Sensitive Periods for the Effects of Childhood Maltreatment on Functional Connectivity in Cognitive Control and Risk Processing Systems

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Thesis submitted to the Faculty of Virginia Polytechnic Institute and State University

in partial fulfillment of the requirements for the degree of

Master of Science

in

Psychology

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September 14, 2023

Blacksburg, Virginia

Keywords: maltreatment, brain connectivity, risk processing, cognitive control

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ABSTRACT

It is well established that childhood adversity is associated with long lasting effects on development including both negative physical and mental health outcomes. Research demonstrates that adverse childhood experiences influence neurodevelopment and propose that this may be a mechanism linking adversity and psychopathology. However, little is known how the timing and type of maltreatment experiences may differentially impact longitudinal changes in neural processes of risk-related decision making. Using conditional growth curve modeling, we examined how abuse and neglect across three developmental periods (early childhood, school age, and adolescence) are associated with longitudinal changes in task-based functional connectivity during risk-processing and cognitive control. The current sample included 167 adolescents (13-14 years old at Time 1; 53% male), assessed annually for six years. At each of the six time points, adolescents completed a lottery choice task and a cognitive control task while blood-oxygen-level-dependent (BOLD) responses were monitored with functional magnetic resonance imaging (fMRI). Adolescents reported on maltreatment experiences occurring during ages 1 to 18. Generalized psychophysiological interactions (gPPI) was used to examine task-based functional connectivity in the insula and dACC (dorsal anterior cingulate cortex) during both risk processing and cognitive control, respectively. Although no sensitive periods emerged for the effects of abuse or neglect on functional connectivity during risk processing, chronic abuse (abuse occurring in more than one developmental period) significantly predicted weaker insula-dACC connectivity in late adolescence. For functional connectivity during cognitive

control, adolescence emerged as a potential sensitive period for neglect, such that those with neglect experiences occurring during ages 13 to 18 showed slower improvements in dACC-insula connectivity across adolescence. Chronic neglect was also associated with slower improvements in dACC-insula connectivity. Additionally, chronic abuse was significantly associated with stronger improvements in dACC-insula connectivity across adolescence. Collectively, these results suggest that abuse may be linked to a delayed maturation in neural connectivity associated with valuation, but an accelerated maturation in neural connectivity associated with cognitive control. Furthermore, neglect may be linked to a delayed maturation in neural connectivity associated with cognitive control. Both sets of findings involved functional connectivity in both the dACC and insula, important regions involved in salience processing. These findings elucidate the distinct effects of abuse and neglect on connectivity in regions involved in risk-related decision making, including valuation and cognitive control. Future work will benefit from examining how these different pathways may lead to outcomes such as health risk behaviors and psychopathology.

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GENERAL AUDIENCE ABSTRACT

Childhood adversity is associated with long lasting effects on development including both negative physical and mental health outcomes. Research shows that adverse childhood experiences influence brain development. However, little is known how the timing and type of maltreatment experiences may differentially impact changes in brain processes of risky decision making across adolescence. We examined how abuse and neglect across three developmental periods (early childhood, school age, and adolescence) are associated with changes in functional connectivity during risk-processing and cognitive control. The current sample included 167 adolescents (13-14 years old at Time 1; 53% male), assessed annually for six years. At each of the six time points, adolescents completed a lottery choice task and a cognitive control task while blood-oxygen-level-dependent (BOLD) responses were monitored with functional magnetic resonance imaging (fMRI). Adolescents reported on maltreatment experiences occurring during ages 1 to 18. Generalized psychophysiological interactions (gPPI) was used to examine task-based functional connectivity in the insula and dACC (dorsal anterior cingulate cortex) during both risk processing and cognitive control, respectively. Results showed that chronic abuse (abuse occurring in more than one developmental period) significantly predicted weaker insula-dACC connectivity in late adolescence. For functional connectivity during cognitive control, those with neglect experiences occurring during ages 13 to 18 showed slower improvements in dACC-insula connectivity across adolescence. Chronic neglect was also associated with slower improvements in dACC-insula connectivity. Additionally, chronic abuse was significantly

associated with stronger improvements in dACC-insula connectivity across adolescence. Both sets of findings involved functional connectivity in both the dACC and insula, important regions involved in salience processing. These findings elucidate the distinct effects of abuse and neglect on connectivity in regions involved in risk-related decision making, including valuation and cognitive control. Future work will benefit from examining how these different pathways may lead to outcomes such as health risk behaviors and psychopathology.

Acknowledgements

This work was supported by grants from the National Institute on Drug Abuse (DA036017) awarded to Jungmeen Kim-Spoon and Brooks Casas. We are thankful for current and former lab members for their help with data collection. We are grateful for both the adolescents and parents who participated in this longitudinal study.

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Introduction

Data indicate that 50% of children in the US will experience at least one type of adversity, which can include exposure to abuse, neglect, violence, separation from caregivers, or household substance use (Giano et al., 2020). These experiences in childhood have lasting effects on development into adulthood. Evidence indicates that multiple early adverse experiences increase risk for both negative mental and physical health outcomes (Hughes et al., 2017). Studies have found that early adversity and maltreatment are associated with risk for early initiation of substance use (Andersen & Teicher, 2009), depression (Pagliaccio & Barch, 2016), and behavioral problems (Hanson et al., 2017). Further, these adverse childhood experiences influence neural development in forms of function, structure, and connecitivty. Reward processing and cognitive control development are particularly salient during adolescence and early adversity may influence the development of related brain regions (Duffy et al., 2008). Identifying mechanisms of adversity related to brain development that may increase risk for negative outcomes will be important to advance prevention and intervention efforts that promote positive development and resilience. This study will focus on addressing how different chacteristics—such as the timing, chronicity, and subtype—of maltreament in childhood influences neurodevelopment in cognitive control and risk processing brain regions during adolescence.

Perspectives on Maltreatment Subtypes

Currently, there are two predominant perspectives addressing how childhood adversity affects brain development and increases risk for negative outcomes. The cumulative approach focuses on number of adverse events rather than the nature of adversity as there is a high rate of co-occurrence and it is assumed exposure to these events have similar effects on neurobiology

(Smith & Pollak, 2020). Alternatively, the dimensional approach proposes that threat and deprivation are separate dimensions that distinctly influence brain development (McLaughlin et al., 2021). Using a developmental psychopathology perspective, this model assumes maltreatment reflects early experiences that involve a lack of expected inputs from the environment (i.e., neglect) or experiences that involve harm or threat (i.e., abuse). This model aligns with the subtype conceptualization of acts of commission (e.g., sexual abuse and physical abuse) and acts of omission (e.g., neglect) as proposed by Rogosch and Cicchetti (1994). These approaches support that experiences of threat are related to changes in emotional development, whereas experiences of deprivation are most related to changes in cognitive development (McLaughlin et al., 2021). Both models were developed as alternatives to the specificity perspective that argues different exposures are associated with completely distinct effects on brain development. The limitations of the specificity models include not being able to account for different types of adversity that can co-occur and for types of adversity that have similar features (e.g., physical abuse and witnessing domestic violence) with common mechanisms (see McLaughlin et al., 2021 for a review of these three models). Given the evidence indicating distinctive effects of threat (exposure to violence) vs. deprivation experiences (poverty) on emotional regulation and cognitive control behavioral performance (Lambert et al., 2016) as well as distinctive abuse versus neglect effects on neural activation during valuation and control (Kim-Spoon et al., 2021), this study evaluates how these abuse (threat) and neglect (deprivation) experiences may differentially predict longitudinal changes in neural connectivity within the cognitive control and valuation neural networks.

Sensitive Periods for the Effects of Adversity

Childhood and adolescence are periods of rapid change related to physical, cognitive, and emotional development. Childhood adversities are negative experiences that represent a deviation from the expected environment (McLaughlin & Sheridan, 2016). Particularly, childhood represents a time in which learning is important for the development of emotion, cognition, and behavior, and adverse experiences during childhood can disrupt these associative learning processes and impact development (McLaughlin & Sheridan, 2016). Sheridan and colleagues (2018) found that prolonged institutional rearing in childhood is associated with deficits in implicit motor learning and reward responsivitity. This finding suggests that there may be periods in development for when the effects of the early environment are most influential. Studies examining timing and senstivie periods for the effects of adversity on neurodevelopment have primarily focused on the effects on brain stucture during childhood or brain function during young adulthood.

From the studies examining the link between adverse experiences and brain structure in childhood, there is evidence suggesting a sensitive period through age 5 for the effects of early stress on brain volume. Specifically, in a sample of 9-13 year olds, stress severity was only assoicated with decreased hippocampal volume if stress occurred before age 5 (Humphreys et al., 2019). Additionally, other evidence suggests different sensitive periods for the effects of abuse on various brain regions. Andersen and colleagues (2008) found that childhood sexual abuse occuring during ages 3-5 and 11-13 was associated with reduced hippocampal volume, whereas abuse occuring during ages 14-16 was associated with reducted frontal cortex volume in a sample of participants ages 18-22 years. In contrast, severe exposure to adversity (including abuse, neglect and witnessing physical violence) that occurred at ages 10-11 was associated with greater right amygdala volume in young adults than in those with lower exposure (Pechtel et al.,

2014). A more recent study examining neural actiavation in response to threat in young adults found that emotional maltreatment during adolescence was associated with increased response to threat in cortico-limbic regions (i.e., amygdala, hippocampus, anterior cingulate, inferior frontal gyrus, and ventromedial PFC and dorsomedial PFC), whereas peer physical abuse and witnessing violence during childhood was associated with increased activation in cortico-limbic regions in response to neutral faces (Zhu et al., 2023). These cross-sectional studies, however, had relatively small sample sizes, and examined brain structure or function during childhood or adulthood, but not adolescence.

It is established that indivudals who experienced chronic stress and adversity are at increased risk for a wide range of negative outcomes and are likely to experience recurring stressors. Prior studies have examined the impact of childhood stress exposure on reward processing while also accounting for current life stress during young adulthood. Hanson and colleagues (2018) found that there was increased functional connectivity between the left VS and mPFC among young adults ages 18-22 with a history of childhood maltreatment (combined experiences of abuse and neglect) who also experienced high levels of recent life stress. Alternatively, high levels of early life stress (e.g., death of close family member, exposure to severe marital conflict), but not high levels of current stress, predicted reduced activation in the putaman and insula during anticipation of potential reward loss among young adults ages 19-23 (Birn et al., 2017). This finding suggests that earlier stress may have a stronger impact on neural functioning than more recent stress during young adulthood.

There are other studies that have attempted to investigate sensitive periods for maltreatment effects across childhood and adolescence, although they examined volume and activation in regions not directly involved in reward processing and cogntive control. For

instance, Teicher et al., (2018) found different effects of abuse and neglect on hippocampal volume among young adults (ages 18-25). Specifically, neglect experienced age 1 through 7 years, but not abuse, predicted male hippocampal volume. In contrast, abuse at 10, 11, 15, and 16 years, but not neglect, predicted female hippocampal volume. Another study found that prepubertal and postpubertal matlreatment were associated with opposite patterns of amygdala activation in response to threatening and salient stimuli, such that there was increased prepubertal bilateral amygdala activity and decreased postpubertal bilateral amygdala activity (Zhu et al., 2019). Collectively, these findings highlight the importance of timing of adversity in predicting changes in neurodevlopment. In a theoretical review, Gee (2021) proposed that individuals who have consistent experiences may lose plasticity earlier in development. It follows that early adverse experiences may cause a continuous sensitive period across childhood to adolescence when environmental cues are unreliable early in life (i.e., responses to environments do not generate reliable, consistent feedback). More work is needed for systematic investigation of adversity timing effects on neurodevelopment across adolescence with specific focus on risky decision-making systems involving valuation and control as these systems are particularly salient during this developmental period.

Nature of Adversity

It is well established that adverse experiences in early life impact brain development. Prior studies used a variety of adverse experiences to examine effects of adversity such as emotional maltreatment, institutional care, abuse, neglect, cumulative stress, and stressful life events. However, most studies did not directly compare types of adversity and how these experiences may impact neurodevelopment differently. Conceptually, dimensional models have defined deprivation as the absence of cognitive and psycho-social stimuli and threat as

experiences of threat or harm. The dimensional approach to adversity argues that deprivation experiences are related to cognitive development and that threat experiences are related to emotional development (McLaughlin et al., 2021; Sheridan et al., 2018). However, recent evidence from functional imaging studies suggests differential effects of abuse and neglect on brain activation, connectivity, and structure among adolescents. First, as for task-based brain activation, neglect (i.e., deprivation) was the main predictor of longitudinal changes of risk-related processing in insula-dACC activation, whereas abuse (i.e., threat) was the main predictor of longitudinal changes of cognitive control related processing in frontoparietal activation (Kim-Spoon et al., 2021). Second, a study examining maltreatment effects on resting-state functional connectivity (rs-fc), neglect (i.e., deprivation) was associated with increased negative amygdala rs-fc with the dlPFC, whereas abuse (i.e., threat) was associated with increased positive amygdala rs-fc with the dACC (Cheng et al., 2021). Finally, studies on brain structure reported that a history of neglect (i.e., deprivation), not abuse (i.e., threat), predicted changes in amygdala and hippocampal volume (Herzog et al., 2020), and particularly, material deprivation, not trauma or emotional deprivation, was associated with reduced volume in frontal regions (Dennison et al., 2019). Thus, emerging evidence suggests that abuse and neglect, or distinct types of adversity in the forms of threat and deprivation, impact brain development differently.

Adversity and Adolescent Brain Development

Adolescence is a particularly vulnerable neurodevelopmental period which includes neural maturation in cognitive control and reward and risk processing systems. Current developmental neuroscience models propose that there is an imbalance between the development of two neural systems of risk-related decision making (valuation and control) during adolescence, such that heighted reward seeking coupled with immature cognitive control

processes may lead to risky decision making (Casey et al., 2008). The valuation system, in particular, is sensitive to rewards and risk as it involves evaluation of reward related stimuli and the risk associated with these rewarding options (Ernst et al., 2006). It is postulated that early life adversity is linked to alterations in neural circuitry involved in cognitive control and reward processing (Duffy et al., 2018), and recent research evidence supports that maltreatment history is associated with alterations in these neural networks through examining brain structure, function, and connectivity.

Study 1: Maltreatment Sensitive Periods and Risk Processing Development.

Introduction

When examining effects of child maltreatment on the valuation system, Teicher and Samson (2016) proposed that maltreatment may lead to experience-dependent changes in the brain that decrease approach responses, such as in the ventral striatum (VS) during reward anticipation. As for empirical findings regarding the effects of adversity on the reward processing system, there is one neuroimaging study that has examined maltreatment associations with brain structure. In a sample of children and adolescents ages 6-19, material deprivation (with food insecurity representing one aspect of deprivation), was associated with decreased reward performance and reduced frontostriatal white matter integrity (Dennison et al., 2019). The effects of maternal deprivation were more pronounced than those of caregiver neglect (representing the other aspect of deprivation) and trauma (representing threat; Dennison et al., 2019). This finding that material deprivation may be particularly linked to altered reward processing in frontal regions appears to be inconsistent with the dimensional model proposal that

deprivation may affect cognitive development and highlights differential effects between material deprivation and caregiver neglect.

Studies examining associations of maltreatment with brain functioning indicate decreased functional response during reward receipt and anticipation in reward related brain regions among those with abuse and neglect experiences. For example, one cross-sectional study comparing maltreated and non-maltreated youth (10-15 years), found those with a history of maltreatment (any experience of abuse, neglect, and intimate partner violence) showed blunted striatum and insula expected value processing during approach and avoidance trials (Gerin et al., 2017). Similarly, in a sample of previously institutionalized children and adolescents, early neglect experiences were associated with reduced nucleus accumbens reactivity compared to a comparison group without a history of early neglect experiences (Goff et al., 2013). In another study, experiences of emotional neglect were associated with blunted reward-related ventral striatum (VS) activity change over two years in adolescence (Hanson et al., 2015). Further, a longitudinal study demonstrated that a history of neglect was associated with slower developmental increases in insula and dorsolateral anterior cingulate cortex (dACC) activation during risk processing across four years during adolescence (Kim-Spoon et al., 2021). This finding suggests that neglect may be associated with a delayed maturation in regions involved in risk processing and valuation.

Turning to studies using functional connectivity, similar patterns of maltreatment effects on reward related connectivity patterns have been reported. Specifically, greater levels of maltreatment (combined experiences of abuse and neglect) were associated with an increase in reward-related functional connectivity (during a card-guessing paradigm where participants have a chance to win monetary rewards) between the VS and medial prefrontal cortex (mPFC) among

young adults (Hanson et al., 2018). Similarly, in a study examining resting-state functional connectivity, there was positive VS-mPFC connectivity in youth (6-18 years) with a history of institutionalized care, whereas there was negative VS-mPFC connectivity in a comparison group (Fareri et al., 2017). Together, these results suggest that maltreatment may be related to an increased connectivity in reward and risk processing regions.

Collectively, findings from prior research suggest that early life adversity may influence neurodevelopment in reward systems. In the current literature, however, the majority of the existing research on the link between maltreatment and brain development is cross-sectional and uses task-based activation. Longitudinal neuroimaging research on child maltreatment in general, and particularly research examining developmental changes in functional connectivity are rare. Given the proposal that there is an accelerated maturation following early life adversity (Callaghan & Tottenham, 2016; Herzberg & Gunnar, 2020), more longitudinal research is needed to understand the development of connectivity in reward processing following early life adversity. Importantly, it is critical to evaluate how maltreatment experiences are related to longitudinal trajectories of neural connectivity within the valuation systems (e.g., risk and reward processing) across adolescence during which neurodevelopmental plasticity is heightened (Fuhrmann et al., 2015; Gee, 2021).

Present Study

Current developmental psychopathology literature postulates that there may be sensitive periods during childhood and adolescence where effects of adversity may be particularly influential for neurodevelopment (Andersen et al., 2008; Fuhrmann et al., 2015). Furthermore, recent findings suggest that early adversity influences development of cognitive control and reward processing systems (Hanson et al., 2018; Cheng et al., 2021). These effects are

particularly important during adolescence as an imbalance between these dual systems may lead to risky decision making. However, literature on adversity and brain development poses some methodological challenges. Most of the studies examining adversity effects on brain structure and functioning are cross-sectional, making it difficult to understand how adversity effects developmental trajectories across time. Also, in the current literature, there is notable variation in defining adversity, contributing to inconsistent findings of adversity effects on the brain. This study examines maltreatment occurring during ages 1 to 18, to investigate three developmental periods, chronicity of maltreatment, and subtypes of maltreatment using the dimensional model to better understand how types and timing of maltreatment are related to developmental changes in task-based neural connectivity.

The current study presents the first prospective longitudinal analysis testing whether maltreatment experiences are related to *changes in connectivity* within valuation regions. According to Casey and colleagues' (2016) theoretical work, the hierarchical developmental shifts in connectivity are expected to change from cortico-subcortical to cortico-cortical circuits throughout adolescence. Indeed, empirical studies indicate shifts between circuitry such that there is more positive cortico-subcortical connectivity in reward and cognitive control networks in early childhood (Gee et al., 2013, Teslovich et al., 2014) and less positive cortico-cortical connectivity (Cohen et al., 2016), compared to adults. Additionally, behavioral performance on these risk processing tasks provides insight into developmental changes across age and associations with neural connectivity. Previous research indicates that adolescents show a decrease in risky choices across two assessments over one year (Lauharatanahirun et al., 2018). The present study investigated how multiple dimensions of adversity (i.e., maltreatment subtype, timing, chronicity) are related to developmental connectivity changes within the neural system of

valuation. Specifically, meta-analytic and longitudinal studies examining trajectories in risk processing brain regions have found changes in the insula (Kim-Spoon et al., 2019; Mohr et al., 2010). Therefore, this study focuses on connectivity changes associated with the insula and related brain regions across development.

The current study has four central aims:

1.) Investigate longitudinal changes in task-related behavioral performance during risk processing across six years from early adolescence to late adolescence.
2.) Investigate longitudinal changes in neural connectivity within valuation system across six years from early adolescence to late adolescence.
3.) Investigate maltreatment related deviations in neural connectivity development to examine accelerated verses delayed maturation.
4.) Investigate how abuse and neglect during three sensitive periods of development (early childhood, school age, and adolescence) may affect functional connectivity changes in valuation regions.

Hypotheses

Consistent with the study aims, we proposed the following hypotheses:

1.) Age-related maturation will be evident through behavioral performance during risk processing such that there will be a decrease in risky choices across time.
2.) Developmental changes in the insula during risk processing will be related to connectivity in the dACC and mPFC. Based on previous evidence, it is expected that connectivity between these regions will be stronger with age as valuation networks becomes more specialized (Rakesh et al., 2023). When examining maltreatment effects

on connectivity, stronger or weaker connectivity strength may indicate accelerated or delayed maturation, depending on task-based behavioral performance development.

3.) Functional connectivity changes related to the insula during risk processing may be primarily predicted by experiences of neglect as supported by recent empirical evidence of neglect effect on task-based brain activation (Kim-Spoon et al., 2021), or by experiences of abuse, as the dimensional model of adversity and psychopathology proposed. These effects may differ by the developmental period during which maltreatment occurred.

Method

Participants

The sample included 167 adolescents (53% male) from a southeastern state in the United States. Adolescents completed annual assessments across six years and were 13 to 14 years of age at Time 1 (M = 14.07, SD = 0.54 for Time 1, M = 15.05, SD = 0.54 for Time 2, M = 16.07, SD = 0.56 for Time 3, M = 17.01, SD = 0.55 for Time 4, M = 18.89, SD = 0.62 for Time 5, M = 20.17, SD = 0.63 for Time 6). About 78% of adolescents identified as White, 14% African American, 2% other, and 6% as more than one race. The median annual family income was in the $35,000-$50,000 range. Inclusion criteria included being age 13 or 14 at Time 1. Exclusion criteria were claustrophobia, history of head injury resulting in loss of consciousness for >10 minutes, orthodontia impairing image acquisition, and other contraindications to magnetic resonance imaging (MRI). At Time 1, 157 families participated, and at Time 2, 10 families were added for a final sample of 167 parent-adolescent dyads. At Time 2 data from 150 participants, at Time 3 data from 147 participants, at Time 4 data from 150 participants, at Time 5 data from 126 participants, and at Time 6 124 participants were collected. Participants did not always

participate in all possible assessments for reasons including ineligibility for tasks (i.e., brain abnormality, not meeting MRI safety criteria), declined participant, and lost contact. Rate of participation was not significantly predicted by demographic backgrounds including sex, race, and family income (p = .083; see Appendix A for demographic information questions).

Procedures

Data included in the current study were collected as part of a larger project. Adolescent participants were recruited via flyers, email announcements, and snowball sampling (word-of-mouth). Data collection was administered at university offices where participants completed self-report questionnaires, behavioral and neuroimaging tasks, and were interviewed by trained research assistants. The study duration was, on average, five hours long and participants were compensated monetarily for their time. All procedures were approved by the institutional review board of the university and written informed consent or assent was received from all participants.

Measures

Maltreatment. The Maltreatment and Abuse Chronology of Exposure (MACE; Teicher & Parigger, 2015; see Appendix B), was used to evaluate severity of exposure to different types of maltreatment during each year of childhood (ages 1-18). Adolescents completed this questionnaire at Time 5 and Time 6 and were asked to retrospectively indicate ages at which they experienced events from 52 items each time. Teicher and Parigger (2015) reported acceptable test-retest reliability (across a range of 5-441 days) for all subtypes (r = .63 - .90). The test-retest reliability across time points (over one year) was acceptable for the current sample (r = .56-.85). A maximum score was calculated between Time 5 and Time 6 reports across ages and subtypes. To examine the presence of maltreatment during three sensitive developmental periods, early childhood is defined as ages 1-5, school age is 6-12 and adolescence is 13-18. Neglect included

two subscales of physical neglect (5 items) and emotional neglect (5 items). Abuse included four subscales of physical abuse (6 items), sexual abuse (7 items), and verbal abuse (4 items), and emotional abuse (6 items). A sample item of physical abuse includes "hit you so hard it left marks for more than a few minutes" and a sample item of physical neglect, reverse coded, includes "were there to take care of you and protect you". These analyses include reports of maltreatment committed by a caregiver (parent, stepparents, or other adults living in the home), except for sexual abuse in which perpetrators included caregiver figures, adults not living in the house, and peers. Severity scores at each age were calculated using an algorithm provided by Teicher and Parigger (2015). Chronicity scores were calculated so that a value of 3 represents maltreatment occurring in all three developmental periods, a value of 2 represents maltreatment occurring in 2 developmental periods, a value of 1 represents maltreatment occurring during 1 developmental period, while a value of 0 represents maltreatment occurring in no developmental periods.

Risk Processing. To measure risk processing and valuation task-based functional connectivity, a modified economic lottery choice task was used (Holt & Laury, 2002; see Appendix C). Adolescents completed this task at Times 1-6 while their blood-oxygen-level-dependent (BOLD) responses were monitored. For each trial, adolescents were asked to choose between two gambles. One gamble was always risker (higher coefficient of variation; CV) than the other. The CV was computed by dividing the standard deviation of a gamble by the expected value (i.e., probability-weighted outcome) of that gamble. There was a high and low monetary outcome for each gamble, and each were associated with a specific probability that varied across 72 trials total (approximately 25 min to complete). To incentivize performance, participants were compensated based on their winnings from four randomly selected trials.

