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Neurodevelopmental Trajectories as an Explanatory Mechanism for Adverse Mental Health Outcomes following Child Maltreatment

Casey Burton Corso
Virginia Commonwealth University

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By: Casey Burton Corso
Bachelor of Arts, University of Virginia, 2019
Master of Education, University of Virginia, 2020
Master of Science, Virginia Commonwealth University, 2022

Director: Marcia A. Winter, PhD
Associate Professor, Department of Psychology

Virginia Commonwealth University
Richmond, Virginia
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"Whatever you do, work heartily, as for the Lord and not for men"

– Colossians 3:23

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Abstract

Affecting one in every seven children in the United States, child maltreatment (CM) is a major public health issue associated with a myriad of adverse outcomes (e.g., alcohol and drug abuse, mental illness, interpersonal violence, sexual risk taking, etc.). While any subtype of CM (e.g., physical abuse, sexual abuse, emotional abuse, and neglect) increases the likelihood of subsequent psychopathology, heterogeneity in psychiatric sequelae of CM is associated with different CM subtypes. Elucidating the underlying mechanisms of this heterogeneity is vital for improving intervention and treatment of CM, and research aimed at accomplishing this is a critical need. One possible explanatory mechanism is structural brain development, though the distinct impact of CM subtype brain morphology has not been thoroughly explored. The goal of this longitudinal study was to examine neurodevelopmental trajectories of the hippocampus, with attention given to the distinct role CM subtype may have, and ascertain if those trajectories act as a mechanistic explanation for anxiety, depression, and substance use following CM. Data were drawn from baseline (ages 9-11 years) as well as 2- and 4-year follow-ups from a large-scale, youth-centered, multi-site dataset: the Adolescent Brain Cognitive Development (ABCD) study. Hippocampus density was found to increase over time in both the left and right hemispheres; however, CM subtypes were not found to have distinct trajectories. Further, CM subtypes mostly did not predict anxiety, depression, and substance use outcomes, and hippocampal morphology did not mediate any present associations. Potential explanations for these null findings are discussed, and directions for future research are outlined.

Neurodevelopmental Trajectories as an Explanatory Mechanism for Adverse Mental Health Outcomes following Child Maltreatment

Child maltreatment (CM) affects at least one in every seven U.S. children each year (Center for Disease Control, 2023; Children's Bureau, 2020; Finkelhor et al., 2015) and is recognized as a major public and global health issue (Anda et al., 2010; Bethell et al., 2019; Gilbert et al., 2009; Larkin et al., 2012; World Health Organization & International Society for Prevention of Child Abuse and Neglect, 2006). The economic burden of CM is comparable to that of other costly health conditions, such as heart disease and diabetes, with a U.S. total lifetime cost of $592 billion in 2018 (Center for Disease Control, 2023; Centers for Disease Control and Prevention, 2022; Kilka et al., 2020; Peterson et al., 2018). Moreover, after considering the cost of low productivity and psychopathology following CM (P. S. Corso et al., 2008), the lifetime loss of income for all those affected is estimated at $2.1 trillion (Smith & Smith, 2010). CM consistently predicts adverse mental health outcomes over and above other known stressors (Arseneault et al., 2011; Ford & Cloitre, 2009). Indeed, CM is considered the most important risk factor for psychopathology (Teicher et al., 2022; Zeanah & Humphreys, 2018), and is specifically associated with the likelihood of developing anxiety (Gardner et al., 2019; Lindert et al., 2014; Norman et al., 2012), depression (Gardner et al., 2019; Lindert et al., 2014; Norman et al., 2012), and substance use problems (Fletcher, 2021; Maniglio, 2011; Norman et al., 2012). In addition, CM survivors experience earlier onset of psychopathology with more severe symptoms and increased rates of comorbidity (Agnew-Blais & Danese, 2016; Alvarez et al., 2011; Leverich et al., 2002; Nanni et al., 2012) and existing mental health treatments are less effective for them compared to peers without CM histories (Nanni et al., 2012; Zeanah & Humphreys, 2018).

Developmental psychopathology frameworks underscore the tenets of multifinality (i.e., diverse outcomes from one experience) and equifinality (i.e., similar outcomes from diverse experiences), which conceptualizes the mental health sequelae of CM as dynamic and evolving (Cicchetti & Rogosch, 1996; Cicchetti & Toth, 2016; Toth & Cicchetti, 2013). The neurocognitive social transactional model further suggests that these complex processes involve transactional, bidirectional relations in which CM is entwined with social processes that shape neurocognition and the subsequent etiology of psychopathology (McCrory et al., 2022). These frameworks highlight the need to further examine CM components, such as subtype, alongside neurobiology and mental health (Cicchetti & Toth, 2015; McCrory et al., 2017; Toth & Cicchetti, 2013). The current study will explore differential impacts of CM subtypes, in the context of a stage-salient neurobiological mechanism potentially underlying mental health vulnerability, using longitudinal, multilevel assessments. Findings will evaluate the neurocognitive social transactional model by testing a specific, stage-salient structure, the hippocampus, through which mental health vulnerability following CM might occur.

Operationalization of Child Maltreatment

Lack of systematic definitions of child maltreatment have historically contributed to challenges with measuring, comparing, and communicating CM-related findings, such as prevalence and adverse effects (Jackson et al., 2019; Warmingham et al., 2019). Ecological, medical, sociological, and legal approaches to defining CM are known to vary according to what criteria are emphasized in order to make a CM judgment, how much context (e.g., environmental conditions, parents' stability, cultural norms) should be considered when evaluating CM, and what consequences exist for the involved child (Barnett et al., 1993). Thus, a multisystem approach to defining CM is advantageous, as the integration allows for multiple perspectives to

be recognized through the use of dimensions (i.e., subtypes, frequency and chronicity, developmental period affected, out-of-home placements involved, and incident perpetrator) (Barnett et al., 1993), though CM is most often operationalized by subtype alone (Barnett et al., 1993; Jackson et al., 2019).

Indeed, while the ever-evolving cultural, economic, political, social, and scientific factors preclude CM from having a static definition (Barnett et al., 1993), there has been a concerted effort to assess CM in more systematic ways over the past three decades using subtype operationalization. For example, the 1999 Consultation on Child Abuse Prevention as well as the U.S. Department of Health and Human Services (U.S. DHHS) consider CM to comprise sexual, physical, or emotional abuse or neglect that results in actual or potential harm to a child before the age of 18 (Consultation on Child Abuse Prevention, 1999; U.S. DHHS, 2023). Similarly, the National Incidence Study (NIS) defines CM as an experience of abuse or neglect that harms or endangers a child, and recognizes emotional, sexual, or physical abuse and physical, emotional, and educational neglect as specific subtypes of CM (Sedlak et al., 2010). These subtypes parallel the Maltreatment Classification System (MCS) definition, which further details that nuances in CM measurement can occur due to differences in research methodology, sampling, state definitions, etc. (Barnett et al., 1993). Thus, the general understanding of CM has become relatively stable, referring to subtypes which result in the actual or potential harm to a child.

Still, subtype classifications and definitions retain more study-specific subtleties. Four main subtypes of CM are generally accepted: physical abuse, sexual abuse, emotional abuse (also called psychological abuse), and neglect. However, recent work advocates for the examination of neglect to be split into two distinct subtypes — physical neglect and emotional neglect — noting their distinct impacts on mental health (Grummitt et al., 2022). While CM

subtype definitions vary according to cultural and subcultural norms (Levinson et al., 1984), overarching definitions (written by myself, based on research and general knowledge) of the CM subtypes are: physical abuse - non-accidental physical injury to a child; sexual abuse - forceful, coerced, or otherwise stimulated sexual behavior towards or involving a child; emotional abuse - manipulating, controlling, and/or punishing a child's using emotions and/or psychological tactics; physical neglect - not fulfilling a child's basic physical needs, such as food and shelter; emotional neglect - not fulfilling a child's basic emotional needs, such as psychological safety and age-appropriate autonomy.

The current study utilizes secondary data analysis from the Adolescent Brain Cognitive Development (ABCD) Study (Volkow et al., 2018), which measures adverse childhood experiences (ACEs) more broadly according to domains from the Center for Disease Control (CDC) - Kaiser ACE Study (Felitti et al., 1998). Therefore, the distinct subtypes of CM utilized in the current study are directly informed by the available measures within the ABCD protocol (Barch et al., 2018; Zucker et al., 2018): physical and sexual abuse. Physical abuse refers to childrens' experience(s) being shot, beated, stabbed, or hit hard enough to leave a bruise. Sexual abuse refers to childrens' experience(s) being touched inappropriately or forced to engage in unwanted sexual behavior.

