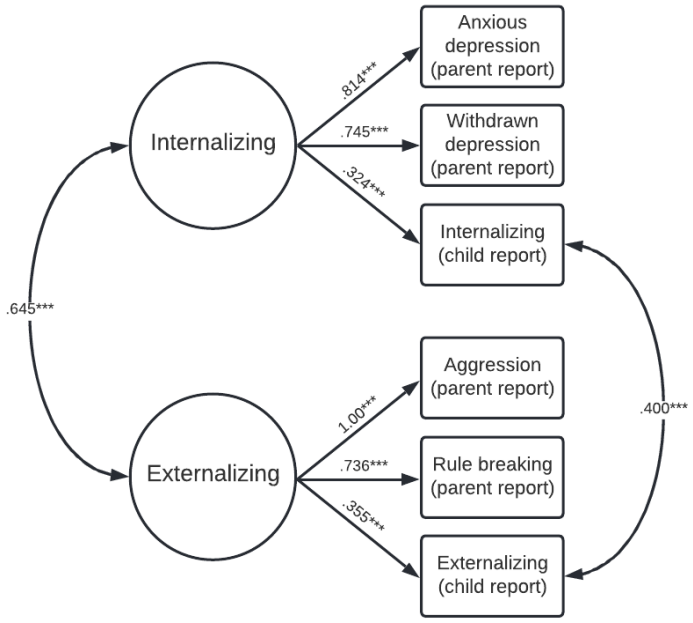

Figure 4.1. CFA of parent- and child-reported internalizing and externalizing symptoms.

Step 0: Repeated Measures LTA

At both T1 and T2, we began with a null 1-class model and increased the profile number by 1 until model fit criteria were reached. For both T1 and T2 data, the 3-profile model indicated optimal fit (Table 4.3, Figure 4.2). Although we initially proposed to include child age and child biological sex as indicators of profiles alongside imaging variables, T2 models that included age and sex indicated convergence difficulties. Beginning at the 3-profile solution for T2, several parameters were automatically fixed to prevent singularity of the information matrix, indicating that the 3- to 6-profile models were not identified. However, this issue was resolved when removing both child age and biological sex as indicators. As such, we re-ran all profile solutions for both T1 and T2 data to test whether they were sensitive to inclusion of child age and biological sex. Because the optimal profile solution and item response probabilities remained consistent without inclusion of child age and biological sex as indicators, we chose to fit all models without these indicators. Details on fit and profile solutions for models including child age and biological sex as indicators may be found in the Appendix.

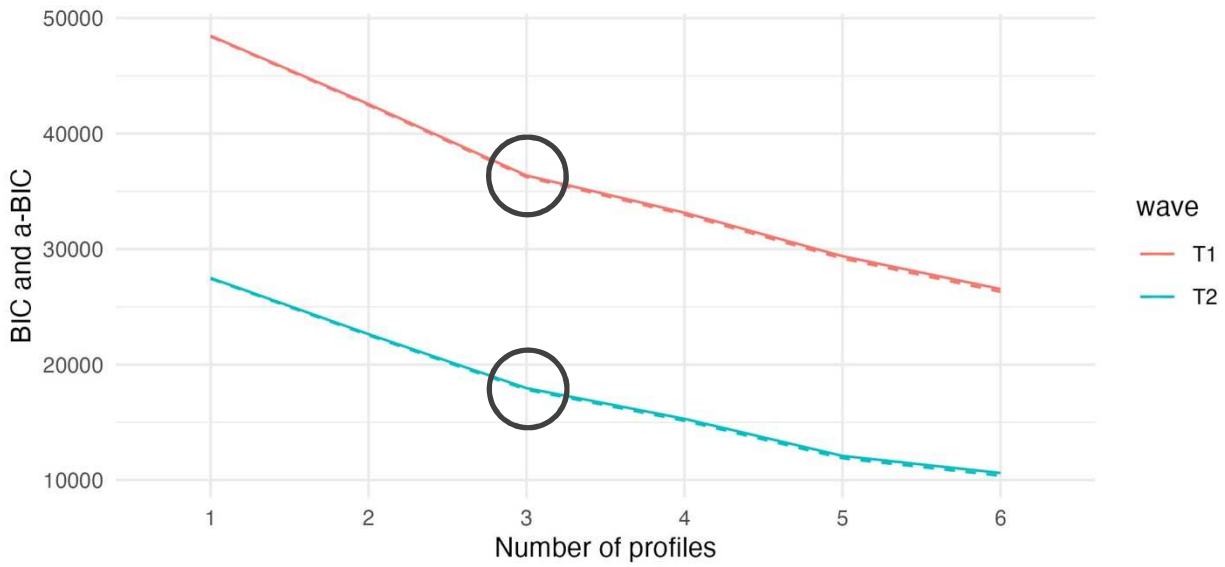

Figure 4.2. Scree plot of models clustering by scanner and family and weighted by propensity scores. Solid line indicates BIC; dashed line indicates adjusted BIC. Grey circles indicate "elbows" of plot.

Table 4.3. LPA fit indices by time point

T1				
Class solution		BIC	a-BIC	Smallest class size
	1	48473.898	48410.432	7928 (100)
	2	42551.489	42452.977	1403 (17.7)
	3	**36369.347**	**36235.879**	**495 (6.3)**
	4	33149.873	32981.45	418 (5.3)
	5	29385.337	29181.957	363 (4.6)
	6	26538.885	26300.55	4 (.05)
T2				
Class solution		BIC	a-BIC	Smallest class size
	1	27496.889	27433.334	6164 (100)
	2	22629.873	22531.363	499 (8.1)
	3	**17955.246**	**17821.781**	**381 (6.2)**
	4	15296.427	15128.007	219 (3.6)
	5	12084.908	11881.533	209 (3.4)
	6	10607.348	10369.018	20 (.3)

At both T1 and T2, the profile solution indicated three distinct profiles largely differentiated by neural response to emotion regulation; neural response to working memory was relatively similar across profiles at both time points (Figure 4.3, Figure 4.4, Table 4.4). The "Typical" profile (n_{T1} = 6661, 84%;

n_{T2} = 5059, 82.1%) was characterized by moderate amygdalar activation and fusiform deactivation to the ER (*faces vs. places*) condition at both T1 and T2. All other ER-related ROIs within this profile exhibited low to negligible response. Within this profile, rostral middle frontal, inferior parietal, and cACC response to the WM (*0 vs. 2-back*) condition were slightly higher than other profiles. The "Emotion hypo-response" profile (n_{T1} = 495, 6.3%; n_{T2} = 724, 11.8%) was characterized by moderate amygdalar deactivation and high cACC, rACC, fusiform, and insula deactivation at T1 to the ER condition, with slight decreases in deactivation across these regions at T2. The "Emotion hyper-response" profile (n_{T1} = 772, 9.7%; n_{T2} = 380, 6.2%) was characterized by high amygdala, cACC, rACC, fusiform, and insula activation at T1 to the ER condition, with slight increases in activation across these regions at T2. Individuals in this profile also exhibited slightly greater T1 deactivation during WM, particularly within the cACC, rACC, and lateral occipital cortex. However, WM activation within this profile increased at T2 to levels similar to other profiles. At both T1 and T2, the WM condition elicited slight deactivation in the rACC across profiles, which became more pronounced at T2.

Table 4.4. T1 and T2 item response probabilities

	Working Memory				
Profile	Rostral middle frontal	Inferior parietal	Caudal anterior cingulate	Rostral anterior cingulate	Lateral occipital
T1					
Emotion hypo-response	-0.051	-0.017	-0.093	-0.201	-0.144
Emotion hyper-response	0.044	0.023	0.027	-0.087	-0.034
Typical	0.099	0.065	0.061	-0.064	-0.023
T2					
Emotion Hypo-response	0.131	0.09	0.056	-0.123	-0.01
Emotion Hyper-response	0.133	0.094	0.072	-0.088	0.043
Typical	0.146	0.084	0.073	-0.104	-0.022
	Emotion Regulation				
	Amygdala	Caudal anterior cingulate	Rostral anterior cingulate	Fusiform	Insula
T1					
Emotion hypo-response	-0.214	-0.688	-0.646	-0.886	-0.526
Emotion hyper-response	0.595	0.509	0.531	0.238	0.48
Typical	0.183	-0.024	-0.005	-0.316	0.005
T2					
Emotion Hypo-response	-0.097	-0.44	-0.406	-0.714	-0.34
Emotion Hyper-response	0.61	0.61	0.603	0.356	0.555
Typical	0.209	0.012	0.043	-0.275	0.035

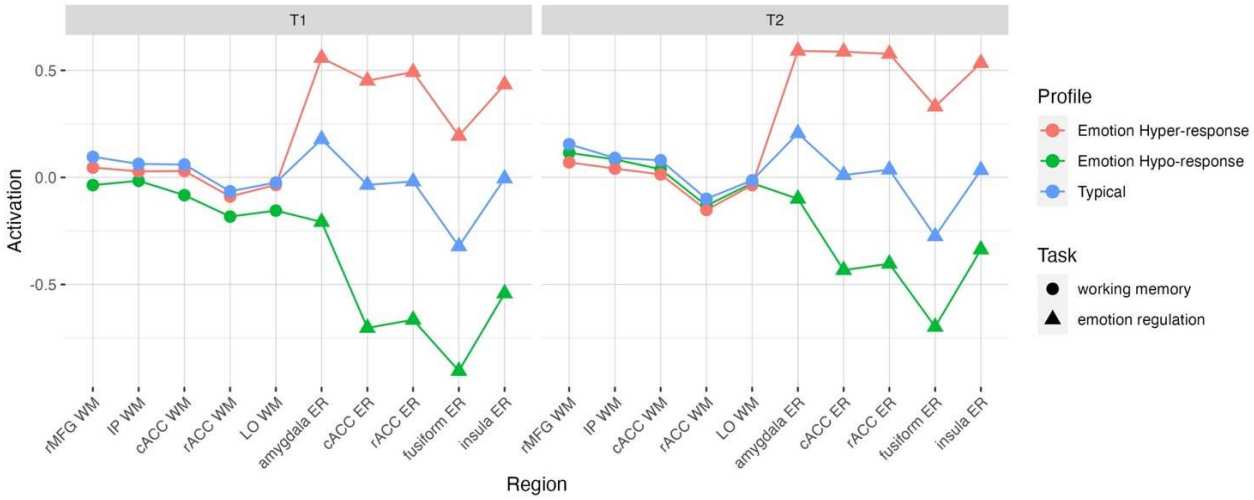

Figure 4.3. T1 and T2 latent profile solutions.

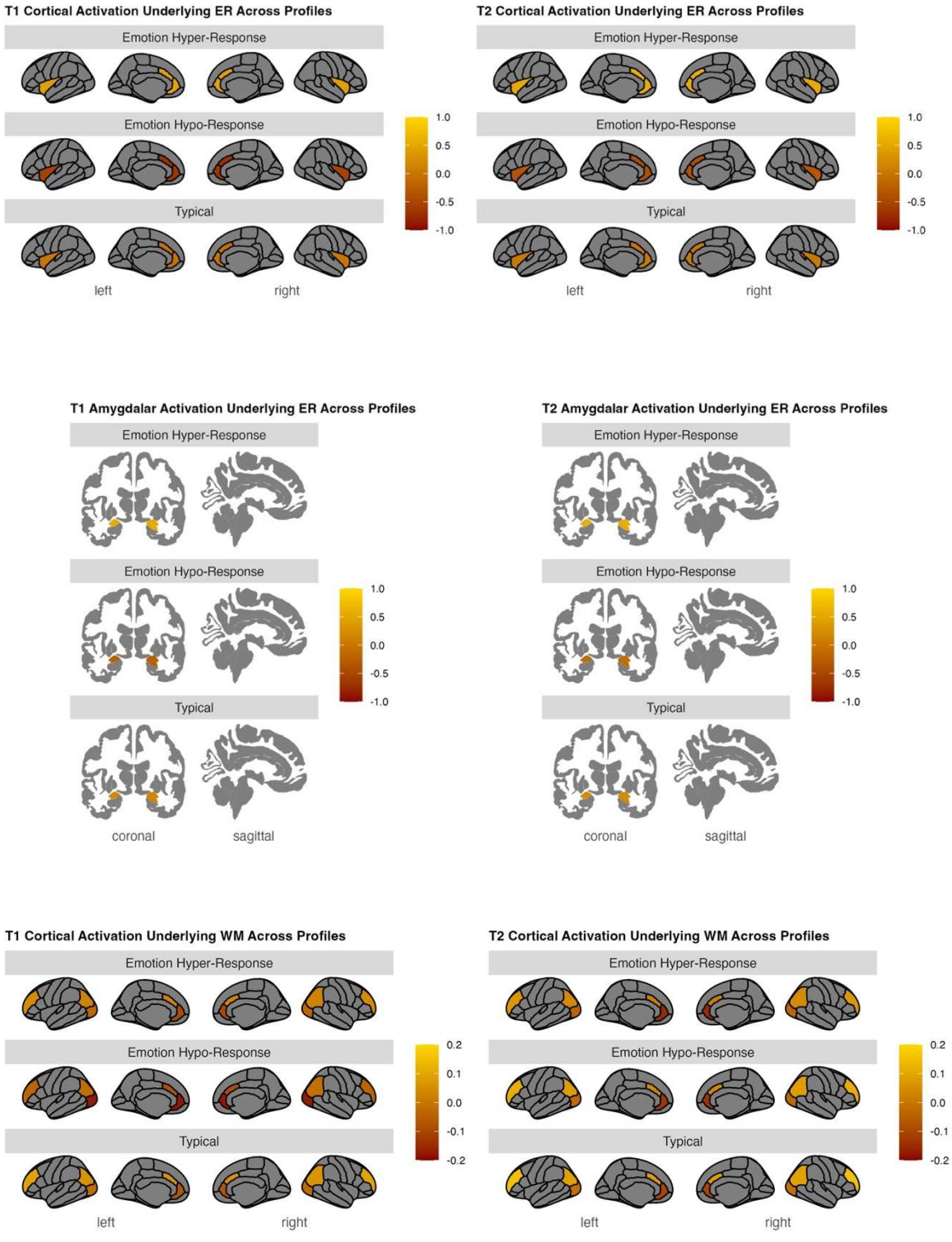

Figure 4.4. Cortical and subcortical activation underlying ER and WM across profiles.

Step 1: Measurement Invariance of LTA

After establishing that the 3-profile solution was optimal at both time points, we compared two different 3-profile LTA models (Table 4.5). The first model tested measurement invariance (MI) by constraining item response probabilities of all indicators across T1 and T2. For example, rostral middle frontal activation within Profile 1 at T1 was constrained to be equal to that within Profile 1 at T2, and this was done for all indicators that corresponded across both profiles and time points. Fit of the MI model was compared to that of a non-MI model, wherein no item response probabilities were constrained. Using a likelihood ratio difference test, we established that the non-MI model exhibited better fit than the MI model, and thus we could not conclude measurement invariance across time points. We followed this comparison with an exploratory comparison of a partial-MI model, in which we constrained only WM indicators that evinced relatively consistent item response probabilities across time and/or profiles. Specifically, we constrained WM items within the Typical and Emotion Hyper-response to be equal at T1, and WM items with all profiles to be equal at T3. After comparing this partial-MI model to the unconstrained non-MI model and fully constrained MI models, significant LRDTs once again indicated a superior fit of the non-MI model.

Table 4.5. Comparison of model fit after implementing measurement invariance

Measurement invariance?	G^2*	*BIC*	*a-BIC*	*DF*	G^2 *diff*	*DF diff*	*LRDT*	*p-value*
Comparison 1: Invariant v. non-invariant								
Yes (fully constrained)	-32655.129	65841.678	65657.363	58				
No (unconstrained)	-26676.155	54158.601	53878.951	88	-5999.267	30	11998.534	<.0001
Comparison 2: Partially invariant v. non-invariant								
Partial (t1 p1 & p3 WM equal; t2 all WM equal)	-26692.012	54052.879	53820.897	73				
No (unconstrained)	-26676.155	54158.601	53878.951	88	-15.857	15	31.714	0.007
Comparison 3: Invariant v. partially invariant								
Yes (fully constrained)	-32655.129	65841.678	65657.363	58				
Partial (t1 p1 & p3 WM equal; t2 all WM equal)	-26692.012	54052.879	53820.897	73	-5963.117	15	11926.234	<.0001

Note. *Likelihood ratio statistic

In our preregistration, we proposed to abandon the LTA in the case that longitudinal MI was not established. However, after investigating the results of the repeated measures LPA and non-MI LTA, we found that T1 and T2 profiles still exhibited strong evidence for stability from T1 to T2, making them readily comparable. As stated by Nylund-Gibson et al. (2022), although longitudinal MI is advantageous in reducing bias (Nylund, 2007) and increasing clarity and ease of LTA interpretation, it is not a required

prerequisite to fitting an LTA model. In the case of a non-MI LTA model, researchers must take care to interpret transition probabilities specific to their respective classes (Nylund-Gibson et al., 2022). Considering these points, we proceeded with our proposed analyses using a non-MI LTA. Given that the following steps were preregistered only for an LTA exhibiting longitudinal MI, the following analyses are considered exploratory.