Imaging Acquisition and Analysis. Neuroimaging data were obtained on a 3T Siemens Tim Trio scanner using a 12-channel head matrix coil. Functional images were obtained with repetition time (TR) = 2 s, slice thickness = 4 mm, 34 axial slices, field of view (FoV) = 220x220 mm, echo time (TE) = 30 ms, flip angle = 90 degrees, voxel size = 3.4x3.4x4 mm, 64x64 grid, and slices were hyperangulated at 30 degrees from anterior-posterior commissure. Anatomical images were acquired with TR = 1.2 s, slice thickness = 1mm, FoV = 245x245 mm, TE = 2.66 ms, flip angle = 8 degrees, and an isotropic $1mm^3$ voxel size across 192 slices. SPM8 (Wellcome Trust Neuroimaging Center) was used to preprocess the MRI data at all time-points. Using a six-parameter rigid body transformation, the functional scans were corrected for motion. The mean functional was then co-registered to the corresponding anatomical image using a rigid-body transformation estimated to maximize the normalized mutual information between the anatomical and mean functional image. Next, the anatomical image was segmented to produce spatial normalization parameters which were then used to normalize the functional images to MNI-152 template. Normalization produced images resliced to an isotropic voxel size of 3 mm^3. Finally, the normalized functional images were smoothed using a 6 mm full-width-half-maximum Gaussian kernel. Six rigid body realignment parameters were included to account for the effect of in-scanner motion, and low-frequency signal was removed using a high- pass filter with cutoff of 0.00781 Hz (128 s which was the default for SPM) for risk processing data to capture the expected signal (see Henson, 2007, pp 200–203).

Neural risk processing. SPM8 (Wellcome Trust Neuroimaging Center) was used to analyze the imaging data. For each individual, a general linear model (GLM) was constructed including decision and outcome events of the task modeled with a duration of four and two seconds, respectively. To measure neural risk processing, a parametric regressor of decision

phase activation representing the risk level (i.e., CV) for chosen gambles was entered into the model. The CV is a scale-free metric and is better in explaining choice behavior compared to other economic measures of risk (i.e., standard deviation or variance) because the outcomes are coded by relative risk as opposed to absolute outcome (Weber et al., 2004). The individual level GLM included a parametric regressor indicating whether participants received high or low monetary outcomes during the outcome phase, one regressor for the button press, and six motion regressors. These regressors were included to characterize the error term in the model.

gPPI. A generalized psychophysiological interactions (gPPI) toolbox in SPM 8 was used to examine task-based functional connectivity (McLaren et al., 2012). A whole brain analysis was conducted using the insula as a seed region. Masks were made using WFU PickAtlas Tool in SPM to create 5mm spheres, centered at the peak coordinates from Time 1 reported in Kim-Spoon et al., 2021 during the decision phase of the lottery choice task. The center of the bilateral insula seed was located at [MNI: 30, 20, -11] and [MNI: -30, 17, -14], and the center of the dACC ROI was located at [MNI: 3, 35, 22], while the mPFC ROI was located at [MNI: 6, 41, 4]. Extracted time-series data were entered into first-level statistical model and included the insula physiological time series, a psychological regressor corresponding to task condition (decision phase for the risk processing task), and the psychophysiological interaction term. A second-level random effects model was conducted and included individual beta images that correspond to the interaction. Values of connectivity strength across hypothesized brain regions (i.e., dACC and mPFC) were extracted for longitudinal analyses.

Data Analytic Plan

Statistical Analysis.

Descriptive statistics were examined for all study variables to determine normality of distributions and outliers. All variable distributions were used to examine skewness, with acceptable levels less than 3, and kurtosis, with acceptable levels less than 10 (Kline, 2011). Additionally, outliers for connectivity values (after parceling) were identified as values that are above 3.29 SD from the mean and they were removed. Multivariate GLM analyses were conducted to determine if demographic variables such as sex (male vs. female), race, (White vs non-White) and family income (income-to-needs-ratios) needed to be added as covariates.

The hypothesized models were tested via Structural Equation Modeling (SEM) using in M*plus* (Muthén & Muthén, 1998-2021). The overall model fit was assessed by χ^2 value, degrees of freedom, corresponding *p*-value, Root Mean Square Error of Approximation (RMSEA), and Confirmatory Fit Index (CFI). RMSEA values less than .08 and CFI values greater than .90 were considered an acceptable fit (Little, 2013). Full information maximum likelihood (FIML; Arbuckle, 1996; Little & Rubin, 2003) estimation procedure was used to handle missing data, given the superiority of FIML estimation to those obtained with listwise deletion or other ad hoc methods (Schafer & Graham, 2002).

First, unconditional growth curve models were tested for risk processing to identify the patterns of both behavioral performance and connectivity changes over time within each construct. The first latent factor is the intercept from Time 6, with all factor loadings fixed to one, such that the level at Time 6 was represented by the intercept. The second latent factor is the slope, indicating growth of the function and change over time. To determine the shape of the trajectories, nested model comparisons were used. The χ^2 difference test is used to compare these nested models and the most parsimonious model with acceptable fits were chosen as the best-fitting model. In the intercept-only model, non-significant change in the slope is assumed. In the

linear growth model, a linear pattern of change is assumed with factor loadings fixed to -5, -4, -3, -2, -1, and 0, from Time 1 through Time 6, respectively. latent basis growth model allows the data to estimate the shape of growth by fixing the first and last time points (to -1 and 0, respectively) and freely estimating the second, third, fourth, and fifth time points.

Next, conditional growth curve modeling were constructed based on the best fitting univariate growth models of neural connectivity and used to examine abuse and neglect during three sensitive periods predicting changes in neural connectivity within risk processing regions. We examined 1) developmental trajectories of functional connectivity associated with the insula during risk processing and 2) the main effects of abuse and neglect during each sensitive period in predicting connectivity patterns associated with the insula during risk processing across adolescence.

Power.

To estimate power, we used an a priori sample size calculator for structural equation modeling (Soper, 2022) to test the minimum sample size needed to detect hypothetical effect sizes of .10, .30, and .50 (small, medium, and large, as described by Cohen, 1988) with a power of .8. The sample sizes needed for effect sizes .10, .30, and .50 were 947, 90, and 23, respectively. For the effect sizes in similar studies (Hanson et al., 2018; Kim-Spoon et al., 2021), the correlations between maltreatment and task-based activation or functional connectivity values ranged from .19-.29. These prior studies demonstrated having small-medium effect size, and our calculations suggested a sample size of 137 for a medium effect size. Therefore, our analyses have sufficient power (>.8) using our sample size of 167.

Results

Bivariate correlations and descriptive statistics for study variables are presented in Table 1. Skewness and Kurtosis values for the two connectivity variables were beyond the acceptable levels (i.e., skewness < 3, and kurtosis < 10), thus outlier data were removed (N = 16). After removing these outliers, skewness and kurtosis problems were fixed. Multivariate GLM analyses were conducted and indicated that demographic covariates were not significant predictors of insula-dACC connectivity variables (p = .719 for sex, p = .751 for race, and p = .980 for family income-to-need ratios) and insula-mPFC connectivity variables (p = .486 for sex, p = .340 for race, and p = .721 for family income-to-need ratios). Therefore, they were not included in the subsequent models.

Confirmatory factor analysis for maltreatment subtypes and developmental periods

A confirmatory factor analyses (CFA) was conducted to create latent factors of neglect and abuse at each developmental period. For neglect at ages 1 to 5, the model was fully saturated (χ^2 = 0, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and both physical neglect and emotional neglect indicators significantly loaded onto one factor (ps <.001). For abuse at ages 1 to 5, the model based on four indictors (sexual abuse, physical abuse, emotional abuse, and verbal abuse) fit was not acceptable fit (χ^2 = 15.37, *df = 2, p* < .001, RMSEA = .22, and CFI = 0.86). Skewness (9.65) and kurtosis (98.67) for the sexual abuse factor was high, and the prevalence (19.2% endorsed) was lower, therefore, this subscale was removed for the following model. The next model was fully saturated (χ^2 = 0, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and all three indicators significantly loaded onto one factor (*p*s <.001). Next, for neglect at ages 6 to 12, the model was fully saturated (χ^2 = 0, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and both indicators significantly loaded onto one factor (*p*s <.001). For abuse at ages 6 to 12, model fit

was acceptable (χ^2 = 0.03, *df = 2, p* = .986, RMSEA = .00, and CFI = 1), and all four indicators significantly loaded onto one factor (*p*s <.001). Finally, for neglect at ages 13 to 18, the model was fully saturated (χ^2 = 0, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and both indicators significantly loaded onto one factor (*p*s <.001). For abuse at ages 13 to 18, model fit was acceptable (χ^2 = 1.45, *df = 2, p* = .474, RMSEA = .00, and CFI = 1.00), and all four indicators significantly loaded onto one factor (*p*s <.001).

Unconditional growth curve models for functional connectivity during risk processing

Three sets (i.e., intercept-only, linear growth, and latent basis growth models) of nested models were fit to determine the shape of the trajectories of functional connectivity across six years for all three hypothesized seed-ROI models: insula-dACC and insula-mPFC. Due to large skewness (4.19-5.77) and kurtosis (11.30-67.54), connectivity values between consecutive ages were parceled (i.e., three separate composites were created for Time 1 and 2, for 3 and 4, for 5 and 6) according to the approach suggested by Little and colleagues (2002).

Insula-dACC

The linear growth model (χ^2 = 16.86, *df = 3, p* = .001, RMSEA = .17, and CFI = 0) provided better fit compared to the intercept-only model (χ^2 = 28.73, *df = 6, p* < .001, RMSEA = .15 and CFI = 0). A latent basis model provided best fit for the data compared to the intercept-only and linear growth models (χ^2 = 0.49, *df = 2, p* = .782, RMSEA = .00, and CFI = 1.00). The slope variance was small yet negative (-.003), so it was set to 0. The resulting model fit was not acceptable (χ^2 = 23.30 *df = 4, p* < .001, RMSEA = .17, and CFI = 0), therefore residual variances were allowed to vary to improve the model fit. The subsequent model provided acceptable fit to the data (χ^2 = 1.42, *df = 2, p* = .493, RMSEA = .00, and CFI = 1.00). In this final model, the mean of the slope was positive and significant (*b* = 0.19, *SE* = .01, *p* = .031), indicating

significant increases in connectivity strength across Times 1 to 6. The means of the intercept (b = 0.00, SE = .01, p = .672) , and the variance of the intercept (b = 0.00, SE = .00, p = .176) were not significant.

Insula-mPFC

The linear growth model (χ^2 = 23.62 *df = 3, p* < .001, RMSEA = .21, and CFI = 0) provided better fit compared to the intercept-only model (χ^2 = 30.60 *df = 6, p* < .001, RMSEA = .16, and CFI = 0). A latent basis model provided best fit for the data compared to the intercept-only and linear growth models (χ^2 = 4.64, *df = 2, p* = .793, RMSEA = .00, and CFI = 1.00). The slope variance was small yet negative (-.003), so the slope variance was set to 0. The resulting model did not have acceptable fit (χ^2 = 28.81, *df = 4, p* < .001, RMSEA = .19, and CFI = 0). The subsequent model with residual variances allowed to vary also provided unacceptable fit to the data (χ^2 = 5.29, *df = 2, p* = .071, RMSEA = .10, and CFI = 0). CFI remained 0, possibly indicating that connectivity values have low correlations across each time point (Muthén, 2013). Therefore, instead of the conditional growth curve models testing the effects of neglect and abuse on the trajectories of the insula-mPFC connectivity, exploratory analyses were conducted to examine overall connectivity change from early adolescence to late adolescence. See exploratory analysis section for more details.

Maltreatment effects on developmental trajectories of neural risk processing

Four models were conducted to examine effects of maltreatment during three different developmental periods (early childhood, school age, and adolescence) on the trajectories of insula-dACC connectivity over time. The first model examining neglect and abuse occurring during ages 1 to 5 had acceptable fit (χ^2 = 1.85, *df = 4, p* = .764, RMSEA = .00, and CFI = 1.00). However, there were no neglect or abuse effects on connectivity changes over time for the early

childhood period. See Figure 1 for all path estimates. The second model examining neglect and abuse occurring during ages 6 to 12 had acceptable fit (χ^2 = 2.21, *df = 4, p* = .687, RMSEA = .00, and CFI = 1.00). However, there were no neglect or abuse effects on connectivity changes over time for the school age period. See Figure 2 for all path estimates. The third model examining neglect and abuse occurring during ages 13 to 18 had acceptable fit (χ^2 = 1.89, *df = 4, p* = .755, RMSEA = .00, and CFI = 1.00). However, there were no neglect or abuse effects on connectivity changes over time for the adolescent period. See Figure 3 for all path estimates. The fourth model examining chronicity of neglect and abuse had good fit (χ^2 = 2.50, *df = 4, p* = .644, RMSEA = .00, and CFI = 1.00). Chronicity of abuse, but not neglect, significantly predicted the intercept (*b* = -0.12, *p* = .028) at the average of Time 5 and Time 6. This finding indicates that those with chronic experiences of abuse exhibited weaker connectivity between insula-dACC during late adolescence. See Figure 4 for all path estimates.

Unconditional growth curve for behavioral performance during risk processing

The linear growth model (χ^2 = 115.77 *df = 21, p* < .001, RMSEA = .16, and CFI = .60) provided better fit compared to the intercept-only model (χ^2 = 292.58 *df = 21, p* < .001, RMSEA = .26, and CFI = 0). Latent basis model provided best fit for the data compared to the intercept-only and linear growth models. Model fit was not acceptable (χ^2 = 73.56, *df = 17, p* < .001, RMSEA = .14, and CFI = .76). To improve model fit, we allowed residuals to vary across time and allowed residuals to be correlated. We kept all significant residual correlations in the model, which resulted in acceptable fit (χ^2 = 16.36, *df = 8, p* = .038, RMSEA = .08, and CFI = .97). The mean of the intercept (*b* = .34, *SE* = 0.04, *p* < .001) and slope (*b* = -0.41, *SE* = 0.05, *p* < .001) factors, as well as the variance of the intercept (*b* = .05, *SE* = 0.01, *p* < .001) and slope (*b* = 0.16,

SE = 0.07, *p* = .018) factors were significant, indicating significant decreases in risky choices over time with significant individual differences in Time 6 and trajectory patterns.

Maltreatment effects on developmental trajectories of behavioral risk processing

Four models were conducted to examine effects of maltreatment during three different developmental periods on the trajectories of behavioral risk processing. The first model examining neglect and abuse occurring during ages 1 to 5 had acceptable fit (χ^2 = 32.62, *df = 16, p* = .008, RMSEA = .08, and CFI = .93). However, there were no neglect or abuse effects on connectivity changes over time for the early childhood period. The second model examining neglect and abuse occurring during ages 6 to 12 had acceptable fit (χ^2 = 19.56, *df = 16, p* = .241, RMSEA = .04, and CFI = .99). However, there were no neglect or abuse effects on connectivity changes over time for the school age period. The third model examining neglect and abuse occurring during ages 13 to 18 had acceptable fit (χ^2 = 29.33, *df = 16, p* = .022, RMSEA = .07 and CFI = .95). However, there were no neglect or abuse effects on connectivity changes over time for the adolescent period. The fourth model examining chronicity of neglect and abuse had acceptable fit (χ^2 = 20.71, *df = 16, p* = .189, RMSEA = .04, and CFI = .98). However, there were no neglect or abuse effects on connectivity changes over time.

Exploratory analyses for maltreatment effects functional connectivity change

To assess mean-level changes between the beginning and the ending time points, rather than the trajectory patterns across six points, we ran a path analysis model with maltreatment variables predicting late adolescent connectivity, while controlling for early adolescent connectivity. The average between Times 1 and 2 connectivity for insula-mPFC represented early adolescent connectivity, while Times 5 and 6 connectivity for insula-mPFC represented late adolescent connectivity. Four models were run to test specific effects of abuse and neglect

for each developmental period, including early childhood, school age, adolescent, and chronicity during multiple developmental periods. All models were fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00 and CFI = 1.00).

Results revealed that chronicity of abuse, but not chronicity of neglect, significantly predicted the average of Time 5 and 6 insula-mPFC connectivity, while controlling for the average of Time 1 and 2 insula-mPFC connectivity (*b* = -0.01, *SE* = .01, *p* = .044). This finding indicates that chronic abuse is associated with weaker insula-mPFC connectivity strength during late adolescence, after controlling for the insula-mPFC connectivity strength during early adolescence. However, neglect and abuse occurring during the three developmental periods (i.e., ages 1 to 5, 6 to 12, and 13 to 18) did not significantly predict changes in the insula-mPFC connectivity strength. See Figures 9-12 for all path estimates.

Additionally, we tested whether late adolescent insula-dACC connectivity is predicted by chronicity of abuse, even after controlling for early adolescent insula-dACC connectivity. All models were fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00 and CFI = 1.00). However, abuse and neglect (during developmental periods or chronicity) did not significantly predict late adolescence insula-dACC connectivity.

Finally, the same models were run for the behavioral risk processing. All models were fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00 and CFI = 1.00). Results indicated that abuse during adolescence (ages 13 to 18) predicted late adolescence risky decision making (*b* = -0.05, *SE* = .02, *p* = .022). This finding suggests that abuse during adolescence may be associated with less behavioral risky decision making. See Figures 13-16 for path estimates.

Discussion

Childhood maltreatment increases risk for negative outcomes such as depression, early substance use initiation, and behavioral problems (Green et al., 2010). These outcomes are especially relevant during adolescence when there is a peak onset of mental illness (Kessler, 2001). Adolescence is also a sensitive period of brain development, particularly in regions related to risk-related decision making (Casey et al., 2011). Theoretical research suggests that early adversity may alter the development of these regions, which increases risk for the development of psychopathology and health risk behaviors (Duffy et al., 2008; McLaughlin et al., 2019). The dimensional model of adversity and psychopathology is particularly useful for examining child maltreatment effects on brain development. Specifically, this model posits that threat (i.e., abuse) and deprivation (i.e., neglect) experiences have different neurobiological mechanisms that lead to risk for psychopathology (McLaughlin et al., 2021). However, most studies examining adversity effects on brain development are cross-sectional with variation in how they define adveristy which leads to inconsistent findings and difficutly in understanding how abuse and neglect experiences contribute to developmental trjeactories of brain functioning. This current prospecive longitudinal study aimed to investiage how different characteristics of maltreatment — such as the timing, chronicity, and subtype — may affect development trajectories of functional connectivity with the insula during risk processing across adolescence.

Our findings contribute to the current literature by leveraging six time points of data encompassing early to late adolescence to analyze differential effects of abuse and neglect on longitudinal changes in functional connectivity during risk processing. Specifically, our findings provide insight to how chronic abuse (i.e., abuse occurring in multiple developmental periods) may alter risk-related connectivity development in adolescence. We hypothesized that we would

see developmental trajectories of functional connectivity over six years between insula with regions such as the dACC and mPFC. Unconditional growth curve modeling of neural connectivity showed that, on average, insula-dACC connectivity strength increased across six years. Our behavioral growth curve models suggest that risky decision-making decreased overtime with significant individual differences in both the levels during late adolescence (at Time 6) and the developmental trajectories across six time points. These findings are consistent with prior research indicating decreases in risky decisions across adolescence (Defoe et al., 2022) and this current study extends these findings by examining developmental trajectories of the same age cohort from early to late adolescence. Together, our findings indicate that insula-dACC connectivity strength increases across adolescence as risky choices decrease over time.

When examining maltreatment effects on connectivity during risk processing, we found significant effects of abuse on the intercept factor of insula-dACC connectivity, such that chronic abuse predicted weaker insula-dACC connectivity during late adolescence. This weaker connectivity in late adolescence may be indicative of delayed maturation during adolescence given the fact that, on average, connectivity strength improved while risky decisions decreased over time. This finding is not consistent with the accelerated neurodevelopment framework (Callaghan & Tottenham, 2016) and previous cross-sectional work suggesting that maltreatment may be associated with stronger connectivity between sub-cortical and cortical brain regions (Hanson et al., 2018; Fareri et al., 2017). However, recent longitudinal resting-state connectivity work has shown that maltreatment history is associated with weakening connectivity strength between neural circuits, such as between the default mode network and salience network, indicating delayed maturation across adolescence (Rakesh et al., 2021b). Importantly, these weaker connectivity associations were predictive of later psychopathology (Rakesh et al.,

2021b), emphasizing the importance of understanding specific maltreatment effects on neurobiological mechanisms. Prior cross-sectional work also indicates that adolescents with higher childhood stress showed lower activation in the insula compared to adolescents with lower childhood stress when presented with stimuli indicating potential reward loss (Birn et al., 2017). Our findings add to this current literature by examining connectivity patterns across adolescence (i.e., ages 13-20) and by examining how the type, timing, and duration of maltreatment may influence deviations in normative connectivity changes over time.

We also aimed to examine connectivity changes between the insula and mPFC during risk processing across adolescence; however, we did not find systematic changes in developmental trajectory patterns reflecting the latent slope factor (i.e., non-significant variance of slope). Instead, we found that chronic abuse (not chronic neglect or abuse/neglect occurring in specific developmental periods) predicted insula-mPFC connectivity in late adolescence, while controlling for early adolescent insula-mPFC connectivity. These findings reflect similar patterns in our results with insula-dACC connectivity patterns, indicating those with chronic abuse experiences show weaker connectivity in late adolescence. Collectively, our findings suggest that abuse occurring across multiple developmental periods is the strongest predicter of weaker connectivity between the insula and other cortical structures (i.e., dACC, mPFC) in late adolescence. The insula and dACC are important regions involved in salience network (SN). Working together, they play a role in detecting external salient stimuli, by filtering and integrating relevant information processing, to influence future goal-directed behavior (Menon & Uddin, 2010; Seeley et al., 2007). This is particularly important during risk processing, as the valuation system is involved in the evaluation of reward related stimuli and the risk associated with these potential rewards (Ernst et al., 2006). Indeed, previous work has shown that elevated

within-SN connectivity is positively associated with lower reward sensitivity (Marusak et al., 2015). Thus, our results may suggest that those with a history of chronic abuse are at greater risk of higher reward sensitivity during risk processing, reflected by weaker insula-dACC connectivity during late adolescence.

Additionally, the mPFC plays an important role in rewarding processing and is consistently activated during reward processing tasks (Ladouceur et al., 2019; Morgan et al., 2016). Specifically, mPFC plays a critical role for value-based goal-directed behaviors and value-based decision making (Laskowski et al., 2016). Our finding is consistent with previous research showing that maltreatment influences structure and connectivity with the mPFC. For example, emotional maltreatment before the age of 16 was associated with reduced dmPFC cortex volume in adulthood (Van Harmelen et al., 2010). Additionally, young adults with a history of child maltreatment showed increased reward-related task-based functional connectivity between the left VS and mPFC (Hanson et al., 2018).

Overall, our findings regarding the effects of chronic abuse experience on insula connectivity with dACC and mPFC highlight that abuse consistently experienced throughout multiple developmental periods impacts functional connectivity in important brain regions involved in salience and valuation processing. Chronic adverse experiences may be particularly detrimental to brain development because the consistent harsh environment causes a continuous sensitive period, leading to a prolonged period of delayed specialization within the brain (e.g., Gee, 2021). The distinctive effects of abuse, but not neglect, on brain connectivity underlying risk processing are also consistent with the dimensional model of adversity and psychopathology, suggesting that abuse (i.e., threat experiences) rather than neglect (i.e., deprivation experiences) influence neurodevelopment in risk and reward related regions.

Developmental neuroscience research suggests that value-based decision making involves neural computations of risk (i.e., variances in the potential outcome) (d'Acremont & Bossaerts, 2008; Mohr et al., 2010). Further, the anterior insular cortex has been identified as a region that is consistently implicated in the processing of risk information. Functionally, such risk processing is important to guide adolescents towards or away from risky choices (Mohr et al., 2010). Evidence from the current study as well as prior research (van Duijvenvoorde et al., 2015) concur that adolescents' hypersensitivity of the insular cortex to risk is related to making safer choices. Importantly, insula activation during risk processing is linked to adolescents' health risk behaviors (Lauharatanahirun et al., in press; Li et al., 2019). As such, the delayed development of insula connectivity observed here may explain why adolescents with abuse experiences show greater risky behaviors including externalizing behaviors and substance use (Cicchetti and Handley, 2019; Oshri et al., 2011). Importantly, abuse experiences may be particularly harmful for brain development related to risk sensitivity due to disruptions in associative learning processes. For example, exposure to physical maltreatment (i.e., abuse) was associated with reduced discrimination between threat and safety cues during fear conditioning in children and adolescents (McLaughlin et al., 2016). These fear conditioning disruptions were also associated with externalizing psychopathology in this sample (McLaughlin et al., 2016). These findings suggest that diminished discrimination between safe and threating stimuli may be a potential mechanism linking abuse and externalizing psychopathology. There is also evidence suggesting that physically maltreated children display more aggression and overly attend to negative cues (Shackman & Pollak, 2014). This pattern of increased detection of negative stimuli in the environment may be adaptive to children who experience threat in their environment, but also comes with a developmental trade-off where poor discrimination of cues may lead to later

aggression and misinterpretation of cues in social contexts (Shackman & Pollak, 2014). It is plausible that the reduction in distinguishing between threat and safety impacts later adolescent neurodevelopment in salience network processing during risky decision making. Harsh, threatening parenting behaviors may deter optimal neurodevelopment related to risk evaluation in adolescence, such that children with abuse exposure become less sensitive to distinguishing between threat and safety. Thus, these adolescents may be more inclined to make riskier decisions even in the presence of safer options.