Child Maltreatment and Psychopathology

CM is a preventable, multidimensional risk factor for psychopathology (McCrory et al., 2017; McLaughlin & Sheridan, 2016; Russotti et al., 2021; Teicher & Samson, 2013; Whiteford et al., 2013). Moreover, the increased risk for psychopathology following CM affects both immediate and long-term development. Indeed, adolescents who have experienced CM display symptoms of internalizing and externalizing disorders (Negriff et al., 2020; Reis et al., 2024)

more than peers with no experiences of CM (Negriff et al., 2020), and CM-related psychopathology often persists into adulthood (Strathearn et al., 2020). Thus, there is a critical need for research aimed at better understanding the underlying mechanisms of the association between CM and adverse outcomes (Agrawal et al., 2022; Cecil et al., 2017; McCrory et al., 2017; McLaughlin et al., 2020; Russotti et al., 2021; Teicher & Khan, 2019).

Differential Impacts of Child Maltreatment Subtypes

Although all forms of CM exposure increase the likelihood of subsequent psychopathology (Fletcher, 2021; Gardner et al., 2019; Jewkes et al., 2010; Lindert et al., 2014; Maniglio, 2011; Norman et al., 2012), there is significant heterogeneity in sequelae that has been attributed to differences in the impact of distinct CM subtypes (Andrews et al., 2004; Cecil et al., 2017; Hildyard & Wolfe, 2002; Norman et al., 2012). For example, physical abuse is most often linked with externalizing outcomes, such as aggression and delinquency (Manly et al., 2001; Warmingham et al., 2019), while sexual abuse is most often associated with serious mental health consequences such as suicide and substance use dependence (Fletcher, 2021; Warmingham et al., 2019). In contrast, neglect is most often associated with internalizing symptomatology and withdrawn behavior (Alkema et al., 2024; Manly et al., 2001). Still, there is a need to examine how CM subtypes, comparative to one another, differentially predict psychopathological outcomes.

Indeed, subtype-specific vulnerabilities following CM have been evidenced across numerous studies. For example, in a sample of 1,367 men and 1,415 women ages 15-26 years from South Africa, Jewkes and colleagues (2010) found sexual abuse to enhance the risk of alcohol abuse for both men and women, while emotional neglect enhanced the risk of depression for both men and women. They also found gender-by-subtype vulnerabilities, such that women –

but not men – who were physically abused had an enhanced risk for depression (Jewkes et al., 2010). Similarly, men who were emotionally abused and those who were physically neglected had an enhanced risk for alcohol abuse, while women who experienced those events did not (Jewkes et al., 2010). In another study of young adults, emotional neglect was associated with depression, anxiety, and stress, while physical neglect was not, and neither form of neglect was associated with alcohol or drug use (Grummitt et al., 2022).

Subtype-specific vulnerabilities have also been found among younger samples. For example, hierarchical regressions were implemented to reveal the unique contributions of developmental timing and subtype on mental health symptomatology in a sample of 814 children ages 5.5-11.5 years (Manly et al., 2001). Physical abuse and sexual abuse were both predictive of externalizing symptoms, but not internalizing symptoms. In contrast, physical neglect predicted internalizing, but not externalizing, symptoms. Both patterns were found to be particularly severe among preschoolers (Manly et al., 2001). Further, in a systematic review and meta-analysis of 124 non-sexual CM studies, physical abuse, emotional abuse, and neglect were all found to be associated with depression, drug use, suicide attempts, and risky sexual behavior (Norman et al., 2012). However, odds ratios varied greatly among subtypes. For example, while emotional abuse had the greatest odds ratio for depression (3.06 compared to 1.54 and 2.11), physical abuse and emotional abuse had the greatest odds ratio for suicide attempts (3.40 and 3.37 compared to 1.95) (Norman et al., 2012). Moreover, emerging literature has found multiple patterns of emotion regulation to exist following CM profiles (Warmingham et al., 2022) as well as differences in social-emotional and behavioral outcomes among latent classes of CM (Warmingham et al., 2019). These unique patterns of psychopathology following CM could partly reflect distinct neurocognitive impacts of CM subtypes.

Potential Role of Brain Development

Given the evidence outlining the subtype-specific differences following CM exposure, the current study posits that such variance among psychopathology may occur via structural changes in the brain. That is, the current study expects physical abuse and sexual abuse to have unique associations with anxiety, depression, and substance use (e.g., physical abuse associated with only depression and anxiety, sexual abuse associated with only substance use), and that those varying associations can be explained by physical changes in adolescents' brains occurring after CM exposure.

There are two processes through which altered brain development may occur: experience-expectant development and experience-dependent development (Greenough et al., 1987; Markham et al., 2007). Experience-expectant development involves critical periods wherein environmental stimuli are required for normal development. Within this, as synaptic connections are overproduced during critical periods of extreme plasticity, the input of environmental stimuli are required to determine which connections to prune and which to strengthen according to the input and demands of the environment. In contrast, experience-dependent development involves sensitive periods wherein environmental stimuli may affect normal development. That is, new synaptic connections are formed following the input of unique environmental stimuli. Experience-dependent development is particularly sensitive to the maturational stage of the brain, such that stimuli-specific effects are incorporated into developing neuronal patterns (Andersen, 2003).

Stratifying CM by its subtypes, then, it is critical when seeking to understand how CM affects the developing brain. While acts of omission (e.g., neglect) are likely to influence experience-expectant processes, acts of commission (e.g., abuse) are likely to influence

experience-dependent processes. Moreover, because each process is sensitive to the specific environmental stimuli involved, it is necessary to consider how differing forms of neglect (e.g., emotional and physical) as well as abuse (e.g., physical, sexual, and emotional) may differentially affect the experience-expectant and experience-dependent development.

In recent work from the ABCD study, Brieant and colleagues (2023) identified that 10 dimensions of early-life adversity held distinct associations with internalizing problems, externalizing problems, cognitive flexibility, and inhibitory control. Further, non-metric multidimensional scaling (NMDS) analysis[1] revealed a non-linear dimension of "acts of commission versus omission" (Brieant et al., 2023). In this dimension, higher, positive scores were indicative of experience-dependent early-life adversities (e.g., physical trauma, family verbal/physical aggression), whereas lower, negative scores indicated experience-expectant early-life adversities (e.g., lack of physical resources, neighborhood safety, caregiver supervision, and caregiver support) (Brieant et al., 2023).

Moreover, when examining functional connectivity following CM exposure, Zhang and colleagues (2022) found CM subtypes to be associated with distinct, atypical neural networks. More specifically, all types of CM were associated with the frontoparietal and default mode networks, such that adults exposed to CM had maladaptive higher-order cognitive functioning (Zhang et al., 2022). However, only experience-dependent commission abuses (i.e., physical, sexual, and emotional abuse) were correlated with the ventral attentional network (Zhang et al., 2022), which mediates stimulus-driven attentional processes (Alves et al., 2022). In contrast, the dorsal attentional network, which mediates goal-driven attentional processes (Alves et al., 2022), was only associated with sexual abuse and the visual network was only correlated with physical

[1] As an exploratory aim, NMDS was used to visualize the similarity/dissimilarity of the 10 identified early-life adversity dimensions.

abuse and emotional neglect (Zhang et al., 2022). Zhang and colleagues (2022) conclude that the unique neurobiological effects of CM subtypes necessitate the stratified study of CM when seeking to understand its impact on brain development.

Trajectory Mapping

Though investigations of mental health sequelae variations have used direct comparison of CM subtypes and implementation of latent class profiles (Andersen, 2003; Hildyard & Wolfe, 2002; Jewkes et al., 2010; Norman et al., 2012), researchers have noted a lack of clarity and highlighted the urgent need for additional investigations to elucidate the underlying mechanisms of mental health sequelae following CM (Hoven et al., 2012; McLaughlin & Sheridan, 2016; Russotti et al., 2021; Warmingham et al., 2022), particularly within the ABCD sample (Brieant et al., 2023; Orendain et al., 2023). This study expanded on existing work by examining neurodevelopment as an explanatory mechanism linking CM subtypes to mental health sequelae.