Step 2: Interpreting LTA Profiles and Transitions

As specified above, the three profiles were similarly characterized at T1 and T2 by patterns of moderate response (the Typical profile), hypo-response (Emotion Hypo-response profile), and hyper-response (Emotion Hyper-response profile). Results of the LTA yielded probabilities of latent statuses and transitions based on posterior probabilities respective to each profile and time point (Figure 4.5, Table 4.8). Individuals most commonly began and remained in the "Typical" profile (n = 6622, 69.5%). The second most common transition was characterized by movement from the Typical profile to the Emotion Hypo-response profile (n = 900, 9.4%). The third most common transition was characterized by movement from the Emotion Hyper-response profile to the Typical profile (n = 698, 7.3%). Those in the atypical profiles were most likely to transition into the Typical profile at T2. All other transitions involving less than 5% of the sample are listed in Table 4.8.

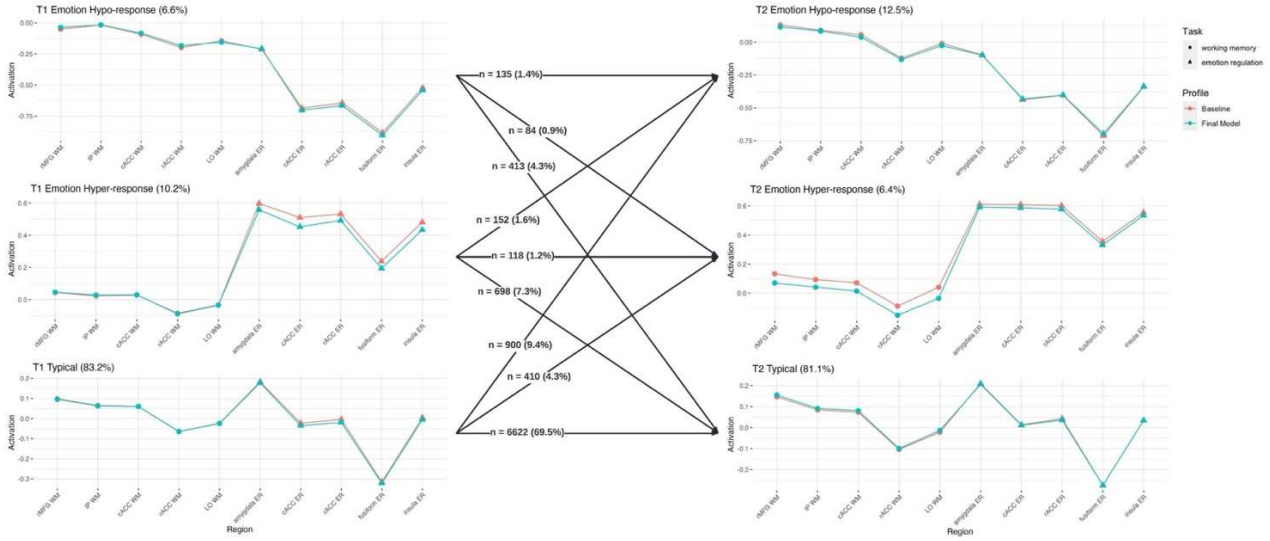

Figure 4.5. Latent transition probabilities

Step 3: Investigating Impacts of Covariates on LTA Parameters

We then investigated the impact of parental support, family history of mental illness, and family income on LTA parameters. Because issues with model identification prevented us from fully probing the effects of child biological sex and age on profiles, we conducted exploratory analyses of two additional covariate models that included child biological sex and age at baseline. In order to assess the unique contributions of each covariate to model fit and LTA parameters, we included them in the model in a stepwise manner (using the order described above), culminating in a final model with all three covariates included. Model fit was compared between each model using a likelihood ratio test.

Reduced G^2, BIC, and a-BIC values paired with significant LRDTs ($p < .0001$) indicated significantly improved model fit after including parental support, family history of mental illness, income, and child biological sex (Table 4.6). The addition of child age, however, did not confer significant changes in model fit, LTA parameters, or prediction of profile membership, and thus was not included in the final model. All LTA parameters, including item response probabilities and likelihood for transition, exhibited relative stability after inclusion of each covariate compared to the baseline model (Table 4.7, Table 4.8). However, both T1 and T2 Emotion Hyper-response profiles exhibited slight changes in item

response probabilities after including parental support and family income in the model (Figure 4.6). In the T1 Emotion Hyper-response profile, activation across WM ROIs increased slightly after including parental support; subsequently, activation of all ROIs decreased as a result of including income in the model. In the corresponding T2 profile, activation during WM decreased slightly in the rostral middle frontal cortex, inferior parietal cortex, and cACC, and deactivation during WM increased slightly in the rACC and lateral occipital cortex, after including parental support and income in the model.

Table 4.6. Comparison of model fit after stepwise addition of covariates

Covariates?	G^2*	*BIC*	*a-BIC*	*DF*	G^2 *diff*	*DF diff*	*LRDT*	*p-value*
Comparison 1								
None	-26676.155	54158.601	53878.951	88				
Support	-24741.692	50319.641	50027.281	92	-1934.463	4	3869.926	< .0001
Comparison 2								
Support	-24741.692	50319.641	50027.281	92				
Support, family history*	-24736.679	50345.974	50040.903	96	-5.013	4	10.026	0.04
Comparison 3								
Support, family history	-24736.679	50345.974	50040.903	96				
Support, family history, family income	-20015.985	40918.375	40600.598	100	-4720.694	4	9441.388	< .0001
Comparison 3								
Support, family history, family income	-20015.985	40918.375	40600.598	100				
Support, family history, family income, sex	-20000.883	40923.627	40593.139	104	-15.102	4	30.204	< .0001
Comparison 4								
Support, family history, family income, sex	-20000.883	40923.627	40593.139	104				
Support, family history, family income, sex, age	-19996.243	40949.802	40606.603	108	-4.64	4	9.28	0.06

Note. *Family history = family history of mental health problems.

Table 4.7. Changes in profile item response probabilities after stepwise inclusion of covariates

	T1									
	Working Memory					Emotion Regulation				
	Rostral middle frontal	Inferior parietal	Caudal anterior cingulate	Rostral anterior cingulate	Lateral occipital	Amygdala	Caudal anterior cingulate	Rostral anterior cingulate	Fusiform	Insula
Emotion Hypo-response										
Baseline	-0.051	-0.017	-0.091	-0.198	-0.144	-0.213	-0.686	-0.644	-0.884	-0.523
+ Support	-0.036	-0.016	-0.083	-0.183	-0.155	-0.208	-0.703	-0.665	-0.904	-0.542
+ Family history	-0.036	-0.016	-0.083	-0.183	-0.155	-0.209	-0.704	-0.666	-0.904	-0.542
+ Family income	-0.035	-0.016	-0.079	-0.177	-0.174	-0.232	-0.708	-0.675	-0.891	-0.54
+ Sex	**-0.037**	**-0.016**	**-0.079**	**-0.178**	**-0.174**	**-0.234**	**-0.709**	**-0.675**	**-0.891**	**-0.54**
+ Age	-0.037	-0.016	-0.079	-0.178	-0.174	-0.234	-0.709	-0.675	-0.891	-0.54
Emotion Hyper-response										
Baseline	0.044	0.023	0.028	-0.085	-0.033	0.597	0.51	0.532	0.238	0.481
+ Support	0.071	0.05	0.049	-0.068	0.005	0.597	0.505	0.539	0.242	0.482
+ Family history	0.071	0.05	0.049	-0.068	0.005	0.597	0.505	0.539	0.242	0.482
+ Family income	0.046	0.028	0.03	-0.089	-0.035	0.558	0.452	0.492	0.194	0.434
+ Sex	**0.046**	**0.028**	**0.03**	**-0.089**	**-0.035**	**0.558**	**0.452**	**0.492**	**0.194**	**0.434**
+ Age	0.046	0.028	0.03	-0.089	-0.035	0.558	0.452	0.492	0.194	0.434
Typical										
Baseline	0.099	0.065	0.061	-0.064	-0.023	0.183	-0.023	-0.004	-0.315	0.005
+ Support	0.094	0.06	0.058	-0.067	-0.03	0.185	-0.025	-0.006	-0.315	0.005
+ Family history	0.094	0.06	0.058	-0.067	-0.03	0.185	-0.025	-0.006	-0.315	0.005
+ Family income	0.096	0.063	0.06	-0.065	-0.024	0.178	-0.035	-0.019	-0.322	-0.005
+ Sex	**0.096**	**0.063**	**0.06**	**-0.065**	**-0.024**	**0.178**	**-0.035**	**-0.019**	**-0.322**	**-0.005**
+ Age	0.096	0.063	0.06	-0.065	-0.024	0.178	-0.035	-0.019	-0.322	-0.005
	T2									
Emotion Hypo-response										
Baseline	0.13	0.09	0.056	-0.122	-0.011	-0.097	-0.439	-0.405	-0.713	-0.339
+ Support	0.121	0.084	0.047	-0.129	-0.023	-0.09	-0.437	-0.403	-0.705	-0.338
+ Family history	0.121	0.084	0.047	-0.129	-0.023	-0.089	-0.436	-0.402	-0.704	-0.337
+ Family income	0.115	0.084	0.037	-0.132	-0.027	-0.102	-0.435	-0.405	-0.699	-0.338
+ Sex	**0.115**	**0.084**	**0.038**	**-0.132**	**-0.027**	**-0.1**	**-0.433**	**-0.403**	**-0.698**	**-0.337**
+ Age	0.115	0.084	0.038	-0.132	-0.027	-0.1	-0.433	-0.403	-0.698	-0.337
Emotion Hyper-response										
Baseline	0.133	0.093	0.071	-0.089	0.04	0.61	0.609	0.602	0.355	0.554
+ Support	0.092	0.064	0.038	-0.121	-0.003	0.612	0.607	0.598	0.349	0.551
+ Family history	0.09	0.063	0.036	-0.122	-0.005	0.612	0.607	0.597	0.35	0.552
+ Family income	0.071	0.041	0.015	-0.15	-0.035	0.589	0.585	0.576	0.329	0.532
+ Sex	**0.07**	**0.041**	**0.014**	**-0.152**	**-0.036**	**0.591**	**0.587**	**0.577**	**0.331**	**0.534**
+ Age	0.07	0.041	0.014	-0.152	-0.036	0.591	0.587	0.577	0.331	0.534
Typical										
Baseline	0.146	0.084	0.073	-0.104	-0.022	0.21	0.013	0.043	-0.274	0.035
+ Support	0.149	0.087	0.078	-0.101	-0.016	0.209	0.012	0.043	-0.276	0.034
+ Family history	0.149	0.087	0.078	-0.101	-0.016	0.209	0.012	0.043	-0.276	0.034
+ Family income	0.155	0.091	0.08	-0.1	-0.014	0.206	0.011	0.036	-0.276	0.034
+ Sex	**0.155**	**0.091**	**0.08**	**-0.1**	**-0.014**	**0.206**	**0.011**	**0.036**	**-0.275**	**0.034**
+ Age	0.155	0.091	0.08	-0.1	-0.014	0.206	0.011	0.036	-0.275	0.034

Note. Bolded lines indicate item response probabilities of final model.

Table 4.8. Changes in latent transitions after stepwise inclusion of covariates

		Model					
		Baseline	+ Support	+ Family History	+ Family Income	+ Sex	+ Age
T1	T2	%	%	%	%	%	%
Hypo	Hypo	1.4	1.4	1.4	1.5	1.5	1.5
Hypo	Hyper	0.9	1	1	1	1	1
Hypo	Typical	4.3	4	4	4	4	4
Typical	**Hypo**	**9.4**	**9.4**	**9.5**	**9.3**	**9.3**	**9.3**
Typical	Hyper	4.3	4.3	4.3	4.4	4.3	4.3
Typical	**Typical**	**69.5**	**70**	**70**	**67.9**	**70**	**67.9**
Hyper	Hypo	1.6	1.6	1.6	2	2	1.9
Hyper	Hyper	1.2	1.3	1.3	1.5	1.5	1.5
Hyper	**Typical**	**7.3**	**7**	**7**	**8.6**	**8.6**	**8.6**

Note. Bolded lines indicate top three most common transitions.

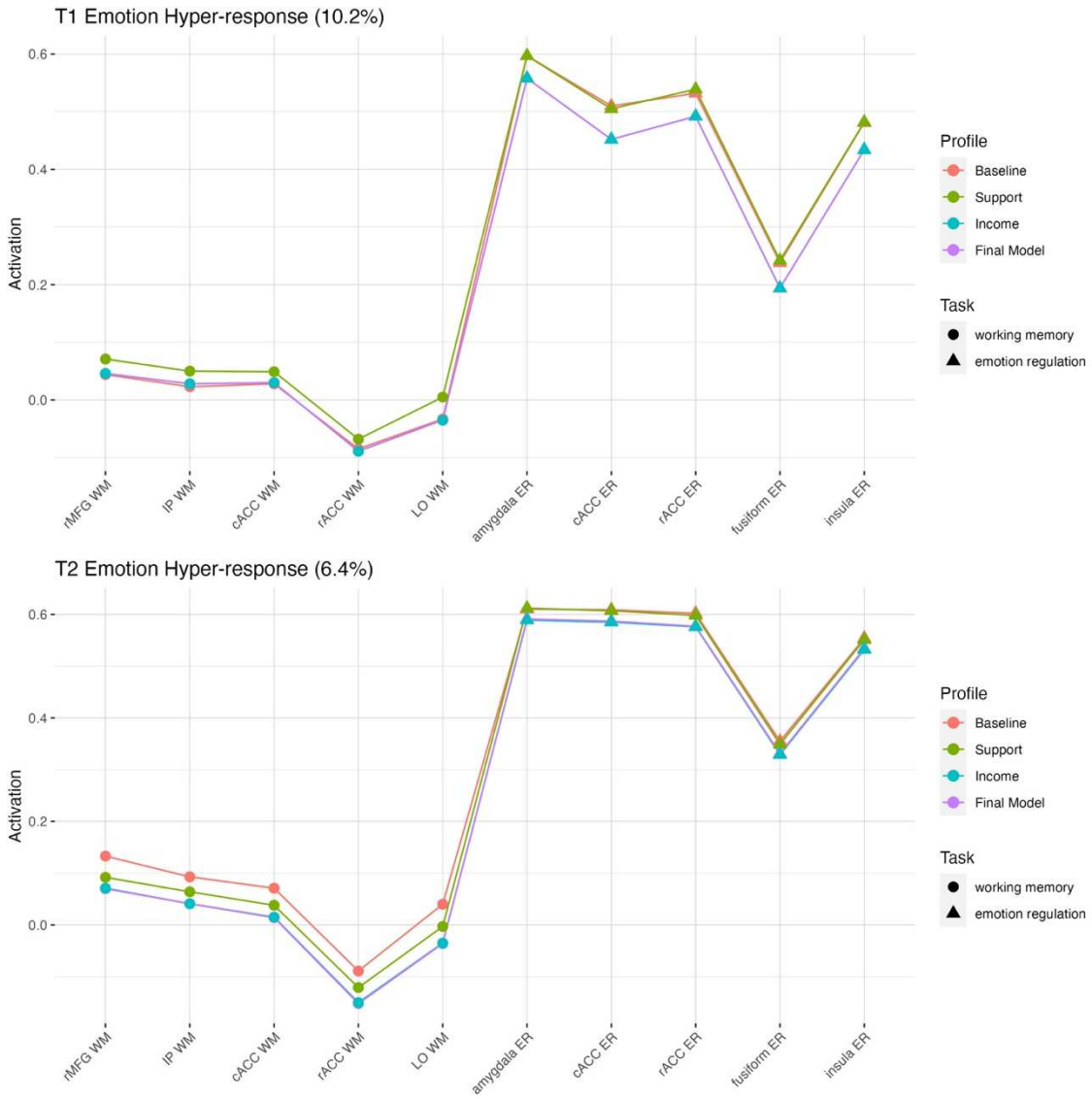

Figure 4.6. Fluctuation in item response probabilities of Emotion Hyper-response profiles after adding covariates.