In addition to observing behavioral changes in risk processing across time, we hypothesized that we would see similar maltreatment effects on behavior development, as we observed in functional connectivity development. Although we did not find significant associations between abuse type and timing on developmental trajectories over time (i.e., slope), we found that abuse occurring during ages 13 to 18 significantly predicted changes in risky choices during late adolescence, while controlling for early adolescent risky decision making. These findings suggest that abuse is associated with a significant decrease in risky decisions from early to late adolescence. While this finding may be contradicting to our insula-dACC and insula-mPFC findings, it might suggest that more current and recent threating experiences may more directly impact behavior. Future work should continue to include both task-based neural and behavioral responses to examine risk processing changes over time to better understand mechanisms that might predict future psychopathology after different types and timing of maltreatment experiences.

One of the main aims of this study was to identify if there are sensitive periods for which abuse or neglect may have the strongest effects on functional connectivity during adolescence. When examining changes in key regions involved in risk processing, we did not find evidence of

sensitive periods for which abuse or neglect predict changes in connectivity strength within valuation brain regions. Previous research on sensitive periods has primarily focused on brain structure including hippocampal volume (Andersen et al., 2008; Pechtel et al., 2014), amygdala volume (Teicher & Parigger, 2015), or pre-frontal gray matter volume (Andersen et al., 2008). It is possible that task-based brain connectivity is less vulnerable to sensitive period effects and that the brain regions involved in risk processing during adolescence may not be as sensitive to the timing effects of stress. Findings from the current study suggest that chronic abuse across developmental periods, regardless of the timing, is a predictor of functional connectivity in key risk processing brain regions. One fruitful direction of future research is to test whether the onset of chronic abuse might impact brain connectivity development.

Overall, the current prospective longitudinal study advances the literature by examining concurrent abuse and neglect within three different developmental periods (i.e., early childhood, school age, and adolescence) as well as the chroncity of this abuse and neglect across developmental periods. We leveraged six years of neuroimaging data to support that there are developmental changes in connectivity strength in insula connectivity to regions such as the dACC and mPFC during risk processing. Importantly, we found that chronic abuse, not neglect, predicts weaker connectivty strength during late adolesncece, which reflects less mature risk processing development. Adoelscence is a vulnerable neurodevelopmental period that involves changes that are associated with increases in sensation seeking and risk-taking behaviors (Casey et al., 2008; Steinberg, 2008). Our findings suggest that those that experience chronic abuse across multiple developmental periods may be at risk for a heightend and prolonged period of risk-taking during adolescence in part due to the impaired neurodevelopment underlying risk processing. Future work should investiage how the functional connectivity development may

predict real-life risk behaviors such as delinquent behaviors and substance use, and examine further if the effects of chronic abuse on brain connectivity may be reversed.

Study 2: Maltreatment Sensitive Periods and Cognitive Control Development.

Introduction

Regarding adversity effects on the control system, there is evidence of adversity related changes in cognitive control regions during adolescence. A few neuroimaging studies have examined individual differences in brain structure varying by maltreatment experiences in the brain regions known to be involved in cognitive control. For example, emotional maltreatment before the age of 16 was associated with reduced dorsal medial prefrontal (dmPFC) cortex volume in adulthood compared to a comparison group with no history of childhood maltreatment (Van Harmelen et al., 2010). Similarly, young adults with a history of childhood maltreatment (combined experiences of abuse and neglect) showed lower gray matter volume in prefrontal regions (Kirsch et al., 2021).

Evidence from functional neuroimaging research using cognitive control tasks supports that cumulative adversity and experiences of abuse and neglect are also associated with altered activation and connectivity in regions involved in cognitive control. A cross-sectional study found associations between maltreatment history (combined experiences of abuse and neglect) and heightened activation in the dorsomedial frontal regions during an inhibitory control task among adolescents (Lim et al., 2015). Further, in a longitudinal study of adolescents, abuse (ages 1-17) was associated with a steeper developmental decrease of frontoparietal activation during a cognitive control task during adolescence (ages 14-17), suggesting that abuse is related to accelerated maturation of regions involved in cognitive control (Kim-Spoon et al., 2021). Similarly, in a sample from the Adolescent Brain Cognitive Development (ABCD) study, greater number of negative life events (e.g., death of family member, victim of crime/violence/assault) assessed at 10-11 years was associated with greater negative cortico-limbic resting-state

functional connectivity over a two-year period (Brieant et al., 2021). This result also suggests an accelerated neurobiological maturation in cortico-limbic circuitry.

Alternatively, there is evidence suggesting that cumulative childhood maltreatment (combined experiences of abuse and neglect) was associated with an increase in resting-state functional connectivity between default mode and frontoparietal networks at age 16 to age 19 (Rakesh et al., 2021b). This finding, in contrast to previous evidence, appears to suggest a developmental delay associated with maltreatment, because maltreatment is positively related to between-network resting-state functional connectivity that decreased with age (Rakesh et al., 2021b). The discrepancy between the two resting-state functional connectivity studies (Brieant et al. 2021; Rakesh et al., 2021b) may be due to the fact that data were collected at different times in ontogeny. That is, it may be that early maltreatment is linked with an accelerated maturation during adolescence (or earlier), whereas it is linked with a developmental delay during late adolescence and in adulthood when the development of the control system approaches its full maturation point. Lastly, in a sample of adolescents ages 11-19 years, neglect, not abuse, was associated with negative amygdala resting-state connectivity with the dorsolateral prefrontal cortex (dlPFC) and positive amygdala resting-state connectivity with the supplementary motor area (SMA), both regions involved in higher-order cognitive processing (Cheng et al., 2021). However, the authors did not discuss whether these results were consistent with accelerated or delayed maturation. These findings highlight the distinct effects of neglect on resting-state connectivity within cognitive processing regions which emphasizes the importance of distinguishing between abuse and neglect when examining pathways from maltreatment to brain development underlying cognitive control.

Collectively, findings from prior research suggest that early life adversity may influence neurodevelopment of cognitive control systems. Given the proposal that there is an accelerated maturation following early life adversity (Callaghan & Tottenham, 2016; Herzberg & Gunnar, 2020) more longitudinal research is needed to understand the development of connectivity during cognitive control processing following early life adversity. Furthermore, it is important to examine how the timing, subtype, and chronicity of maltreatment experiences are associated with longitudinal trajectories of neural connectivity within these cognitive control systems across adolescence during which neurodevelopmental plasticity is heightened (Fuhrmann et al., 2015; Gee, 2021)

Present Study

Developmental psychopathology literature proposes that there may be sensitive periods during childhood and adolescence where the effects of maltreatment are most influential for brain development (Andersen et al., 2008; Fuhrmann et al., 2015). Recent evidence suggests that early adversity impacts development of cognitive control and reward processing systems (Hanson et al., 2018; Cheng et al., 2021). These effects of adversity are especially relevant during adolescence as an imbalance between these two systems may be associated with risky decision making. However, the current literature on adversity and brain development poses some critical methodological challenges. Many of the existing studies examining adversity effects on brain structure and function are cross-sectional, making it difficult to understand how adversity is related to brain development over time. In addition, the notable variation in adversity definition (i.e., maltreatment, exposure to violence, negative life events), contributes to inconsistent findings of adversity effects on the brain. Thus, this study considers maltreatment occurring during ages 1 to 18 to investigate three developmental periods, chronicity of maltreatment, and

subtypes of maltreatment to better understand how types and timing of maltreatment are related to developmental changes in task-based neural connectivity.

The current study presents the first prospective longitudinal analysis investigating how maltreatment experiences are related to *changes in connectivity* within cognitive control regions. According to Casey and colleagues' (2016) theoretical work, the hierarchical developmental shifts in connectivity are expected to change from cortico-subcortical to cortico-cortical circuits throughout adolescence. Indeed, empirical studies suggest shifts between circuitry representing more positive cortico-subcortical connectivity in cognitive control networks in early childhood (Gee et al., 2013; Teslovich et al., 2014) and less positive cortico-cortical connectivity (Cohen et al., 2016), compared to adults. Additionally, behavioral performance on cognitive control tasks provides insight into developmental changes across age and associations with neural connectivity. Previous research based on the current sample indicated increased age-related improvements on performance during a cognitive control task across four years from early to middle adolescence (Kim-Spoon et al., 2021).

The present study investigates multiple dimensions of adversity (subtype, timing, and chronicity) and their effects on developmental connectivity changes within cognitive control systems. Longitudinal studies examining trajectories of brain activation during cognitive control across adolescence have found developmental changes in regions such as the dorsal anterior cingulate cortex (dACC; Kim-Spoon et al., 2021; Ordaz et al., 2013). This study focuses on connectivity changes associated with the dACC and related brain regions during cognitive control across adolescence.

The proposed study has four central aims:

1.) Investigate longitudinal changes in task-related behavioral performance during cognitive control across six years from early adolescence to late adolescence.
2.) Investigate longitudinal changes in neural connectivity within cognitive control system across six years from early adolescence to late adolescence.
3.) Investigate maltreatment related deviations in neural connectivity development to examine accelerated versus delayed maturation.
4.) Investigate how abuse and neglect during three sensitive periods of development (early childhood, school age, and adolescence) may affect functional connectivity changes in cognitive control brain regions.

Hypotheses

Consistent with the study aims, we proposed the following hypotheses:

1.) Age-related maturation will be evident through behavioral performance during cognitive control such that there will be an increase in performance during cognitive control
2.) Developmental changes in the dACC during cognitive control will be related to connectivity within frontoparietal regions such as the medial prefrontal cortex, supplementary motor area (SMA), and the insula. Based on previous evidence, it is expected that connectivity between these regions will be stronger with age as cognitive control networks becomes more specialized (Stevens et al., 2009, Rakesh et al., 2021a). When examining maltreatment effects on connectivity, stronger or weaker connectivity strength which may indicate accelerated or delayed maturation, depending on task-based behavioral performance development.
3.) Functional connectivity changes related to dACC activation during cognitive control may be primarily predicted by experiences of abuse as supported by recent empirical evidence

of abuse on task-based brain activation (Kim-Spoon et al., 2021), or by experiences of neglect, as the dimensional model of adversity and psychopathology proposed. These effects may differ by the developmental period during which maltreatment occurred.

Method

Participants

The sample included 167 adolescents (53% male) from a southeastern state in the United States. Adolescents completed annual assessments across six years and were 13 to 14 years of age at Time 1 (M = 14.07, SD = 0.54 for Time 1, M = 15.05, SD = 0.54 for Time 2, M = 16.07, SD = 0.56 for Time 3, M = 17.01, SD = 0.55 for Time 4, M = 18.89, SD = 0.62 for Time 5, M = 20.17, SD = 0.63 for Time 6). About 78% of adolescents identified as White, 14% African American, 2% other, and 6% as more than one race. The median annual family income was in the $35,000-$50,000 range. Inclusion criteria included being age 13 or 14 at Time 1. Exclusion criteria were claustrophobia, history of head injury resulting in loss of consciousness for >10 minutes, orthodontia impairing image acquisition, and other contraindications to magnetic resonance imaging (MRI). At Time 1, 157 families participated, and at Time 2, 10 families were added for a final sample of 167 parent-adolescent dyads. At Time 2 data from 150 participants, at Time 3 data from 147 participants, at Time 4 data from 150 participants, at Time 5 data from 126 participants, and at Time 6 124 participants were collected. Participants did not always participate in all possible assessments for reasons including ineligibility for tasks (i.e., brain abnormality, not meeting MRI safety criteria), declined participant, and lost contact. Rate of participation was not significantly predicted by demographic backgrounds including sex, race, and family income (p = .083; see Appendix A for demographic information questions).

Procedures

Data included in the current study were collected as part of a larger project. Adolescent participants were recruited via flyers, email announcements, and snowball sampling (word-of-mouth). Data collection was administered at university offices where participants completed self-report questionnaires, behavioral and neuroimaging tasks, and were interviewed by trained research assistants. The study duration was, on average, five hours long and participants were compensated monetarily for their time. All procedures were approved by the institutional review board of the university and written informed consent or assent was received from all participants.

Measures

Maltreatment. The Maltreatment and Abuse Chronology of Exposure (MACE; Teicher & Parigger, 2015; see Appendix B), was used to evaluate severity of exposure to different types of maltreatment during each year of childhood (ages 1-18), was used. Adolescents completed this questionnaire at Time 5 and Time 6 and were asked to retrospectively indicate ages at which they experienced events from 52 items each time. Teicher and Parigger (2015) reported acceptable test-retest reliability (across a range of 5-441 days) for all subtypes (r = .63 - .90). The test-retest reliability across time points (over one year) was acceptable for the current sample (r = .56-.85). A maximum score was calculated between Time 5 and Time 6 reports across ages and subtypes. To examine the presence of maltreatment during three sensitive developmental periods, early childhood is defined as ages 1-5, school age is 6-12 and adolescence is 13-18. Neglect included two subscales of physical neglect (5 items) and emotional neglect (5 items). Abuse included four subscales of physical abuse (6 items), sexual abuse (7 items), and verbal abuse (4 items), and emotional abuse (6 items). A sample item of physical abuse includes “hit you so hard it left marks for more than a few minutes” and a sample item of physical neglect, reverse coded, includes “were there to take care of you and protect you”. These analyses

included reports of maltreatment committed by a caregiver (parent, stepparents, or other adults living in the home), except for sexual abuse in which perpetrators included caregiver figures, adults not living in the house, and peers. Severity scores at each age were calculated using an algorithm provided by Teicher and Parigger (2015). Chronicity scores were calculated so that a value of 3 represents maltreatment occurring in all three developmental periods, a value of 2 represents maltreatment occurring in 2 developmental periods, a value of 1 represents maltreatment occurring during 1 developmental period, while a value of 0 represents maltreatment occurring no developmental periods.

Cognitive Control. Adolescents completed the Multi-Source Interference Task (MISIT; Bush, Shin, Holmes, Rosen, & Vogt, 2003; see Appendix D) while completing a functional MRI scan at Times 1-5. Adolescents were presented with three digits in each trial and were asked to report the identity of the different digit (unlike the other two) by pressing a button. In neutral trials, the target number's identity matched the digit's presented location, whereas in interference trials, the target number's identity was not congruent with the digit's presented location. We used intraindividual variability in response time to assess task performance. This was indexed as intraindividual standard deviations (ISD; MacDonald et al., 2012) for correct responses in the interference condition.

Imaging Acquisition and Analysis. Neuroimaging data were obtained on a 3T Siemens Tim Trio scanner using a 12-channel head matrix coil. Functional images were obtained with repetition time (TR) = 2 s, slice thickness = 4 mm, 34 axial slices, field of view (FoV) = 220x220 mm, echo time (TE) = 30 ms, flip angle = 90 degrees, voxel size = 3.4x3.4x4 mm, 64x64 grid, and slices were hyperangulated at 30 degrees from anterior-posterior commissure. Anatomical images were acquired with TR = 1.2 s, slice thickness = 1mm, FoV = 245x245 mm, TE = 2.66

ms, flip angle = 8 degrees, and an isotropic 1mm^3 voxel size across 192 slices. SPM8 (Wellcome Trust Neuroimaging Center) was used to preprocess the MRI data at all time-points. Using a six-parameter rigid body transformation, functional scans were corrected for motion. Then, the mean functional was co-registered to the corresponding anatomical image using a rigid-body transformation estimated to maximize the normalized mutual information between the anatomical and mean functional image. Next, the anatomical image was segmented to produce spatial normalization parameters which were then used to normalize the functional images to MNI-152 template. Normalization produced images resliced to an isotropic voxel size of 3 mm^3. Finally, the normalized functional images were smoothed using a 6 mm full-width-half-maximum Gaussian kernel. Six rigid body realignment parameters were included to account for the effect of in-scanner motion, and low-frequency signal was removed using a high- pass filter with cutoff of 0.006 Hz (168 s) for cognitive control data to better capture the expected signal (see Henson, 2007, pp 200–203).

Neural cognitive control. Preprocessed MRI data were analyzed by entering them into a first-level analysis General Linear Model (GLM) in SPM8. Interference and neutral blocks were modeled using boxcars convolved with the canonical haemodynamic response function (HRF) with six motion regressors. Framewise displacement (FD) was calculated from the realignment parameters, with rotational displacement converted to millimeters using the surface of a sphere of radius 50 mm (Power et al., 2012; Siegel et al., 2014). Volumes with FD > 0.9 mm were censored by adding a volume-specific regressor for each scrubbed volume in the GLM. This frame censoring approach was used because it appeared to be beneficial to analyzing the repeated measures data simultaneously. An interference greater than neutral contrast map was generated for each GLM by subtracting the neutral beta map from the positive beta map.

gPPI. A generalized psychophysiological interactions (gPPI) toolbox in SPM 8 was used to examine task-based functional connectivity (McLaren et al., 2012). A whole brain analysis was run using the dorsolateral prefrontal cortex (dACC) as a seed region. Masks were made using WFU PickAtlas Tool in SPM to create 5mm spheres, centered at the peak coordinates from Time 1 reported in Kim-Spoon et al., 2021 during the interference minus neutral condition of the MSIT task. The center of the dACC seed was located at [MNI: -3, 8, 25], and the center of the bilateral insula ROI was located at [MNI: -30, 14, 13] and [MNI: 33, 20, 7], the mPFC ROI was located at [MNI: -39, 38, 28] and the center of the SMA ROI was located at [MNI: -6, 14, 49]. Extracted time-series data was entered into first-level statistical model and include the dACC physiological time series, a psychological regressor corresponding to task condition (interference versus neutral for the cognitive control task) and the psychophysiological interaction term. A second-level random effects model was conducted and included individual beta images that correspond to the interaction. Values of connectivity strength across hypothesized brain regions (i.e., insula, mPFC, and SMA) were extracted for longitudinal analyses.

Data Analytic Plan

Statistical Analysis.

For all study variables, descriptive statistics were examined to determine normality of distributions and outliers. All variable distributions were used to examine skewness, with acceptable levels less than 3, and kurtosis, with acceptable levels less than 10 (Kline, 2011). Additionally, outliers were identified as values that are above 3.29 SD from the mean and removed. A multivariate GLM analyses was conducted to determine if demographic variables such as sex, race, and family income need to be added as covariates.

The hypothesized models were tested via Structural Equation Modeling (SEM) using in M*plus* (Muthén & Muthén, 1998-2021). The overall model fit was assessed by χ^2 value, degrees of freedom, corresponding *p*-value, Root Mean Square Error of Approximation (RMSEA), and Confirmatory Fit Index (CFI). RMSEA values less than .08 and CFI values greater than .90 were considered an acceptable fit (Little, 2013). Full information maximum likelihood (FIML; Arbuckle, 1996; Little & Rubin, 2003) estimation procedure was used to handle missing data, given the superiority of FIML estimation to those obtained with listwise deletion or other ad hoc methods (Schafer & Graham, 2002).

First, unconditional growth curve models for cognitive control were used to identify the patterns of both behavioral performance and connectivity changes over time within each construct. The first latent factor is the intercept from Time 6, with all factor loadings fixed to one, such that the level at Time 6 was represented by the intercept. The second latent factor is the slope, indicating growth of the function and change over time. To determine the shape of the trajectories, nested model comparisons were used. The χ^2 difference test was used to compare these nested models and the most parsimonious model with acceptable fits were chosen as the best-fitting model. In the intercept-only model, non-significant change in the slope is assumed. In the linear growth model, a linear pattern of change is assumed with factor loadings fixed to -5, -4, -3, -2, -1, and 0, from Time 1 through Time 6, respectively. The latent basis growth model allows the data to estimate the shape of growth by fixing the first and last time points (to -1 and 0, respectively) and freely estimating the second, third, fourth, and fifth time points.

Next, conditional growth curve modeling was constructed based on the best fitting univariate growth models of neural connectivity and was used to examine abuse and neglect during three sensitive periods predicting changes in neural connectivity cognitive control

regions. We examined 1) developmental trajectories of functional connectivity associated with the dACC during cognitive control and 2) the main effects of abuse and neglect during each sensitive period in predicting connectivity patterns associated with the dACC during cognitive control across adolescence.

Power.

To estimate power, we used an a priori sample size calculator for structural equation modeling (Soper, 2022) to test the minimum sample size needed to detect hypothetical effect sizes of .10, .30, and .50 (small, medium, and large, as described by Cohen, 1988) with a power of .8. The sample sizes needed for effect sizes .10, .30, and .50 were 947, 90, and 23, respectively. For the effect sizes in similar studies (Hanson et al., 2018; Kim-Spoon et al., 2021), the correlations between maltreatment and task-based activation or functional connectivity values ranged from .19-.29. These prior studies demonstrated having small-medium effect size, and our calculations suggested a sample size of 137 for a medium effect size. Thus, our analyses have sufficient power (>.8) using our sample size of 167.

Results

Bivariate correlations and descriptive statistics for study variables are presented in Table 2. Skewness and Kurtosis values for the three connectivity variables were within range (i.e., skewness < 3, and kurtosis < 10). Multivariate GLM analyses were conducted and indicated that sex (*p*s > .350), race (*p*s > .074), and family income (*p*s > .242) were not significant predictors of the three connectivity variables, and thus were not included in the subsequent models.

Confirmatory factor analysis for maltreatment subtypes and developmental periods

A confirmatory factor analyses (CFA) was conducted to create latent factors of neglect and abuse at each developmental period. For neglect at ages 1 to 5, the model was fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and both physical neglect and emotional neglect significantly loaded onto one factor (*p*s <.001). For abuse at ages 1 to 5, the model based on four indicators (sexual abuse, physical abuse, emotional abuse, verbal abuse) had a model fit that was not acceptable ($\chi^2 = 15.37$, *df = 2, p* < .001, RMSEA = .22, and CFI = 1.86). Skewness (9.65) and kurtosis (98.67) for the sexual abuse factor was high, and the prevalence (19.2% endorsed) was notably low, therefore this subscale was removed for the following model. The next model was fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and all three indicators significantly loaded onto one factor (*p*s <.001). Next, for neglect at ages 6 to 12, the model was fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and both indicators significantly loaded onto one factor (*p*s <.001). For abuse at ages 6 to 12, model fit was acceptable ($\chi^2 = 0.03$, *df = 2, p* = .986, RMSEA = .00, and CFI = 1), and all four indicators significantly loaded onto one factor (*p*s <.001). Finally, for neglect at ages 13 to 18, the model was fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00, and CFI = 1.00) and both indicators significantly loaded onto one factor (*p*s <.001). For abuse ages at 13 to 18, model fit was acceptable ($\chi^2 = 1.45$, *df = 2, p* = .474, RMSEA = .00, and CFI = 1), and all four indicators significantly loaded onto one factor (*p*s <.001).

Unconditional growth curve for functional connectivity during cognitive control

Three sets (i.e., intercept-only, linear growth, and latent basis growth models) of nested models were fit to determine the shape of the trajectories of functional connectivity across six years for all three hypothesized seed-ROI models: dACC-mPFC, dACC-insula, and dACC-SMA.

dACC-insula

The linear growth model (χ^2 = 37.99, *df = 21, p* = .013 RMSEA = .07 and CFI = 0) provided better fit compared to the intercept-only model (χ^2 = 40.43 *df = 24, p* = .091, RMSEA = .07, and CFI = 0). The latent basis model provided best fit for the data compared to the intercept-only and linear growth models. Model fit was not acceptable (χ^2 = 24.76, *df = 17, p* = .100, RMSEA = .05, and CFI = 0). To improve model fit, residuals of the manifest indicators were correlated, and one significant residual correlation was retained. The resulting model fit was acceptable (χ^2 = 15.36, *df = 16, p* = .499, RMSEA = .0, and CFI = 1). The variance (*b* = .05, *SE* = 0.02, *p* = .035) of the intercept was significant, indicating there were significant individual differences in connectivity strength at Time 6. The variance of the slope (*b* = .03, *SE* = 0.02, *p* = .224), and the means of the intercept (*b* = .01, *SE* = 0.03, *p* = .583) and slope (*b* = .02, *SE* = 0.02, *p* = .380) were not significant.

dACC-mPFC

The linear growth model (χ^2 = 42.01, *df = 21, p* = .004 RMSEA = .08 and CFI = 0) provided better fit compared to the intercept-only model (χ^2 = 43.61 *df = 24, p* = .001, RMSEA = .07, and CFI = 0). A latent basis model provided best fit for the data compared to the intercept-only and linear growth models. However, model fit was not acceptable (χ^2 = 25.28, *df = 17, p* = .089, RMSEA = .06 and CFI = 0). Both intercept (-.01) and slope (-.00) had negative variance, so the intercept and slope variance were set to 0. This subsequent model did not provide acceptable fit to the data (χ^2 = 31.76, *df = 20, p* = .046, RMSEA = .06 and CFI = 0). CFI remained 0, possibly indicating low correlations among connectivity variables (Muthén, 2013). Therefore, instead of the conditional growth curve models testing the effects of abuse and neglect on the trajectories of dACC-mPFC, exploratory analyses were conducted to examine

overall change from connectivity from early adolescence to late adolescence. See exploratory analysis section for more details.

dACC-SMA

The linear growth model (χ^2 = 28.85, *df = 21, p* = .118 RMSEA = .08 and CFI = 0) provided better fit compared to the intercept-only model (χ^2 = 30.19 *df = 24, p* = .178, RMSEA = .04, and CFI = 0). A latent basis model provided best fit for the data compared to the intercept-only and linear growth models. Model fit was acceptable (χ^2 = 11.81, *df = 17, p* = .812, RMSEA = .00 and CFI = 1). However, both intercept (-.01) and slope (-.03) had negative variance so both the intercept and slope variance were set to 0. This subsequent model did not provide acceptable fit to the data (χ^2 = 120.05, *df = 20, p* < .00, RMSEA = .18, and CFI = 0). CFI remained 0, possibly indicating low correlations among connectivity variables (Muthén, 2013). Therefore, instead of the conditional growth curve models testing the effects of abuse and neglect on the trajectories of dACC-SMA, exploratory analyses were conducted to examine overall change from connectivity from early adolescence to late adolescence. See exploratory analysis section for more details.