Precise, developmentally informed, mechanistic models in neurocognitive research using trajectory mapping of structural brain development can help identify malleable targets for intervention (McCrory et al., 2022; Rakesh et al., 2021; Riem et al., 2015). Some researchers have suggested that neurocognitive alterations following CM might place individuals at risk of increased stress exposure and diminished social relationships, which could explain subsequent outcomes; however, these mechanisms have not been empirically explored (McCrory et al., 2022). It is unknown how CM subtypes might differentially affect neurodevelopmental trajectories (McLaughlin et al., 2015) and how these trajectories, in turn, relate to subsequent mental health outcomes (Cecil et al., 2017; Russotti et al., 2021). Recent work which focused on the ABCD sample specifically calls for the continued examination of CM subtype nuances as a means of more precisely understanding neurobiological and neurobehavioral outcomes without

obscuring potentially critical heterogeneity within the associations (Brieant et al., 2023; Orendain et al., 2023). Indeed, leaders in MRI-derived brain metrics and CM research recommend prioritizing research aimed at preventing and treating CM and related psychopathology (Teicher et al., 2022). This project will contribute to this goal by increasing knowledge of mental health etiology following CM, and elucidating distinct effects of CM subtypes on brain development. Identifying neurodevelopmental mechanisms linking CM to mental health sequelae is critical for developing more effective interventions (Kavanaugh et al., 2017; McLaughlin et al., 2020); with this knowledge, interventions and treatments could be better customized to target the cognitions, processes, and skills associated with specific neurocognitive structures known to underpin psychiatric sequelae of CM.

Hippocampal Development

The hippocampus plays an important role in learning, memory, and emotional behavior (Anand & Dhikav, 2012) and is involved in a range of social processes and flexible cognition, including behavior and adaptation (Montagrin et al., 2018; Rubin et al., 2014). In particular, the hippocampus is critically involved in conscious knowledge: encoding experiences as memories as well as recollecting them (Anand & Dhikav, 2012). It is involved in reinforcement learning in adolescents (Davidow et al., 2016) and its cognitive and emotional processes have been implicated as influencing moral understanding and behavior (Anand & Dhikav, 2012). Moreover, the hippocampus plays a critical role in processing emotions. For example, it has been implicated in the recognition of emotional faces (Gennatas et al., 2017) and in processing other- and self-referential emotional processing (Immordino-Yang & Singh, 2013). Importantly, cognitive processes of the hippocampus, such as memory, have been suggested to be critical components of emotional development (S. Pollak et al., 1998).

Hippocampal Processes and Child Maltreatment

The current study recognizes that the etiological processes following CM as entwined with the social-emotional dynamics of the maltreatment experience (Brugha et al., 2005; Matthews et al., 2019). Of particular relevance to the current study, physical and sexual abuse are distinctly different experiences, with unique impacts on the developing child. CM disrupts typical development of emotion processing (Young & Widom, 2014), and is associated with robust memory for emotionally distressing material (Goodman et al., 2010). However, the role of memory and emotion varies across subtypes. Indeed, children with a history of physical abuse were found to be less accurate in identifying neutrally-valenced pictures, while children with a history of sexual abuse were found to be less accurate in identifying positively-valenced pictures (Young & Widom, 2014). Further, children with a history of physical abuse have been found to be better at identifying angry faces (S. D. Pollak & Sinha, 2002), and to overattend to anger cues (Shackman et al., 2007). In contrast to this hypersensitivity associated with physical abuse, sexual abuse has been associated with slower emotional processing as a whole (van Hoof et al., 2017).

In addition to facilitating the CM and emotion processing association, cognitive processes of the hippocampus (e.g., memory) have been implicated as linking emotion with psychopathology (S. Pollak et al., 1998) and thus may be a crucial factor in understanding mental health sequelae of CM. Indeed, molecular memory (DNA methylation)[2] has been evidenced as mediating maladaptive behavioral patterns and psychopathological risk associated with CM (Lutz et al., 2015) and deficits in memory performance have been associated with trauma-related psychopathology (McWilliams et al., 2014). Compared to children who have not experienced

[2] DNA methylation is involved in memory formation across multiple brain regions, including the hippocampus (Kupke et al., 2024).

CM, abused children had generally poorer memory performance when answering questions about their experiences; however, the groups did not differ in memory accuracy of abuse-specific questioning, though sexual abuse was associated with more errors of omission (Goodman et al., 2001). Still, research on memory functioning following CM remains mixed, and researchers suggest memory and attention work in tandem with emotional processing, rather than alone, to predict psychopathology following CM (McWilliams et al., 2014). These findings may suggest the underlying neurocognitive structure as being differentially impacted by CM subtypes. Thus, the hippocampus is an excellent mechanistic candidate potentially underlying the association between CM and mental health outcomes.

Adolescence as a Critical Period for Hippocampal Development

Neuroimaging studies have continually highlighted adolescence as a critical neurodevelopmental period (Eiland & Romeo, 2013; Giedd, 2008), particularly for the hippocampus (Anand & Dhikav, 2012; Eiland & Romeo, 2013; Hueston et al., 2017). Limbic system structures, including the hippocampus, increase in volume during childhood and adolescence (Canada et al., 2020; Tamnes et al., 2018), peaking around age 16 (Giedd et al., 1999). Adolescence is also considered a sensitive period for CM, due to the structural changes which may occur following the stress response (Brieant et al., 2023). Researchers have noted the need for work examining stage-salient neurobiological mechanisms in adolescence (Schulenberg et al., 2004; Toth & Cicchetti, 2013). Thus, adolescence offers a critical period for examining hippocampal development, particularly in the context of CM.

Impact of Stress on the Hippocampus

Consistent with the notion that adolescence is a sensitive period for hippocampal development, subcortical structures (e.g., hippocampus) undergoing rapid maturation during this

developmental period are likely to be more heavily impacted by environmental stimuli, such as stress (Andersen, 2003). Further, the hippocampus is considered the most stress-sensitive brain structure (Riem et al., 2015). During times of extreme plasticity (e.g., adolescence), stress-response systems and their associated regions (e.g., hippocampus) are especially vulnerable to stress-related alterations in development (Anand & Dhikav, 2012; Rakesh et al., 2021; Riem et al., 2015; Shonkoff et al., 2012). Long-term elevation of stress hormones leads to overactivation of the stress-response systems that ultimately hinder development and subsequent functioning (National Scientific Council on the Developing Child, 2014; Shonkoff et al., 2012). These changes in social and emotional processing may serve as transdiagnostic mechanisms linking CM to psychopathology (McLaughlin et al., 2020). Indeed, stress is known to alter developmental trajectories underlying various mental health sequelae (Andersen, 2003).

Of particular interest is how stress in the form of CM exposure impacts hippocampal development. CM is known to impact neurobiological development (Cabrera et al., 2020; Kavanaugh et al., 2017; Norman et al., 2012; Teicher et al., 2022), and blunted hippocampal development has been linked to the onset of mental health sequelae (Amico et al., 2011; Anand & Dhikav, 2012; Hueston et al., 2017; Teicher et al., 2012). Still, little is known about the true developmental trajectory of the hippocampus following CM (Riem et al., 2015; Shrivastava et al., 2017). Adults with a history of CM tend to have lower hippocampal volume (Carrion & Wong, 2012; Dannlowski et al., 2012; Hart & Rubia, 2012; McLaughlin et al., 2020; Teicher et al., 2012, 2016), though youth show inconsistencies in hippocampal volume following exposure to adversity. One meta-analysis of 49 studies found CM broadly to be associated with reduced hippocampal volume, particularly for CM experienced in early childhood or adolescence (Riem et al., 2015). However, a systematic review of 109 studies found youth's hippocampal volume to

be reduced following some, but not all, exposures to adversity (McLaughlin et al., 2019). Consistent with experience-expectant versus experience-dependent processes, McLaughlin and colleagues (2019) outline exposure to threat-related-adversity (e.g., physical abuse, sexual abuse) as being associated with reduced hippocampal volume whereas exposure to deprivation-related-adversity (e.g., neglect) was not.