When using the T1 and T2 Typical profiles as reference classes, child biological sex and family history of mental illness predicted profile membership (Table 4.9). Namely, male youths were significantly more likely to belong to the Emotion Hypo-response profile than the Typical profile at T1 (OR = 1.649, SE = 0.244, p = .008). Those with increased family history of mental illness were less likely to belong to the Emotion Hyper-response profile than the Typical profile at T2 (OR = .924, SE = .038, p =

.047). However, after correcting all p-values for family-wise error (16 tests total), these ORs were no longer significant (adjusted p-values, respectively: .128, .229).

Table 4.9. Logistic regression odds ratio results (Reference class: Typical)

	OR	SE	*p*	Adjusted *p*
T1 Emotion Hypo-response				
Support	0.849	0.166	0.363	0.449
Family history	0.954	0.033	0.171	0.327
Family income	0.953	0.03	0.118	0.27
Biological sex*	1.649	0.244	0.008	0.128
T1 Emotion Hyper-response				
Support	1.039	0.142	0.786	0.838
Family history	0.977	0.025	0.365	0.449
Family income	0.981	0.025	0.442	0.505
Biological sex	1.186	0.175	0.289	0.449
T2 Emotion Hypo-response				
Support	1.023	0.161	0.888	0.888
Family history	0.975	0.026	0.34	0.449
Family income	0.967	0.025	0.184	0.327
Biological sex	1.242	0.141	0.086	0.229
T2 Emotion Hyper-response				
Support	1.622	0.36	0.084	0.229
Family history	0.924	0.038	0.047	0.229
Family income	0.946	0.029	0.066	0.229
Biological sex	1.392	0.227	0.085	0.229

Note. *Female = 0, Male = 1. Odds ratios that are greater than 1 indicate that the event is more likely to occur as the predictor increases. Odds ratios that are less than 1 indicate that the event is less likely to occur as the predictor increases.

Step 4: Evaluating Mean Differences in T2 Psychopathology Across Latent Transitions

In the final step of model building, we used several Wald tests to determine whether mean differences in T2 internalizing and externalizing symptoms were significantly different across T2 latent profiles (Tables 4.10 and 4.11, Figure 4.7). Three comparisons were conducted for both internalizing and externalizing symptoms (six comparisons in total). According to results of each Wald test, factor scores of internalizing and externalizing did not differ significantly based on profile membership at T2. Although those belonging to the Emotional Hyper-response profile at T2 exhibited the highest factor scores of both

internalizing and externalizing, the non-significant Wald test indicates that the difference between psychopathology of the Emotion Hyper-response profile and other profiles was not significantly different from zero.

We followed this step with an exploratory analysis of mean differences in T2 EN-back task behavior (Tables 4.10 and 4.11, Figure 4.7). Specifically, we tested whether total accuracy rate (reported as a percentage) and response time (in milliseconds) differed significantly across T2 profiles. We found that total accuracy but not response time differed between the Typical and non-typical profiles at T2, such that those in the Emotion Hypo- and Hyper-response profiles showed significantly lower accuracy (86.8% and 86.5%, respectively) than those in the Typical profile (89%). These significant differences remained after correcting for multiple comparisons.

Table 4.10. Mean values of outcome variables across latent trajectories in final model

	*n**	%	Internalizing	Externalizing	Accuracy Rate	Response Time
Hypo to Hypo	105	1.5	0.019	0.077	0.868	889.601
Hypo to Hyper	69	1	0.04	0.084	0.865	887.899
Hypo to Typical	280	4	0.008	0.01	0.89	883.037
Hyper to Hypo	138	2	0.019	0.077	0.868	889.601
Hyper to Hyper	106	1.5	0.04	0.084	0.865	887.899
Hyper to Typical	606	8.6	0.008	0.01	0.89	883.037
Typical to Hypo	658	9.3	0.019	0.077	0.868	889.601
Typical to Hyper	306	4.3	0.04	0.084	0.865	887.899
Typical to Typical	4805	67.9	0.008	0.01	0.89	883.037

Note. Estimated *n* based on posterior probabilities rounded up to nearest whole number.

Table 4.11. Wald χ^2 tests of equality in outcome variables

	χ^{2}*	*p*	Adjusted *p*
Internalizing			
Hypo v. Hyper	0.077	0.7812	-
Hypo v. Typical	0.046	0.8301	-
Hyper v. Typical	0.256	0.6126	-
Externalizing			
Hypo v. Hyper	0.005	0.9439	-
Hypo v. Typical	0.895	0.3441	-
Hyper v. Typical	0.701	0.4024	-
Accuracy rate			
Hypo v. Hyper	0.077	0.7815	0.8656

Hypo v. Typical	8.13	0.004	0.012
Hyper v. Typical	13.959	0.0002	0.0012
Response time			
Hypo v. Hyper	0.029	0.8656	0.8656
Hypo v. Typical	1.045	0.3067	0.6134
Hyper v. Typical	0.333	0.5642	0.8463

Note. All tests have 1 degree of freedom. Hypo = Emotion Hypo-response, Hyper = Emotion Hyper-response.

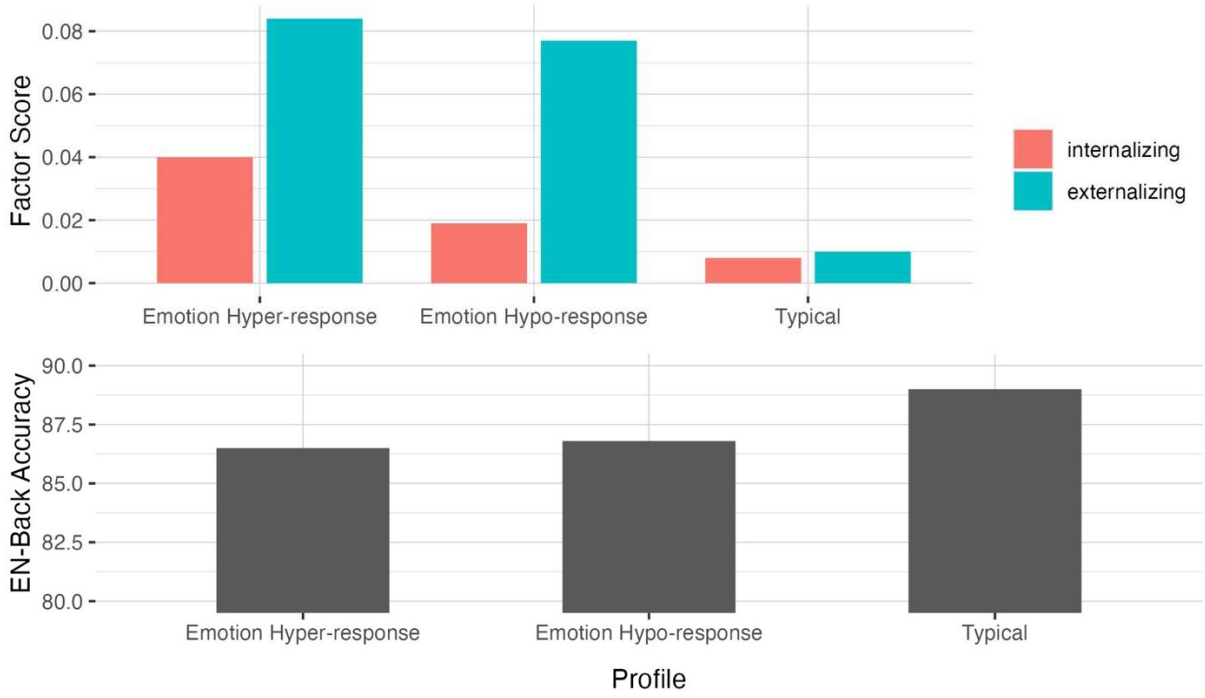

Figure 4.7. Mean differences in psychopathology and WM accuracy across profiles.

Discussion

Extant research suggests that neural maturation evinces high inter-individual variability and intra-individual change, and this variability may underlie vulnerability for psychopathology in adolescence. Using latent transition analysis, the current preregistered study employed a large, two-wave sample of early adolescents to test whether neural underpinnings of working memory and emotion regulation may be characterized by distinct patterns of function both at fixed time points and across 24 months. Moreover, we explored whether parental emotional support and other demographic variables at the family and individual levels significantly contributed to inter- and intra-individual variability across time (e.g.,

latent transitions). Finally, we investigated the relevance of homogenous subgroups of neural function to behavior by testing mean differences of internalizing and externalizing psychopathology and (in a follow-up analysis) EN-back task performance across T2 latent profiles.

Both latent profile and latent transition analyses yielded three distinct profiles of neural function at T1 and T2, which were characterized primarily by high, low, and moderate neural response (respectively labeled Emotion Hyper-response, Emotion Hypo-response, and Typical) during an ER task (e.g., the *faces vs. places* condition of the EN-back). Although our results did not fully support our hypotheses, many aspects of them were confirmatory. We anticipated a "low-regulation" profile characterized by attenuated WM response of the rostral middle frontal and ER response of the ACC. Although the Emotion Hypo-response profile did exhibit these qualities, most other regions in the profile also exhibited low response (e.g., greater deactivation). It should be noted that BOLD activation to *faces* stimuli within this profile was calculated in contrast to the *places* stimuli. As such, it could be that individuals within this profile are showing particularly attenuated neural responses to images of faces *or* particularly elevated neural responses to images of places. Nonetheless, healthy individuals tended to show attentional bias and elevated neural response viewing human facial stimuli compared to non-face stimuli (Royuela-Colomer et al., 2022; Xu et al., 2014). Lack of attention to facial stimuli, as well as blunted prefrontal, visual, and amygdala response to facial stimuli, has been previously linked to an array of maladaptive psychosocial and psychiatric outcomes, including depression, anxiety, schizophrenia, and psychopathic traits (Royuela-Colomer et al., 2022; Rubo et al., 2021; Kimonis et al., 2012; Park et al., 2016; Huffman & Oshri, 2022). Although we did not find those within this profile to evince greater frequency of psychopathology, this profile may highlight a group of youths particularly vulnerable to future maladaptation stemming from disruptions in emotion recognition.

We anticipated that youth who evince relatively higher levels of amygdalar response to ER would be paired with lower levels of response within the ACC. Instead—and similar to the Emotion Hypo-response profile—we found within the Emotion Hyper-response profile that activity other ER-related ROIs corresponded closely to that of the amygdala. The ACC in particular nearly matched the level of

activation of the amygdala at both T1 and T2. This may suggest that among youths with very elevated amygdala response to emotion, prefrontal regulatory regions such as the ACC have to "work harder" to modulate subcortical activity and facilitate emotion regulation (Oschner & Gross, 2014; Yang et al., 2020). However, the close positive association between amygdala and ACC function in the Hyper-response profile, as well as the relatively elevated amygdala function in other profiles, may be normative within the developmental period of the current sample (Silvers et al., 2017; Gee et al., 2013). In a study focusing on ER development from childhood to early adulthood, Silvers et al. (2017) found that increasing age predicted decreasing amygdala response and increased inverse coupling between the vmPFC and amygdala during a reappraisal task. Indeed, vmPFC-amygdala coupling did not become inverse in the cohort studied by Silvers et al. until approximately age 16. As more waves of ABCD data and other large longitudinal imaging cohorts are released, additional research will be necessary to identify the developmental periods at which high positive coupling between the amygdala and prefrontal regions is normative and, conversely, maladaptive.

Counter to our hypotheses, parental support did not predict profile membership at either time point. However, inclusion of both parental support and family income in the model significantly impacted patterns of brain activation within the Emotion Hyper-response profile. Although the limitations of this statistical evidence prevent us from drawing directional conclusions between parental support and WM- and ER-related neural function, it may indicate that parental support and family income are particularly relevant to neural function among youth evincing heightened response to emotional stimuli, particularly within the amygdala and ACC. Indeed, a large body of evidence using both functional MRI and physiological data suggests that neurobiologically "reactive" youths may be more sensitive to contextual inputs via the rearing environment than their less reactive counterparts (Liu et al., 2021) (Huffman et al., 2021; Roberts & Lopez-Duran, 2019; Guyer, 2020). Using data-driven longitudinal methods such as LTA may be a crucial next step to identifying profiles of neurobiological sensitivity to environmental inputs and their relevance to developmental outcomes among youths.

There are numerous established links between normative variations in parenting behavior, including parental warmth and support, and youth emotion regulation (Tottenham et al., 2018; Zeanah et al., 2009). However, studies on parenting as a precursor to child neurobiological development have largely focused on parenting behaviors outside of the normative range (e.g., neglect, abuse, psychiatric illness, and addiction; Green et al., 2010; McLaughlin et al., 2015; Herringa et al., 2013; McLaughlin et al., 2016). In a scoping review, Farber et al. (2020) identified ten studies that investigated normative range parenting and functional neurodevelopment among youths. Several studies identified by the authors found significant associations between positive parenting behaviors and neural function, including amygdala response to threat (Romund 2016; Farber 2018), striatal response to reward (Telzer et al., 2018), and prefrontal response during cognitive control tasks (Kim Spoon et al., 2017; McCormick et al., 2016; Telzer et al. 2013). Although invaluable, these studies are limited by a predominance of cross-sectional designs (six out of ten) and small sample sizes (eight out of ten under 200; all under 1000). Moreover, these studies focus on measurements of parenting and neural function in middle to late adolescence (13 to 18 years). The current study builds upon this nascent literature by highlighting a particular pattern of neural response among early adolescents that may confer additional sensitivity to the parenting and socioeconomic contexts.

When assessing mean differences in T2 behavior, we found no significant difference in internalizing and externalizing symptoms across T2 latent profiles. However, exploratory analyses suggested modest but significant differences in EN-back behavioral performance between the Typical and non-typical profiles, indicating that neural response to WM and ER conditions within the Hypo- and Hyper-response profiles may undermine performance during affective WM tasks. Although the current study did not find these same profiles to evince greater psychopathology, a large body of evidence suggests that WM performance in the context of affective information is itself a consistent correlate of psychopathology. Individuals exhibiting psychopathology often exhibit lower WM performance when simultaneously processing emotional information (Schwiezer et al. 2019; Huang Pollock et al., 2017). Conversely, interventions aimed at improving both affective and non-affective WM capacity are often

effective in improving emotion regulation and mitigating psychopathological symptoms (Xiu et al., 2018; Jopling et al., 2020). Should later studies find that those evincing similar patterns of neural response found in the current study are at greater risk for psychopathology, affective WM may be a compelling target for preventive intervention.

Given the highly variable nature of associations between brain function patterns and psychological phenotypes, the fact that we did not detect significant differences between psychopathology symptoms is not entirely surprising. First of all, this may be an example of equifinality, in which unique mechanisms precipitate a similar outcome. Examples of equifinality have been previously documented in brain-behavior associations, such as between patterns of aberrant reward processing and multiple types of psychiatric outcomes (e.g., mood disorders, schizophrenia, and addiction; Nusslock & Alloy, 2016; Pine & Fox, 2015). However, the null finding may also stem from increased methodological rigor. Recent studies and commentaries (Marek et al., 2022) have pointed out that many extant studies identifying direct links between neural function and psychopathology are limited by small sample size and unreliable phenotypic measurements, which in turn inflate effect sizes and increase risk for irreproducibility (Nikolaidis et al., 2022). When sample size is large, as in the current study, brain-behavior associations fail to reproduce or at the very least, show smaller effect sizes than comparable less-powered studies (Marek et al., 2022). That we did not find strong associations between neural function and psychopathology might also be a picture of the complex pathways underlying not only psychopathology but resilience as well. Evidence that fails to support direct developmental links between atypical neural function and psychopathology arguably undergirds the idea of resilience: individuals and their behaviors are greater than a sum of their biological and contextual "parts," however extensively those parts have been measured. Indeed, the complexity and inscrutability of the mediating and moderating processes that prevent inherent or inherited risk from developing into psychopathology cannot be overstated, as decades of developmental studies have confirmed.