Maltreatment effects on developmental trajectories of neural cognitive control

Four models were conducted to examine effects of maltreatment on dACC-insula connectivity during different developmental periods. The first model examining neglect and abuse occurring during ages 1 to 5 had acceptable fit (χ^2 = 20.45 *df = 24, p* = .671, RMSEA = .00, and CFI = 1.00). However, there were no neglect or abuse effects on connectivity changes over time for the early childhood period. See Figure 5 for all path estimates. The second model examining neglect and abuse occurring during ages 6 to 12 had acceptable fit (χ^2 = 24.13, *df = 24, p* = .454, RMSEA = .01, and CFI = .98). However, there were no neglect or abuse effects on

connectivity changes over time for the school age period. See Figure 6 for all path estimates. The third model examining neglect and abuse occurring during ages 13 to 18 had acceptable fit (χ^2 = 21.51, *df = 24, p* = .608, RMSEA = .00, and CFI = 1.00). Neglect during adolescence significantly predicted the intercept factor, which indicated the connectivity strength at Time 6 (*b* = -0.09, *SE* = .04, *p* = .030), and also predicted the slope factor with marginal significance (*b* = -0.08, *SE* = .04, *p* = .052). These results indicate that neglect predicts weaker connectivity strength at Time 6, and slower connectivity strength improvement from Time 1 to Time 6. See Figure 7 for all path estimates. The fourth model examining chronicity of neglect and abuse had acceptable fit (χ^2 = 26.09, *df = 24, p* = .352, RMSEA = .02, and CFI = .89). Chronicity of neglect significantly predicted the intercept (*b* = -0.06, *p* = .008) at Time 6 and the slope of dACC-insula connectivity strength over time (*b* = -0.04, *p* = .044), indicating that chronic neglect is associated with weaker dACC-insula connectivity strength at Time 6, and slower dACC-insula connectivity improvement from Time 1 to Time 6. Chronicity of abuse significantly predicted the slope of dACC-insula connectivity strength over time (*b* = 0.06, *p* = .045), indicating that for those with chronic abuse, connectivity strength increases, and becomes stronger over 6 years. See Figure 8 for all path estimates.

Growth curve for behavioral performance during cognitive control

The linear growth model (χ^2 = 236.49, *df = 21, p* < .001 RMSEA = .25 and CFI = 0) provided better fit compared to the intercept-only model (χ^2 = 560.04 *df = 24, p* < .001, RMSEA = .37, and CFI = 0). Latent basis model provided best fit for the data compared to the intercept-only and linear growth models. Model fit was not acceptable (χ^2 = 44.25, *df = 7, p* < .001, RMSEA = .09, and CFI = .89). To improve model fit, residual variances were correlated, and two residual correlations were retained. The subsequent model had acceptable fit acceptable (χ^2 =

24.45, *df = 15, p* = .058, RMSEA = .06, and CFI = .96). The mean of the intercept (*b* = -0.17, *SE* = 0.00 *p* < .001) and slope (*b* = 0.07, *SE* = 0.00, *p* < .001), as well as the variance of the intercept (*b* = .00, *SE* = 0.00, *p* < .001) was significant. Variance of the slope was not significant (*b* = .00, *SE* = 0.00, *p* = .140). This finding indicates significant increases in cognitive control over time with significant individual differences in Time 6.

Maltreatment effects on developmental trajectories of behavioral cognitive control

Four models were conducted to examine effects of maltreatment during different developmental periods. The first model examining neglect and abuse occurring during ages 1 to 5 had acceptable fit (χ^2 = 31.48, *df = 23, p* = .114, RMSEA = .05, and CFI = .96). However, there were no neglect or abuse effects on connectivity changes over time for the early childhood period. The second model examining neglect and abuse occurring during ages 6 to 12 had acceptable fit (χ^2 = 40.88, *df = 23, p* = .012, RMSEA = .07, and CFI = .92). However, there were no neglect or abuse effects on connectivity changes over time for the school age period. The third model examining neglect and abuse occurring during ages 13 to 18 had acceptable fit (χ^2 = 41.29, *df = 23, p* = .011, RMSEA = .07, and CFI = .92). However, there were no neglect or abuse effects on connectivity changes over time for the adolescent period. The fourth model examining chronicity of neglect and abuse had acceptable fit (χ^2 = 45.89, *df = 23, p* = .003, RMSEA = .08, and CFI = .90). However, there were no neglect or abuse effects on connectivity changes over time.

Exploratory analyses for examining connectivity change over-time

To assess average change from early adolescence to late adolescence rather than developmental trajectory patterns across six-time points, we ran a path analysis model with maltreatment variables predicting late adolescent connectivity, while controlling for early

adolescent connectivity. As such, Times 1 and 2 connectivity values for dACC-mPFC were averaged to represent early adolescent time levels, and Times 5 and 6 connectivity values for dACC-mPFC were averaged to represent late adolescent time levels. Four models were run for three developmental periods as well as for chronicity of maltreatment. All models were fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00 and CFI = 1.00). Neglect and abuse occurring during the three developmental periods (i.e., age 1 to 5, 6 to 12, and 13 to 18) and chronicity did not significantly predict changes in connectivity strength from early adolescence to late adolescence.

Similarly, Times 1 and 2 connectivity values for dACC-SMA were averaged to create a composite for early adolescence, and Times 5 and 6 connectivity values for dACC-SMA were averaged to create a composite for late adolescence. Four models were run for three developmental periods as well as for chronicity of maltreatment. All models were fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00 and CFI = 1.00). Neglect and abuse occurring during the three developmental periods (i.e., age 1 to 5, 6 to 12, and 13 to 18) and chronicity did not significantly predict changes in connectivity strength from early adolescence to late adolescence.

Additionally, we tested whether late adolescent dACC-insula connectivity is predicted by chronicity of abuse, while controlling for the average early adolescent dACC-insula connectivity. Early and late adolescent composite scores were created similar to dACC-SMA and dACC-mPFC connectivity. Four models were run for three developmental periods as well as for chronicity of maltreatment. All models were fully saturated ($\chi^2 = 0$, *df = 0, p* < .001, RMSEA = .00 and CFI = 1.00). Results revealed that neglect, not abuse, occurring between ages 13 and 18 significantly predicted the average of Time 5 and 6 dACC-insula connectivity, while controlling for the average of Time 1 and 2 connectivity (*b* = -0.09, *SE* = .04, *p* = .018). This finding

indicates that neglect during adolescence is associated with weaker dACC-insula connectivity strength during late adolescence. Additionally, chronic abuse significantly predicted the average of Time 5 and 6 dACC-insula connectivity, while controlling for the average of Time 1 and 2 connectivity (b = 0.06, SE = .03, p = .029). This finding suggests that chronic abuse is associated with stronger dACC-insula connectivity strength during late adolescence. However, neglect and abuse occurring during the earlier developmental periods (ages 1 to 5 and 6 to 12) did not significantly predict changes in the dACC-insula connectivity strength. See Figures 17-20 for all path estimates.

Discussion

Childhood maltreatment is associated with an increased risk for multiple negative mental health outcomes such as depression, early substance use initiation, and behavioral problems (Green et al., 2010). Adolescence is a peak period for the onset of mental illness (Kessler, 2001) and sensitive period of brain development for regions related to risk-related decision making (Casey et al., 2011). Theoretical work suggests that early adversity may alter the development of these regions, which are associated with an increased risk for the development of psychopathology and health risk behaviors (Duffy et al., 2008; McLaughlin et al., 2019). The dimensional model of adversity and psychopathology proposes that threat (i.e., abuse) and deprivation (i.e., neglect) experiences have different neurobiological mechanisms that lead to psychopathology (McLaughlin et al., 2021). However, the majority of prior studies on adversity effects on brain development used cross-sectional data (i.e., age differences across different age-cohorts) and examined broadly defined adveristy (i.e., low parental education, exposure to violence), resulting in difficutly in understanding the link between maltreatment experiences and

brain development trajectories across time. This prospecive longitudinal study aimed to investiage how different characteristics of maltreatment —such as subtype, timing, and chronicity— may explain development trajectories of functional connectivity with the dACC during cognitive control across adolescence.

Our findings contribute to the current literature by using 6-time points to analyze how abuse and neglect effects longitudinal changes in functional connectivity during cognitive control. Specifically, our findings provide insight to how both chronic neglect and abuse (i.e., maltreatment occurring in multiple developmental periods) may alter connectivity development into late adolescence. We hypothesized that we would see developmental trajectories of functional connectivity over six years between dACC with regions such as the insula, SMA and mPFC. Unconditional growth curve modeling showed that there were significant individual differences in dACC-insula connectivity strength during late adolescence (at Time 6). Thus, this finding provides evidence for significant individual differences in connectivity strength during cognitive control processing in late adolescence rather than significant developmental increases or decreases in connectivity strength across time.

We hypothesized that there would be sensitive periods of maltreatment effects in the sense that maltreatment effects on functional connectivity are particularly pronounced depending on the developmental period during which maltreatment occurred. Our findings revealed that neglect occurring between ages 13 and 18 (i.e., adolescence) may be most influential in predicting developmental changes of dACC-insula connectivity throughout adolescence, compared to neglect in earlier developmental periods or abuse experienced in any developmental period. Specifically, neglect occurring during adolescence significantly predicted both decreases in dACC-insula connectivity over time and weaker dACC-insula connectivity during late

adolescence, indicating adolescence as a sensitive period for neglect effects on cognitive control related functional connectivity. This finding is consistent with theoretical perspectives suggesting that adolescent brains are particularly sensitive to socio-environmental contexts (Blakemore, 2008). Furthermore, theoretical papers posit that the impacts of specific dimensions of adversity on brain development may be particularly pronounced when it is experienced during specific periods of development (Cohodes, et al., 2021). Given that cortical areas typically have a protracted development during adolescence (Casey & Jones, 2010), there are increasing opportunities for brain developmental trajectories to be altered by concurrent environmental input (e.g., Sapolsky, 2017).

Additionally, neglect occurring across multiple developmental periods also significantly predicted intercept and slope of the growth trajectories of dACC-insula connectivity, such that neglect chronicity was associated with weaker connectivity strength in late adolescence and slower connectivity strength improvement across adolescence. Both of our findings are consistent with the dimensional model of adversity and psychopathology which suggests that deprivation (i.e., experiences of reduced expected cognitive and social inputs from the environment, such as neglect), rather than threat, influences neurodevelopment in cognitive regions. A previous structural imaging study found that deprivation experiences are associated with reduced cortical thickness in areas such as the insula and ACC (Machlin et al., 2023). Similarly, studies have found similar findings in task-based activation during cognitive control by using socioeconomic disadvantage to examine neglect impacts on altered neural activation in frontoparietal regions (e.g., Sheridan et al., 2012). Our findings suggest that neglect exposure during adolescence and chronic neglect occurring across multiple developmental periods predicts weaker connectivity between the dACC and insula during cognitive control, which may indicate

delayed maturation across adolescence after experiences of neglect. This finding of neglect effects on delayed maturation is consistent with recent longitudinal resting-state connectivity work showing that maltreatment is associated with weaker connectivity strength between circuits such as the default mode network and salience network, which implies delayed maturation across two years of adolescence (Rakesh et al., 2021b). Our findings extend this literature by including task-based functional connectivity development across six years and clarifying the distinctive effect of neglect apart from abuse effects.

In addition to our findings regarding associations between neglect exposure and weaker dACC-insula connectivity, we found that chronic abuse predicted stronger dACC-insula connectivity strength improvement across six years. These findings are consistent with the framework posited by Callaghan and Tottenham (2016) that there may be accelerated neural development after adversity. Similarly, a previous longitudinal study examining task-based activation changes during cognitive control found that abuse was associated with steeper developmental decreases in frontoparietal activation (indicating accelerated maturation) during adolescence (Kim-Spoon et al., 2021). Further, a recent meta-analysis examining interpersonal adversity (e.g., family-based maltreatment) and brain volume found that interpersonal adversity was primarily associated with larger fronto-limbic volumes earlier in development (indicating accelerated maturation), and then smaller volumes in adolescence (Vannucci et al., 2023). According to the accelerated neurodevelopment model, altered neurodevelopment may be a response that occurs after interpersonal adversities, such that these are adaptations after signals from proximal environments indicate safety from a caregiver is unreliable (Callaghan & Tottenham, 2016). Together, our findings indicate that associations between adversity and

neurodevelopment may depend both on the type of adversity, such as abuse, and the timing, such as chronicity.

The dACC and insula are two main regions involved in the salience network (SN). They play a role in detecting external salient stimuli and filtering and integrating relevant information, which influences future goal-directed behavior (Menon & Uddin, 2010; Seeley et al., 2007). Further, literature suggests that these two regions are especially important in multiple cognitive control processes including conflict monitoring, motor planning, and inhibition (Botvinick et al., 2004; Bush et al., 2003; Molnar-Szakacs & Uddin, 2022). In addition to hypothesizing developmental changes in the insula and dACC, we expected to see both longitudinal changes and maltreatment effects on dACC connectivity with the SMA and mPFC. However, we did not find significant changes in developmental trajectory patterns of connectivity for these regions. Exploratory path analyses did not show significant effects of abuse and neglect on mean levels of dACC-mPFC and dACC-SMA connectivity during cognitive control. It is possible that abuse and neglect may affect these regions earlier in development, or that maltreatment may affect regions involved in other circuity during adolescence (Herzberg et al., 2021; Holz et al., 2023). Altered functional connectivity changes in the brain after adversity may be more region-specific as the period of neuroplasticity is prolonged after maltreatment exposure (Holz et al., 2023). For example, circuit-specific differences in frontolimbic connectivity indicative of accelerated maturation were found in youth with a history of institutionalization; however, findings were less clear when examining changes in dorsal attention network connectivity (Herzberg et al., 2021). Thus, future studies may consider examining network based or whole-brain connectivity when examining the effects of maltreatment on adolescent brain development.

Regarding behavioral cognitive control, latent basis model provided the best fit with a significant mean of intercept and slope and significant variance in intercept, suggesting improvement in cognitive control over time with significant individual differences at Time 6. The finding of significant individual differences of the intercept (Time 6) is consistent with the finding regarding dACC-insula connectivity development, as the findings indicate significant individual differences in late adolescence (at Time 6) at both a behavioral and neural level. However, we did not find significant effects of abuse and neglect on longitudinal behavioral changes during cognitive control across adolescence. This non-significant finding was surprising. However, it could indicate neurobiological embedding of adversity overtime. We examined abuse and neglect over three different developmental periods spanning over multiple years. It is possible that task-based behavior changes following abuse and neglect are more immediate, as opposed to long-lasting yearly changes. Future work should continue to include both task-based behavioral and neural indicators to examine changes in cognitive control over time with different time intervals to better capture dynamic effects of different types and timing of maltreatment experiences.

Overall, the current prospective longitudinal study advances the literature by examining concurrent abuse and neglect within three different developmental periods (i.e., early childhood, school age, and adolescence) as well as the chroncity of abuse and neglect across developmental periods. We leveraged six years of neuroimaging data to support that there are developmental changes in connectivity strength in dACC connectivity with the insula during cognitive control. Importantly, we found that both neglect exposure during ages 13 to 18 years and chronic neglect are predicters of weaker connectivty strength during late adolescence, which reflects less mature cognitive control development. Additionally, we found that chronic abuse exposure predicted

stronger connectivity strenght development across adolescence, which reflects a more mature cognitive development. Adoelscence is a vulnerable neurodevelopmental period that involves changes that have implications for increases in sensation seeking and risk-taking behaviors (Casey et al., 2008; Steinberg, 2008), as well as social problems and depression (Pagliaccio & Barch, 2016; Sheridan et al., 2018). Our findings suggest that the timing of abuse and neglect may exert differential effects on neurobiogloical mechanmisms related to cognitive processing. Future work should investiage how these functional connectivity changes may have implications for different mental health pathways leading to psychopathology in adulthood.

Study 1 and Study 2 Conclusions

In the two prospective longitudinal studies, we found evidence for both accelerated and delayed maturation associated with maltreatment type and timing across childhood and adolescence. First, we found that chronic abuse occurring across multiple developmental periods was associated with weaker insula connectivity improvement with both the dACC and mPFC into late adolescence, suggesting delayed maturation during risk processing after abuse. These findings also support the current dimensional model of adversity and psychopathology by demonstrating that abuse, not neglect was associated with functional connectivity changes in risk-related valuation processing. Second, we found that neglect occurring during ages 13 and 18 and chronic neglect were associated with weaker dACC-insula connectivity improvement into late adolescence, suggesting delayed maturation during cognitive control processing after neglect. This finding is also consistent with the dimensional model of adversity and psychopathology by illustrating that neglect, not abuse was associated with functional connectivity changes in cognitive control processing. Finally, we found that those who

experienced chronic abuse showed stronger dACC-insula connectivity improvement during cognitive control across adolescence, which supports the accelerated neurodevelopment framework (Callaghan & Tottenham, 2016). These findings emphasize the importance of examining childhood and adolescent maltreatment type, timing, and duration on distinct associations with longitudinal functional connectivity changes during risk processing and cognitive control across adolescence.

Both present studies should be interpreted in light of a few limitations. First, although data were longitudinal, these are correlational and prevent us from inferring causality. Second, adolescents reported retrospectively on experiences of maltreatment across 18 years. However, we had good reliability in retrospective reporting of maltreatment over 1 year. Additionally, research suggests that subjective, self-reporting of adversity are the strongest predictors of psychopathology (Francis et al., 2023). These strong associations with psychopathology have important implications when linking neurobiological mechanisms with early adversity and mental health outcomes. Furthermore, we were not able to examine the role that pubertal timing or tempo may have in the link between adversity and brain development across adolescence. One study found that maltreatment occurring pre and post puberty were associated with opposite patterns of amygdala activation in response to threatening and salient stimuli (Zhu et al., 2019). Theories of cognitive-affective brain development posit that earlier pubertal timing increases risk for psychopathology through changes in emotion regulation networks before the prefrontal cognitive networks mature (Ladouceur, 2012). It is possible that sensitive periods for which abuse, or neglect may have strongest effects on neurodevelopment may depend on pubertal timing or tempo, as this has been shown to be associated with accelerated brain development evident through white matter tracts (Chahal et al., 2018). Lastly, our longitudinal functional

connectivity analyses focused on specific regions of interest selected based on prior research examining brain regions involved in task-based activation during valuation and cognitive control. Other research suggests that abuse and neglect may have more broad effects on other neural circuitry (Herzberg et al., 2021; Holz et al., 2023). Future studies should consider using whole-brain functional connectivity along with resting-state connectivity, as these approaches together may offer advantages of evaluating between and within network connectivity which may add additional clarification on delayed versus accelerated development during adolescence (Chahal et al., 2022; Rakesh et al., 2021a).

Despite some of these limitations, there are significant strengths in the present study. For instance, we used six time points of neuroimaging data to examine developmental trajectories of functional connectivity changes in hypothesized regions. Additionally, we leveraged our measure of maltreatment by examining potential sensitive periods for the effects of maltreatment on brain development. By examining maltreatment subtypes during ages 1 to 18, we were also able to investigate abuse and neglect chronicity across developmental periods such as early childhood (ages 1 to 5), school age (ages 6 to 12) and adolescence (ages 13 to 18). Within each sensitive period model, we included both abuse and neglect predictors, so that we could understand abuse vs. neglect effects relative to the other. This approach was suitable as the goal of the current study was to examine if deprivation and threat experiences might have different effects on connectivity during cognitive control and risk processing. Furthermore, using latent basis growth modeling we were able to examine both between and within person change patterns of connectivity strength across six years. This also allowed us to examine specific maltreatment effects on trajectories of connectivity strength over time.

Overall, we found evidence for a potential sensitive period for the effects of neglect experienced in adolescence on dACC-insula connectivity during cognitive control. In our sample, chronic abuse and chronic neglect were the strongest predictors of developmental changes in functional connectivity. Understanding these neurobiological mechanisms that link chronic abuse or neglect to negative outcomes will help inform and target interventions to specific populations that are most at risk. It is important to establish pathways from maltreatment to psychopathology outcomes, as our findings suggest there are different neurobiological mechanisms associated with abuse and neglect exposure throughout development. These were specifically predicted by chronic maltreatment occuring across multiple developmental periods. It will be important for for future intervention research to consider focusing on children and adolescents who are most at risk for experiencing recurrent and chronic maltreatment.

References

Andersen, S. L., & Teicher, M. H. (2009). Desperately driven and no brakes: Developmental stress exposure and subsequent risk for substance abuse. *Neuroscience and Biobehavioral Reviews*, *33*(4), 516–524. https://doi.org/10.1016/j.neubiorev.2008.09.009.Desperately

Andersen, S. L., Tomada, A., Vincow, E. S., Valente, E., Polcari, A., & Teicher, M. H. (2008). Preliminary evidence for sensitive periods in the effect of childhood sexual abuse on regional brain development. *Journal of Neuropsychiatry and Clinical Neurosciences*, *20*(3), 292-301. https://doi.org/10.1176/jnp.2008.20.3.292

Arbuckle, J. (1996). Full information estimation in the presence of incomplete data. In G.A Marcoulides & R.E Shumacker (Eds.), *Advanced structural equation modeling: Issues and Techniques*. Mahwah, NJ: Erlbaum.

Blakemore, S.-J. (2008). The social brain in adolescence. *Nature Reviews Neuroscience*, *9*(4), 267–277. https://doi.org/10.1038/nrn2353

Birn, R. M., Roeber, B. J., Pollak, S. D., & Reyna, V. F. (2017). Early childhood stress exposure, reward pathways, and adult decision making. *Proceedings of the National Academy of Sciences of the United States of America*, *114*(51), 13549–13554. https://doi.org/10.1073/pnas.1708791114

Botvinick, M.M., Cohen, J.D., & Carter C.S (2004). Conflict monitoring and anterior cingulate cortex: an update. *Trends in Cognitive Science* 8: 539-546.

Brieant, A. E., Sisk, L. M., & Gee, D. G. (2021). Associations among negative life events, changes in cortico-limbic connectivity, and psychopathology in the ABCD Study. *Developmental Cognitive Neuroscience*, *52*, 101022. https://doi.org/10.1016/j.dcn.2021.101022

Bush, G., Shin, L. M., Holmes, J., Rosen, B. R., & Vogt, B. A. (2003). The multi-source interference

task: Validation study with fMRI in individual subjects. *Molecular Psychiatry, 8*(1*)*, 60–70. https://doi.org/10.1038/sj.mp.4001217

Callaghan, B. L., & Tottenham, N. (2016). The Stress Acceleration Hypothesis: Effects of early-life adversity on emotion circuits and behavior. *Current Opinion in Behavioral Sciences*, *7*, 76–81. https://doi.org/10.1016/j.cobeha.2015.11.018

Casey, B. J., Galván, A., & Somerville, L. H. (2016). Beyond simple models of adolescence to an integrated circuit-based account: A commentary. *Developmental Cognitive Neuroscience, 17*, 128–130. https://doi.org/10.1016/j.dcn.2015.12.006

Casey, B. J., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review*, *28*(1), 62–77. https://doi.org/10.1016/j.dr.2007.08.003

Casey, B., & Jones, R. M. (2010). Neurobiology of the Adolescent Brain and Behavior. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*(12), 1189–1285. https://doi.org/10.1016/j.jaac.2010.08.017.Neurobiology

Casey, B. J., Jones, R. M., & Somerville, L. H. (2011). Braking and accelerating of the adolescent brain. *Journal of Research on Adolescence*, *21*(1), 21–33. https://doi.org/10.1111/j.1532-7795.2010.00712.x

Chahal, R., Vilgis, V., Grimm, K. J., Hipwell, A. E., Forbes, E. E., Keenan, K., & Guyer, A. E. (2018). Girls' pubertal development is associated with white matter microstructure in late adolescence. *NeuroImage*, *181*, 659–669. https://doi.org/10.1016/j.neuroimage.2018.07.050

Cicchetti, D., & Handley, E. D. (2019). Child maltreatment and the development of substance use and disorder. *Neurobiology of Stress*, *10*, 100144. https://doi.org/10.1016/j.ynstr.2018.100144

Cohodes, E. M., Kitt, E. R., Baskin-Sommers, A., & Gee, D. G. (2021). Influences of early-life stress on frontolimbic circuitry: Harnessing a dimensional approach to elucidate the effects of

heterogeneity in stress exposure. *Developmental Psychobiology*, *63*(2), 153–172. https://doi.org/10.1002/dev.21969

Cheng, T. W., Mills, K. L., Miranda Dominguez, O., Zeithamova, D., Perrone, A., Sturgeon, D., Feldstein Ewing, S. W., Fisher, P. A., Pfeifer, J. H., Fair, D. A., & Mackiewicz Seghete, K. L. (2021). Characterizing the impact of adversity, abuse, and neglect on adolescent amygdala resting-state functional connectivity. *Developmental Cognitive Neuroscience*, *47*, 100894. https://doi.org/10.1016/j.dcn.2020.100894

Cohen, J. (1988). Statistical Power Analysis for the Behavioral Sciences (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers.