Less is known about the impact of CM on hippocampal density, in either adult or adolescent samples. Generally, developmental studies have focused primarily on examining brain volume and cortical thickness, rather than on gray matter density (Gennatas et al., 2017). However, there is a pressing need to consider multiple brain metrics simultaneously, as size (e.g., volume) and composition (e.g., gray matter density) of the brain have been found to develop in unique ways, particularly throughout adolescence (Gennatas et al., 2017; Im et al., 2008).

Application to the Current Study

Overall, stressors such as CM activate the limbic system, including the hippocampus (da Silva Ferreira et al., 2014). However, consistent with experience-dependent processes, the incorporation of these stimuli and subsequent developmental effects of this activation vary. The hippocampus undergoes rapid alterations throughout adolescence and is evidenced as being particularly vulnerable to stress, such as CM (Anand & Dhikav, 2012; Eiland & Romeo, 2013; Hueston et al., 2017; Riem et al., 2015). Thus, CM subtypes might differentially impact the underlying social and emotional processing occurring in the hippocampus, affecting both hippocampal morphology and the subsequent mental health outcomes. Therefore, the hippocampal region is an important area to study during adolescence and stress exposure.

The current study posits that these inconsistent findings in youth hippocampal volume and density following adverse exposures are attributable to the existence of multiple trajectories

and subtype-based differences not yet explicated in previous work. That is, because the hippocampus is likely to change during adolescence, its structure (i.e., volume and density) is likely to be heavily impacted by a stress experience such as CM. Therefore, examining hippocampal morphology as a mechanism of the CM and adverse outcome association will elucidate whether these adverse outcomes following CM are occurring as a result of neurobiological risk processes.

Social Determinants of Health

Examining social determinants of health as potential influences on the neurodevelopment following CM is paramount. Numerous findings have evidenced the negative health consequences of social determinants of health, such as poverty, race/ethnicity, and education, and in particular the intersection between CM and social determinants of health (Lane & Dubowitz, 2021; Mehta et al., 2023). Further, extant findings from ABCD literature suggest social determinants of health to potentially complicate developmental sequelae of CM (Brieant et al., 2023; Orendain et al., 2023). Specifically, characteristics of sex, race and ethnicity, family income, and caregiver's education have been found to differentiate youth with early life adversity from non-adversity comparison groups in the ABCD sample (Orendain et al., 2023). However, other findings from ABCD suggest that these sociodemographic factors do not influence associations between early adversity and later mental health (Stinson et al., 2021). Thus, it is necessary to explore the potential, though unclear, impact of social determinants of health in the current study.

Limitations of Previous Work

To address critical research gaps noted in the literature and ascertain if hippocampal trajectories are related to CM subtype, variability of neurodevelopmental change will be tested

by using a longitudinal investigation. Neuroimaging studies as well as research on mental health sequelae of CM have been critiqued for their lack of high-quality, sufficiently-powered studies (Becht & Mills, 2020; Braithwaite et al., 2017; Jung & Wickrama, 2008; Marek et al., 2022). Additionally, much of the work looking at CM and neurobiology has used adult, cross-sectional data (Becht & Mills, 2020; Rakesh et al., 2021). The use of adult scans and retroactive report of CM presents issues in causality, as it lacks the ability to establish temporal precedence, resulting in masked effects (Rakesh et al., 2021; Vijayakumar et al., 2018). Thus, longitudinal work is especially important when examining deviations among neurodevelopmental trajectories, like those that may occur due to CM (Di Martino et al., 2014; Ho, 2019). Recent work calls for the use of longitudinal data as a means to examine individual developmental trajectories (Becht & Mills, 2020; Rakesh et al., 2021), specifically when looking at CM-based neurodevelopmental deviations (Rakesh et al., 2021; Teicher & Khan, 2019).

Moreover, trajectory mapping often uses variable-centered approaches, despite their inability to adequately assess individual patterns (Russotti et al., 2021). Most longitudinal MRI studies to date have focused on establishing group-level trajectories of brain development, rather than testing within-group variability in developmental change (Becht & Mills, 2020), and recent work has specifically called for the use of person-centered approaches to examine how isolated adversities affect isolated behavioral outcomes (Cecil et al., 2017; Gard, 2021). In order to address these outlined limitations of previous work, the current study will utilize longitudinal, adolescent data, which is specifically mentioned as an excellent source for addressing issues of temporal precedence, model flexibility, and precision growth (Becht & Mills, 2020; Marek et al., 2022), and multilevel structural equation modeling (ML-SEM) to map adolescents' neurodevelopmental trajectories of the hippocampus.

Current Study

The current study will examine developmental trajectories of hippocampal volume and density during adolescence as a function of time and child maltreatment (CM) subtype (i.e., physical abuse, sexual abuse, and both physical and sexual abuse). Secondary data analyses will examine de-identified data from the Adolescent Brain Cognitive Development (ABCD) study at baseline (9-11 y/o), as well as the year two (11-13 y/o) and year four (13-15 y/o) follow-ups. The ultimate goal of this work is to investigate the role of hippocampal development (baseline through year four) as a mechanism between CM (baseline) and anxiety, depression, and substance use (year four). Examining hippocampal morphology throughout adolescence, with consideration for the potentially differential impact of CM subtype, will provide insight into the neurobiological processes following CM.

This line of research will increase knowledge about where to intervene, and in particular whether such interventions are best suited to specific CM subtypes and/or hippocampal trajectories. Findings will provide insight into the physical brain disruptions caused by CM. Notably, Teicher and Khan conceptualized changes in neurodevelopment following CM as induced adaptation rather than damage (Teicher & Khan, 2019). This reframing helps combat negative stigma surrounding CM and allows for all brain changes to be understood as potentially adaptive for children experiencing abusive conditions (Teicher et al., 2022), but with maladaptive long-term consequences (Teicher & Khan, 2019). Where brain damage is generally seen as irreversible, like a lesion, this language shift also connotes remaining plasticity that can be targeted in prevention and treatment efforts. If the hippocampus is indicated as a neurological mechanism shaping outcomes of CM, results would provide support for the neurocognitive social transactional model (McCrory et al., 2022) underpinning this proposal. Because

neuroimaging can help identify malleable targets for intervention (McCrory et al., 2022; Rakesh et al., 2021; Riem et al., 2015), findings will allow for a deeper understanding of how CM affects the developing brain and hippocampal-driven processes and behaviors such as learning, memory, emotion, and social processing. Ultimately, this line of work could inform interventions and treatments that consider the optimal developmental timing to preempt trajectories leading to adverse outcomes. Future work could then further target the cognitions, processes, and skills associated with specific neurocognitive structures to alleviate vulnerability to mental health sequelae, with respect to CM subtype, allowing for better customization and potentially more effective intervention and treatment overall. Study aims are detailed below and a conceptual model of study aims is provided in Figure 1. Additionally, statistical models of study aims are provided in Figures 2-5.

Aim 1. Hippocampal Trajectories

Identify trajectories in adolescent hippocampal development (Figure 2), and test whether there are differences in these trajectories as a function of different CM exposures (Figure 3).

- Hypothesis 1a: Hippocampal volume and gray matter density will increase over time (i.e., with age) for all adolescents.
- Hypothesis 1b: All CM subtypes, compared to a non-CM control group, will exhibit less hippocampus development (i.e., less increase over time in hippocampal volume and gray matter density). Additionally, although there is not enough previous research to make slope predictions for each CM subtype, it is hypothesized that each CM subtype will have a distinct slope.

- Hypothesis 1c: An interaction of time and CM on brain development will exist, such that unique trajectories of hippocampal structural development will emerge for each of the CM subtypes as well as the non-CM control group.

Aim 2. Mediating Effects of Hippocampal Morphology

Investigate the mediating role of brain development on adolescents' mental health outcomes following child maltreatment (Figure 4).

- Hypothesis 2a: Incidence of CM at baseline will be associated with greater anxiety, depression, and substance use at follow-up waves.
- Hypothesis 2b: Hippocampal morphology will mediate the direct relationship between CM and mental health outcomes, such that less developed hippocampal trajectories will be associated with higher rates of anxiety, depression, and substance use.

Aim 3. Moderating Effects of Social Determinants of Health

Test the moderating effects of child age, sex, and race/ethnicity, as well as parental educational attainment and household income on the association between CM and hippocampal morphology (Figure 5).

- Hypothesis 3a: Social determinants of health will moderate the association between CM and hippocampal morphology, such that known demographic risk factors (i.e., younger, female, racial/ethnic minority, lower parental educational attainment, and lower household income), will exacerbate the negative impact of CM on hippocampal development.