The current study has several limitations. First, the use of only two time points limits our ability to draw conclusions beyond the brief period studied. Considering our predictors and outcomes were

assessed at T1 and T2, respectively, we were able to investigate cross-sectional associations only between covariates of interest and T1 neural function, as well as outcomes of interest and T2 neural function. Additionally, our use of mean values of regional activation across hemispheres prevented us from investigating lateralized contributions of neural response to LTA parameters. This choice was made after establishing within the current sample the high positive inter-hemispheric correlations of all ROIs to prevent model-overfitting and convergence difficulties. However, given that lateralization during WM and ER processes has been previously documented, additional research is necessary to determine how hemispheric differences impact the current profiles of neural function. Despite these limitations, our findings offer valuable insight into person-centered patterns of neural function underlying key cognitive processes across a crucial period of adolescent development. We hope that the current study establishes a framework by which others may investigate, replicate, and expound upon the profiles generated within later waves of the ABCD Study and other large neuroimaging cohorts.

CHAPTER 5

DISCUSSION

Introduction

The overall aim of this dissertation was to examine the development of adolescent externalizing behavior as a product of interactions between the parenting environment and the individual neurobiological context. I used the Research Domain criteria as a framework by which the complex etiological pathways of externalizing behavior might be elucidated in a methodological and cohesive way. Specifically, I employed multiple forms of measurement to conceptualize the RDoC domains of social processes, arousal, and cognition as contributors to externalizing behavior. The studies included in this dissertatation are further bolstered by use of longitudinal methods (all studies), latent variable modeling (all studies), large, population-based samples (*Study 2, Study 3*), and person-centered modeling approaches (*Study 3*). These methods further contribute to the field of developmental psychopathology and developmental cognitive neuroscience by characterizing specific mechanisms and contexts which both increase and mitigate risk for development of externalizing behavior across early adolescence. This section will include discussions of findings and central constructs therein; findings in context of the RDoC framework and the utility of the RDoC for psychopathology research; implications of findings as they relate to prevention and policy; and finally, methodological limitations and future directions.

Summary of Findings

Associations Between Parent-Child Social Processes and Child Outcomes

Chapters 2 and 3 assessed processes of affiliation and attachment by measuring effects of normative variations in harsh and supportive parenting behaviors, respectively, on youth externalizing behavior, while *Study 3* assessed effects of supportive parenting behaviors on development of neural function. Findings from all studies suggest that the construct of affiliation and attachment within the RDoC *social processes* domain may not fully explain the variation in externalizing behavior or functional

neural development among adolescents; additional domains of *arousal* and *negative valence* reflecting the child neurobiological context must also be considered. Chapter 2 showed that among a sample of low-income young adolescents, mothers' harsh parenting alone did not significantly predict development of aggressive and rule-breaking behavior 18 months later. Rather, harsh parenting interacted with youth sympathetic function at rest (basal pre-ejection period, or PEP) and parasympathetic function (resting sinus arrhythmia, or RSA) during acute stress to differentially predict aggressive and rule-breaking behaviors. Specifically, harsh parenting predicted increased rule-breaking among youth with high SNS activity (shortened PEP) at rest. Harsh parenting also predicted increase rule-breaking and aggression among youth with high PNS activity (RSA withdrawal) during acute stress. These findings partially confirm the BSC theory, indicating that parental harshness increases risk for low self-regulation and externalizing behaviors among those who already exhibit heightened autonomic function. However, because we did not test the interactions between physiological function and *positive* contexts (e.g., supportive parenting) and their effect on adaptive behavioral responses (e.g., prosociality), we were not able to conclusively determine whether the observed patterns of autonomic function reflect heightened "sensitivity" to the environment (for better *and* for worse) or simply dysregulated autonomic function. Notwithstanding, this study suggests that the effects of harsh parenting are inextricable from those of the child physiological context underlying arousal and consequent behavioral dysregulation. Youth exhibiting disruptions within the *arousal* domain (e.g., dysregulated autonomic function) may be more susceptible difficulties in EF development *particularly* when experiencing harsh parenting.

Findings from Chapter 3 suggest that the impact of supportive parenting on child antisocial behaviors (AB) is similarly moderated by child neural function within the *negative valence* domain. Contrary to our hypotheses, parental support did not directly predict development of child AB at either high or low levels of CU traits. Rather, parental support exacerbated the negative link between neural response to fearful faces within the visual cortex (inferior parietal cortex) and development of AB among high-CU youth. Namely, among those reporting low parental support, low inferior parietal response to fearful faces predicted greater AB 24 months later. These findings build upon the growing evidence that

suggests high-CU youth are malleable to parental inputs and socialization. In fact, in this study, high-CU youth reflected *more* malleability to parental support than low-CU youth, for whom we did not find any moderating effects of parenting on the link between neural response to emotion and AB development. In Chapter 3, we found that in the context of a parent who offers little emotional support (and presumptively little positive socialization), high-CU youth who display attenuated response within the visual cortex to potential threat (e.g., fearful faces) are more likely to develop heightened AB even across a relatively brief span of 24 months.

In Chapter 4, we identified three distinct latent profiles of neural function underlying working memory (WM) and implicit emotion regulation (ER) at ages 10 and 12. We then identified the likelihood of individuals transitioning from one latent profile to another (e.g., latent transitions). Similar to Studies 1 and 2, we found that parental support did not significantly predict profile membership at either time point or change individuals' likelihood for latent transition. It should be noted, however, that inclusion of parental support as a covariate conferred significant changes in mean ROI response within the Emotion Hyper-response profile. Although we were unable to conclude directional associations from this finding, it is notable that this particular sensitivity to parental support was limited to the Emotion Hyper-response profile, which was characterized by elevated prefrontal, amygdala, and insula response to facial expressions. The two other profiles identified—one characterized by moderate response to facial expressions (Typical), and another by high *de*activation to facial expressions (Emotion Hypo-response)—exhibited relative consistency after adding parental support to the model. As such, findings from Chapter 4 suggest parental support may be particularly relevant to youths exhibiting elevated neural response to not only fearful faces, as found in Chapter 3, but facial expressions in general. These three studies converge to suggest that normative variations in parenting impact development of child self-regulation *particularly* among those who evince elevated neurobiological reactivity to stressful or emotionally salient stimuli (Gard et al., 2018; Rudolph et al., 2018).

Associations Between Youth Neurobiological Function and Youth Outcomes

In Chapter 2, we found that disruptions within the *arousal* domain—namely, attenuated basal RSA—predicted youth delinquent behaviors 18 months later irrespective of variations in harsh parenting. This salient link between low parasympathetic function at baseline and dysregulated behaviors such as rule breaking is consistent with a number of studies that have found parasympathetic function at rest to proxy self-regulation, emotion regulation, and higher-order cognitive processes such as Theory of Mind and empathy (Dyer 2016; Eisenberg 2012; Belsky & Pluess 2009; Frith and Frith 1999). The well-documented link between dysregulated psychopathology and low basal RSA (Beauchaine 2015, Black 2021; Nguyen 2022) is underlied by the anatomical and functional connections between the medial prefrontal cortex and the parasympathetic nervous system (Beauchaine, 2015). Parasympathetic function is indeed closely linked to prefrontal activity in response to emotion (Lane et al., 2009) and prefrontal modulation of the amygdala (Sakaki et al., 2016). Results of Chapter 2 indicate that while effects of certain constructs within the *arousal* domain (e.g., basal sympathetic function, RSA reactivity) may be malleable to parenting behaviors, others, such as basal RSA, may evince a more severe and persistent neurobiological risk factor for behavioral dysregulation.

Similar to Chapter 2, in Chapter 3 we found that disruptions in neural function underlying the *negative valence* domain led to increased AB despite variations in parental emotional support. Interestingly, these direct effects were observed only among low-CU youth. Among this group, attenuated response to potential threat (e.g., fearful faces) within the left superior temporal sulcus and left fusiform gyrus (components of the face and emotion processing networks) predicted greater AB 24 months later. The specificity of these effects to fearful faces suggests that antisocial behaviors are a product of disruptions within the *negative valence* domain, evinced by low attentiveness to and recognition of fear in others, but not the *positive valence* domain. Previous research indicates that lack of attendance to visual cues of distress in others are indeed symptomatic of low empathy and low emotion regulation characteristic of those with AB (Sarker et al., 2011).

Individual Variability in Neural Function in Early Adolescence

In Chapter 4, the three latent profiles identified exhibited distinct patterns of neural response to facial stimuli (*faces vs. places* condition, conceptualized as emotion regulation in the study) but relatively similar patterns of response to working memory stimuli (*0-back vs. 2-back* condition). In late childhood and early adolescence, neural response to emotion is characterized by elevated activation within threat- and motivation-related circuitry (e.g., amygdala, insula, fusiform gyrus) and lower activation within prefrontal areas (e.g., medial prefrontal, anterior cingulate; Del Piero et al., 2016), which decreases and increases respectively throughout adolescence and adulthood. Chapter 4 builds upon these findings, showing that as a result of these normative patterns of maturation, neural response to facial stimuli evinced the greatest amount of individual variability at ages 10 and 12. Interestingly, these patterns of neural response underlying ER were not linked to differences in internalizing or externalizing symptoms, as we hypothesized. Rather, we observed significant differences in WM task performance (e.g., accuracy on the Emotion N-back task) between the Typical emotion response profile and the two atypical profiles. Whereas prior evidence among adults suggests that improved WM capacity leads to better ER (Xiu et al., 2016; Pe et al., 2013; Barkus, 2020), our findings indicate that neural underpinnings of ER are associated with—and possibily precede—WM ability in early adolescence. Although further studies are necessary to replicate and extend these findings across development, they offer preliminary evidence that ER-oriented treatments may be efficacious among children and early adolescents evincing delays in higher-order cognition.

Utility of RDoC in Studying Externalizing Behavior Development

Strengths of the RDoC Framework

RDoC's emphasis on specific neurobiological systems underlying psychopathology may be useful in identifying, in the words of Pasion et al. (2019), a "hierarchy of priority to intervention." RDoC-informed studies have great potential for intervention research, as they often identify neural, behavioral, and cognitive targets included in domains salient to development of specific forms of psychopathology. Two intervention programs to date have used RDoC methodology (Pasion, 2019). The first, Training for Awareness Resilience and Action (TARA), targets RDoC domains relevant to depression and anxiety,

including negative and positive valence and social processes, in order to mitigate neural and behavioral symptoms among adolescents (Blom et al., 2014; 2017). Similarly, another program called Engage targets constructs within the domains of negative valence, positive valence, and arousal to increase neurocognitive and emotional response to reward and mitigate depressive symptoms among adults (Alexopoulos et al., 2015; 2017). Both programs have yielded promising results, finding that both targeted interventions decrease depressive symptoms by increasing self-regulatory abilities (TARA) and reward responsiveness (Engage).

These programs highlight the potential practical utility of RDoC domains for translational and intervention research that may be extended to the current dissertation. For example, Chapter 3 highlighted the particular salience of neurocognitive disruptions within the *negative valence* domain in the development of AB and CU traits. In this same study, disruptions within the *positive valence* domain were not found to be predictive of AB, indicating that *negative valence* disruptions are more relevant to development of AB and thus more appropriate to target in a behavioral intervention program for youth with AB. Intervention researchers may compare this study to several others that, although employing unique measures (e.g., a behavioral task eliciting frustrative non-reward, or an imaging paradigm showing dangerous scenes), all identify constructs within the *negative valence* domain as central risk factors for AB (Dugré et al., 2020). In this way, the generality and flexibility of RDoC domains may facilitate cross-study and cross-method comparison to help identify overarching trends (e.g., significant domains) in etiological pathways for externalizing behaviors (Pacheco et al, 2022). Identification of these relevant RDoC domains may in turn allow intervention researchers to maximize the effectiveness of intervention targets with greater confidence.

Limitations of the RDoC Framework

A central limitation of the RDoC framework as it currently stands is its lack of attention to and inclusion of development across the lifespan. Although RDoC recommends inclusion of developmental processes in RDoC-informed studies, it fails to offer specific methodological recommendations. This ambiguity has led to a scarcity of RDoC-informed research that considered long-term developmental

processes in psychopathology (Beauchaine & Hinshaw, 2020; Mittal and Wakschlag 2018; Casey 2014; Ostlund 2021). Although RDoC studies reflect a wide range of developmental periods when taken together, most individual RDoC studies are confined to short periods of development (e.g., 1-3 years, Carcone & Ruocco, 2017).

The studies included in the current dissertation demonstrate this limitation as well: although our findings indicate that several patterns of aberrant neurobiological function are linked to externalizing behavior 18-24 months later, measurement of these same functions from early childhood to late adolescence/early adulthood is necessary to determine the full course of time-dependent neurobiological change across development. With a full developmental picture of even one specific neurobiological construct (e.g., working memory), researchers will be better equipped to determine (1) when aberrant or risky neurobiological function begins, (2) at what point aberrant neurobiological function is *most* predictive of psychopathology, and (3) ways in which continuities or discontinuities of neurobiological development are linked to time-specific environmental inputs (Franklin et al., 2015; Mittal and Wakschlag 2017; Rutter and Sroufe 2000).

Within the field of temperament research are a number of studies that have modeled development of RDoC-related constructs from early childhood through adolescence (Ostlund et al., 2021). Using multi-level and age-specific assessment methods, several temperament studies have identified as early as infancy moderately stable traits that reflect differences in emotionality and behavior. Traits such as inhibition (Kagan, 2018; Perez-Edgar & Fox, 2018) and irritability (Damme 2021; Beauchaine 2017; Wakschlag 2015; 2020) robustly confer risk for both internalizing and externalizing psychopathology in adolescence and adulthood. The advantages of these developmental studies are many: (1) disentangling trait level versus environmentally-initiated risk, (2) identifying whether risk factors exhibit continuity throughout childhood—and if not, identifying time-specific relevance of risk factors for psychopathology, and (3) leveraging ontogenetic data to create data-driven systems of vulnerability to psychopathology. These "mental health risk calculators" (MacNeill et al., 2021) may ultimately be used by clinicians and caretakers to identify and prevent psychopathology during critical periods of development.

Given financial and temporal constraints of researchers, it is unsurprising that psychopathology research often falls into two methodological camps. The first trades off breadth of measures for length and depth of assessment, measuring very few constructs across long spans of time, and often in multiple ways (Wakschlag). This camp may be better suited to answer the question, *When and how does psychopathology happen?* The second, which includes the studies within this dissertation, trades temporal depth in order to assess multiple aspects of the external and internal environment during a relatively brief period of time. This camp may be better suited to answer the question, *For whom does psychopathology happen?* Although merging these two methodological camps is clearly a goal of developmental research, it may be helpful to consider their unique practical advantages. Studies that address long-term development provide valuable information regarding timing and specificity of psychopathology development. However, multi-measure studies confined to shorter time periods, such as those in the present dissertation, are able to assess "snapshots" of contextual risk. For example, Chapter 2 indicates that youth evincing patterns of parasympathetic dysregulation during acute stress are particularly vulnerable to harsh parenting. Although addressing a relatively short time frame, findings like these may be equally helpful to clinicians and intervention researchers who aren't able to access or study a child's life history but still need evidence-based approaches for mitigating risk and improving mental health.