Cohen, A. O., Breiner, K., Steinberg, L., Bonnie, R. J., Scott, E. S., Taylor-Thompson, K., Rudolph, M. D., Chein, J., Richeson, J. A., Heller, A. S., Silverman, M. R., Dellarco, D. V., Fair, D. A., Galvan, A., & Casey, B. J. (2016). When is an adolescent an adult? Assessing cognitive control in emotional and nonemotional contexts. *Psychological Science, 27*(4), 549–562. https://doi.org/10.1177/0956797617690343

D'Acremont, M., & Bossaerts, P. (2008). Neurobiological studies of risk assessment: A comparison of expected utility and mean-variance approaches. *Cognitive, Affective, & Behavioral Neuroscience, 8*(4), 363-374. https://doi.org/10.3758/CABN.8.4.363

Dennison, M. J., Rosen, M. L., Sambrook, K. A., Jenness, J. L., Sheridan, M. A., & McLaughlin, K. A. (2019). Differential associations of distinct forms of childhood adversity with neurobehavioral measures of reward processing: A developmental pathway to depression. *Child Development*, *90*(1), e96–e113. https://doi.org/10.1111/cdev.13011

Duffy, K. A., Mclaughlin, K. A., & Green, P. A. (2018). Early life adversity and health-risk behaviors: Proposed psychological and neural mechanisms. *Annals of the New York Academy of*

Sciences, 151–169. https://doi.org/10.1111/nyas.13928

Ernset, M., Pine, D.S., & Hardin, M. (2006). Triadic model of the neurobiology of motivated behavior in adolescence. *Psychological Medicine, 36*, 299-312

Fareri, D. S., Gabard-durnam, L., Goff, B., Flannery, J., Gee, D. G., Lumian, D. S., Caldera, C., & Tottenham, N. (2017). Altered ventral striatal-medial prefrontal cortex resting-state connectivity mediates adolescent social problems after early institutional care. *Developmental Psychopathology*, *29*(5), 1865–1876. https://doi.org/10.1017/S0954579417001456.Altered

Francis, E. R., Tsaligopoulou, A., Stock, S. E., Pingault, J., & Baldwin, J. R. (2023). Subjective and objective experiences of childhood adversity: A meta-analysis of their agreement and relationships with psychopathology. *Journal of Child Psychology and Psychiatry*, *64*(8), 1185–1199. https://doi.org/10.1111/jcpp.13803

Fuhrmann, L., Knoll, L.J., & Blakemore, S. (2015). Adolescences as a sensitive period of brain development. *Trends in Cognitive Sciences, 19*(10),558-566. http://dx.doi.org/10.1016/j.tics.2015.07.008

Gee, D. G. (2021). When do sensitive periods emerge later in development? Trends in Cognitive Sciences, In press.

Gee, D. G., Humphreys, K. L., Flannery, J., Goff, B., Telzer, E. H., Shapiro, M., Hare, T. A., Bookheimer, S. Y., & Tottenham, N. (2013). A developmental shift from positive to negative connectivity in human amygdala-prefrontal circuitry. *Journal of Neuroscience, 33*(10), 4584–4593. https://doi.org/10.1523/JNEUROSCI.3446-12.2013

Gerin, M. I., Puetz, V. B., Blair, R. J. R., White, S., Sethi, A., Hoffmann, F., Palmer, A. L., Viding, E., & McCrory, E. J. (2017). A neurocomputational investigation of reinforcement-based decision making as a candidate latent vulnerability mechanism in maltreated children.

Development and Psychopathology, *29*(5), 1689–1705. https://doi.org/10.1017/S095457941700133X

Giano, Z., Wheeler, D. L., & Hubach, R. D. (2020). The frequencies and disparities of adverse childhood experiences in the U.S. *BMC Public Health*, *20*(1), 1327. https://doi.org/10.1186/s12889-020-09411-z

Goff, B., Gee, D. G., Telzer, E. H., Humphreys, K. L., Gabard-Durnam, L., Flannery, J., & Tottenham, N. (2013). Reduced nucleus accumbens reactivity and adolescent depression following early-life stress. *Neuroscience*, *249*, 129–138. https://doi.org/10.1016/j.neuroscience.2012.12.010

Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychopathology in the National Comorbidity Survey Replication (NCS-R) 1: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, *67*(2), 113. https://doi.org/10.1001/archgenpsychiatry.2009.186.Childhood

Hanson, J. L., Hariri, A. R., & Williamson, D. E. (2015). Blunted ventral striatum development in adolescence reflects emotional neglect and predicts depressive symptoms. *Biological Psychiatry*, *78*(9), 598–605. https://doi.org/10.1016/j.biopsych.2015.05.010

Hanson, J. L., Knodt, A. R., Brigidi, B. D., & Hariri, A. R. (2018). Heightened connectivity between the ventral striatum and medial prefrontal cortex as a biomarker for stress-related psychopathology: Understanding interactive effects of early and more recent stress. *Psychological Medicine*, *48*(11), 1835–1843. https://doi.org/10.1017/S0033291717003348

Hanson, J. L., van den Bos, W., Roeber, B. J., Rudolph, K. D., Davidson, R. J., & Pollak, S. D. (2017). Early adversity and learning: implications for typical and atypical behavioral

development. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 58*(7), 770–778. https://doi.org/10.1111/jcpp.12694

Henson, R. (2007). Efficient experimental design for fMRI. In Penny, W., Firston, K., Ashburner, J., Keibel, S., Nichols, T. (Eds.). Statistical Parametric Mapping. *Elsevier*. (1st ed., pp. 193-210). https://doi.org/10.1016/B978-012372560-8/50015-2

Herzberg, M. P., & Gunnar, M. R. (2020). Early life stress and brain function: Activity and connectivity associated with processing emotion and reward. *NeuroImage, 209,* 116493. https://doi.org/10.1016/j.neuroimage.2019.116493

Herzberg, M. P., McKenzie, K. J., Hodel, A. S., Hunt, R. H., Mueller, B. A., Gunnar, M. R., & Thomas, K. M. (2021). Accelerated maturation in functional connectivity following early life stress: Circuit specific or broadly distributed? *Developmental Cognitive Neuroscience*, *48*, 100922. https://doi.org/10.1016/j.dcn.2021.100922

Herzog, J. I., Thome, J., Demirakca, T., Koppe, G., Ende, G., Lis, S., Rausch, S., Priebe, K., Müller-Engelmann, M., Steil, R., Bohus, M., & Schmahl, C. (2020). Influence of severity of type and timing of retrospectively reported childhood maltreatment on remale amygdala and hippocampal volume. *Scientific Reports*, *10*(1), 1–10. https://doi.org/10.1038/s41598-020-57490-0

Holt, C. A., & Laury, S. K. (2002). Risk Aversion and Incentive Effects. *The American Economic Review, 92*(5), 1644–1655. https://www.jstor.org/stable/3083270

Holz, N. E., Berhe, O., Sacu, S., Schwarz, E., Tesarz, J., Heim, C. M., & Tost, H. (2023). Early Social Adversity, Altered Brain Functional Connectivity, and Mental Health. *Biological Psychiatry*, *93*(5), 430–441. https://doi.org/10.1016/j.biopsych.2022.10.019

Hughes, K., Bellis, M. A., Hardcastle, K. A., Sethi, D., Butchart, A., Mikton, C., Jones, L., & Dunne, M. P. (2017). The effect of multiple adverse childhood experiences on health: a systematic

review and meta-analysis. *The Lancet Public Health*, *2*(8), e356–e366. https://doi.org/10.1016/S2468-2667(17)30118-4

Humphreys, K. L., King, L. S., Sacchet, M. D., Camacho, M. C., Colich, N. L., Ordaz, S. J., Ho, T. C., & Gotlib, I. H. (2019). Evidence for a sensitive period in the effects of early life stress on hippocampal volume. *Developmental Science*, *22*(3), 1–11. https://doi.org/10.1111/desc.12775

Kessler, R. C., Avenevoli, S., & Merikangas, K. R. (2001). Mood disorders in children and adolescents. *Biological Psychiatry*, *49*, 1002–1014. https://doi.org/10.1007/978-1-4939-2528-5_19

Kim-Spoon, J., Lauharatanahirun, N., Peviani, K. M., Brieant, A., Deater-Deckard, K., Bickel, W. K., & King-Casas, B. (2019). Longitudinal pathways linking family risk, neural risk processing, delay discounting, and adolescent substance use. *Journal of Child Psychology and Psychiatry*, *60*(6), 655–664. https://doi.org/10.1111/jcpp.13015.Longitudinal

Kim-Spoon, J., Herd, T., Brieant, A., Peviani, K., Deater-Deckard, K., Lauharatanahirun, N., Lee, J., & King-Casas, B. (2021). Maltreatment and brain development: The effects of abuse and neglect on longitudinal trajectories of neural activation during risk processing and cognitive control. *Developmental Cognitive Neuroscience*, *48*. https://doi.org/10.1016/j.dcn.2021.100939

Kirsch, D. E., Tretyak, V., Radpour, S., Weber, W. A., Nemeroff, C. B., Fromme, K., Strakowski, S. M., & Lippard, E. T. C. (2021). Childhood maltreatment, prefrontal-paralimbic gray matter volume, and substance use in young adults and interactions with risk for bipolar disorder. *Scientific Reports*, *11*(1), 1–12. https://doi.org/10.1038/s41598-020-80407-w

Kline, R. B., (2011). *Principles and practice of structural equation modeling* (3rd ed.). New York: Guildford Press.

Ladouceur, C. D. (2012). Neural systems supporting cognitive-affective interactions in adolescence:

The role of puberty and implications for affective disorders. *Frontiers in Integrative Neuroscience*, *6*. https://doi.org/10.3389/fnint.2012.00065

Ladouceur, C. D., Kerestes, R., Schlund, M. W., Shirtcliff, E. A., Lee, Y., & Dahl, R. E. (2019). Neural systems underlying reward cue processing in early adolescence: The role of puberty and pubertal hormones. *Psychoneuroendocrinology*, *102*, 281–291. https://doi.org/10.1016/j.psyneuen.2018.12.016

Lambert, H. K., King, K. M., Monahan, K. C., & McLaughlin, K. A. (2016). Differential associations of threat and deprivation with emotion regulation and cognitive control in adolescence. *Development and Psychopathology, 29*(3), 929–940. https://doi.org/10.1017/S0954579416000584

Lauharatanahirun, N., Maciejewski, D., Kim-Spoon, J., & Casas. B. (in press). Risk-related insular cortex activation is linked to longitudinal changes in adolescent health risk behaviors. *Developmental Cognitive Neuroscience.*

Laskowski, C. S., Williams, R. J., Martens, K. M., Gruber, A. J., Fisher, K. G., & Euston, D. R. (2016). The role of the medial prefrontal cortex in updating reward value and avoiding perseveration. *Behavioural Brain Research*, *306*, 52–63. https://doi.org/10.1016/j.bbr.2016.03.007

Li, M., Lauharatanahirun, N., Steinberg, L., King-Casas, B., Kim-Spoon, J., & Deater-Deckard, K. (2019). Longitudinal link between trait motivation and risk-taking behaviors via neural risk processing. *Developmental Cognitive Neuroscience*, *40*, 100725 https://doi.org/10.1016/j.dcn.2019.100725

Lim, L., Hart, H., Mehta, M. A., Simmons, A., Mirza, K., & Rubia, K. (2015). Neural correlates of error processing in young people with a history of severe childhood abuse: An fMRI study.

American Journal of Psychiatry, *172*(9), 892–900. https://doi.org/10.1176/appi.ajp.2015.14081042

Little, T. D. (2013). Longitudinal structural equation modeling. The Guilford Press.

Little, R. J., & Rubin, D. B. (2003). Statistical analysis with missing data. John Wiley & Sons, Inc. https://doi.org/10.1002/9781119013563

MacDonald, S. W. S., Karlsson, S., Rieckmann, A., Nyberg, L., & Bäckman, L. (2012). Aging-related increases in behavioral variability: Relations to losses of dopamine D 1 receptors. *Journal of Neuroscience*, *32*(24), 8186–8191. https://doi.org/10.1523/JNEUROSCI.5474-11.2012

Machlin, L., Egger, H. L., Stein, C. R., Navarro, E., Carpenter, K. L. H., Goel, S., Patel, K. K., Copeland, W. E., & Sheridan, M. A. (2023). Distinct Associations of Deprivation and Threat With Alterations in Brain Structure in Early Childhood. *Journal of the American Academy of Child & Adolescent Psychiatry*, S0890856723000618. https://doi.org/10.1016/j.jaac.2023.02.006

Marusak, H. A., Martin, K. R., Etkin, A., & Thomason, M. E. (2015). Childhood Trauma Exposure Disrupts the Automatic Regulation of Emotional Processing. *Neuropsychopharmacology*, *40*(5), 1250–1258. https://doi.org/10.1038/npp.2014.311

McLaren, D. G., Ries, M. L., Xu, G., & Johnson, S. C. (2012). A generalized form of context-dependent psychophysiological interactions (gPPI): A comparison to standard approaches. *NeuroImage, 61*(4), 1277–1286.https://doi.org/10.1016/j.neuroimage.2012.03.068

McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood advrsity. *Current Directions in Psychological Science*, *25*(4), 239–245. https://doi.org/10.1177/0963721416655883.Beyond

McLaughlin, K. A., Sheridan, M. A., Humphreys, K. L., Belsky, J., & Ellis, B. J. (2021). The value of dimensional models of early experience: Thinking clearly about concepts and categories.

Perspectives on Psychological Science. *16*(6), 1463-1472. https://doi.org/10.1177/1745691621992346

McLaughlin, K. A., Weissman, D., & Bitrán, D. (2019). Childhood Adversity and Neural Development: A Systematic Review. *Annual Review of Developmental Psychology*, *1*(1), 277–312. https://doi.org/10.1146/annurev-devpsych-121318-084950

Menon, V., & Uddin, L. Q. (2010). Saliency, switching, attention and control: A network model of insula function. *Brain Structure and Function*, *214*(5–6), 655–667. https://doi.org/10.1007/s00429-010-0262-0

Mohr, P. N. C., Biele, G., & Heekeren, H. R. (2010). *Neural processing of risk. Journal of Neuroscience, 30*(19), 6613–6619. https://doi.org/10.1523/JNEUROSCI.0003-10.2010

Molnar-Szakacs, I., & Uddin, L. Q. (2022). Anterior insula as a gatekeeper of executive control. *Neuroscience & Biobehavioral Reviews*, *139*, 104736. https://doi.org/10.1016/j.neubiorev.2022.104736

Morgan, J. K., Shaw, D. S., Olino, T. M., Musselman, S. C., Kurapati, N. T., & Forbes, E. E. (2016). History of Depression and Frontostriatal Connectivity During Reward Processing in Late Adolescent Boys. *Journal of Clinical Child & Adolescent Psychology*, *45*(1), 59–68. https://doi.org/10.1080/15374416.2015.1030753

Muthén, L [Linda K. Muthen]. 2013, February 14. *CFI Fit Indices*. [Online forum post]. StatModel. http://www.statmodel.com/discussion/messages/9/7450.html?1513463765

Muthén, L., & Muthén, B. . (1998-2021.). Mplus User's Guide. (Seventh). Muthén & Muthén.

Ordaz, S. J., Foran, W., Velanova, K., & Luna, B. (2013). Longitudinal growth curves of brain function underlying inhibitory control through adolescence. *Journal of Neuroscience, 33*(46), 18109–18124. https://doi.org/10.1523/JNEUROSCI.1741-13.2013

Oshri, A., Rogosch, F. A., Burnette, M. L., & Cicchetti, D. (2011). Developmental pathways to adolescent cannabis abuse and dependence: Child maltreatment, emerging personality, and internalizing versus externalizing psychopathology. *Psychology of Addictive Behaviors*, *25*(4), 634–644. https://doi.org/10.1037/a0023151

Pagliaccio, D., & Barch, D. M. (2016). Early life adversity and risk for depression: Alterations in cortisol and brain structure and function as mediating mechanisms. *Systems Neuroscience in Depression.* 29-77. https://doi.org/10.1016/B978-0-12-802456-0.00002-9

Pechtel, P., Lyons-ruth, K., Andersen, C. M., & Teicher, M. H. (2014). Sensitive periods of amygdala development: The role of maltreatment in preadolescence. *NeuroImage*, *97*, 236–244. https://doi.org/10.1016/j.neuroimage.2014.04.025.Sensitive

Power, J. D., Barnes, K. A., Snyder, A. Z., Schlaggar, B. L., & Petersen, S. E. (2012). Spurious but systematic correlations in functional connectivity MRI networks arise from subject motion. *NeuroImage, 1*(59), 2142–2154. https://doi.org/10.1016/j.neuroimage.2011.10.018.Spurious

Rakesh, D., Allen, N. B., & Whittle, S. (2021a). Longitudinal changes in within-salience network functional connectivity mediate the relationship between childhood abuse and neglect, and mental health during adolescence. *Psychological Medicine*, 1-13. https://doi.org/10.1017/S0033291721003135

Rakesh, D., Kelly, C., Vijayakumar, N., Zalesky, A., Allen, N. B., & Whittle, S. (2021b). Unraveling the consequences of childhood maltreatment: Deviations from typical functional neurodevelopment mediate the relationship between maltreatment history and depressive symptoms. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *6*(3), 329–342. https://doi.org/10.1016/j.bpsc.2020.09.016

Rakesh, D., Whittle, S., Sheridan, M. A., & McLaughlin, K. A. (2023). Childhood socioeconomic

status and the pace of structural neurodevelopment: Accelerated, delayed, or simply different? *Trends in Cognitive Sciences*, S1364661323000736. https://doi.org/10.1016/j.tics.2023.03.011

Rogosch, F.A., & Chiccetti, D. (1994). Illustrating the interface of family and peer relations through the study of maltreatment. *Social Development, 3*, 291-308. https://doi.org/10.1111/j.1467-9507.1994.tb00046.x

Sapolsky, R. M. (2017). *Behave: The biology of humans at our best and worst.*: New York: Penguin Press.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods, 7*(2), 147–177. https://doi.org/10.1037/1082-989X.7.2.147

Shackman, J. E., & Pollak, S. D. (2014). Impact of physical maltreatment on the regulation of negative affect and aggression. *Development and Psychopathology*, *26*(4pt1), 1021–1033. https://doi.org/10.1017/S0954579414000546

Sheridan, M. A., McLaughlin, K. A., Winter, W., Fox, N., Zeanah, C., & Nelson, C. A. (2018). Early deprivation disruption of associative learning is a developmental pathway to depression and social problems. *Nature Communications*, *9*(1), 1–8&. https://doi.org/10.1038/s41467-018-04381-8

Sheridan, M. A., Sarsour, K., Jutte, D., D'Esposito, M., & Boyce, W. T. (2012). The Impact of Social Disparity on Prefrontal Function in Childhood. *PLoS ONE*, *7*(4), e35744. https://doi.org/10.1371/journal.pone.0035744

Siegel, J. S., Power, J. D., Dubis, J. W., Vogel, A. C., Church, J. A., Schlaggar, B. L., & Petersen, S. E. (2014). Statistical improvements in functional magnetic resonance imaging analyses produced by censoring high-motion data points. *Human Brain Mapping, 35*(5), 1981–1996. https://doi.org/10.1002/hbm.22307

Smith, K. E., & Pollak, S. D. (2020). Rethinking concepts and categories for understanding the neurodevelopmental effects of childhood adversity. *Perspectives on Psychological Science*. *16*(1),67-93. https://doi.org/10.1177/1745691620920725

Soper, D.S. (2022). A-priori Sample Size Calculator for Multiple Regression [Software]. Available from https://www.danielsoper.com/statcalc

Steinberg, L. (2008). A Social Neuroscience Perspective on Adoelscent Risk-Taking. *Developmental Rev Rev*, *28*(1), 78–106. https://doi.org/10.1016/j.dr.2007.08.002.A

Stevens, M. C., Pearlson, G. D., & Calhoun, V. D. (2009). Changes in the interaction of resting-state neural networks from adolescence to adulthood. *Human Brain Mapping*, *30*(8), 2356–2366. https://doi.org/10.1002/hbm.20673

Teicher, M. H., Andersen, C. M., Ohashi, K., Khan, A., McGreenery, C. E., Bolger, E. A., Rohan, M. L., & Vitaliano, G. D. (2018). Differential effects of childhood neglect and abuse during sensitive exposure periods on male and female hippocampus. *NeuroImage*, *169*, 443–452. https://doi.org/10.1016/j.neuroimage.2017.12.055.Differential

Teicher, M. H., & Parigger, A. (2015). The "Maltreatment and Abuse Chronology of Exposure" (MACE) scale for the retrospective assessment of abuse and neglect during development. *PLoS ONE, 10*(2), 1–37. https://doi.org/10.1371/journal.pone.0117423

Teicher, M. H., & Samson, J. A. (2016). Annual research review: enduring neurobiological effects of childhood abuse and neglect. *Journal of Child Psychology and Psychiatry*, *57*(3), 241–266. https://doi.org/10.1111/jcpp.12507.Annual

Teslovich, T., Mulder, M., Franklin, N. T., Ruberry, E. J., Millner, A., Somerville, L. H., Simen, P., Durston, S., & Casey, B. J. (2014). Adolescents let sufficient evidence accumulate before making a decision when large incentives are at stake. *Developmental Science, 17*(1), 59–70.

https://doi.org/10.1111/desc.12092

Van Duijvenvoorde, A. C. K., Huizenga, H. M., Somerville, L. H., Delgado, M. R., Powers, A., Weeda, W. D., Casey, B. J., Weber, E. U., & Figner, B. (2015). Neural Correlates of Expected Risks and Returns in Risky Choice across Development. *The Journal of Neuroscience*, *35*(4), 1549–1560. https://doi.org/10.1523/JNEUROSCI.1924-14.201

Van Harmelen, A. L., Van Tol, M. J., Van Der Wee, N. J. A., Veltman, D. J., Aleman, A., Spinhoven, P., Van Buchem, M. A., Zitman, F. G., Penninx, B. W. J. H., & Elzinga, B. M. (2010). Reduced medial prefrontal cortex volume in adults reporting childhood emotional maltreatment. *Biological Psychiatry*, *68*(9), 832–838. https://doi.org/10.1016/j.biopsych.2010.06.011

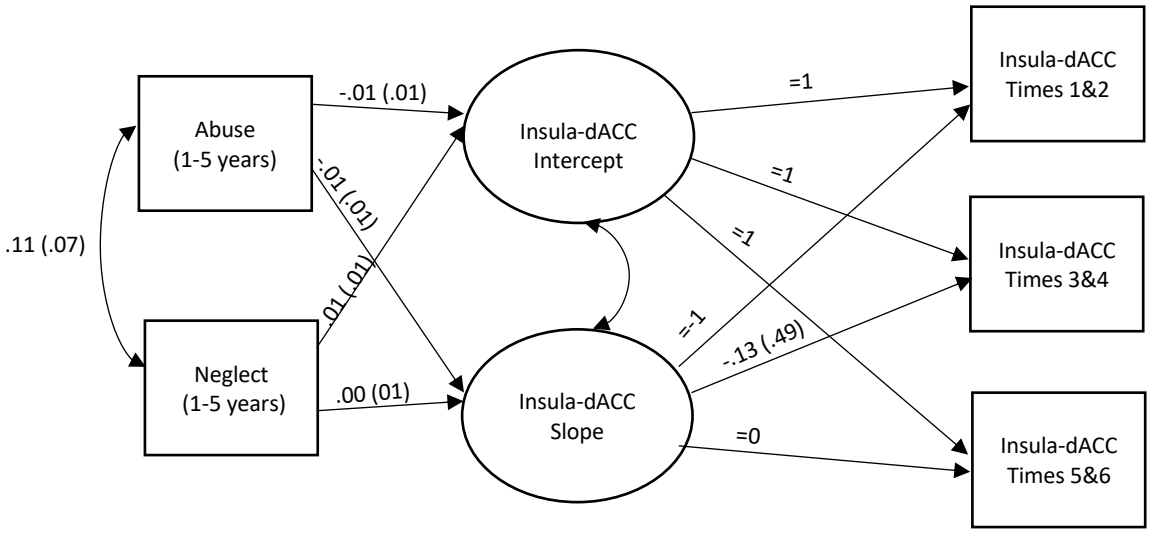
Vannucci, A., Fields, A., Hansen, E., Katz, A., Kerwin, J., Tachida, A., Martin, N., & Tottenham, N. (2023). Interpersonal early adversity demonstrates dissimilarity from early socioeconomic disadvantage in the course of human brain development: A meta-analysis. *Neuroscience & Biobehavioral Reviews*, *150*, 105210. https://doi.org/10.1016/j.neubiorev.2023.105210