Method

Participants

The Adolescent Brain Cognitive Development (ABCD) study is an ongoing, multi-site, longitudinal investigation of the brain development and mental health of approximately 12,000 U.S. youth born between 2006 and 2008 (Jernigan et al., 2018; Volkow et al., 2018). Recruited in 2016–2018,[3] youth enrolled at ages 9 to 11 years across 21 U.S. sites; comprehensive demographic, clinical, psychosocial, and neurobiological information data are collected each year, for 10 years. The current study utilizes a subset of de-identified ABCD data from baseline (9-11 y/o), as well as the year two (11-13 y/o) and four (13-15 y/o) follow-ups (see Table 1).

Descriptive statistics are provided in Table 2. Child maltreatment (CM) subtypes were endorsed as follows: physical abuse $n = 199$, sexual abuse $n = 220$, both physical and sexual abuse $n = 35$, no abuse $n = 11{,}082$. Overall, youth were aged 9.5 years at baseline, split relatively evenly on sex (male/female), and the majority were White. Parents were mostly educated, with the majority indicating their education to be at or above the level of some college. Further, ABCD is a relatively affluent sample, though among CM subtypes the annual household income was generally reported as lower than within the whole sample or no abuse category.

Ethics Approval

De-identified data used in this study were obtained from the National Institute of Mental Health (NIMH) Data Archive (NDA) and came from NDA Release 4.0 (DOI:10.15154/1523041). This project was determined not to qualify as human subjects research on December 12, 2023, and was thus exempt from review by the IRB review committee at

[3] Please see Garavan et al. (2018) for additional description of design and procedures used for ABCD sample recruitment.

Virginia Commonwealth University. The ABCD IRB-of-Record (HM20007469) was approved by full-board review at University of California San Diego (UCSD).

Procedures and Measures

At baseline, youth completed a battery of surveys and tasks measuring physical and mental health, neurocognition, substance use, and cultural and environmental exposures. Structural and functional brain imaging data (T1 structural scans, diffusion-weighted imaging, and resting-state and task-based functional imaging) (Casey et al., 2018; Hagler et al., 2019) and biospecimen data were also collected. Participants' parents completed a survey battery concerning their own and their child's physical and mental health, substance use, and cultural and environmental exposures. The annual follow-up assessment strategy includes a biennial survey battery with neuroimaging that mirrors the baseline assessment (with complementary data provided by both adolescents and their parents) alternating with shorter "off-year" assessments of mental health and substance use updates. Data collection is harmonized across study sites, and de-identified data are released annually through the NIMH Data Archive (NDA).

Demographics

Parents reported child age, sex, race, ethnicity, parental educational attainment, and household income at study baseline (Barch et al., 2018). For the current study, sex was coded as male/female. Race/ethnicity was coded as White, Black, Hispanic, Asian, or Other and parental educational attainment categories included less than a HS diploma, HS diploma or GED, some college, Bachelor's degree, and postgraduate degree. Lastly, household income was split into three categories of less than $50,000 annually, between $50,000 and $100,000 annually, and more than $100,000 annually.

Child Maltreatment

Generally, adverse experience domains were based on the classic CDC - Kaiser ACE study (Felitti et al., 1998). At baseline (youth ages 9-10 years), parent-report from the Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5 (KSADS-5) post-traumatic stress disorder modules (Kaufman et al., 2017) was used to measure physical and sexual abuse experiences (see Appendix A).[4] Additional subtypes of CM were not assessed in the ABCD study and are therefore not included in the current analyses.

Physical abuse was measured by five items which ask if the child has been shot, stabbed or beaten brutally by a non-family member, shot, stabbed or beaten brutally by a grown up in the home, beaten to the point of having bruises by a grown up in the home, death threatened by a non-family member, or death threatened by a family member. Sexual abuse was measured by three items asking parents if their child has ever been touched inappropriately by or forced to engage in unwanted sexual behavior by a grown up in the home, adult outside of the family, or a peer. Each item is coded as *no* (0) or *yes* (1) to indicate whether it is applicable to the child. Physical and sexual abuse will each have their items combined into separate binary (yes/no) measures of physical or sexual abuse, respectively, wherein any indication of abuse (i.e., a code of 1 on any individual item) is coded as a yes for that CM subtype.

Binary coding of CM was based on prior analyses using the ABCD sample (Brieant et al., 2023; Chu et al., 2022; Hoffman et al., 2019; Orendain et al., 2023; Stinson et al., 2021). Additionally, it is intended to reflect current best practices. Indeed, though experiencing an adverse event increases the likelihood that another will occur (Dong et al., 2004; K. T. Putnam et al., 2013), emerging literature argues against the use of cumulative scores and risk thresholds,

[4] Please see ABCD protocol (Barch et al., 2018; Zucker et al., 2018) for more in-depth information about why these measures were selected to measure traumatic experiences of youth across study waves.

asserting that assuming adverse experiences hold equal weight or that cumulative effects are linear is problematic (Anda et al., 2020; F. W. Putnam et al., 2020). Recent findings highlight the presence of differential impacts among adverse childhood experiences; for example, the presence of any sexual abuse, physical abuse, or neglect better accounts for variability among mental health outcomes, compared to the use of cumulative scores (F. W. Putnam et al., 2020). Moreover, leaders in MRI-derived brain metrics and CM research suggest brain morphology following CM is highly specific and profound (Teicher et al., 2016). Together, this suggests that hippocampal development during adolescence may be uniquely impacted by each CM subtype, thus varying trajectories exist.

To account for the potential of multiple CM exposure, while also accounting for the differential impacts of subtype on outcomes, an additional "physical and sexual abuse" binary (yes/no) variable was created. All three dummy coded variables (physical abuse, sexual abuse, and physical and sexual abuse) were run as individual predictors in analyses. The influence of other adverse experiences (e.g., household substance use; parental separation/divorce; domestic violence) were controlled in analyses, as warranted, using individual items also taken from the larger KSADS-5 measure.

Hippocampal Morphology

High-resolution structural scans are acquired from participants at baseline and biennially, permitting examination of morphology across the entire brain. Acquired using state-of-the-art imaging protocols on 3-Tesla scanner platforms (i.e., Siemens Prisma, Philips Achieva, and GE MR750), as set forth by the ABCD Neuroimaging Workgroup (Auchter et al., 2018; Casey et al., 2018),[5] structural MRI scans gather data on subcortical gray matter volumes, cortical thickness,

[5] Please see Casey and colleagues (2018) for a detailed description of image acquisition methods within the ABCD study.

and cortical surface area from regions defined by the Desikan atlas (Desikan et al., 2006). Before being scanned, all participants were screened for contraindications and a "prescan" used to teach about how motion may impact MRI results (Casey et al., 2018; Hagler et al., 2019). All structural MRI data (raw T1-weighted and T2-weighted data) are then preprocessed (e.g., grad warp and bias field correction) using FreeSurfer v. 5.30 (Fischl et al., 2002) and standardized into Brain Imaging Data Structure (BIDS) format (Hagler et al., 2019). Images were then processed, including correcting for head motion, segmentation, and extraction, with region-of-interest (ROI) analysis results compiled and summarized into tabulated form (Hagler et al., 2019).[6]

An automated, atlas-based, volumetric segmentation procedure was used to label all subcortical structures, including the hippocampus (Hagler et al., 2019). The left and right hippocampus were measured individually. Because cortical contrast measures, such as morphometric (e.g., volume) and image intensity (e.g., density) measures, may be a more sensitive cortical marker (Hagler et al., 2019) when examining brain development (Lewis et al., 2018) and psychopathology (Norbom et al., 2019), both volume and density were used as cortical markers in the current study. Hippocampal volume was measured in mm^3 and density was measured using the average intensity of the normalized T1-weighted subcortical segmentation (ASEG) image.