Implications for Prevention and Policy

Prevention and intervention programs most frequently target the endogenous, within-person causes of externalizing behaviors. This is understandable, considering the extensive evidence for neurocognitive vulnerabilities underlying externalizing psychopathology (as highlighted by this dissertation and elsewhere). As researchers learn more regarding the specific domains, constructs, and patterns of development that confer risk for externalizing behavior, intervention and prevention programs can better target these endogenous factors and ideally become more effective. However, as long as these programs *exclusively* target endogenous factors, they are limited in their ability to precipitate long-term change. As posited by the developmental psychopathology framework and confirmed by extensive research, externalizing behaviors are not only a product of how the individual responds to their

environment *but also of how the environment responds to the individual* (Baskin-Sommers, 2022). The current disseratation exemplifies this point in its study of both harsh and supportive parenting behaviors. The behavioral maladaptation of youths who exhibit neurocognitive vulnerabilities is often significantly heightened due to parents' negative responses (or lack of positive responses) to their children. Extant research among justice-involved adolescents indicates a similar pattern: offending youth report more frequent negative experiences among family members and peers, which increases their risk for negative emotionality and low self-regulation **(CITE)**. Evidence suggests that these negative interactions extend to mechanisms of formal social control as well: in schools, students evincing dysregulated behavior receive less direct instruction, more frequent negative feedback from teachers, less frequent praise from teachers, and fewer opportunities to respond in classroom discussion and activities (Sutherland et al., 2008; Kaufman & Brigham, 2009; Scott et al., 2011). Moreover, U.S. police officers are often untrained to deal constructively with those exhibiting dysregulated psychopathology (Rogers et al., 2019; Seo et al., 2021). As a result, conflicts between law enforcement and those exhibiting externalizing behaviors are more likely to become escalated and end in arrest (Livingston, 2016). Correctional facilities, too, overwhelmingly fail to create a constructive environment for dysregulated youth. Lack of or misuse of funding within juvenile corrections often leads to undertrained mental health professionals who fail to employ evidence-based treatment (Skowyra & Cocozza, 2007). Moreover, those exhibiting heightened dysregulated behavior in correctional facilities are placed in solitary confinement more often than those without (Whitley et al., 2016). It is well-documented that solitary confinement does not mitigate problem behaviors, but rather leads to increases in anxiety, depression, and psychiatric symptoms in adults and, to an even greater extent, adolescents (Clark, 2017).

If a gardener plants a seed in hard Georgia clay and waters the soil but does not fertilize it, they cannot expect the budding plants to thrive—they must address both aspects of the plants' environment, water *and* soil quality, to encourage healthy growth. Similarly, interventions that address only endogenous factors are inherently limited in their efficacy, because they fail to address the full scope of externalizing behavior development. The most effective measures will target exogenous change by

improving how the external environment responds to youth exhibiting externalizing behavior. These may look like programs to improve parent and teacher education and skills, especially regarding deescalating negative exchanges and providing emotional support (Tully & Hunt, 2016). Ideally, these measures will also include structural-level change. Currently, only 55% of schools in the U.S. provide diagnostic mental health assessments, and only 42% provide mental health treatment (NCES). In order to more effectively address dysregulated behaviors and mental health problems as a whole, a clear step is to equip both teachers and students with high-quality tools for identification and treatment that are free and consistently accessible (August et al., 2018). It is also paramount to improve law enforcement training to incorporate conflict resolution measures and work to eliminate punitive measures such as solitary confinement, especially within juvenile correctional facilities.

Limitations, Methodological Considerations, and Future Directions

Complexity of the Parenting and Family Context

Parenting is a multidimensional, complex, and dynamic process that includes a wide array of behaviors (Lindhiem & Shaffer, 2017). A specific parenting behavior, such as positive discipline, may be more adaptive within specific contexts and developmental periods (e.g., childhood versus adolescence; Bi et al., 2018). Outside of the challenges of defining which parenting behaviors are "positive" and "negative" across development, there is also the significant challenge of measuring parenting so that it reflects the true nature of the parent-child environment (Hurley et al., 2014). Although Chapter 2 included a novel assessment of harsh parenting behavior via coded observations of parent-child interaction, Chapter 3 and 4 both employed a single child-reported measure of parental support. Previous studies have indicated that more attention to measuring the complexity and dynamic change of parenting behaviors is necessary to properly inform parenting-oriented prevention efforts (Lindhiem et al., 2014; Serbin et al., 2015; Bennetts et al., 2016). As such, further study is necessary to assess the impact of multiple parenting behaviors, multiple reporters, and change in parenting behaviors across time on development of externalizing among youth.

Assessment of Additional Developmental Contexts

From the inception of Bonfenbrenner's Ecological Systems Theory (1979), processes within additional proximal contexts such as the neighborhood, community, and school have been linked to development of psychopathology (Steinberg et al., 2015; Hastings et al., 2020; Busso & McLaughlin, 2017). Recent studies have also indicated that factors within the neighborhood and school contexts may interact with and even precipitate youth neurobiological function underlying externalizing behaviors (Hyde et al., 2020; Gard et al., 2021; Rakesh et al., 2021; Gonzalez et al., 2020). These findings indicate that a more comprehensive assessment of the youth developmental context is necessary to further illuminate the etiology of externalizing behaviors, especially considering the increased relevance of the social context during adolescence.

A nascent body of research has begun to reveal associations between structural-level adversity and neurobiological development among youth. In a national study of adolescents by Weissman et al. (2021), associations between low income and small hippocampal volume were mitigated in states that offered greater financial support to low-income families. Associations between low income and psychopathology were similar, suggesting that anti-poverty policies may impact both structural brain development and associated mental health. In a meta-analysis of 22 studies, Hatzenbuehler et al. (2021) found that neural response to racial out-group members was elevated among those living in communities with higher levels of racial prejudice. Again, these findings suggest that structural differences in not only socioeconomic policy but race relations are linked individual neural function. Although additional research is necessary to replicate and explore the longitudinal implications of findings, these studies offer preliminary evidence that variations in policy, culture, and structural inequality have developmental ramifications for brain development underlying psychopathology.

Conclusions

This dissertation utilized longitudinal, multi-method, person-centered approaches to explore contributions of parenting and youth neurobiological function to development of dysregulated psychopathology in early adolescence. Informed by the frameworks of developmental psychopathology and Research Domain Criteria, our findings suggest several pathways by which parenting behaviors

interact with neurobiological function underlying arousal, negative valence processing, and social processing to predict externalizing behaviors. In Chapter 2, we showed that youths experiencing harsh parenting were more likely to develop aggressive and delinquent behaviors when exhibiting elevated parasympathetic stress reactivity and sympathetic function at rest. In Chapter 3, we found that CU trait severity underlied distinct associations between neural response to fearful faces and development of AB. Our results suggested that youths with high CU traits and blunted visual response to fearful faces exhibited the greatest vulnerability to negative effects of low parental support. In Chapter 4, our longitudinal person-centered analysis showed high within- and between-person variability in neural underpinnings of ER across early adolescence. Similar to Chapters 2 and 3, we found that a subset of youth evincing neural hyper-response to emotion were most sensitive to effects of parental support and family income on neural function over time. Dissimilar to the previous studies, however, we found that distinct neural function underlying ER was not linked to dysregulated psychopathology but rather to WM abilities. The findings of these three studies converge to suggest complex associations between multiple social and emotional processes that precipitate not only development of self-regulation but higher-order cognition as well. Given the rise of large-scale and publicly accessible neuroimaging cohorts such as the ABCD Study, as well as improvements in biological and phenotypic measurements, methodological rigor and reproducibility are at the forefront of developmental cognitive neuroscience. In my own and others' future work, I hope to see these results replicated and extended longitudinally, with greater focus on potential targets for intervention and prevention among at-risk youth.

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APPENDICES

Chapter 3 Appendix

Neuroimaging Methods

Image Acquisition. Each MRI scanning session included T1w and T2w sMRI, one dMRi, and four rs-fMRI series, as well as 2 task fMRI series for each of three tasks (MID, SST, EN-back). Scans were ordered as follows: localizer, 3D T1-weighted images, 2 runs of resting state fMRI, diffusion-weighted images, 3D T2-weighted images, 2 runs of resting state, and 3 fMRI tasks. FMRI tasks were randomized across subjects, except for siblings (who received tasks in the same order), but task order remained consistent across each wave within subjects. Scan sessions lasted about 2 hours but were sometimes split into 2 sessions that took place within a week of each other (3.5% of participants). FMRI acquisitions (2.4 mm isotropic, TR = 800 ms) used multi band EPI with slice acceleration factor 6. FMRI acquisition blocks included fieldmap scans for B0 distortion correction.

Protocol Compliance & Preprocessing (direct quote from Hagler et al., 2019). *Using a combination of automated and manual methods, we review datasets for problems such as incorrect acquisition parameters, imaging artifacts, or corrupted data files. Automated protocol compliance checks providing information about the completeness of the imaging series and the adherence to the intended imaging parameters. Out-of-compliance series are reviewed by DAIC staff, and sites are contacted if corrective action is required. Protocol compliance criteria include whether key imaging parameters, such as voxel size or repetition time, match the expected values for a given scanner. For dMRI and fMRI series, the presence or absence of corresponding B_0 distortion field map series is checked. Each imaging series is also checked for completeness to confirm that the number of files matches what was expected for each series on each scanner. Missing files are typically indicative of either an aborted scan or incomplete data transfer, of which the latter can usually be resolved through re-initiating the data transfer. Errors in the unpacking and processing of the imaging data at various stages are tracked, allowing for an assessment*

of the number of failures at each stage and prioritization of efforts to resolve problems and prevent future errors.

Automated quality control procedures include the calculation of metrics such as signal-to-noise ratio (SNR) and head motion statistics. For sMRI series, metrics include mean and SD of brain values. For dMRI series, head motion is estimated by registering each frame to a corresponding image synthesized from a tensor fit, accounting for variation in image contrast across diffusion orientations (Hagler et al., 2009). Overall head motion is quantified as the average of estimated frame-to-frame head motion, or FD. Dark slices, an artifact indicative of abrupt head motion, are identified as outliers in the root mean squared (RMS) difference between the original data and data synthesized from tensor fitting. The total numbers of the slices and frames affected by these motion artifacts are calculated for each dMRI series. For fMRI series, measures include mean FD, the number of seconds with FD less than 0.2, 0.3, or 0.4 mm (Power et al., 2012), and temporal SNR (tSNR) (Triantafyllou et al., 2005) computed after motion correction.

Trained technicians visually review image series as part of our manual QC procedures, including T_1w, T_2w, dMRI, dMRI field maps, fMRI, and fMRI field maps. Reviewers inspect images for poor image quality, noting various imaging artifacts and flagging unacceptable data, typically those with the most severe artifacts or irregularities. For example, despite the use of prospective motion correction for sMRI scans, which greatly reduces motion-related image degradation (Brown et al., 2010; Kuperman et al., 2011; Tisdall et al., 2016), images of participants with excessive head motion may exhibit severe ghosting, blurring, and/or ringing that makes accurate brain segmentation impossible. Reviewers are shown several pre-rendered montages for each series, showing multiple slices and views of the first frame, and multiple frames of individual slices if applicable. For multi-frame images, linearly spaced subsets of frames are shown as a 9 × 9 matrix of 81 frames. For dMRI and fMRI, derived images are also shown. For dMRI series, derived images include the average b = 0 image, FA, MD, tensor fit residual error, and DEC FA map. For fMRI series, derived images include the average across time and the temporal SD (computed following motion correction). All series are consensus rated by two or more

reviewers. In the case of a rejection, the reviewer is required to provide notes indicating the types of artifacts observed using a standard set of abbreviations for commonly encountered artifacts. Series rejected based on data quality criteria are excluded from subsequent processing and analysis.

FMRI Preprocessing. Head motion was corrected by registering each frame to the first with AFNI's 3dvolreg (Cox, 1996). This method provides estimates of head motion time courses that are then incorporated into task- and resting-state-fMRI single subject analyses. B_0 distortions were corrected by aligning pairs of non-diffusion weighted images with opposite phase encoding polarities using a nonlinear registration procedure; the estimated displacement field volume was then used to correct distortions in each frame (Holland et al. 2010). To avoid signal "drop-out" due to within-voxel field gradients in gradient-echo acquisitions, the displacement field was estimated from separate spin-echo calibration scans, adjusted for estimated between-scan head motion, and applied to the series of gradient-echo images. Images were then corrected for distortions due to gradient nonlinearities (Jovicich 2006), with each scan resampled with cubic interpolation into alignment with a reference scan (e.g., the scan nearest to the middle of the set of the participant's fMRI scans). Registration between spin-echo field maps and T1w structural images was completed using mutual information (Wells, 1996) with coarse pre-alignment based on within-modality registration to atlas brains. These methods yielded fMRI images with 2.4 mm isotropic resolution.

Behavioral measures: EN-back (direct quote from Hagler et al., 2019). *With a block design of 0-back and 2-back working memory tasks, participants indicate by button press the repeated presentation of images. Image types include emotionally positive, negative, or neutral faces or pictures of places. Behavioral metrics include: total number of trials presented, number of correct responses, and accuracy (correct responses divided by total trials), and mean and SD of reaction times for correct responses. Following the imaging session, the EN-Back Recognition Memory task asks the participant to decide if pictures presented were seen previously in the EN-Back task. For each stimulus type (old and new), hit rates and false alarm rates are calculated. Additional metrics include corrected accuracy (hit rate from working memory task minus false alarm rate from recall task), response bias, and d-prime. Poor*

performance is indicated if the overall response accuracy for the 0-back or 2-back blocks is less than 60%.

Preprocessing of task fMRI (direct quote from Hagler et al., 2019). *Estimates of task-related activation strength are computed at the individual subject level using a general linear model (GLM) and an ROI-based approach.* ***Pre-analysis processing:*** *Processing steps subsequent to fMRI preprocessing include the removal of initial frames[4] to ensure equilibration of the T_1w signal and normalization of voxel time series by dividing by the mean across time of each voxel.* ***Surface sampling:*** *Preprocessed time courses are sampled onto the cortical surface for each individual subject. Voxels containing cortical gray matter are projected onto the surface by sampling values 1 mm from the gray/white boundary, into cortical gray matter, along the surface normal vector at each vertex (using FreeSurfer's mri_vol2surf with "white" surface, "-projdist 1" option, and default "nearest" interpolation).* ***ROI values extraction:*** *Average time courses are calculated for cortical surface-based ROIs using FreeSurfer's anatomically-defined parcellations (Desikan et al., 2006; Destrieux et al., 2010) and subcortical ROIs (Fischl et al., 2002).* ***Nuisance regressors:*** *Baseline and quadratic trends in the time-series data are included in the analysis. Motion estimates and their derivatives are also included as regressors (Power et al., 2014). Time points with FD greater than 0.9 mm are censored (Siegel et al., 2014). Estimated motion time courses used for regression and censoring are temporally filtered using an infinite impulse response (IIR) notch filter to attenuate signals in the range of 0.31–0.43 Hz. This frequency range corresponds to empirically observed oscillatory signals in motion estimates linked to respiration and the dynamic changes in magnetic susceptibility due to lung movement in the range of 18.6–25.7 respirations/minute. With the removal of these fast oscillations linked to respiration, the filtered motion estimates and FD values more accurately reflect actual head motion (Fair et al., 2018).* ***General linear model:*** *Estimates of task-related activation strength are computed at the individual subject level using a general linear model (GLM) implemented in AFNI's 3dDeconvolve (Cox, 1996). Hemodynamic response functions are modelled with two parameters using a gamma variate basis function plus its temporal derivative (using AFNI's 'SPMG' option within 3dDeconvolve). Task models include stimulus timing for*

each condition and linear contrasts of conditions (see Supp. Tables 8-10). For MID and SST analyses, events are modelled as instantaneous; for EN-back, the duration of cues (~3 s) and trial blocks (~24 s) are modelled as square waves convolved with the two parameter gamma basis function (i.e., block duration specified when using AFNI's 'SPMG' option). Outputs include GLM beta coefficients and standard errors of the mean (SEM; calculated from the ratio of the beta and t-statistic) calculated for each voxel, vertex, or ROI time series. ***Averaging across runs:*** *ROI average beta coefficients and standard errors are computed for each of two runs. We compute the average across runs for each participant weighted by the nominal degrees of freedom (number of frames remaining after motion censoring minus number of model parameters, but not accounting for temporal autocorrelation), which differs between runs due to motion censoring. Runs with fewer than 50 degrees of freedom are excluded from the average between runs.* ***Censoring invalid contrasts:*** *The frequency and magnitude of head movements varies widely in children. Some participants exhibit frequent periods of motion resulting in greatly reduced numbers of time points used to estimate model parameters. Depending on when supra-threshold head movements (FD > 0.9 mm) occur relative to instances of a given event type, rare conditions may be under-represented in some participants, or lack representation entirely. For unrepresented conditions, beta and SEM values are undefined and shared as empty cells in the tabulated data. If conditions are under-represented, the design matrix of the GLM analysis becomes ill-conditioned, making estimated beta weights unreliable for those conditions and the contrasts that include them. In rare cases, this results in extreme values for the beta and SEM estimates, as much as several orders of magnitude different from typical values for a given contrast. The presence of extreme outliers violates standard parametric assumptions, so group-level statistical analyses can produce invalid and nonsensical results. To prevent this, we censor the beta and SEM values if they are identified as having extremely high SEM values and therefore low reliability beta estimates. For a given subject with an extreme value for a particular contrast and ROI, there are typically outliers in other brain regions for the same subject and contrast and generally greater variation across brain regions. We censor the beta and SEM values for all ROIs for those contrasts that have RMS of SEM values across the cortical surface greater than 5% signal*

change. This represents less than 0.5% of all subject-task-contrast-run combinations. The censored values are replaced with empty cells.