Weber, E.U., Shafir, S., Blais, A.R., 2004. Predicting risk sensitivity in humans and lower animals: Risk as variance or coefficient of variation. *Psychological Review 111*(2), 430-445. https://doi.org/10.1037/0033-295X.111.2.430

Zhu, J., Lowen, S. B., Anderson, C. M., Ohashi, K., Khan, A., & Teicher, M. H. (2019). Association of prepubertal and postpubertal exposure to childhood maltreatment with adult amygdala function. *JAMA Psychiatry*, *76*(8), 843–853. https://doi.org/10.1001/jamapsychiatry.2019.0931

Figure 1

Conditional Growth Curve Model of Abuse and Neglect Ages 1 to 5 on Neural Risk Processing Connectivity in the Insula-dACC

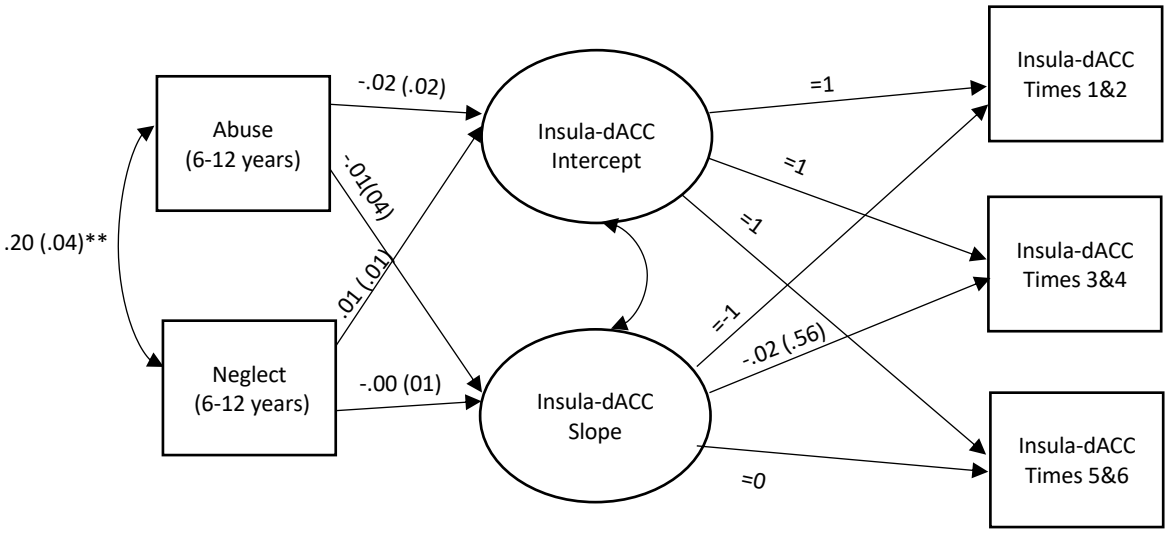


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 2

Conditional Growth Curve Model of Abuse and Neglect Ages 6 to 12 on Neural Risk Processing Connectivity in the Insula-dACC

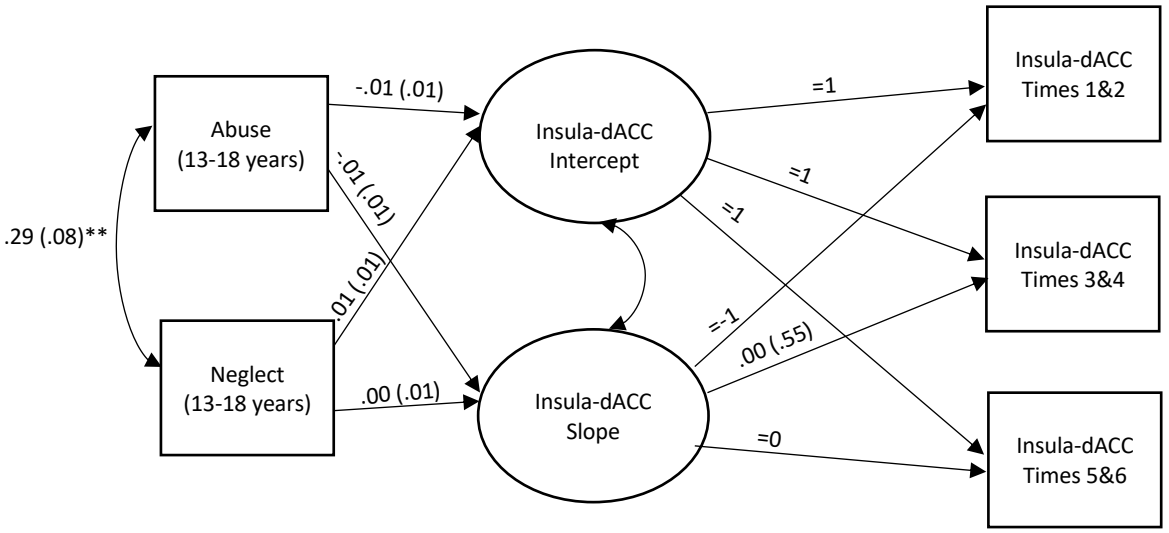


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 3

Conditional Growth Curve Model of Abuse and Neglect Ages 13 to 18 on Neural Risk Processing Connectivity in the Insula-dACC

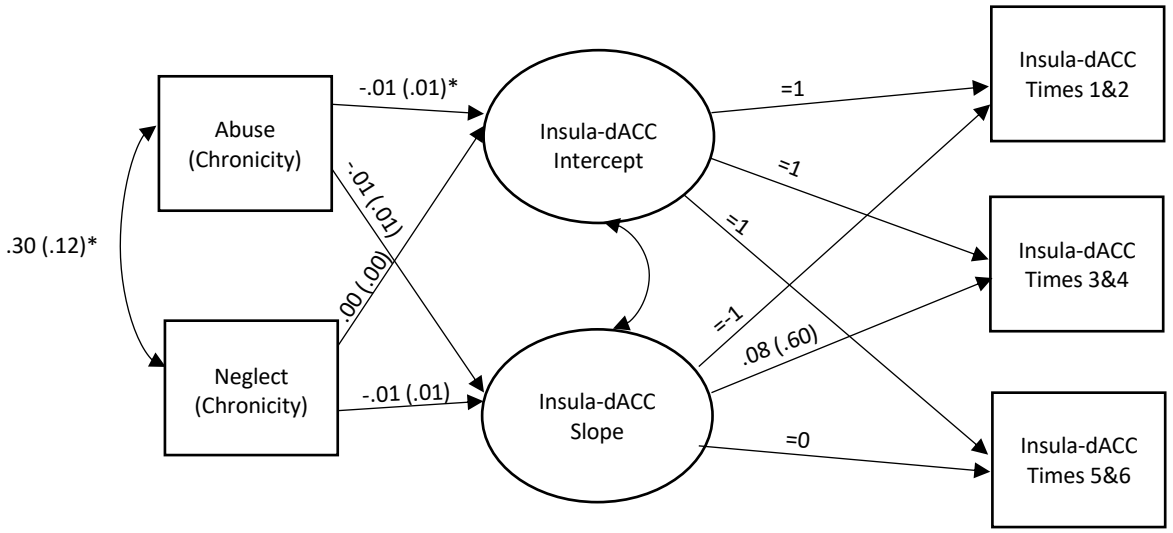


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 4

Conditional Growth Curve Model of Abuse and Neglect Chronicity on Neural Risk Processing Connectivity in the Insula-dACC

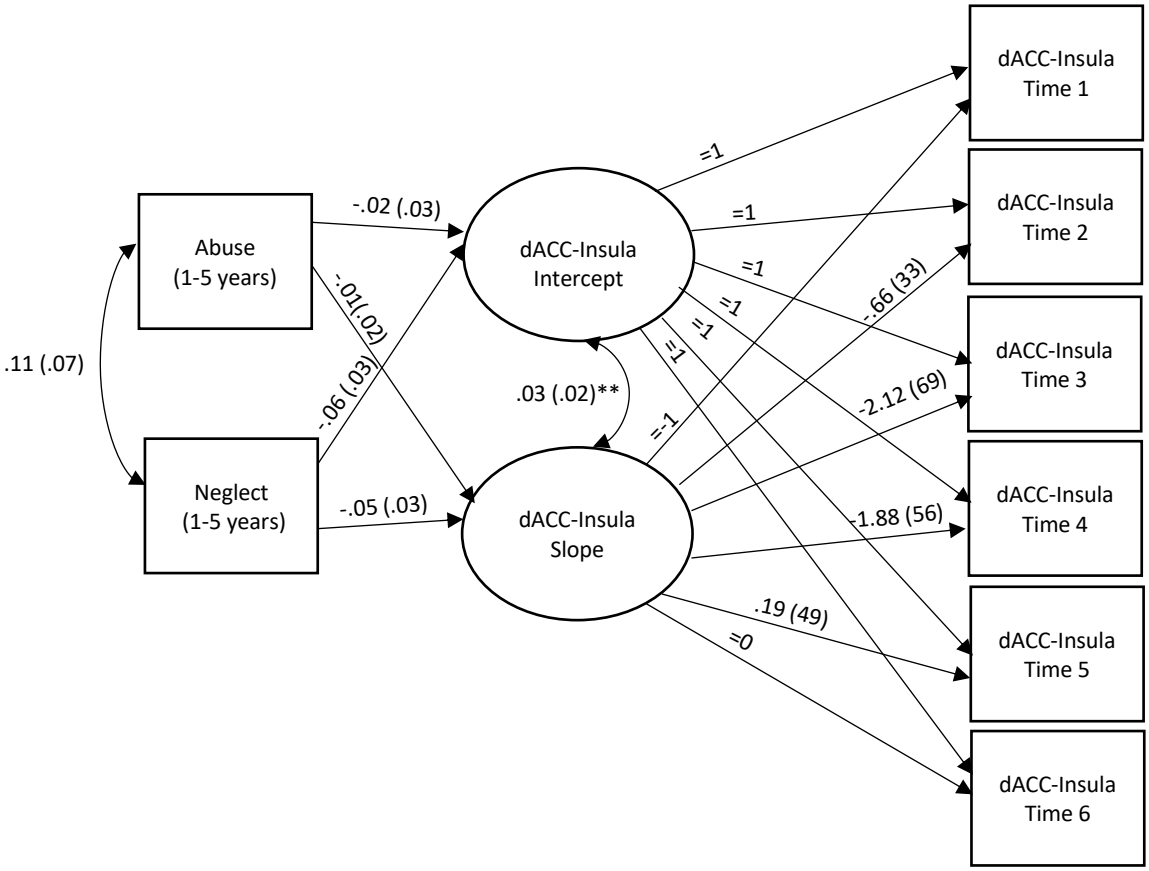


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 5

Conditional Growth Curve Model of Abuse and Neglect Ages 1 to 5 on Neural Cognitive Control Connectivity in the dACC-Insula

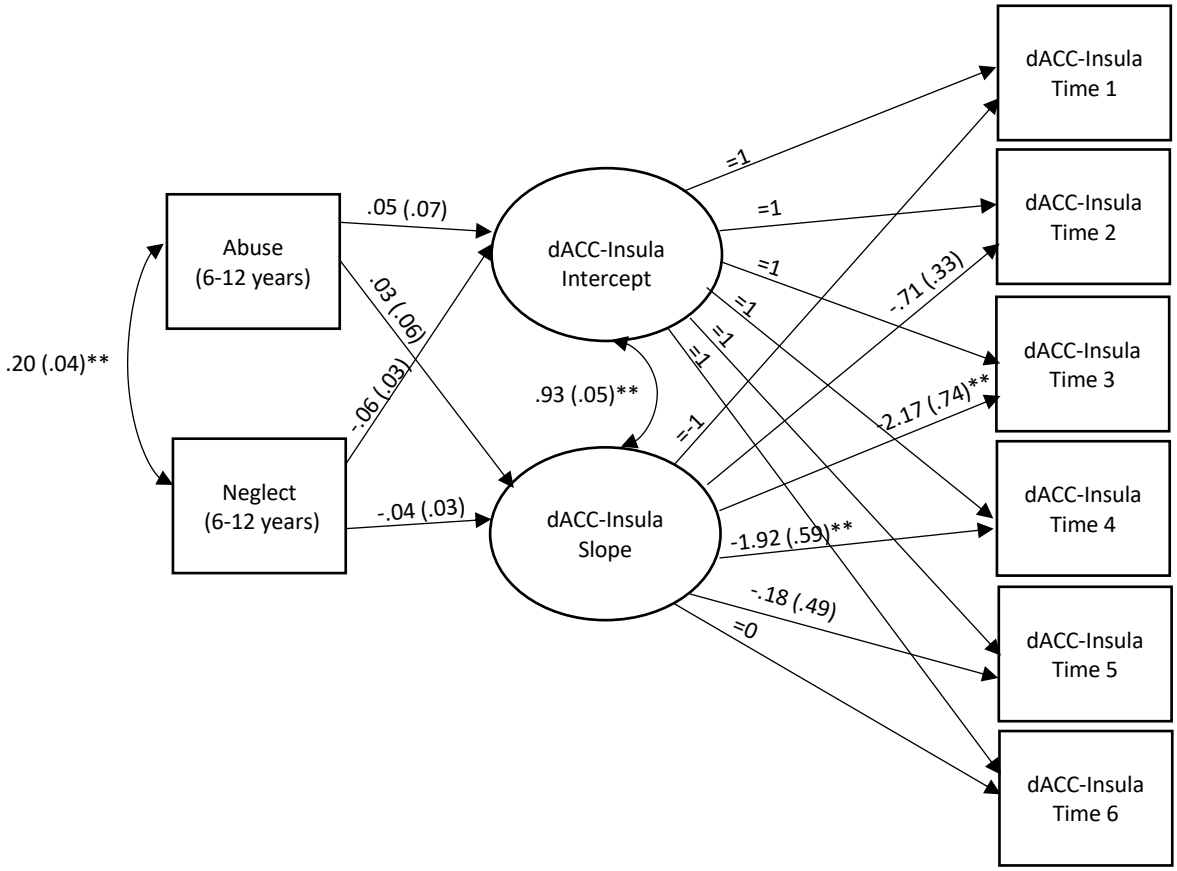


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 6

Conditional Growth Curve Model of Abuse and Neglect Ages 6 to12 on Neural Cognitive Control Connectivity in the dACC-Insula

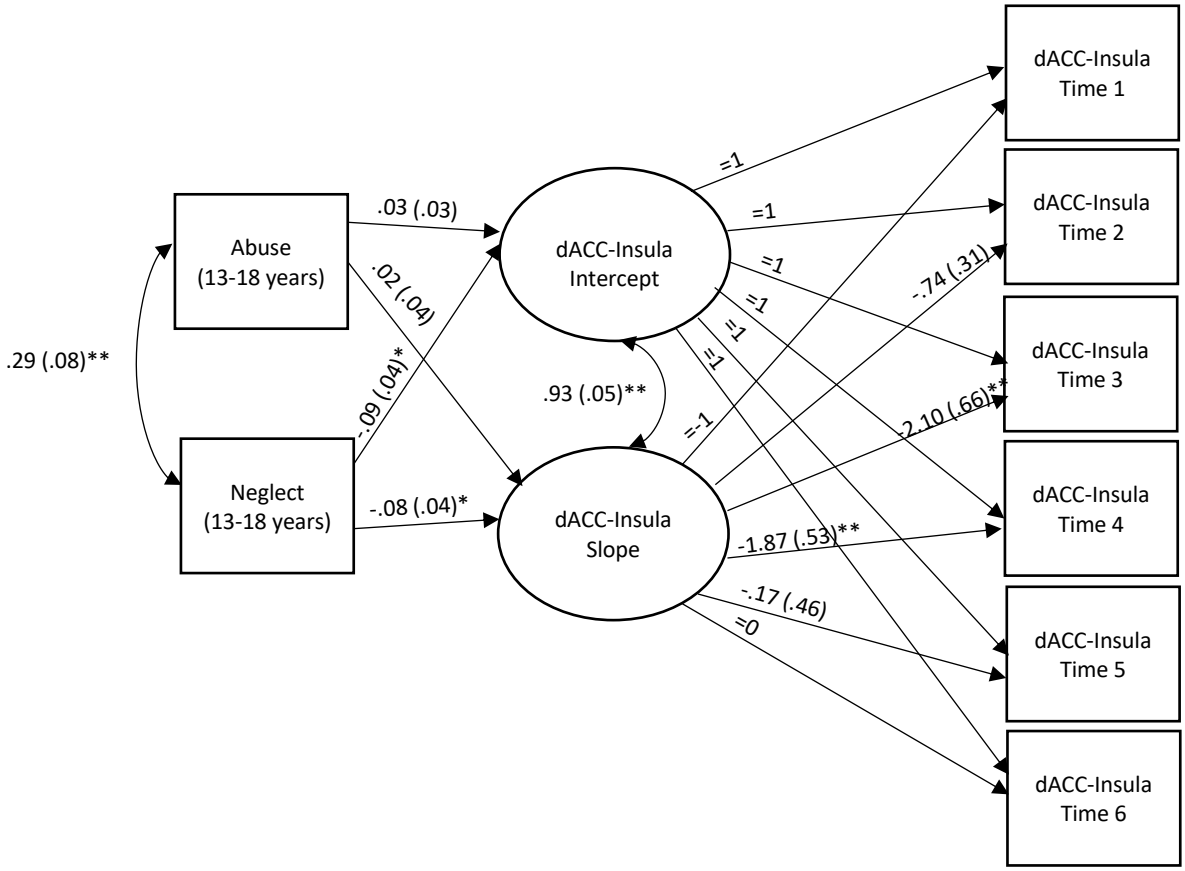


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 7

Conditional Growth Curve Model of Abuse and Neglect Ages 13 to 18 on Neural Cognitive Control Connectivity in the dACC-Insula

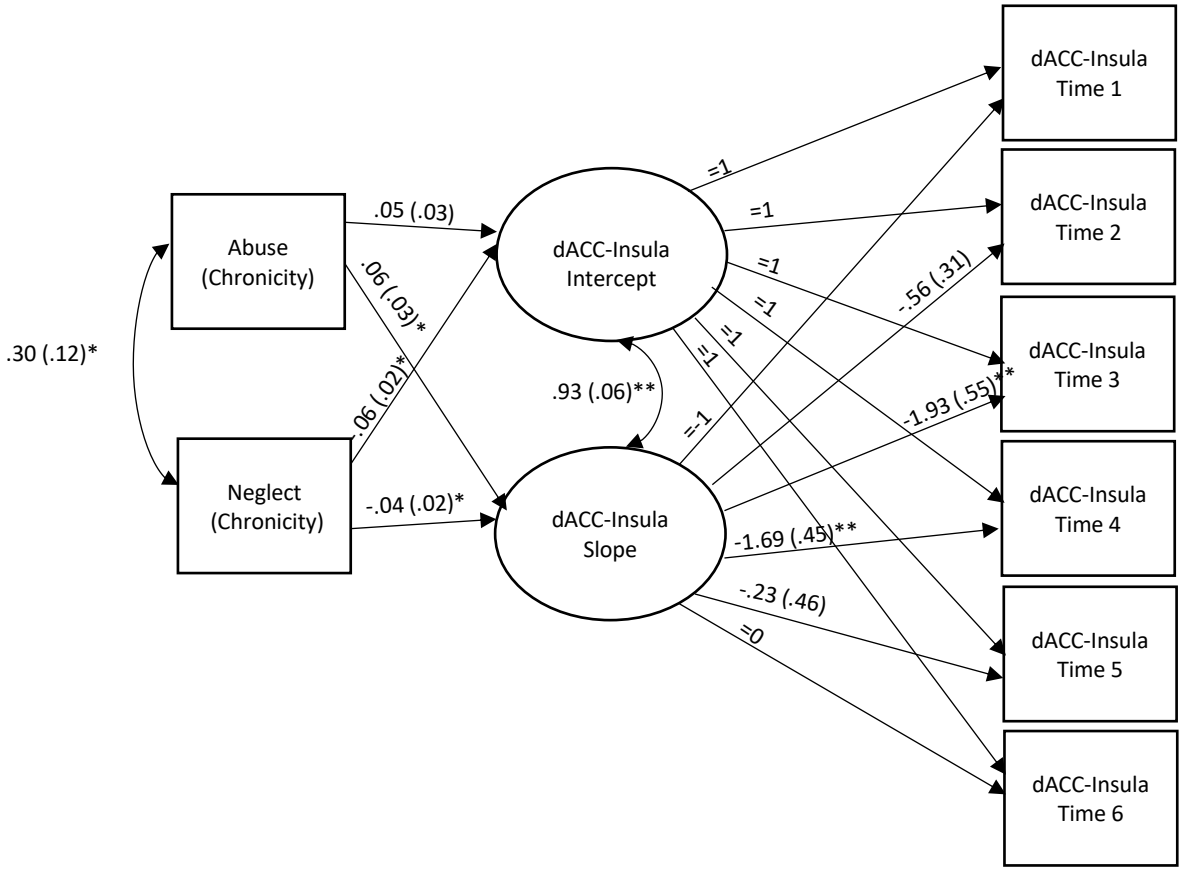


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 8

Conditional Growth Curve Model of Abuse and Neglect Chronicity on Neural Cognitive Control Connectivity in the dACC-Insula

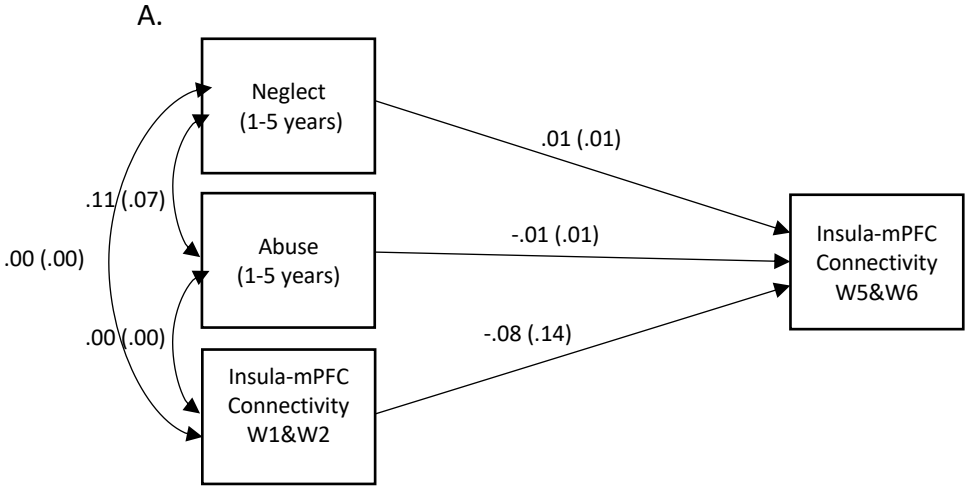


Note. Unstandardized Estimates (standard error) are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 9

Path Model of Abuse and Neglect Ages 1 to 5 on Insula-mPFC connectivity changes from early to late adolescence

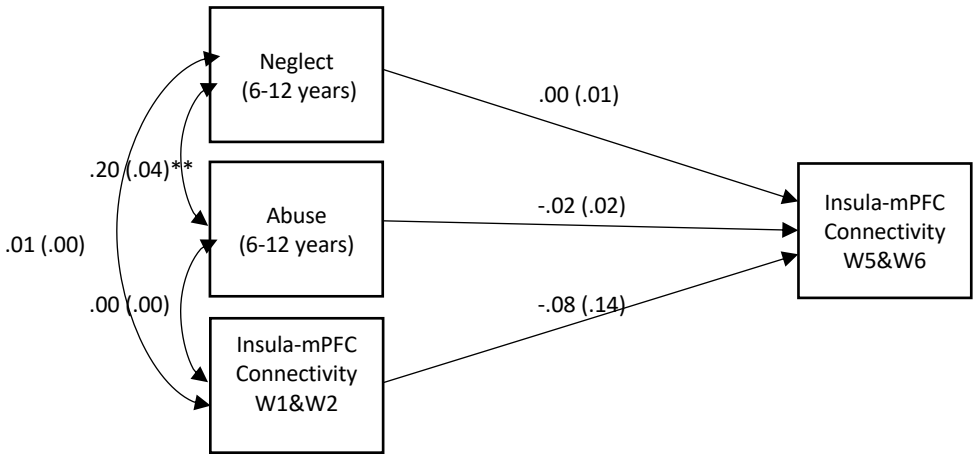


Note. Unstandardized Estimates are presented. mPFC = medial prefrontal cortex.

*$p < .05$, ** $p < .001$.