Anxiety and Depression

The Child Behavior Checklist (CBCL) (Achenbach, 2011; Achenbach & Ruffle, 2000) includes items consistent with the DSM-5 criteria for anxiety and depression in youth (ages 6-18) from parent reports. The CBCL consists of 113 items used to assess children's internalizing,

[6] Please see Hagler and colleagues (2019) for a detailed description of image processing and analysis methods within the ABCD study.

externalizing, and total problems from the prior 6 months, inclusive of eight syndrome scales: anxious/depressed, depressed, somatic complaints, social problems, thought problems, attention problems, rule-breaking behavior, and aggressive behavior. Only the anxious/depressed (13 items) and depressed (8 items) scales will be used in the current analyses (see Appendix B). Parents rate each item as *not true* (0), *somewhat/sometimes true* (1), or *very/often true* (2). The CBCL is one of the most widely used measures of emotional problems and provides standardized scores based on national norms. Raw summative scores, wherein lower scores indicate less anxiety or depression, are converted to t scores. T scores at or above 60 represent non-normative symptomatology. The current study used t scores, but maintained continuous scoring rather than clinical cutoffs.

Substance Use

Scales from the substance use battery (Lisdahl et al., 2018) assessed substance use and intent in youth. The Lifetime Use Interview (Lisdahl & Price, 2012) assessed patterns of substance use across all drug categories via youth self-report (see Appendix C). Questions about substance use were only administered if the youth indicated that they have heard of the substance before. If so, use was assessed using a binary *no* (0) and *yes* (1). Following previous work which utilized the ABCD Lifetime Use Interview data (Sullivan et al., 2022), the current study only included items from major drug categories[7] (i.e., will exclude caffeine use). Additionally, only the binary item assessing whether or not youth have used the substance before was utilized.

The PATH Intention to Use scale (Hyland et al., 2017) measured curiosity and intention to use substances (i.e., tobacco, cigarettes, electronic nicotine, alcohol, and marijuana; see

[7] Major drug categories include: alcohol, nicotine products, cannabis products, synthetic cannabinoids, cocaine, cathinones, methamphetamine, ecstasy/MDMA, ketamine, gamma-hydroxybutyrate (GHB), heroin, psilocybin, salvia, other hallucinogens, anabolic steroids, inhalants, and prescription misuse of stimulants, sedatives, opioid pain relievers, and over-the-counter (OTC) cough/cold medicine. See Lisdahl et al. (2018) for a comprehensive list of drug categories included in the original questionnaire.

Appendix D). Questions were only administered if the youth indicated that they have heard of the substance before but have not yet tried it. This 10-item scale measured youths' likelihood to begin using substances. For each substance, youth were asked if they think they will try the substance soon and if they would try the substance if a friend offered it to them. Youth ranked each item on a 4-point Likert scale: *definitely yes* (1), *probably yes* (2), *probably not* (3), and *definitely not* (4). Youth were also able to answer *don't know* (5) or *refuse to answer* (6).

For the current study, one or more endorsed substances from the Lifetime Use Interview were coded as substance use (2) and no endorsed categories were coded as no substance use (0). Any youth who indicated that they definitely or probably would try a substance or would if a friend offered it to them on the PATH Intention to Use scale had their original score of 0 recoded to (1) as indication that they are high-risk for substance use.

Analytic Plan

Secondary data from the ABCD study were downloaded and scored at three key timepoints: baseline, two-, and four-year follow-up assessments. Study hypotheses were pre-registered on the Open Science Framework (C. B. Corso, 2024).

Preliminary Analyses

Data were cleaned, including checks for outliers, heteroscedasticity, and multicollinearity. Data were also screened for multivariate normality (PP and QQ plots) and deemed to meet the assumption. Measure descriptives, including means, standard deviations, skewness, and kurtosis, were calculated in SPSS version 29.0 (IBM Corp, 2023) and are reported in Table 3. Zero-order correlations among variables are also provided in Table 4. Further, to determine potential covariates for hippocampal morphology and anxiety, depression, and substance use outcomes, Pearson correlations, independent samples t-tests, one-way ANOVAs, and cross-tabs with

Pearson chi-square tests were conducted.[8] Coefficients from these tests are provided in Table 5. Child age, sex, race/ethnicity, parent education, and household income were all determined to be necessary covariates for models in Aim 2 and Aim 3.

Power Analysis. It is suggested that research employing structural equation modeling approaches achieve a sample size of 200 individuals (Kenny, 2020). The current study consists of 11,466 individuals. Still, the pwrSEM utility (Wang & Rhemtulla, 2021) was used to conduct a simulation-based power analysis. For the most complex model proposed, with $\alpha = 0.05$ and standardized regression and indirect effect sizes set conservatively at 0.10, the sample size (N = 11,466) had sufficient power (>.99). Additional *post hoc* analyses were conducted with the *semPower 2* package (Moshagen & Bader, 2024) in version 4.3.1 of R Statistical Computing Environment (Posit Team, 2024), which revealed that there was sufficient power (0.80) to detect Root Mean Square Error of Approximation (RMSEA) effects as small as 0.026, 0.016, 0.013, and 0.011 for Aim 1 (hypothesis 1a and 1b/1c), Aim 2, and Aim 3 (see Figure 6).

Missing Values Analysis. Data were observed to have considerable missingness across year 2 and year 4 waves. In order to determine whether data were missing at random (MAR), a Little's test was performed (Little, 1988). Values were determined to be not MAR, and instead suggested a pattern of non-random missingness, $\chi^2(84) = 255.445$, $p = 0.000$. Missing values were imputed using full information likelihood (FIML) estimation, which is considered a more effective method than listwise or pairwise deletion (Kaplan, 2001). Unlike traditional imputation methods, FIML simultaneously estimates the specified model and missing values, based on the model and all observed data. Additionally, FIML was selected given its consistent advantages in

[8] Pearson correlations were utilized when both variables were continuous. Independent samples t-tests were utilized when one variable was categorical with two groups and the other variable was continuous. One-way ANOVAs were utilized when one variable was categorical with three or more groups and the other variable was continuous. Cross-tabs with Pearson chi-square tests were utilized when both variables were categorical.

structural equation models, such as yielding low convergence failure, minimizing bias in parameter estimates, producing efficient standard errors and more accurate Type I error rates, and maintaining statistical power (Enders & Bandalos, 2001).

Main Analyses

This study examined trajectories of hippocampal development as a function of time (hypothesis 1a), CM subtype (hypothesis 1b), and the interaction of time and CM subtype (hypothesis 1c) using latent growth curve modeling (LGCM). Once latent intercepts and slopes which fit the data well were established with LGCM, the direct relationships among CM subtypes and anxiety, depression, and substance use (hypothesis 2a), potential mediating role of hippocampal morphology (hypothesis 2b), and potential a-path moderating role of social determinants of health (hypothesis 3a) were assessed. A conceptual model of study aims is provided (Figure 1), as well as statistical models (Figures 2-5).

Latent Growth Curve Modeling (LGCM). A structural equation modeling (SEM) approach, latent growth curve modeling (LGCM), was utilized to relax the assumption of homogeneous population and allow for the identification of homogeneous subgroups with distinct developmental trajectories and intraindividual variability to emerge (Becht & Mills, 2020; Bollen & Curran, 2006; Felt et al., 2017; Howard & Curran, 2014; Preacher et al., 2008). Utilizing both variable- and person-centered analyses, advantages associated with SEM also apply to LGCM, such as effectively accounting for measurement error and dealing with missing data (Preacher et al., 2008). This method was necessary for better understanding subgroup effects and mechanistic processes (Cecil et al., 2017; Cicchetti & Toth, 2015; Krull & MacKinnon, 2001; Russotti et al., 2021).

In particular, LGCM allows for each sample case to have an individual trajectory, representing that individual's change over time (Bollen & Curran, 2006; Howard & Curran, 2014). LGCMs model repeated measures as latent variables composed of random slopes and random intercepts, which accounts for clustering at the individual level and best allows for variability within the trajectory data to be maintained (Bollen & Curran, 2006; Howard & Curran, 2014). In this study, trajectories of hippocampal volume and density were examined. Additionally, LGCM allows for the examination of both predictors of and outcomes related to latent variables representing trajectories (Bollen & Curran, 2006; Howard & Curran, 2014; Preacher et al., 2008). That is, this approach allows for mediated effects to be tested within a clustered dataset (Krull & MacKinnon, 2001). In this study, CM subtypes (physical abuse, sexual abuse, and physical and sexual abuse) were examined as potential predictors of differences in hippocampal trajectories, and anxiety, depression, and substance use were examined as potential outcomes related to those trajectories.