Quality Control Methods

To average BOLD responses across runs, ROI average beta coefficients and standard errors were computed for each of the two task runs. The average across runs were then computed for each participant weighted by the nominal degrees of freedom (frames remaining after motion censoring minus the number of model parameters, but not accounting for temporal autocorrelation), which differs between runs due to motion censoring. Runs with less than 200 degrees of freedom were recoded as missing in the analyses.

Although techniques were used to mitigate movement in the scanner (e.g., real-time motion correction and monitoring), the young age of the subjects and the length of the scan resulted in some scans failing the quality control assessment as established by Hagler et al. (2019). Available MRI images were passed through mandatory filtering (Figure 2) that excluded incomplete or poor-quality data prior to creation of minimally processed data, leading to EN-back tabulated data for 9,504 participants. Next, minimally processed data were passed through further filtering to exclude imaging data for subjects with incidental findings, unacceptable FreeSurfer reconstruction, excessive head motion, and poor behavioral performance (Table A1). Performance was deemed "poor" if overall response accuracy for the 0-back or 2-back blocks was less than 60%. Consequently, full EN-back imaging data remained for 7,472 participants.

Table A1. N of EN-back data before and after quality control

Subjects with complete QC data		Subjects without incidental findings		Subjects who pass Freesurfer QC		Subjects with at least one complete series		Subjects with acceptable performance		Subjects with acceptable degrees of freedom	
T1	T5	T1	T5	T1	T5	T1	T5	T1	T5	T1	T5
7,472	4,258	11,235	5,404	11,265	5,583	10,190	5,186	8,573	4,843	9,505	4,814

Missing data. In addition to data missing from imaging assessments, data was missing on variables from both T1 (n = 11,881) and T5 (n = 6,571). Specifically, data was missing for age ($n_{missing}$ = 11), household income ($n_{missing}$ = 1018), parent education ($n_{missing}$ = 14), parental support ($n_{missing}$ = 34), baseline CU trait indicators ($n_{missing}$ = 64), baseline and T5 aggression and rule-breaking (respectively: $n_{missing}$ = 8, $n_{missing}$ = 5331), baseline and T5 ODD (respectively: $n_{missing}$ = 3895, $n_{missing}$ = 9906), and baseline and T5 CD

(respectively: $n_{missing} = 4426$, $n_{missing} = 10528$). Although previous studies using the current sample have employed listwise deletion techniques (e.g., participants missing data on any variables are excluded from analyses), this method can yield biased parameter estimates when data does not meet missing-completely-at-random (MCAR) assumptions (Enders, 2001, 2006). As such, the current study employs the full-information maximum likelihood with robust standard errors (MLR) method, which effectively and accurately estimates missing data under the assumption of MAR (Enders & Bandalos, 2001).

Latent Variable Modeling of Antisocial Behaviors

Four separate but corresponding confirmatory factor analyses (CFA) of AB were conducted: within the Low CU group at T1 and T5 and within the High CU group at T1 and T5. All models exhibited acceptable fit at both time points (Low CU, T1: χ^2 (2) = 106.529, p < .000; RMSEA = 0.066; CFI = 0.98; SRMR = 0.025; Low CU, T5: χ^2 (2) = 35.847, p < .000; RMSEA = 0.051; CFI = 0.98; SRMR = 0.031; High CU, T1: χ^2 (2) = 17.101, p = .0002; RMSEA = 0.103; CFI = 0.98; SRMR = 0.023; High CU, T5: χ^2 (2) = 2.801, p = .247; RMSEA = 0.031; CFI = 0.99; SRMR = 0.021). All factor loadings were significant and above .30, as suggested by Brown [55].

Direct Effects of Predictors on CU Traits

Table A2. Direct effects of predictors on continuous CU traits*

	b	*SE*	*p*	*β*	*SE*	*p*	Adjusted *p*	*95% CI*
Support	-0.203	0.052	0	-0.043	0.011	0	0	[-.061, -.024]
Conduct problems	0.018	0.005	0.001	0.07	0.02	0.001	0.001	[.037, .104]
Oppositional defiance	0.028	0.005	0	0.107	0.019	0	0	[.076, .137]
Youth age	0.012	0.002	0	0.063	0.01	0	0	[.047, .079]
Youth biological sex	0.122	0.029	0	0.041	0.01	0	0	[.025, .058]
Parent education	0.027	0.017	0.11	0.022	0.014	0.11	0.11	[-.001, .045]
Household income	-0.025	0.009	0.005	-0.042	0.015	0.005	0.007	[-.066, -.018]
Right fusiform gyrus	-0.097	0.043	0.024	-0.029	0.013	0.022	0.176	[-.050, -.008]

Note. All models controlled for CU traits at T1.

Results of Moderation Models with Covariates

Table A3. Results of stepwise moderation analyses without family-wise error correction

			Fearful vs. neutral, Low CU				
Left Hemisphere	*b*	*SE*	*p*	*B*	*SE*	*p*	*95% CI*
Right LOFC (n = 7199)	-0.807	0.295	0.006	-0.053	0.02	0.008	[-.086, -.020]
Right LOFC (n = 10606)	-1.061	0.39	0.007	-0.065	0.025	0.01	[-.107, -.024]
Right LOFC (n = 7199) + ANX/ADHD	-0.785	0.284	0.006	-0.052	0.019	0.007	[-.119, -.015]
Right LOFC (n = 10606) + ANX/ADHD	-1	0.351	0.004	-0.059	0.023	0.011	[-.098, -.014]
			Fearful vs. neutral, High CU				
Right insula (n = 401)	6.112	3.107	0.049	0.165	0.084	0.049	[.027, .303]
Right insula (n = 684)	5.388	4.435	0.224	0.13	0.105	0.215	[-.043, .303]
Right insula (n = 401) + ANX/ADHD	6.064	2.802	0.03	0.14	0.066	0.033	[.032, .248]
Right insula (n = 684) + ANX/ADHD	8.476	4.121	0.04	0.184	0.091	0.043	[.035, .333]
Left cMFG (n = 401)	7.692	3.549	0.03	0.185	0.083	0.026	[.048, .322]
Left cMFG (n = 684)	7.377	4.444	0.097	0.159	0.097	0.101	[0.00, .319]
Left cMFG (n = 401) + ANX/ADHD	8.318	3.522	0.018	0.171	0.072	0.017	[.053, .289]
Left cMFG (n = 684) + ANX/ADHD	11.303	4.686	0.016	0.219	0.094	0.019	[.065, .373]
Right IPS (n = 401)	6.686	3.273	0.041	0.174	0.083	0.037	[.037, .311]
Right IPS (n = 684)	6.598	4.154	0.112	0.154	0.096	0.109	[-.004, .312]
Right IPS (n = 401) + ANX/ADHD	7.108	3.429	0.038	0.177	0.085	0.036	[.038, .317]
Right IPS (n = 684) + ANX/ADHD	9.762	4.374	0.026	0.205	0.092	0.026	[.053, .357]
Left IPS (n = 401)	9.149	3.66	0.012	0.218	0.084	0.01	[.079, .356]
Left IPS (n = 684)	9.538	4.604	0.038	0.204	0.1	0.041	[.040, .204]
Left IPS (n = 401) + ANX/ADHD	9.774	3.525	0.006	0.199	0.071	0.005	[.082, .315]
Left IPS (n = 684) + ANX/ADHD	13.636	4.563	0.003	0.262	0.091	0.004	[.112, .412]

Results Prior to Correcting for Multiple Comparisons

Table A4. Direct effects

				Low CU					
	b	*SE*	*p*	*β*	*SE*	*p*	Adj. *p*	*95% CI*	R^2
Support	-0.065	0.206	0.754	-0.006	0.019	0.754		[-.038, .026]	0
				Fearful vs. Neutral					
L superior temporal sulcus	-0.541	0.198	0.006	-0.05	0.018	0.006	0.048	[-.08, -.02]	0.088
Left fusiform gyrus	-0.481	0.195	0.014	-0.062	0.025	0.014	0.056	[-.103, -.021]	0.088
				Happy vs. Neutral					
R pars triangularis	-0.267	0.137	0.052	-0.038	0.019	0.046	0.828	[-.068, -.007]	0.086
				High CU (Fearful versus. Neutral)					
	b	***SE***	***p***	***β***	***SE***	***p***		***95% CI***	
Support	0.729	0.757	0.336	0.053	0.055	0.334		[-.037, .143]	0.003
L rostral ACC	-1.917	0.696	0.006	-0.18	0.068	0.008	0.144	[-.292, -.069]	27.5

Note. All effects control for ADHD and anxiety disorders. No statistically significant demographic predictors were found for LCU or HCU groups.

Table A5. Results of moderation analyses

	Fearful vs. neutral, Low CU (n = 10,606)								
	b	*SE*	*p*	*B*	*SE*	*p*	Adj. *p*	*95% CI*	R^2
R lateral orbitofrontal	-1	0.351	0.004	-0.059	0.023	0.011	0.198	[-.098, -.014]	0.089
	Fearful vs. neutral, High CU (n = 684)								
R insula	8.476	4.121	0.04	0.184	0.091	0.043	0.387	[.035, .333]	0.272
L caudal middle frontal	11.303	4.686	0.016	0.219	0.094	0.019	0.342	[.065, .373]	0.293
R inferior parietal	9.762	4.374	0.026	0.205	0.092	0.026	0.016	[.053, .357]	0.289
L inferior parietal	13.636	4.563	0.003	0.262	0.091	0.004	0.052	[.112, .412]	0.309

Note. All models control for effects of ADHD and anxiety disorders. Non-significant demographic covariates were trimmed from the models.