Figure 10

Path Model of Abuse and Neglect Ages 6 to 12 on Insula-mPFC connectivity changes from early to late adolescence

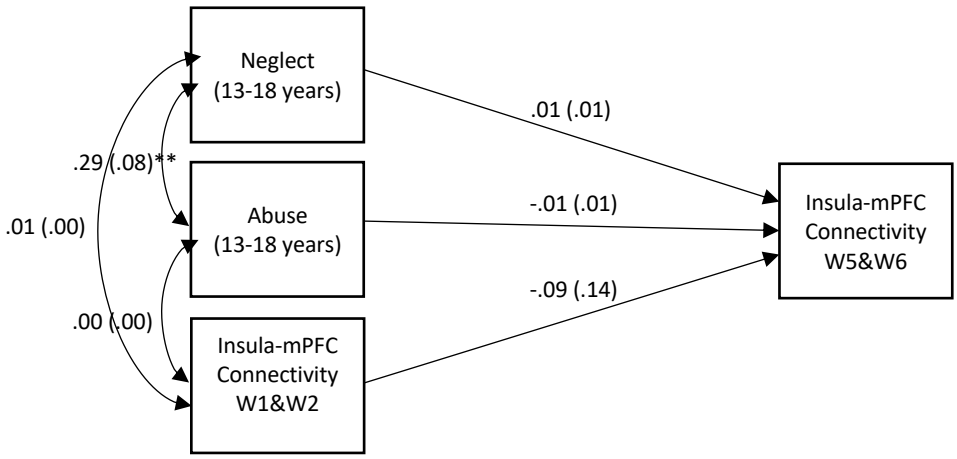


Note. Unstandardized Estimates are presented. mPFC = medial prefrontal cortex.

*$p < .05$, ** $p < .001$.

Figure 11

Path Model of Abuse and Neglect Ages 13 to 18 on Insula-mPFC connectivity changes from early to late adolescence

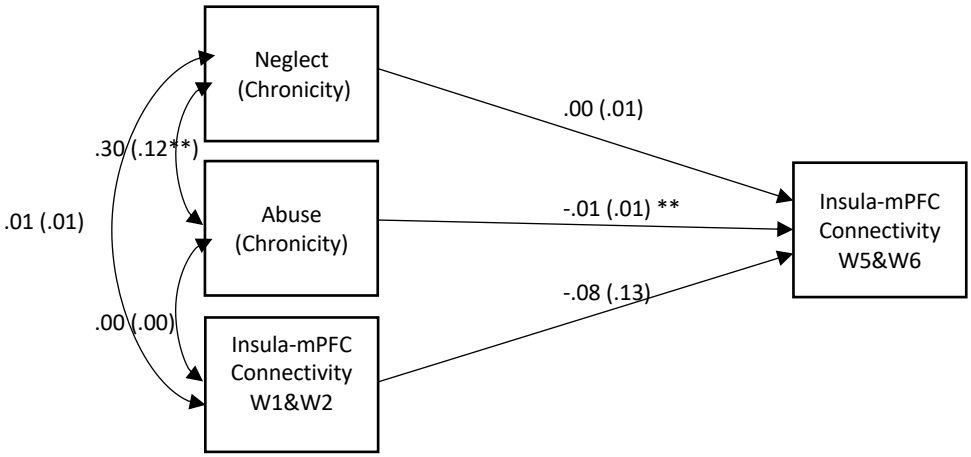


Note. Unstandardized Estimates are presented. mPFC = medial prefrontal cortex.

*$p < .05$, ** $p < .001$.

Figure 12

Path Model of Abuse and Neglect Chronicity on Insula-mPFC connectivity changes from early to late adolescence

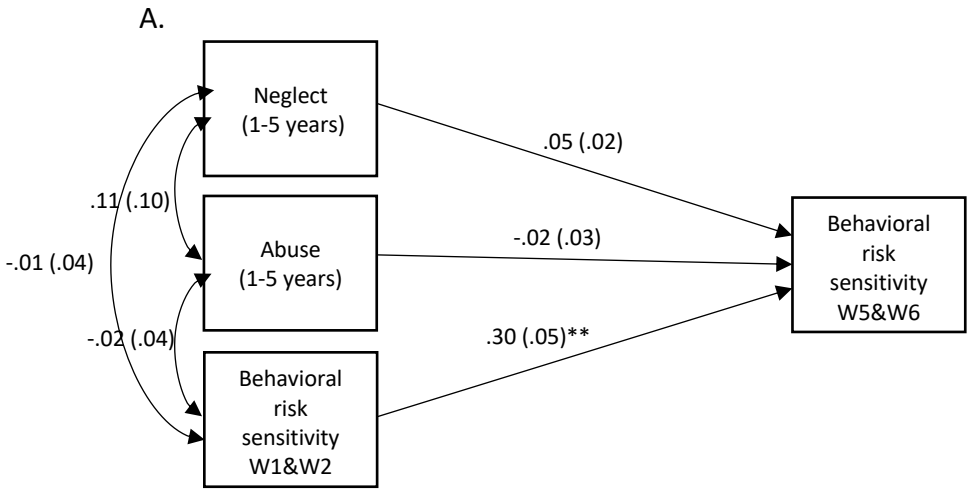


Note. Unstandardized Estimates are presented. mPFC = medial prefrontal cortex.

*$p < .05$, ** $p < .001$.

Figure 13

Path Model of Abuse and Neglect Ages 1 to 5 on risky decision-making changes from early to late adolescence

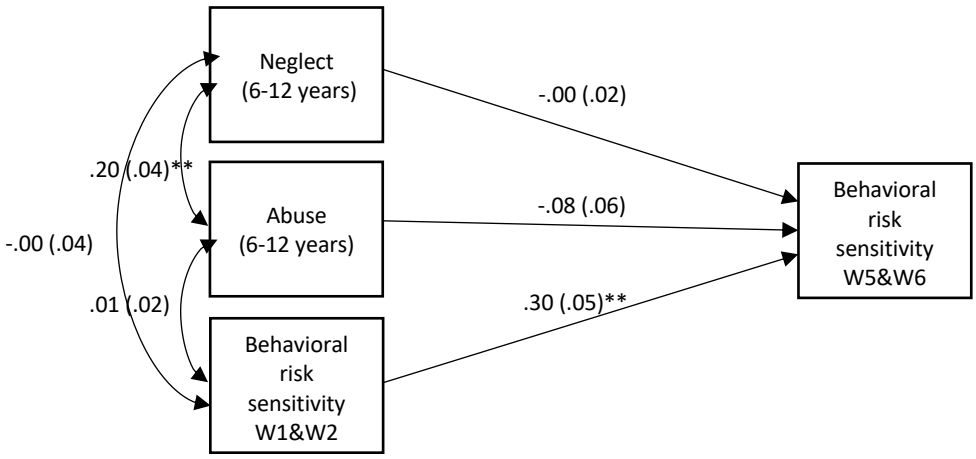


Note. Unstandardized Estimates are presented.

*$p < .05$, ** $p < .001$.

Figure 14

Path Model of Abuse and Neglect Ages 6 to 12 on risky decision-making changes from early to late adolescence



Note. Unstandardized Estimates are presented.

*$p < .05$, ** $p < .001$.

Figure 15

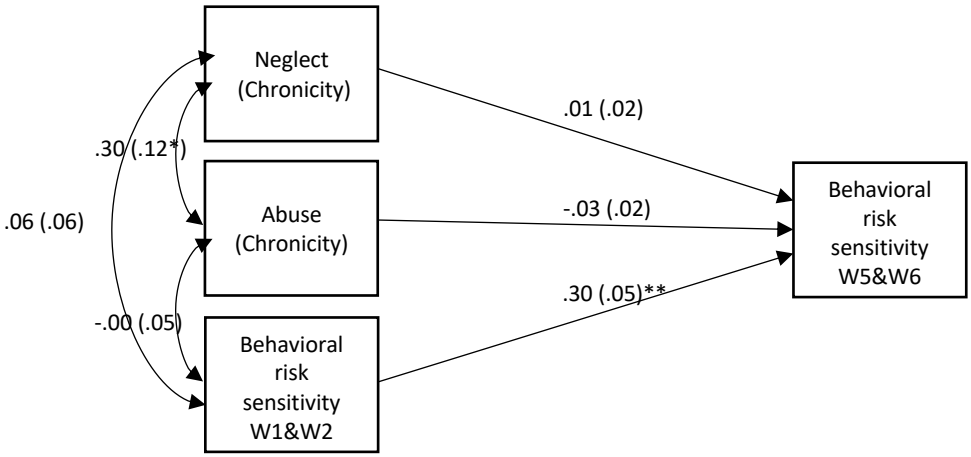
Path Model of Abuse and Neglect Ages 13 to 18 on risky decision-making changes from early to late adolescence

Note. Unstandardized Estimates are presented.

*$p < .05$, ** $p < .001$.

Figure 16

Path Model of Abuse and Neglect Chronicity on risky decision-making changes from early to late adolescence

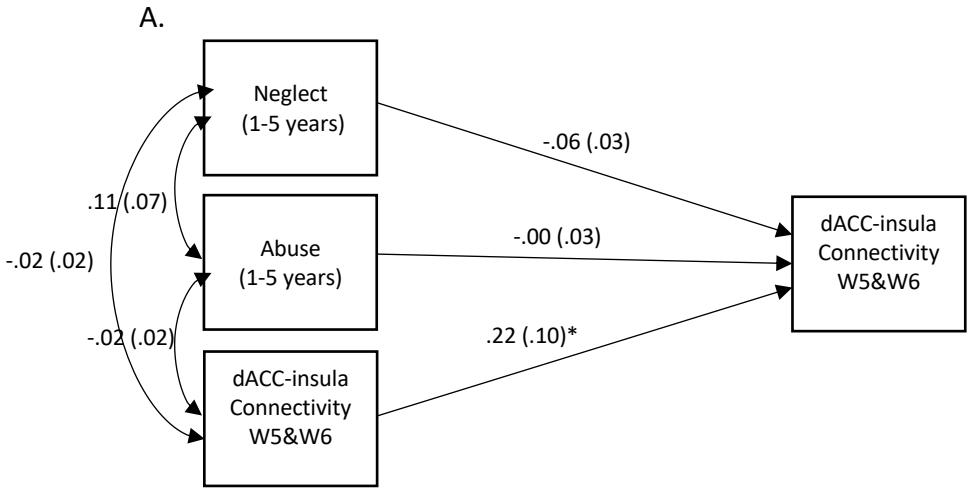


Note. Unstandardized Estimates are presented.

*$p < .05$, ** $p < .001$.

Figure 17

Path Model of Abuse and Neglect Ages 1 to 5 on dACC-insula changes from early to late adolescence

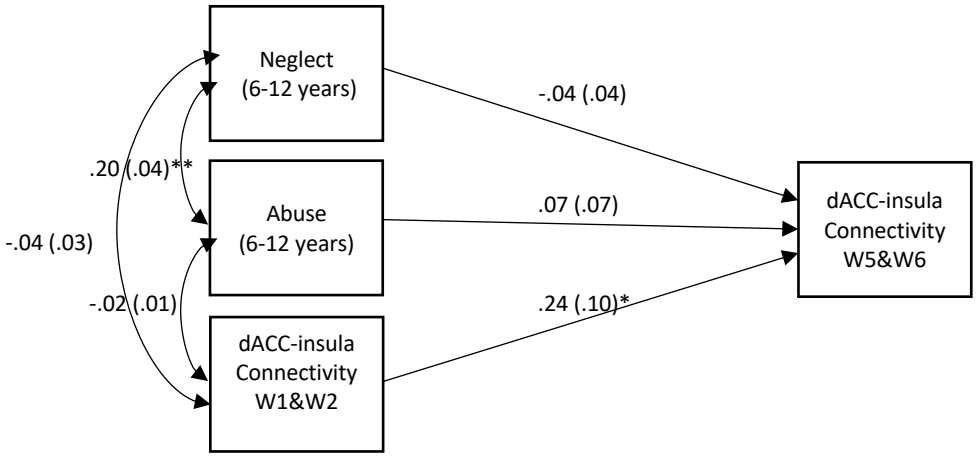


Note. Unstandardized Estimates are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 18

Path Model of Abuse and Neglect Ages 6 to 12 on dACC-insula changes from early to late adolescence

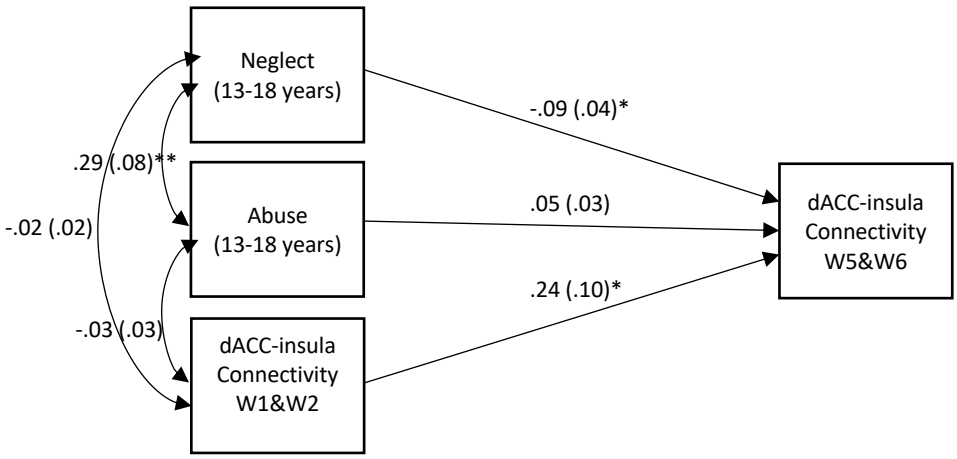


Note. Unstandardized Estimates are presented. dACC = dorsal anterior cingulate cortex

*$p < .05$, ** $p < .001$.

Figure 19

Path Model of Abuse and Neglect Ages 13 to 18 on dACC-insula changes from early to late adolescence

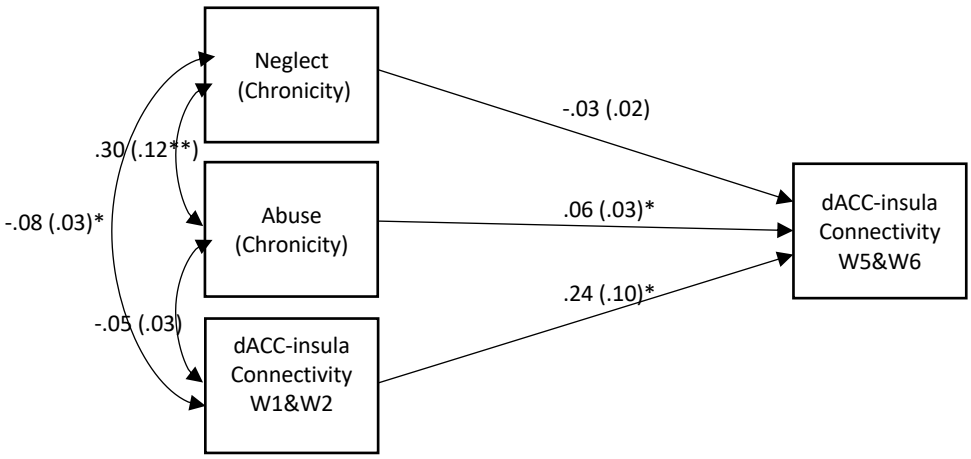


Note. Unstandardized Estimates are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Figure 20

Path Model of Abuse and Neglect Chronicity on dACC-insula changes from early to late adolescence



Note. Unstandardized Estimates are presented. dACC = dorsal anterior cingulate cortex.

*$p < .05$, ** $p < .001$.

Table 1

Descriptive Statistics and Correlations for Maltreatment and Risk Processing Variables, Part 1 (Maltreatment, insula-dACC connectivity, insula-mPFC connectivity)

	1.	***2.***	***3.***	***4.***	***5.***	***6.***	***7.***	***8.***	***9.***	***10.***	***11.***	***12.***	***13.***	***14.***
1. *Abuse age 1-5*	-													
2. *Neglect age 1-5*	.14	-												
3. *Abuse age 6-12*	.71**	.04	-											
4. *Neglect age 6-12*	.36**	.57**	.43**	-										
5. *Abuse age 13-18*	.69**	.08	.86**	.42**	-									
6. *Neglect age 13-18*	.32**	.75**	.28**	.69**	.31**	-								
7. *Abuse Chronicity*	.50**	-.07	.54**	.08	.54**	.03	-							
8. *Neglect Chronicity*	.37**	.69**	.46**	.78**	.49**	.74**	.21*	-						
9. *Insula-dACC T1&2*	-.05	.10	.02	.15	.07	.10	-.08	.17	-					
10. *Insula-dACC T3&4*	-.12	.13	-.08	-.00	-.09	.06	-.21*	.01	.16	-				
11. *Insula-dACC T5&6*	-.11	.01	-.01	.08	-.02	.04	-.06	.03	.02	.04	-			
12. *Insula-mPFC T1&2*	.06	.07	.10	.19*	.07	.12	.04	.02	.60**	.11	-.09	-		
13. *Insula-mPFC T3&4*	-.05	.09	-.04	-.08	-.06	-.00	-.12	-.04	.14	.53**	-.23*	.16	-	
14. *Insula-mPFC T5&6*	-.05	.12	-.09	-.01	-.07	.12	-.19*	-.05	-.02	-.00	.39**	-.05	-.13	-
M	-.00	.00	-.00	-.00	-.00	.00	2.20	1.36	-.02	.00	.00	-.01	.00	.00
SD	.85	.91	.45	1.01	1.06	.87	1.08	1.32	.06	.09	.09	.05	.07	.07

Note. T = Time; dACC = dorsal anterior cingulate cortex; mPFC = medial prefrontal cortex.

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

Table 1

Descriptive Statistics and Correlations for Maltreatment and Risk Processing Variables, Part 2 (insula-dACC connectivity, insula-mPFC connectivity, behavioral risk sensitivity)

	1.	***2.***	***3.***	***4.***	***5.***	***6.***	***7.***	***8.***	***9.***	***10.***	***11.***	***12.***
1. *Insula-dACC T1&2*	-											
2. *Insula-dACC T3&4*	.16	-										
3. *Insula-dACC T5&6*	.02	.04	-									
4. *Insula-mPFC T1&2*	.60**	.11	-.09	-								
5. *Insula-mPFC T3&4*	.14	.53**	-.23*	.16	-							
6. *Insula-mPFC T5&6*	-.02	-.00	.39**	-.05	-.13	-						
7. *BRS T1*	.12	.07	.06	.07	.01	.02	-					
8. *BRS T2*	.26**	-.07	.14	.07	-.10	-.05	.43**	-				
9. *BRS T3*	.11	.11	.16	.08	-.03	-.12	.35**	.57**	-			
10. *BRS T4*	.21*	.02	.04	-.00	-.00	.02	.33**	.45**	.56**	-		
11. *BRS T5*	.09	-.09	-.15	-.05	-.12	-.02	.34**	.46**	.40**	.60**	-	
12. *BRS T6*	.03	-.12	.04	-.08	-.07	.10	.22*	.45**	.31**	.46**	.55**	-
M	-.02	.00	.00	-.01	.00	.00	.77	.56	.46	.37	.40	.33
SD	.06	.09	.09	.05	.07	.07	.56	.50	.42	.36	.31	.32

Note. T = Time; dACC = dorsal anterior cingulate cortex; mPFC = medial prefrontal cortex; BRS = behavioral risk sensitivity.

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

Table 1

Descriptive Statistics and Correlations for Maltreatment and Risk Processing Variables, Part 3 (Maltreatment, behavioral risk sensitivity)

	1.	***2.***	***3.***	***4.***	***5.***	***6.***	***7.***	***8.***	***15.***	***16.***	***17.***	***18.***	***19.***	***20.***
1. *Abuse age 1-5*	-													
2. *Neglect age 1-5*	.14	-												
3. *Abuse age 6-12*	.71**	.04	-											
4. *Neglect age 6-12*	.36**	.57**	.43**	-										
5. *Abuse age 13-18*	.69**	.08	.86**	.42**	-									
6. *Neglect age 13-18*	.32**	.75**	.28**	.69**	.31**	-								
7. *Abuse Chronicity*	.50**	-.07	.54**	.08	.54**	.03	-							
8. *Neglect Chronicity*	.37**	.69**	.46**	.78**	.49**	.74**	.21*	-						
15. *BRS T1*	-.01	-.08	.07	-.01	.01	-.06	-.01	.04	-					
16. *BRS T2*	-.04	.08	.01	.06	-.04	.08	.03	.14	.43**	-				
17. *BRS T3*	-.10	-.12	.01	-.02	.01	-.08	.01	.02	.35**	.57**	-			
18. *BRS T4*	-.06	.03	.02	-.05	.02	-.12	.09	.08	.33**	.45**	.56**	-		
19. *BRS T5*	.09	.02	-.02	-.02	-.06	-.09	-.06	.02	.34**	.46**	.40**	.60**	-	
20. *BRS T6*	-.16	.13	-.12	-.07	-.18*	.04	-.06	.08	.22*	.45**	.31**	.46**	.55**	-
M	-.00	.00	-.00	-.00	-.00	.00	2.20	1.36	.77	.56	.46	.37	.40	.33
SD	.85	.91	.45	1.01	1.06	.87	1.08	1.32	.56	.50	.42	.36	.31	.32

Note. T = Time; BRS = behavioral risk sensitivity

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

Table 2.

Descriptive Statistics and Correlations for Maltreatment and Cognitive Control Variables, part 1 (Maltreatment and dACC-insula connectivity)

	1.	***2.***	***3.***	***4.***	***5.***	***6.***	***7.***	***8.***	***9.***	***10.***	***11.***	***12.***	***13.***
1. *Abuse age 1-5*	-												
2. *Neglect age 1-5*	.14	-											
3. *Abuse age 6-12*	.71**	.04	-										
4. *Neglect age 6-12*	.36**	.57**	.43**	-									
5. *Abuse age 13-18*	.69**	.08	.86**	.42**	-								
6. *Neglect age 13-18*	.32**	.75**	.28**	.69**	.31**	-							
7. *Abuse Chronicity*	.50**	-.07	.54**	.08	.54**	.03	-						
8. *Neglect Chronicity*	.37**	.69**	.46**	.80**	.48**	.74**	.21*	-					
9. *dACC- insula T1*	-.06	.08	-.12	-.02	-.06	-.01	-.19*	.00	-				
10. *dACC-insula T2*	-.11	-.18	-.11	-.20*	-.09	-.11	-.02	-.28**	.15	-			
11. *dACC-insula T3*	.03	.05	.10	.07	.06	.17	-.16	.04	-.03	-.04	-		
12*. dACC-insula T4*	.04	.10	.02	.06	.08	.11	-.09	.07	.06	-.04	.17	-	
13. *dACC-insula T5*	-.12	-.15	-.03	-.06	.02	-.15	.15	-.16	.18	.21	-.06	-.21	-
14*. dACC-insula T6*	.03	-.11	.10	-.09	.12	-.10	.06	-.10	.14	-.03	-.17	-.16	-.07
M	-.00	.00	-.00	-.00	-.00	.00	-.00	.00	-.03	.03	.01	-.05	.01
SD	.85	.91	.45	1.01	1.06	.87	.85	.91	.32	.35	.40	.32	.42

Note. T = Time; dACC = dorsal anterior cingulate cortex; mPFC = medial prefrontal cortex.

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

Table 2.

Descriptive Statistics and Correlations for Maltreatment and Cognitive Control Variables, part 2 (Maltreatment, dACC-mPFC connectivity, dACC-mPFC connectivity, ISD)

	1	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***	***11***	***12***	***13***	***14***
15. *dACC-mPFC T1*	.05	-.04	-.03	-.19	.02	-.15	-.07	-.09	.43**	-.15	.05	-.05	-.03	.11
16. *dACC-mPFC T2*	.08	.16	.05	.10	.02	.12	-.03	.06	-.14	.33**	-.01	-.02	-.00	-.18
17. *dACC-mPFC T3*	-.09	.05	-.00	.12	-.06	.13	-.32**	.02	-.09	-.02	.59**	.21*	-0.14	.08
18. *dACC-mPFC T4*	-.07	.07	.12	.05	.14	.13	.03	.08	-.16	-.03	.21*	.48**	-0.1	-.00
19. *dACC-mPFC T5*	-.03	-.04	-.00	-.04	.04	-.05	.02	-.09	.08	.10	-.10	.03	.52**	-.17
20. *dACC-mPFC T6*	.01	-.04	.03	.05	.06	-.08	-.05	-.04	.06	-.08	.11	-.06	-.05	.59**
21. *dACC-SMA T1*	-.03	.02	-.02	-.09	-.01	-.04	-.13	-.03	.44**	.05	.09	-.06	-.01	-.08
22. *dACC-SMA T2*	-.12	-.01	-.05	-.00	-.10	.07	-.13	-.07	-.01	.58**	.06	-.21*	.02	-.02
23. *dACC-SMA T3*	-.16	-.09	-.02	-.02	-.05	.05	-.15	-.05	-.20	-.07	.59**	.01	-.06	-.09
24. *dACC-SMA T4*	-.02	-.08	-.01	-.03	.05	-.02	-.03	-.01	.07	-.02	.10	.57**	-.10	.06
25. *dACC-SMA T5*	-.13	-.07	.02	-.01	.03	-.06	.05	-.09	-.02	.18	-.00	-.08	.61**	.23
26. *dACC-SMA T6*	.06	-.04	.06	.06	.05	-.07	.03	-.05	-.03	-.20	-.06	-.19	-.12	.61**
27. *ISD T1*	.07	-.16	.13	-.04	.08	-.01	.01	-.01	-.07	-.06	.00	.03	-.17	-.11
28. *ISD T2*	-.15	-.18*	-.07	-.21*	-.15	-.21*	-.11	-.19*	-.20*	-.07	.07	-.02	-.02	.01
29. *ISD T3*	-.04	-.06	.03	-.02	.01	-.08	.13	-.02	-.11	-.01	-.18	.04	-.07	.09
30. *ISD T4*	-.02	-.04	.04	-.08	.05	-.06	-.11	-.06	-.07	-.06	-.01	.04	.01	-.07
31. *ISD T5*	-.06	-.03	.03	.07	-.06	-.01	-.14	.10	-.18	-.18	.08	.08	-.15	-.27*
32. *ISD T6*	-.08	-.14	.02	-.06	0.08	-.21*	-.05	-.05	-.05	-.18	.09	.00	-.10	-.06
M	-.00	.00	-.00	-.00	-.00	.00	-.00	.00	-.03	.03	.01	-.05	.01	-.00
SD	.85	.91	.45	1.01	1.06	.87	.85	.91	.32	.35	.40	.32	.42	.40

Note. T = Time; dACC = dorsal anterior cingulate cortex; mPFC = medial prefrontal cortex;

SMA = supplementary motor area; ISD = cognitive control performance during MSIT.