Model Specification. Latent growth curve models (LGCM) were used to examine hippocampal trajectories (Aim 1) and to test the hypothesized pathway (Aim 2) and moderated pathway (Aim 3). The intercept and slope of the LGCM were allowed to covary, with the latent slope[9] estimated linearly (i.e., $\lambda = 0$, 1, and 2, for waves 0, 2, and 4, respectively)[10] and latent intercept[11] fixed to 1. Directional regressions were specified, such that latent intercept and slope were regressed onto the three dummy-coded CM variables (i.e., physical abuse, sexual abuse, both physical and sexual abuse) simultaneously. An additional directional regression was specified for Aim 2, such that outcomes of interest (i.e., anxiety, depression, substance use) were regressed onto latent intercept and slope, in separate models. For Aim 3, moderators were

[9] Latent slope represents the rate at which the measure changes over time.
[10] Factor loadings describe trends over time in the measure.
[11] Latent intercept represents the level of the measure when time equals 0.

specified on the mediational *a*-path via regressing latent intercept and slope onto the covariate of interest as well as calculated interaction terms between the covariate and predictors.

Model Fit. Model fit cutoffs were not pre-registered. Model fit statistics which indicate good model fit, described below, are also available in Table 6. Model fit was measured using Chi-Square (χ^2), Root Mean Square Error of Approximation (RMSEA), Standardized Root Mean Square Residual (SRMR), Tucker-Lewis Index (TLI), and Comparative Fit Index (CFI). Chi-Square (χ^2) values compare expected values to observed data/model results, where higher values indicate greater differences. The associated *p*-value indicates whether these differences are significantly different from one another, statistically. Good model fit is typically indicated by a low, non-significant χ^2 (Pearson, 1900) statistic; however, χ^2 values are particularly affected by large sample size (Kenny, 2020), such that larger samples are more biased towards type I (false positive) errors. Given the large sample size of the present study, reported χ^2 values were considered in light of this bias. The RMSEA (Steiger, 1990; Steiger & Lind, 1980) is an absolute measure of fit, based on the non-centrality parameter, which assesses how far the hypothesized model is from perfect (Kenny, 2020). RMSEA tends towards being too large (i.e., positive bias) and is currently the most popular measure of fit (Kenny, 2020). Good model fit is indicated by RMSEA value less than or equal to .06 (Hu & Bentler, 1999) with an upper confidence interval value of less than .08 (Kenny, 2020). The SRMR (Maydeu-Olivares, 2017) is also a positively-biased absolute measure of fit, and indicates the standardized difference between the observed and predicted correlation (Kenny, 2020). Good fit of the model is indicated by SRMR value less than or equal to .08 (Hu & Bentler, 1999). The TLI (Bentler & Bonett, 1980; Tucker & Lewis, 1973) and CFI (Bentler, 1990) are incremental fit indices, which assess how the hypothesized model fits the data relative to a baseline model with the worst possible fit (Kenny,

2020). Good model fit is indicated by CFI and TLI values greater than or equal to .95 (Hu & Bentler, 1999). Additionally, the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC), both comparative fit indices, were used to compare models. Lower AIC (Akaike, 1974, 1987) and BIC (Schwarz, 1978) values are indicative of a better fitting model (Kenny, 2020). Model fit statistics selected for use satisfy all four categories of fit statistics: residual-based (SRMR), independence-model based (CFI, TLI), root mean square area of approximation (RMSEA), and information-criterion-based (AIC, BIC) (Hayashi et al., 2011).

Results

Secondary data analyses were conducted on a subsample of ABCD youth from whom there was complete data on CM-related items (N = 11,466). Missing values were imputed using full information likelihood (FIML) estimation. All analyses were run using version 0.6-16 of the *lavaan* package (Rosseel, 2012) in version 4.3.1 of R Statistical Computing environment (Posit Team, 2024). Measure descriptives are located in Table 3 and R code for analyses is located in Appendix E.

A general summary of results is provided in Table 7. The full results for each hypothesis tested are described below and presented in Tables 8-14.

Aim 1. Hippocampal Trajectories

All unconditional (hypothesis 1a) and conditional (hypothesis 1b) model fit statistics for Aim 1 can be found in Table 8, with relevant models reported below.

Hypothesis 1a: Hippocampal Trajectories

Unconditional LGCM were conducted to examine hippocampal trajectories and establish models that fit the data well. Initially, a “whole” model was run, wherein the left and right hemispheres of both hippocampal volume and density were combined into a single measure of

hippocampal development (LGCM 1). However, due to poor fit statistics, models examining left- (LGCM 2) and right- (LGCM 3) hippocampal development (combined volume and density) were conducted. Again, due to poor fit statistics, additional models were tested; separate volume and density models were examined. First, a combined left and right model of volume (LGCM 4) and a combined left and right model of density (LGCM 7) were run. However, due to poor model fit, it was determined that examining the left and right measures of both volume and density were necessary. While left (LGCM 5) and right (LGCM 6) volume models retained poor fit, unconditional models of left (LGCM 8; $\chi^2(1) = 2.027$, p = .155, RMSEA = .009 [90% CI = .000, .029], SRMR = .003, TLI = 1.000, CFI = 1.000, AIC = 97,808.315, and BIC = 97,867.093) and right (LGCM 9; $\chi^2(1) = 6.770$, p = .009, RMSEA = .022 [90% CI = .009, .040], SRMR = .005, TLI = 0.999, CFI = 1.000, AIC = 98,551.149, and BIC = 98,609.926) density both had acceptable model fit. Therefore, only left density and right density models were used in subsequent analyses. Variances (Ψ_α and Ψ_β) and covariance ($\Psi_{\alpha*\beta}$) of the latent variables (i.e., intercept and slope) for unconditional left and right density models were significant (see Table 9), indicating that proceeding with conditional model testing was an appropriate next step.

Hypothesis 1b and 1c: Hippocampal Trajectories Predicted by CM Subtype

Conditional LGCM model fit was acceptable for left (LGCM 10; $\chi^2(4) = 3.121$, p = .538, RMSEA = .000 [90% CI = .000, .013], SRMR = .002, TLI = 1.000, CFI = 1.000, AIC = 97,815.204, and BIC = 97,918.064) and right (LGCM 11; $\chi^2(4) = 7.390$, p = .117, RMSEA = .009 [90% CI = .000, .018], SRMR = .003, TLI = .999, CFI = 1.000, AIC = 98,557.183, and BIC = 98,660.043) density. However, model fit did not improve substantially from the unconditional models, with some comparative indices (e.g., AIC, BIC) revealing conditional models (LGCMs 10 and 11) to fit more poorly than their unconditional counterparts (LGCMs 8 and 9,

respectively). Additionally, parameter estimates of the conditional models revealed no significant associations between CM subtype and density slope or intercept. Moreover, variance in latent intercept and slope for the conditional models (LGCM 10 and 11) remain unchanged from that of the respective unconditional models (LGCM 8 and 9), further indicating that CM subtype had no effect on the latent growth of the hippocampus. Model parameter estimates are provided in Table 10 (LGCM 10, left density model) and Table 11 (LGCM 11, right density model). Because the effect of CM on hippocampal morphology was not supported, no covariates were added to the models for further testing.

Aim 2. Mediating Effects of Hippocampal Morphology

Latent hippocampal intercept and slope from Aim 1 were used as the mediator variable in all subsequent models. Given the lack of support for Aim 1, the mediational *a*-path in Aim 2 did not have a reasonable statistical basis; that is, CM subtypes were not found to predict hippocampal trajectories in Aim 1, thus nullifying the *a*-path for Aim 2. Literature is mixed concerning whether mediation without support for an *a*-path should be performed. For example, while the causal steps approach suggests the *a*-path to be a prerequisite to mediation (Baron & Kenny, 1986), more recent work suggests that indirect pathways remain plausible in spite of non-significant *a* or *b* paths (Hayes, 2022). Thus, to thoroughly test the proposed hypotheses, Aim 2 was conducted. Six mediator models were run (LGCMs 12-17): one per outcome variable (i.e., anxiety, depression, substance use) per each side of the hippocampus (i.e., left and right). Model fit across mediational models was relatively poor, particularly in comparison (e.g., AIC/BIC) to LGCMs performed in Aim 1. Model fit indices for LGCMs 12-17 are provided in Table 12 and effect estimates are provided in Tables 13 and 14.