Chapter 4 Appendix

Mplus Code for Steps 1 Through 4

```
TITLE: step 0 repeated measures lpa 3 class
DATA: FILE = "lta.dat";
VARIABLE:
NAMES = subid siteid wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1
      emo_rac_m_1 emo_ff_m_1 emo_ins_m_1 emo_amy_m_1 emo_cac_m_1
          wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2
          emo_amy_m_2 emo_rac_m_2 emo_cac_m_2 emo_ff_m_2 emo_ins_m_2
          ext int beh_mean_rt_2 beh_tot_cor_2 beh_rate_cor_2
          rel_family_id ppensity serial support income famhx ysex yage;
MISSING=.;

!run time 1 and time 2 separately

!time 1:
usevar =
wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1
emo_rac_m_1 emo_ff_m_1 emo_ins_m_1 emo_amy_m_1 emo_cac_m_1;

!time 2
!wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2
!emo_amy_m_2 emo_rac_m_2 emo_cac_m_2 emo_ff_m_2 emo_ins_m_2;


classes = c1(3);
!classes = c2(3);

weight = ppensity;                          !weight with propensity scores
cluster is rel_family_id;                   !nest within families
stratification is serial;                   !nest within scanner

analysis:
type = complex;
type = mixture;
estimator = mlr;
starts = 500 20;
```

```
TITLE: step 1-2 measurement invariance and LTA 3 class solution
DATA: FILE = "lta.dat";
NAMES = subid siteid wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1
      emo_rac_m_1 emo_ff_m_1 emo_ins_m_1 emo_amy_m_1 emo_cac_m_1
         wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2
         emo_amy_m_2 emo_rac_m_2 emo_cac_m_2 emo_ff_m_2 emo_ins_m_2
         ext int beh_mean_rt_2 beh_tot_cor_2 beh_rate_cor_2
         rel_family_id ppensity serial support income famhx ysex yage;
MISSING=.;

!time 1:
usevar =
wm_rmf_m_1
wm_rac_m_1
wm_cac_m_1
wm_ip_m_1
wm_lo_m_1
emo_rac_m_1
emo_ff_m_1
emo_ins_m_1
emo_amy_m_1
emo_cac_m_1

!time 2
wm_rmf_m_2
wm_ip_m_2
wm_cac_m_2
wm_rac_m_2
wm_lo_m_2
emo_amy_m_2
emo_rac_m_2
emo_cac_m_2
emo_ff_m_2
emo_ins_m_2
;

classes = c1(3) c2(3);

weight = ppensity;                          !weight with propensity scores
cluster is rel_family_id;                   !nest within families
stratification is serial;                   !nest within scanner

analysis:
type = complex;
type = mixture;
estimator = mlr;
starts = 500 20;

model:
%overall%
```

```
c2 on c1;

model c1:
%c1#1%
! for measurement non-invariance remove parameter constraints
! [wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1 emo_rac_m_1 emo_ff_m_1
!  emo_ins_m_1 emo_amy_m_1 emo_cac_m_1];
[wm_rmf_m_1] (1);
[wm_rac_m_1] (2);
[wm_cac_m_1] (3);
[wm_ip_m_1] (4);
[wm_lo_m_1] (5);
[emo_rac_m_1] (6);
[emo_ff_m_1] (7);
[emo_ins_m_1] (8);
[emo_amy_m_1] (9);
[emo_cac_m_1] (10);


%c1#2%
! [wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1 emo_rac_m_1 emo_ff_m_1
!  emo_ins_m_1 emo_amy_m_1 emo_cac_m_1];
[wm_rmf_m_1] (13);
[wm_rac_m_1] (14);
[wm_cac_m_1] (15);
[wm_ip_m_1] (16);
[wm_lo_m_1] (17);
[emo_rac_m_1] (18);
[emo_ff_m_1] (19);
[emo_ins_m_1] (20);
[emo_amy_m_1] (21);
[emo_cac_m_1] (22);


%c1#3%
! [wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1 emo_rac_m_1 emo_ff_m_1
!  emo_ins_m_1 emo_amy_m_1 emo_cac_m_1];
[wm_rmf_m_1] (25);
[wm_rac_m_1] (26);
[wm_cac_m_1] (27);
[wm_ip_m_1] (28);
[wm_lo_m_1] (29);
[emo_rac_m_1] (30);
[emo_ff_m_1] (31);
[emo_ins_m_1] (32);
[emo_amy_m_1] (33);
[emo_cac_m_1] (34);

model c2:
%c2#1%
![wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2 emo_amy_m_2 emo_rac_m_2
```

```
! emo_cac_m_2 emo_ff_m_2 emo_ins_m_2];
[wm_rmf_m_2] (1);
[wm_ip_m_2] (2);
[wm_cac_m_2] (3);
[wm_rac_m_2] (4);
[wm_lo_m_2] (5);
[emo_amy_m_2] (6);
[emo_rac_m_2] (7);
[emo_cac_m_2] (8);
[emo_ff_m_2] (9);
[emo_ins_m_2] (10);


%c2#2%
![wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2 emo_amy_m_2 emo_rac_m_2
! emo_cac_m_2 emo_ff_m_2 emo_ins_m_2];
[wm_rmf_m_2] (13);
[wm_ip_m_2] (14);
[wm_cac_m_2] (15);
[wm_rac_m_2] (16);
[wm_lo_m_2] (17);
[emo_amy_m_2] (18);
[emo_rac_m_2] (19);
[emo_cac_m_2] (20);
[emo_ff_m_2] (21);
[emo_ins_m_2] (22);


%c2#3%
![wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2 emo_amy_m_2 emo_rac_m_2
! emo_cac_m_2 emo_ff_m_2 emo_ins_m_2];
[wm_rmf_m_2] (25);
[wm_ip_m_2] (26);
[wm_cac_m_2] (27);
[wm_rac_m_2] (28);
[wm_lo_m_2] (29);
[emo_amy_m_2] (30);
[emo_rac_m_2] (31);
[emo_cac_m_2] (32);
[emo_ff_m_2] (33);
[emo_ins_m_2] (34);

!!!!!!!! code below is for partial measurement invariance !!!!!!!!

!model c1:
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![wm_rmf_m_1] (1);
![wm_rac_m_1] (2);
![wm_cac_m_1] (3);
![wm_ip_m_1] (4);
![wm_lo_m_1] (5);
```

```
![emo_rac_m_1];
![emo_ff_m_1];
![emo_ins_m_1];
![emo_amy_m_1];
![emo_cac_m_1] ;

!%c1#2%
![wm_rmf_m_1] ;
![wm_rac_m_1] ;
![wm_cac_m_1] ;
![wm_ip_m_1] ;
![wm_lo_m_1] ;
![emo_rac_m_1];
![emo_ff_m_1];
![emo_ins_m_1];
![emo_amy_m_1];
![emo_cac_m_1];

!%c1#3%
![wm_rmf_m_1] (1);
![wm_rac_m_1] (2);
![wm_cac_m_1] (3);
![wm_ip_m_1] (4);
![wm_lo_m_1] (5);
![emo_rac_m_1];
![emo_ff_m_1];
![emo_ins_m_1];
![emo_amy_m_1];
![emo_cac_m_1] ;

!model c2:
!%c2#1%
![wm_rmf_m_2] (11);
![wm_ip_m_2] (12);
![wm_cac_m_2] (13);
![wm_rac_m_2] (14);
![wm_lo_m_2] (15);
![emo_amy_m_2];
![emo_rac_m_2];
![emo_cac_m_2];
![emo_ff_m_2];
![emo_ins_m_2];

!%c2#2%
![wm_rmf_m_2] (11);
![wm_ip_m_2] (12);
![wm_cac_m_2] (13);
![wm_rac_m_2] (14);
![wm_lo_m_2] (15);
![emo_amy_m_2];
![emo_rac_m_2];
```

```
![emo_cac_m_2];
![emo_ff_m_2];
![emo_ins_m_2];

!%c2#3%
![wm_rmf_m_2] (11);
![wm_ip_m_2] (12);
![wm_cac_m_2] (13);
![wm_rac_m_2] (14);
![wm_lo_m_2] (15);
![emo_amy_m_2];
![emo_rac_m_2];
![emo_cac_m_2];
![emo_ff_m_2];
![emo_ins_m_2];
```

```
TITLE: step 3 test covariates
DATA: FILE = "lta.dat";
VARIABLE:
NAMES = subid siteid wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1
      emo_rac_m_1 emo_ff_m_1 emo_ins_m_1 emo_amy_m_1 emo_cac_m_1
          wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2
          emo_amy_m_2 emo_rac_m_2 emo_cac_m_2 emo_ff_m_2 emo_ins_m_2
          ext int beh_mean_rt_2 beh_tot_cor_2 beh_rate_cor_2
          rel_family_id ppensity serial support income famhx ysex yage;
MISSING=.;

!time 1:
usevar =
wm_rmf_m_1
wm_rac_m_1
wm_cac_m_1
wm_ip_m_1
wm_lo_m_1
emo_rac_m_1
emo_ff_m_1
emo_ins_m_1
emo_amy_m_1
emo_cac_m_1

!time 2
wm_rmf_m_2
wm_ip_m_2
wm_cac_m_2
wm_rac_m_2
wm_lo_m_2
emo_amy_m_2
emo_rac_m_2
emo_cac_m_2
emo_ff_m_2
```

```
emo_ins_m_2

!covs
support famhx income ysex
;

classes = c1(3) c2(3);

weight = ppensity;                          !weight with propensity scores
cluster is rel_family_id;                   !nest within families
stratification is serial;                   !nest within scanner

analysis:
type = complex;
type = mixture;
estimator = mlr;
starts = 500 20;

model:
%overall%
c2 on c1;
c1 on support famhx income ysex ;
c2 on support famhx income ysex ;

!set thresholds so that typical profile is reference profile

model c1:
%c1#1% !hypo-response
 [wm_rmf_m_1*-0.037
  wm_rac_m_1*-0.178
  wm_cac_m_1*-0.079
  wm_ip_m_1*-0.016
  wm_lo_m_1*-0.174
  emo_rac_m_1*-0.675
  emo_ff_m_1*-0.891
  emo_ins_m_1*-0.540
  emo_amy_m_1*-0.234
  emo_cac_m_1*-0.709];

%c1#2% !hyper-response
 [wm_rmf_m_1*0.046
  wm_rac_m_1*-0.089
  wm_cac_m_1*0.030
  wm_ip_m_1*0.028
  wm_lo_m_1*-0.035
  emo_rac_m_1*0.492
  emo_ff_m_1*0.194
  emo_ins_m_1*0.434
  emo_amy_m_1*0.558
  emo_cac_m_1*0.452];
```

```
%c1#3% !typical (reference class)
 [wm_rmf_m_1*0.096
  wm_rac_m_1*-0.065
  wm_cac_m_1*0.06
  wm_ip_m_1*0.063
  wm_lo_m_1*-0.024
  emo_rac_m_1*-0.019
  emo_ff_m_1*-0.322
  emo_ins_m_1*-0.005
  emo_amy_m_1*0.178
  emo_cac_m_1*-0.035];

model c2:
%c2#1% !hypo-response
[wm_rmf_m_2*0.115
 wm_ip_m_2*0.084
 wm_cac_m_2*0.038
 wm_rac_m_2*-0.132
 wm_lo_m_2*-0.027
 emo_amy_m_2*-0.1
 emo_rac_m_2*-0.403
 emo_cac_m_2*-0.433
 emo_ff_m_2*-0.698
 emo_ins_m_2*-0.337];

%c2#2% !hyper-response
[wm_rmf_m_2*0.07
 wm_ip_m_2*0.041
 wm_cac_m_2*0.014
 wm_rac_m_2*-0.152
 wm_lo_m_2*-0.036
 emo_amy_m_2*0.591
 emo_rac_m_2*0.577
 emo_cac_m_2*0.587
 emo_ff_m_2*0.331
 emo_ins_m_2*0.534];

%c2#3% !typical (reference class)
[wm_rmf_m_2*0.155
 wm_ip_m_2*0.091
 wm_cac_m_2*0.08
 wm_rac_m_2*-0.1
 wm_lo_m_2*-0.014
 emo_amy_m_2*0.206
 emo_rac_m_2*0.036
 emo_cac_m_2*0.011
 emo_ff_m_2*-0.275
 emo_ins_m_2*0.034];

 output: svalues;
```

```
TITLE: step 4 test outcomes
DATA: FILE = "/Users/landryhuffman/Documents/abcd/abcd_analysis/data_out/lta_trimmed.dat";
VARIABLE:
NAMES = subid siteid wm_rmf_m_1 wm_rac_m_1 wm_cac_m_1 wm_ip_m_1 wm_lo_m_1
      emo_rac_m_1 emo_ff_m_1 emo_ins_m_1 emo_amy_m_1 emo_cac_m_1
          wm_rmf_m_2 wm_ip_m_2 wm_cac_m_2 wm_rac_m_2 wm_lo_m_2
          emo_amy_m_2 emo_rac_m_2 emo_cac_m_2 emo_ff_m_2 emo_ins_m_2
          ext int beh_mean_rt_2 beh_tot_cor_2 beh_rate_cor_2
          rel_family_id ppensity serial support income famhx ysex yage;
MISSING=.;

!time 1:
usevar =
wm_rmf_m_1
wm_rac_m_1
wm_cac_m_1
wm_ip_m_1
wm_lo_m_1
emo_rac_m_1
emo_ff_m_1
emo_ins_m_1
emo_amy_m_1
emo_cac_m_1

!time 2
wm_rmf_m_2
wm_ip_m_2
wm_cac_m_2
wm_rac_m_2
wm_lo_m_2
emo_amy_m_2
emo_rac_m_2
emo_cac_m_2
emo_ff_m_2
emo_ins_m_2

!covs
support famhx income ysex

!outcomes
int ext
!beh_mean_rt_2 beh_rate_cor_2
;

classes = c1(3) c2(3);

weight = ppensity;                          !weight with propensity scores
cluster is rel_family_id;                   !nest within families
stratification is serial;                   !nest within scanner
```

```
analysis:
type = complex;
type = mixture;
estimator = mlr;
starts = 0;

model:
    %OVERALL%

    c2#1 ON c1#1*0.97116;
    c2#1 ON c1#2*0.50097;
    c2#2 ON c1#1*1.31110;
    c2#2 ON c1#2*0.99389;
    c2#1 ON support*0.02228;
    c2#1 ON famhx*-0.02535;
    c2#1 ON income*-0.03312;
    c2#1 ON ysex*0.21647;
    c2#2 ON support*0.48371;
    c2#2 ON famhx*-0.07904;
    c2#2 ON income*-0.05507;
    c2#2 ON ysex*0.33040;
    c1#1 ON support*-0.16385;
    c1#1 ON famhx*-0.04674;
    c1#1 ON income*-0.04821;
    c1#1 ON ysex*0.50033;
    c1#2 ON support*0.03787;
    c1#2 ON famhx*-0.02284;
    c1#2 ON income*-0.01943;
    c1#2 ON ysex*0.17029;

    [ c1#1*-2.42817 ];
    [ c1#2*-2.07998 ];
    [ c2#1*-2.07608 ];
    [ c2#2*-3.99701 ];


  MODEL C1:
    %C1#1%

    [ wm_rmf_m_1*-0.03709 ] (1);
    [ wm_rac_m_1*-0.17847 ] (2);
    [ wm_cac_m_1*-0.07933 ] (3);
    [ wm_ip_m_1*-0.01628 ] (4);
    [ wm_lo_m_1*-0.17395 ] (5);
    [ emo_rac_m_1*-0.67479 ] (6);
    [ emo_ff_m_1*-0.89116 ] (7);
    [ emo_ins_m_1*-0.53984 ] (8);
    [ emo_amy_m_1*-0.23395 ] (9);
    [ emo_cac_m_1*-0.70925 ] (10);

    %C1#2%
```

```
  [ wm_rmf_m_1*0.04576 ] (61);
  [ wm_rac_m_1*-0.08928 ] (62);
  [ wm_cac_m_1*0.02955 ] (63);
  [ wm_ip_m_1*0.02828 ] (64);
  [ wm_lo_m_1*-0.03545 ] (65);
  [ emo_rac_m_1*0.49210 ] (66);
  [ emo_ff_m_1*0.19375 ] (67);
  [ emo_ins_m_1*0.43421 ] (68);
  [ emo_amy_m_1*0.55794 ] (69);
  [ emo_cac_m_1*0.45218 ] (70);

  %C1#3%

  [ wm_rmf_m_1*0.09605 ] (71);
  [ wm_rac_m_1*-0.06505 ] (72);
  [ wm_cac_m_1*0.06004 ] (73);
  [ wm_ip_m_1*0.06286 ] (74);
  [ wm_lo_m_1*-0.02403 ] (75);
  [ emo_rac_m_1*-0.01863 ] (76);
  [ emo_ff_m_1*-0.32231 ] (77);
  [ emo_ins_m_1*-0.00459 ] (78);
  [ emo_amy_m_1*0.17831 ] (79);
  [ emo_cac_m_1*-0.03490 ] (80);

MODEL C2:
  %C2#1%

  [ wm_rmf_m_2*0.11511 ] (11);
  [ wm_ip_m_2*0.08409 ] (12);
  [ wm_cac_m_2*0.03754 ] (13);
  [ wm_rac_m_2*-0.13187 ] (14);
  [ wm_lo_m_2*-0.02705 ] (15);
  [ emo_amy_m_2*-0.10039 ] (16);
  [ emo_rac_m_2*-0.40326 ] (17);
  [ emo_cac_m_2*-0.43341 ] (18);
  [ emo_ff_m_2*-0.69809 ] (19);
  [ emo_ins_m_2*-0.33667 ] (20);
  [int](i1);
  [ext](e1);

  %C2#2%

  [ wm_rmf_m_2*0.06978 ] (41);
  [ wm_ip_m_2*0.04057 ] (42);
  [ wm_cac_m_2*0.01431 ] (43);
  [ wm_rac_m_2*-0.15151 ] (44);
  [ wm_lo_m_2*-0.03588 ] (45);
  [ emo_amy_m_2*0.59080 ] (46);
  [ emo_rac_m_2*0.57710 ] (47);
  [ emo_cac_m_2*0.58664 ] (48);
```

```
    [ emo_ff_m_2*0.33132 ] (49);
    [ emo_ins_m_2*0.53356 ] (50);
    [int](i2);
    [ext](e2);

    %C2#3%

    [ wm_rmf_m_2*0.15509 ] (51);
    [ wm_ip_m_2*0.09083 ] (52);
    [ wm_cac_m_2*0.08040 ] (53);
    [ wm_rac_m_2*-0.09973 ] (54);
    [ wm_lo_m_2*-0.01407 ] (55);
    [ emo_amy_m_2*0.20621 ] (56);
    [ emo_rac_m_2*0.03624 ] (57);
    [ emo_cac_m_2*0.01131 ] (58);
    [ emo_ff_m_2*-0.27535 ] (59);
    [ emo_ins_m_2*0.03432 ] (60);
    [int](i3);
    [ext](e3);

model test:                !wald test for each comparison run separately!
!i1 = i2;
!i1 = i3;
!i2 = i3;

!e1 = e2;
!e1 = e3;
!e2 = e3;
```

Models With and Without Child Age and Sex as Indicators: Comparison of Model Solution and Probabilities for Membership

Table A6. T1 and T2 model fit of LPA profile solutions including age and sex as indicators

	T1		
Profiles	BIC	a-BIC	Smallest class size (%)
1	114012.076	113938.987	7928 (100)
2	108106.911	107991.724	1400 (17.7)
3	**101921.841**	**101766.129**	**494 (6.2)**
4	98674.757	98477.733	414 (5.2)
5	94963.152	94724.817	369 (4.7)

6	92138.048	91858.401	4 (.05)

T2

Profiles	BIC	a-BIC	Smallest class size (%)
1	79447.821	79374.733	6182 (100)
2	74604.533	74490.135	487 (7.9)
3	**69901.204**	**69745.496**	**378 (6.1)**
4	67262.407	67065.388	220 (3.6)
5	64063.259	63824.929	211 (3.4)
6*	62600.81	62321.169	20 (.30)

Note. *Best loglikelihood was not replicated.

Table A7. Comparison of models with and without age and sex as covariates

T1	*n*	%
Emotion Hypo-response[SA]	494	6.20
Emotion Hypo-response	495	6.3
Emotion Hyper-response[SA]	769	9.7
Emotion Hyper-response		
Typical[SA]	6665	84.1
Typical	6661	84.00
T2		
Emotion Hypo-response[SA]	714	11.6
Emotion Hypo-response	707	11.4
Emotion Hyper-response[SA]	378	6.1
Emotion Hyper-response	380	6.2
Typical[SA]	5089	82.3
Typical	5095	82.4

Note. [SA]Indicates model including child age and sex as covariates.

Sensitivity Analyses: Comparison of Final Model to Non-Winsorized and Non-Clustered/Weighted Model

Table A8. LPA fit indices, final model

T1

Class solution	BIC	a-BIC	Smallest class size
1	48473.898	48410.432	7928 (100)

2	42551.489	42452.977	1403 (17.7)
3	**36369.347**	**36235.879**	**495 (6.3)**
4	33149.873	32981.45	418 (5.3)
5	29385.337	29181.957	363 (4.6)
6	26538.885	26300.55	4 (.05)

T2

Class solution	BIC	a-BIC	Smallest class size
1	27496.889	27433.334	6164 (100)
2	22629.873	22531.363	499 (8.1)
3	**17955.246**	**17821.781**	**381 (6.2)**
4	15296.427	15128.007	219 (3.6)
5	12084.908	11881.533	209 (3.4)
6	10607.348	10369.018	20 (.3)

Table A9. LPA fit indices, model without clustering or weighting

T1

Class solution	BIC	a-BIC	Smallest class size
1	47086.501	47022.945	7930 (100)
2	40895.379	40796.867	1359 (17.1)
3	**35312.355**	**35178.887**	**532 (6.7)**
4	31669.155	31500.731	451 (5.7)
5	28143.915	27940.535	378 (4.8)
6	25082.08	24843.745	5 (.06)

T2

Class solution	BIC	a-BIC	Smallest class size
1	28690.127	28626.572	6184 (100)
2	23841.058	23742.548	525 (8.5)
3	**19043.475**	**18910.01**	**381 (6.2)**
4	16547.344	16378.924	185 (3.0)
5	13276.358	13072.983	193 (3.1)
6	11672.373	11434.043	38 (.6)

Table A10. LPA fit indices, model without winsorized ROIs

T1

Class solution	BIC	a-BIC	Smallest class size
1	51590.957	51527.401	7930 (100)
2*	-	-	-
3	-	-	-

4	-	-	-
5	-	-	-
6	-	-	-

T2

Class solution		BIC	a-BIC	Smallest class size
	1	29451.892	29388.337	6166 (100)
	2	24654.54	24556.03	471 (7.6)
	3	**20076.282**	**19942.817**	**375 (6.1)**
	4	17507.249	17338.83	183 (3.0)
	5	14605.678	14402.304	186 (3.0)
	6	12859.012	12620.682	15 (0.2)

Note. *T1 profile solutions beyond a 1-profile solution did not converge.