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

Table 2.

Descriptive Statistics and Correlations for Maltreatment and Cognitive Control Study, part 3 (dACC-mPFC connectivity, dACC-mPFC connectivity, ISD)

	1.	***2.***	***3.***	***4.***	***5.***	***6.***	***7.***	***8.***	***9.***	***10.***	***11.***	***12.***	***13.***	***14.***	***15.***	***16.***	***17.***	***18.***
1. *dACC-mPFC T1*	-																	
2. *dACC-mPFC T2*	-.14	-																
3. *dACC-mPFC T3*	-.15	.07	-															
4. *dACC-mPFC T4*	-.05	.05	.08	-														
5. *dACC-mPFC T5*	.07	.11	-.22	-.04	-													
6. *dACC-mPFC T6*	-.03	-.18	.15	.01	-.27*	-												
7. *dACC- SMA T1*	.46**	-.00	.03	-.02	-.06	-.05	-											
8. *dACC- SMA T2*	-.00	.48**	.17	-.15	-.12	-.04	.08	-										
9. *dACC- SMA T3*	-.06	-.10	.49**	.13	-.11	.16	-.02	.04	-									
9. *dACC- SMA T4*	.02	.06	.12	.47**	.07	.11	-.14	-.13	.09	-								
11. *dACC- SMA T5*	.00	-.11	-.05	-.01	.44**	.05	-.05	.04	.06	-.05	-							
12. *dACC- SMA T6*	-.00	-.06	.03	-.07	-.19	.58**	-.13	-.04	-.12	-.16	.00	-						
13. *ISD T1*	.03	-.12	-.08	-.06	.03	-.06	.01	.04	-.08	-.07	-.04	-.04	-					
14. *ISD T2*	.06	-.14	.00	-.01	.09	.00	.05	-.02	.20*	-.08	.13	.01	.49**	-				
15. *ISD T3*	.06	-.19*	-.17	-.11	.09	-.06	-.02	.03	.02	-.06	.09	.01	.47**	.59**	-			
16. *ISD T4*	.11	-.16	-.10	.03	.22*	-.14	.00	-.11	.15	-.01	.25*	-.13	.26**	.34**	.30**	-		
17. *ISD T5*	-.02	-.12	.08	-.06	.15	-.20	-.12	-.18	.19	-.07	.02	-.21	.45**	.50**	.49**	.34**	-	
18. *ISD T6*	.19	-.10	.13	-.07	.10	.02	.03	-.15	.07	.00	.02	-.05	.42**	.38**	.36**	.37**	.63**	-
M	-.04	-.01	.11	-.11	.06	.04	-.12	.02	.01	-.07	-.02	-.03	-.24	-.21	-.19	-.15	-.17	-.16
SD	.52	.43	.50	.45	.45	.57	.41	.45	.54	.44	.47	.53	.04	.04	.04	.04	.04	.04

Note. T = Time; dACC = dorsal anterior cingulate cortex; mPFC = medial prefrontal cortex; SMA = supplementary motor area; ISD = cognitive control performance during MSIT.

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

Appendix A

Demographic Interview

1. What is your sex?

0-Male 1-Female

4. How old are you? (Record age in years.) _____

13a. How would you describe your own race?

1 = American Indian/Alaska Native
2 = Asian
3 = Native Hawaiian or Other Pacific Islander
4 = Black or African American
5 = White
6 = More than one race
7 = Other _____

13c. How would you describe your own ethnicity?

1. 1 Hispanic or Latino
2. 2 Not Hispanic or Latino

21. What is your total annual family income before taxes for all the adults in your household? Please include all (including TANF, AFDC, food stamps, SSI, rent voucher, fuel assistance and child support). If you are not sure about the amount, please estimate.

A	None	or	$0 per month
B	Less than 1,000	or	Less than $83 per month
C	$1,000 - $2,999	or	$83 - $249 per month
D	$3,000 - $4,999	or	$250 - $416 per month

E	$5,000 - $7,499	or	$417 - $624 per month
F	$7,500 - $9,999	or	$625 - $833 per month
G	$10,000 - $14,999	or	$834 - $1,249 per month
H	$15,000 - $19,999	or	$1,250 - $1,666 per month
I	$20,000 - $24,999	or	$1,667 - $2,083 per month
J	$25,000 - $34,999	or	$2,084 - $2,916 per month
K	$35,000 - $49,999	or	$2,917 - $4,167 per month
L	$50,000 - $74,999	or	$4,168 - $6,249 per month
M	$75,000 - $99,999	or	$6, 250 - $8,333 per month
N	$100,000 - $199,999	or	$8,334 - $16,666 per month
O	$200,000 or more	or	$16,667 or more per month

Appendix B

Maltreatment and Abuse Chronology of Exposure scale

MACE

Sometimes parents, stepparents or other adults living in the house do hurtful things.
If this happened during your childhood (first 18 years of your life) please provide your best estimate of your age at the time(s) of occurrence.
Please check all ages that apply.

For example item 1. Swore at you, called you names, said insulting things like your "fat", "ugly", "stupid", etc. more than a few times a year.

● Yes ❍ No

If at ages 6-8 your father swore at you and at ages 8-10 your mother insulted you, and at age 17 your mother's new live-in boyfriend called you names; you would check off as follows:

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
					✓	✓	✓	✓	✓							✓	

1. Swore at you, called you names, said insulting things like your "fat", "ugly", "stupid", etc. more than a few times a year.
Please check all ages that apply.

❍ Yes_1 ❍ No_0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

2. Said hurtful things that made you feel bad, embarrassed or humiliated more than a few times a year.
Please check all ages that apply.

❍ Yes_1 ❍ No_0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

3. Acted in a way that made you afraid that you might be physically hurt.
Please check all ages that apply.

❍ Yes_1 ❍ No_0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

4. Threatened to leave or abandon you.
Please check all ages that apply.

❍ Yes ❍ No_0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

5. Locked you in a closet, attic, basement or garage. ❍ Yes1 ❍ No0
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

6. Intentionally pushed, grabbed, shoved, slapped, pinched, punched or kicked you. ❍ Yes1 ❍ No0
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

7. Hit you so hard that it left marks for more than a few minutes. ❍ Yes1 ❍ No0
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

8. Hit you so hard, or intentionally harmed you in some way, that you received or should have received medical attention. ❍ Yes1 ❍ No0
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

9. Spanked you on your buttocks, arms or legs. ❍ Yes1 ❍ No
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

10. Spanked you on your bare (unclothed) buttocks. ❍ Yes1 ❍ No0
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

11. Spanked you with an object such as a strap, belt, brush, paddle, rod, etc. ❍ Yes1 ❍ No0
Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

12. Made inappropriate sexual comments or suggestions to you. Please check all ages that apply.

❍ Yes$_1$ ❍ No$_0$

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

13. Touched or fondled your body in a sexual way. Please check all ages that apply.

❍ Yes$_1$ ❍ No$_0$

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

14. Had you touch their body in a sexual way. Please check all ages that apply.

❍ Yes$_1$ ❍ No$_0$

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Sometimes parents, stepparents or other adults living in the house do hurtful things to your siblings (brother, sister, stepsiblings). If this happened during your childhood (first 18 years of your life) please provide your best estimates of your age at the time(s) of occurrence.

Please check all ages that apply.

15. Hit your sibling (stepsibling) so hard that it left marks for more than a few minutes.

Please check all ages that apply.

❍ Yes1 ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

16. Hit your sibling (stepsibling) so hard, or intentionally harmed him/her in some way, that he/she received or should have received medical attention.

Please check all ages that apply.

❍ Yes. ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

17. Made inappropriate sexual comments or suggestions to your sibling (stepsibling).

Please check all ages that apply.

❍ Yes1 ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

18. Touched or fondled your sibling (stepsibling) in a sexual way.

Please check all ages that apply.

❍ Yes1 ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Sometimes adults or older individuals NOT living in the house do hurtful things to you.
If this happened during your childhood (first 18 years of your life) please provide your best estimates of your age at the time(s) of occurrence.

Please check all ages that apply.

19. Had you touch their body in a sexual way.
Please check all ages that apply.

❍ Yes1 ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

20. Actually had sexual intercourse (oral, anal or vaginal) with you.
Please check all ages that apply.

❍ Yes1 ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Sometimes intense arguments or physical fights occur between parents, stepparents or other adults (boyfriends, girlfriends, grandparents) living in the household.
If this happened during your childhood (first 18 years of your life) please provide your best estimates of your age at the time(s) of occurrence.

Please check all ages that apply.

21. Saw adults living in the household push, grab, slap or throw something at your mother (stepmother, grandmother).
Please check all ages that apply.

❍ Yes1 ❍ No.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

22. Saw adults living in the household hit your mother (stepmother, grandmother) so hard that it left marks for more than a few minutes.
Please check all ages that apply.

❍ Yes1 ❍ No0

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

23. Saw adults living in the household hit your mother (stepmother, grandmother) so hard, or intentionally harm her in some way, that she received or should have received medical attention.
Please check all ages that apply.

❍ Yes. ❍ No.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

24. Saw adults living in the household push, grab, slap or throw something at your father (stepfather, grandfather). ❍ Yes1 ❍ No.

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

25. Saw adults living in the household hit your father (stepfather, grandfather) so hard that it left marks for more than a few minutes. ❍ Yes1 ❍ No.

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Sometimes children your own age or older do hurtful things like bully or harass you.
If this happened during your childhood (first 18 years of your life) please provide your best estimates of your age at the time(s) of occurrence.
Please check all ages that apply.

26. Swore at you, called you names, said insulting things like your "fat", "ugly", "stupid", etc. more than a few times a year. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

27. Said hurtful things that made you feel bad, embarrassed or humiliated more than a few times a year. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

28. Said things behind your back, posted derogatory messages about you, or spread rumors about you. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

29. Intentionally excluded you from activities or groups. ❍ Yes$_1$ ❍ No$_0$

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

30. Acted in a way that made you afraid that you might be physically hurt. ❍ Yes ❍ No

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

31. Threatened you in order to take your money or possessions. ❍ Yes$_1$ ❍ No$_0$

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

32. Forced or threatened you to do things that you did not want to do. ❍ Yes$_1$ ❍ No$_0$

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

33. Intentionally pushed, grabbed, shoved, slapped, pinched, punched, or kicked you. ❍ Yes$_1$ ❍ No$_0$

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

34. Hit you so hard that it left marks for more than a few minutes. ❍ Yes$_1$ ❍ No$_0$

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

35. Hit you so hard, or intentionally harmed you in some way, that you received or should have received medical attention. ❍ Yes$_1$ ❍ No$_0$

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

36. Forced you to engage in sexual activity against your will. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

37. Forced you to do things sexually that you did not want to do. ❍ Yes1 ❍ No

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Please indicate if the following happened during your childhood (first 18 years of your life). Please provide your best estimates of your age at the time(s) of occurrence.
Please check all ages that apply.

38. You felt that your mother or other important maternal figure was present in the household but emotionally unavailable to you for a variety of reasons like drugs, alcohol, workaholic, having an affair, heedlessly pursuing their own goals. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

39. You felt that your father or other important paternal figure was present in the household but emotionally unavailable to you for a variety of reasons like drugs, alcohol, workaholic, having an affair, heedlessly pursuing their own goals. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

40. A parent or other important parental figure was very difficult to please. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

41. A parent or other important parental figure did not have the time or interest to talk to you. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

42. One or more individuals in your family made you feel loved. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

43. One or more individuals in your family helped you feel important or special. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

44. One or more individuals in your family were there to take care of you and protect you. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

45. One or more individuals in your family were there to take you to the doctor or Emergency Room if the need ever arose, or would have if needed. ❍ Yes1 ❍ No0

Please check all ages that apply.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18

Please indicate if the following statements were true about you and your family during your childhood, and your age at the time(s) you felt this to be true.

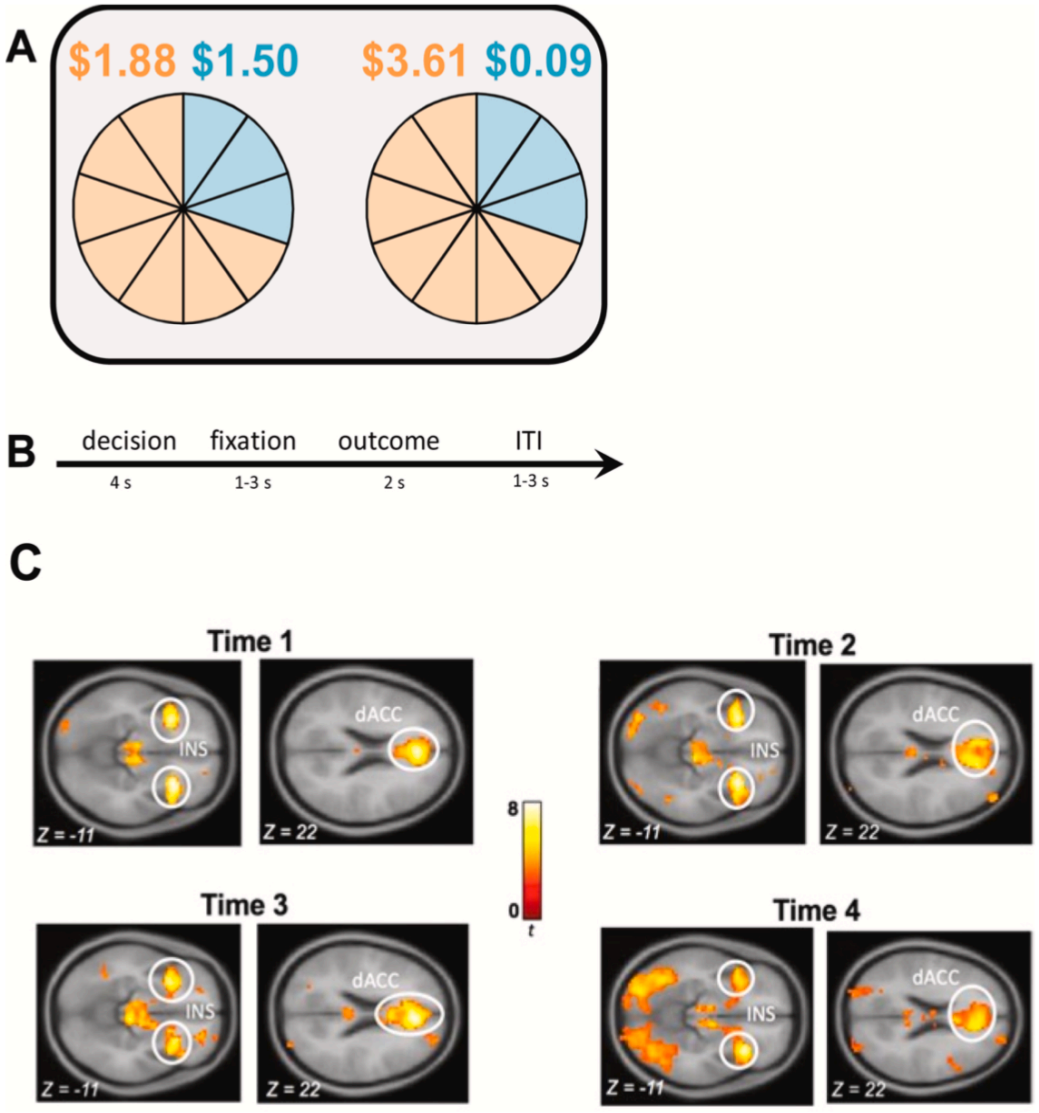
Please check all ages that apply.

46.	You didn't have enough to eat. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$
47.	You had to wear dirty clothes. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$
48.	You felt that you had to shoulder adult responsibilities. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$
49.	You felt that your family was under severe financial pressure. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$
50.	One or more individuals kept important secrets or facts from you. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$
51.	People in your family looked out for each other. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$
52.	Your family was a source of strength and support. Please check all ages that apply. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18	❍ Yes$_1$	❍ No$_0$

Appendix C

Figure 1.

Schematic Display of the Lottery Choice Task and Blood-Oxygen-Level-Dependent Responses to Risk (Coefficient of Variation)



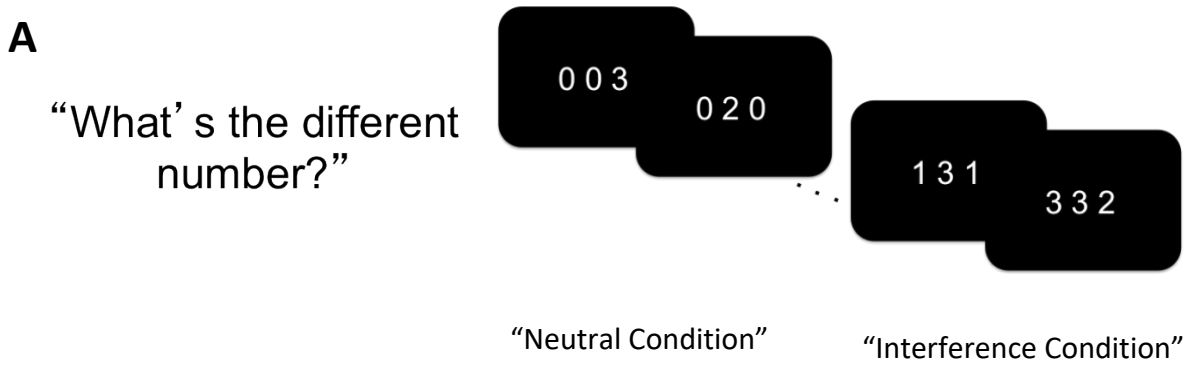
Note. (A) In the lottery choice task, adolescents were asked to choose between pairs of uncertain gambles. For each gamble, there was a high and low monetary outcome, each associated with a specific probability. The associations between outcomes and probabilities are represented with corresponding colors (orange or blue).

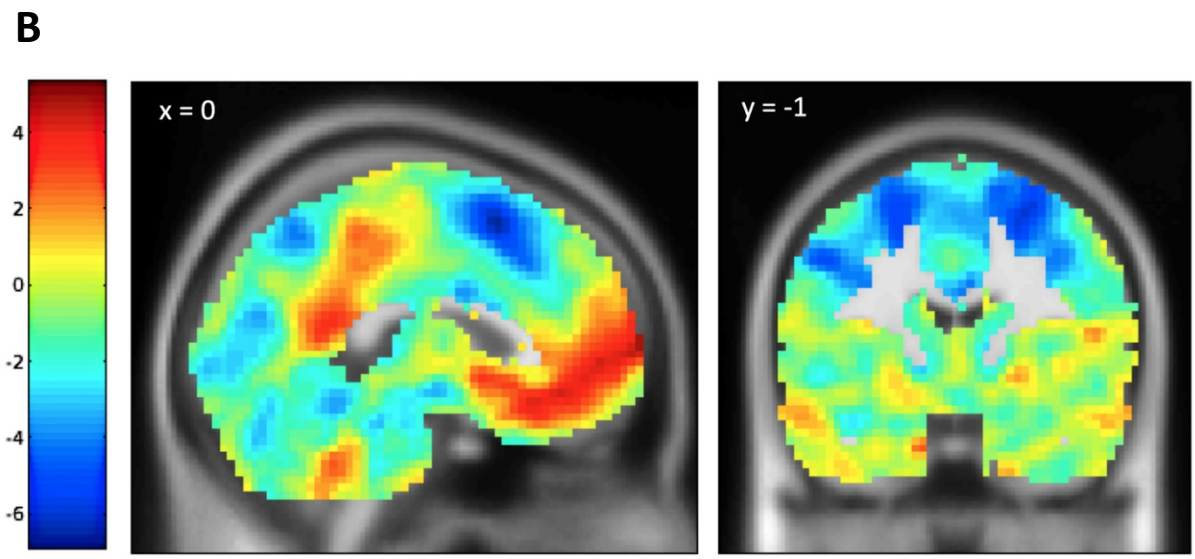
(B) Each trial consisted of a decision phase, a fixation phase, an outcome phase, and an inter-trial-interval (ITI). (C) During the decision phase increased activation was found in the insula (INS) and dorsal anterior cingulate cortex (dACC) during riskier gambles as was indicated by the coefficient of variation (CV). Figure reprinted with permission from Kim-Spoon, J., Herd, T., Brieant, A., Peviani, K., Deater-Deckard, K., Lauharatanahirun, N., Lee, J., & King-Casas, B. (2021). Maltreatment and brain development: The effects of abuse and neglect on longitudinal trajectories of neural activation during risk processing and cognitive control. *Developmental Cognitive Neuroscience 48,* 100939.

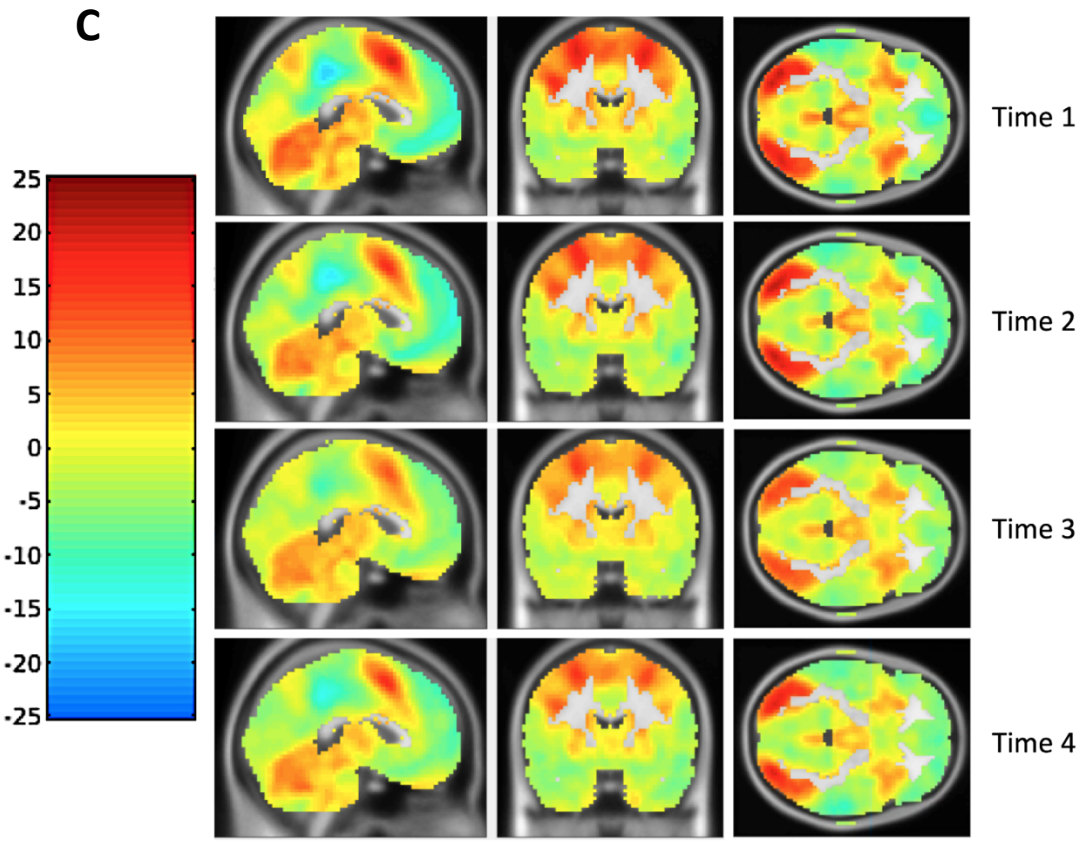
Appendix D

Figure 2.

Schematic Display of the Multi-Source Interference Task (MSIT) and Activation Maps Showing Significant Activation for the Interference-Neutral Contrast







Note: A) Adolescents were instructed to identify the different digit while ignoring its position. B) Statistical *T* map showing regions of positive and negative linear change in the interference effect on BOLD responses with time point using the Sandwich Estimator Toolbox after applying a gray matter mask. C) Statistical *T* maps showing regions of positive (interference > neutral) and negative (neutral > interference) interference effect for each time point after applying a gray matter mask. Figure reprinted with permission from Kim-Spoon, J., Herd, T., Brieant, A., Elder, J., Lee, J., Deater-Deckard, K., & King-Casas, B. (2021). A 4-year longitudinal neuroimaging study of cognitive control using latent growth modeling: Developmental changes and brain-behavior associations. *Neuroimage,* 237, 118134.