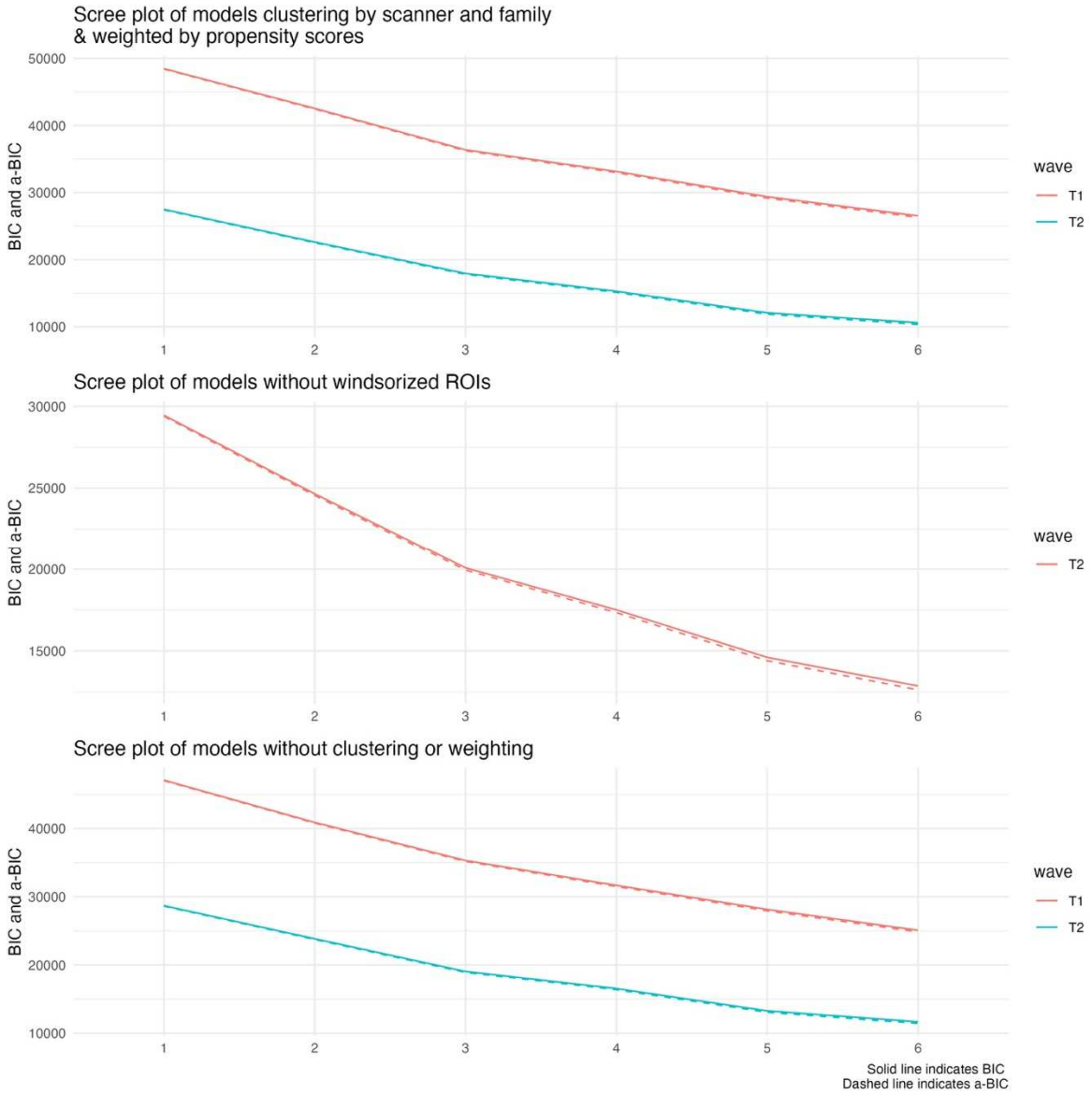

Figure A1. Scree plots comparing model solutions of final model, non-winsorized model, and non-clustered/weighted model.

Table A11. Comparison of baseline and final covariate models item response probabilities across models

	T1									
	Working Memory					Emotion Regulation				
	Rostral middle frontal	Inferior parietal	Caudal anterior cingulate	Rostral anterior cingulate	Lateral occipital	Amygdala	Caudal anterior cingulate	Rostral anterior cingulate	Fusiform	Insula
Emotion Hypo-response										
Baseline	-0.051	-0.017	-0.091	-0.198	-0.144	-0.213	-0.686	-0.644	-0.884	-0.523
Baseline^{NCW}	-0.026	-0.006	-0.066	-0.173	-0.133	-0.201	-0.652	-0.617	-0.862	-0.497
Final	-0.037	-0.016	-0.079	-0.178	-0.174	-0.234	-0.709	-0.675	-0.891	-0.54
Final^{NCW}	-0.011	0	-0.059	-0.165	-0.143	-0.207	-0.665	-0.635	-0.87	-0.503
Emotion Hyper-response										
Baseline	0.044	0.023	0.028	-0.085	-0.033	0.597	0.51	0.532	0.238	0.481
Baseline^{NCW}	0.056	0.035	0.045	-0.073	-0.023	0.606	0.541	0.561	0.262	0.506
Final	0.046	0.028	0.03	-0.089	-0.035	0.558	0.452	0.492	0.194	0.434
Final^{NCW}	0.042	0.021	0.028	-0.086	-0.044	0.564	0.496	0.54	0.227	0.465
Typical										
Baseline	0.099	0.065	0.061	-0.064	-0.023	0.183	-0.023	-0.004	-0.315	0.005
Baseline^{NCW}	0.097	0.063	0.059	-0.069	-0.024	0.187	-0.017	0	-0.309	0.011
Final	0.096	0.063	0.06	-0.065	-0.024	0.178	-0.035	-0.019	-0.322	-0.005
Final^{NCW}	0.098	0.063	0.061	-0.071	-0.025	0.183	-0.025	-0.009	-0.314	0.005
	T2									
Emotion Hypo-response										
Baseline	0.13	0.09	0.056	-0.122	-0.011	-0.097	-0.439	-0.405	-0.713	-0.339
Baseline^{NCW}	0.133	0.091	0.062	-0.12	0.002	-0.111	-0.456	-0.418	-0.735	-0.35
Baseline^{NW}	0.128	0.089	0.054	-0.122	-0.012	-0.102	-0.445	-0.412	-0.721	-0.345
Final	0.115	0.084	0.038	-0.132	-0.027	-0.1	-0.433	-0.403	-0.698	-0.337
Final^{NCW}	0.125	0.086	0.049	-0.129	-0.009	-0.106	-0.447	-0.414	-0.714	-0.343
Emotion Hyper-response										
Baseline	0.133	0.093	0.071	-0.089	0.04	0.61	0.609	0.602	0.355	0.554
Baseline^{NCW}	0.152	0.102	0.08	-0.083	0.042	0.62	0.607	0.589	0.332	0.548
Baseline^{NW}	0.14	0.1	0.079	-0.088	0.046	0.623	0.614	0.613	0.364	0.56
Final	0.07	0.041	0.014	-0.152	-0.036	0.591	0.587	0.577	0.331	0.534
Final^{NCW}	0.115	0.069	0.045	-0.125	-0.002	0.602	0.587	0.564	0.31	0.525
Typical										
Baseline	0.146	0.084	0.073	-0.104	-0.022	0.21	0.013	0.043	-0.274	0.035
Baseline^{NCW}	0.146	0.083	0.072	-0.109	-0.022	0.209	0.009	0.038	-0.273	0.034
Baseline^{NW}	0.146	0.084	0.073	-0.104	-0.022	0.209	0.012	0.042	-0.275	0.035
Final	0.155	0.091	0.08	-0.1	-0.014	0.206	0.011	0.036	-0.275	0.034
Final^{NCW}	0.153	0.088	0.078	-0.108	-0.017	0.206	0.008	0.033	-0.275	0.033

Note. ^{NCW}Indicates model without clustering or weighting; ^{NW}Indicates model without winsorized ROIs (only for T2)

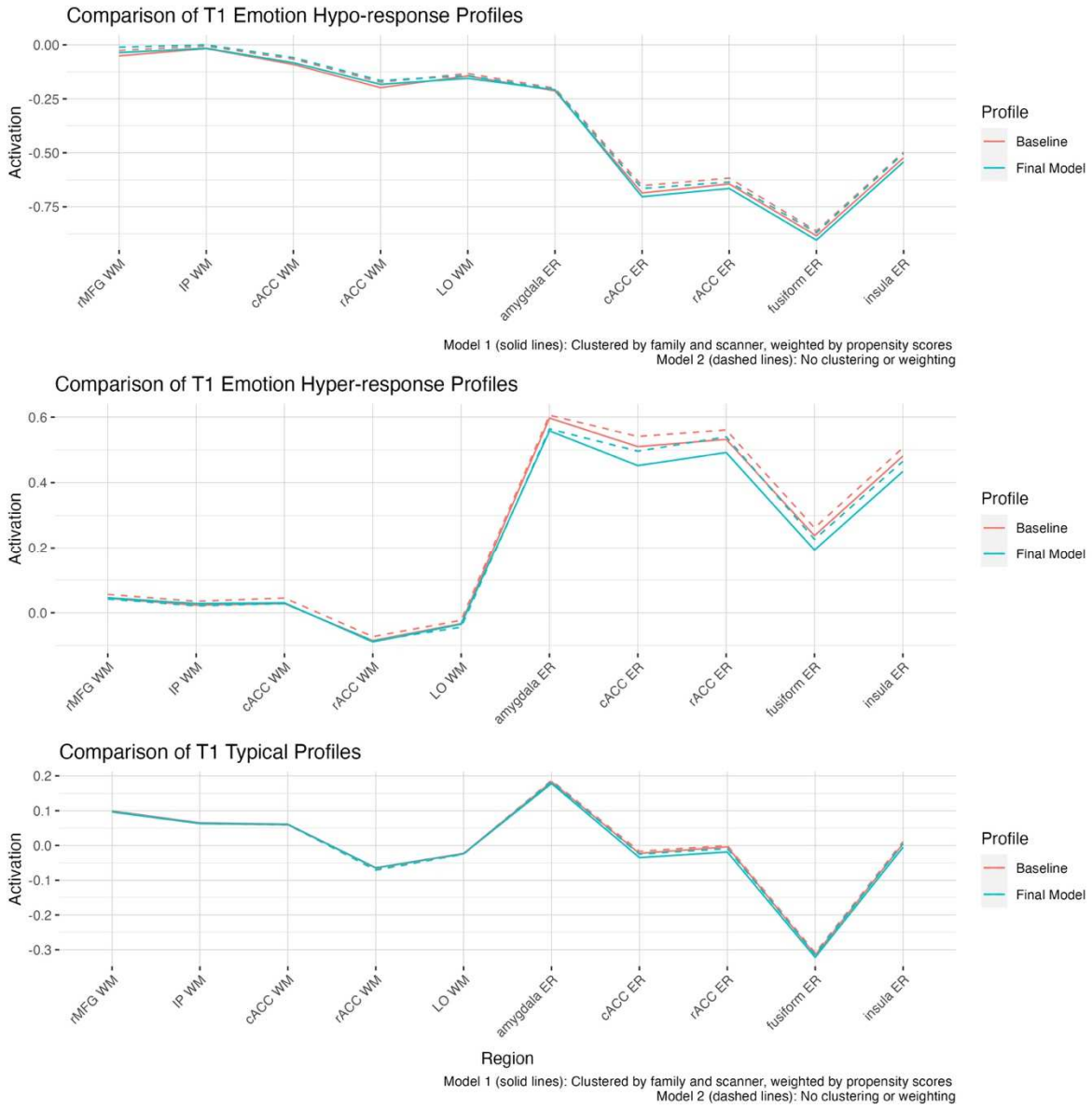

Figure 2A. T1 item response probabilities across models.

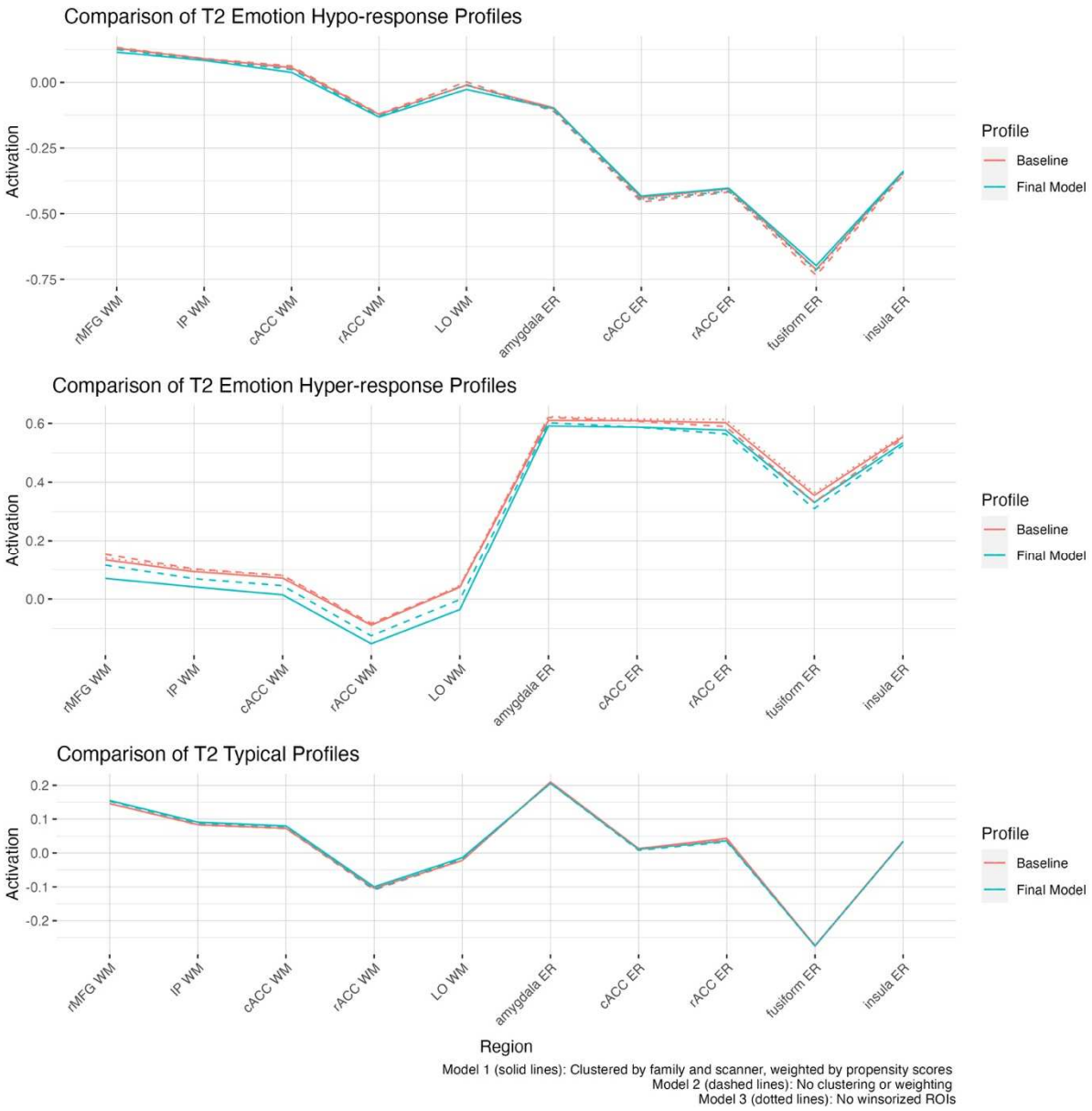

Figure 3A. T2 item response probabilities across models.