Hypothesis 2a: CM Predicts Anxiety, Depression, and Substance Use (X → Y)

Direct effects for left-hippocampal and right-hippocampal mediations are reported as path *c'* in Tables 13 and 14, respectively. Contrary to the hypothesized direct effect, none of the CM subtypes were associated with depression or substance use. Additionally, no CM subtypes were associated with anxiety in the right-brain model. However, all CM subtypes were associated with anxiety in the left-brain model. As hypothesized, physical abuse (β = 0.034, *B* = 1.741, *SE* = 0.806, *p* = 0.031) and sexual abuse (β = 0.066, *B* = 3.225, *SE* = 0.783, *p* = 0.000) were each associated with greater anxiety symptoms. In contrast, experiencing both physical and sexual abuse was associated with fewer anxiety symptoms (β = -0.057, *B* = -6.889, *SE* = 2.116, *p* = 0.001). Still, all three significant standardized effects are marginal.

Hypothesis 2b: Hippocampal Morphology as a Mediator (X → M → Y)

Effects for left-hippocampal and right-hippocampal mediations are reported in Tables 13 and 14, respectively. Unsurprisingly, given the results from Aim 1, none of the mediational models had a significant *a*-path (i.e., path from predictor to mediator). Five *b*-paths (i.e., paths from meditator to outcome) were statistically significant: left-density intercept predicted anxiety (β = 0.348, *B* = 0.727, *SE* = 0.002, *p* = 0.000), left-density slope predicted depression (β = 0.275, *B* = 370.342, *SE* = 31.679, *p* = 0.000), right-density slope predicted anxiety (β = 0.573, *B* = 248.099, *SE* = 16.415, *p* = 0.000), and right-density intercept (β = -0.030, *B* = -0.053, *SE* = 0.027, *p* = 0.048) and slope (β = 0.723, *B* = 269.113, *SE* = 17.613, *p* = 0.000) predicted depression. Aside from right-density intercept on depression, which had marginal, negative standardized effect, these significant *b*-paths had positive, medium-to-large effects. Overall, albeit with mixed support, this suggests that greater density is associated with greater anxiety and depression. There were no statistically significant indirect effects or total effects. Because direct

and indirect pathways were not supported, no covariates were added to the models for further testing.

Aim 3. Moderating Effects of Social Determinants of Health

Due to the lack of statistical grounding for the mediational models in Aim 2, Aim 3 (moderated mediation) was not conducted. Still, sample code for these models is included in Appendix E as part of the overall R code.

Discussion

It is unclear whether heterogeneity in psychopathology following CM is due to specific aversive stimuli encountered or differences in behavioral adaptations to traumas (Cecil et al., 2017; Russotti et al., 2021). The purposes of this study were to examine neurodevelopmental trajectories of the hippocampus, with attention given to the distinct role CM subtype may have, and ascertain if those trajectories act as a mechanistic explanation for anxiety, depression, and substance use following CM. In elucidating distinct effects of CM subtype on brain development, I sought to advance etiological understanding of adverse outcomes following CM. Ultimately, this research intended to inform interventions and treatments, such that the optimal developmental timing, respective to each CM subtype, could be used to preempt trajectories leading to adverse outcomes.

Study Findings

Study results are summarized in Table 7 and detailed below and in subsequent tables.

Aim 1. Hippocampal Trajectories

Hypothesis 1a was partially supported, such that hippocampal gray matter density increased over time (i.e., with age) for all adolescents; this is unsurprising given the extreme plasticity and expected growth of the adolescent brain, particularly the hippocampus.

Interestingly, the effect of the latent slope of the right hippocampus had a large effect, while the effect was small for that of the left hippocampus, which suggests the slope, or change over time, of the right hippocampus to be larger than that of the left. These findings are consistent with right-greater-than-left laterality effects of normal hippocampal development (Burgess et al., 2002; Giedd et al., 1999; Maguire & Frith, 2003; Tamnes et al., 2014, 2018). Findings were also consistent with prior literature on variability within the hippocampus (Burgess et al., 2002; Leuner & Gould, 2010; Tamnes et al., 2018). Indeed, there was significant variance in both the latent intercept and slope factors of the unconditional models, indicating that the hippocampal density of adolescents in the sample varied greatly at baseline as well as in growth rate. However, this variability was not explained by CM subtype. Contrary to hypotheses 1b or 1c, CM subtype was not found to predict hippocampal morphology, nor were subtypes (i.e., physical abuse, sexual abuse, or both physical and sexual abuse) found to have distinct slopes. Rather, only a single hippocampal trajectory emerged across all participants (i.e., CM and non-CM).

Aim 2. Mediating Effects of Hippocampal Morphology

Moreover, hypothesis 2a was only partially supported: physical abuse and sexual abuse predicted higher anxiety scores, but only in the left hippocampus mediation model. Unexpectedly, experiencing both physical and sexual abuse was associated with lower anxiety scores, which was also exclusive to the left hippocampus mediation model. However, all effects were marginal in size. Against study predictions, CM did not predict anxiety in the right hippocampus mediation model, and models from both hemispheres did not evidence an association between CM and depression or substance use. Research does suggest the left hippocampus as being more involved with autobiographical memory and context-dependent episodic memory, while the right is more involved with memory for locations within an

environment (Burgess et al., 2002). Further, the significance of time on memory recollection varies according to laterality, such that the left hippocampus has more time invariant involvement with memory while the right tends to be less involved as memories become more remote (Maguire & Frith, 2003). These left hippocampus models, then, somewhat align with this previous work in laterality, assuming the role of memory in the current models. However, hypothesis 2b was not supported, as hippocampal morphology did not mediate associations between CM and anxiety, depression, or substance use.

Still, albeit with mixed support, greater hippocampal density[12] was associated with greater anxiety and depression symptoms (path *b*) in the present study. Though initially counterintuitive, this association reflects normative patterns of adolescent development: both gray matter density (Gennatas et al., 2017), particularly in the hippocampus (Brouwer et al., 2015; Suzuki et al., 2005), and psychopathology (Cohen et al., 1993; Costello et al., 2003, 2011) increase throughout adolescence. Powers and Casey (2015) highlight how adolescent-specific brain morphology partially explains the increased risk for psychopathology seen throughout adolescence. While this finding could be a spurious association, it could also be indicative of more nuanced associations between the hippocampus and psychopathology, as this finding mirrors how hippocampal density and activity are negatively associated with connectivity. That is, hippocampi with less density generally have less spontaneous network activity, but greater connectivity, compared to hippocampi with greater density (Ivenshitz & Segal, 2010). Researchers note that this pattern implies synaptic strength to be inversely proportional hippocampal network size, so while spontaneous activity occurs more frequently in denser

[12] Importantly, density is a size-independent measure, such that it represents an item's mass relative to its size. That is, a large-in-size hippocampus does not necessarily have a greater density than a small-in-size hippocampus, though it does have greater maximum density potential. Thus, literature concerning the overall size of the hippocampus, such as volume, are not directly applicable to this finding and will not be discussed.

networks, its impact is stronger in less dense networks (Ivenshitz & Segal, 2010). Moreover, spontaneous network activity is critical in the development and maintenance of brain network plasticity and adaptability (Kerschensteiner, 2014; Latham, Richmond, Nelson, et al., 2000; Latham, Richmond, Nirenberg, et al., 2000; Opitz et al., 2002), which is vital to the brain's stress response processes (Hermans et al., 2014). Thus, this heightened density may indicate less impactful activity occurring, resulting in less adaptable brain networks and, consequently, greater anxiety and depression. Notably, however, hippocampal activity was not included in the present analyses. Therefore, associations between density, activity, and connectivity and how they relate to mental health symptomatology can only be theorized. Future work should seek to test these relationships. Results could yield important information concerning the etiology of psychopathology, particularly during periods of brain morphology and increased risk for mental health problems, such as adolescence. Further, considering the potentially confounding role of normative development (i.e., age) within these endeavors is paramount.

Aim 3. Moderating Effects of Social Determinants of Health

Aim 3 intended to examine social determinants of health as moderators of the *a*-path from aim 2 (hypothesis 3a). However, these conditional indirect effects were not assessed due to the lack of evidence that indirect effects were present (Aim 2). Indeed, moderated mediation (i.e., conditional indirect effects) assesses the strength of a mediation (i.e., indirect effect) and whether those indirect effects remain constant across different contexts or groups (Preacher et al., 2007). Given the absence of indirect effects in the present study, moderated mediation models were not statistically warranted.

Explanation of Null Findings

There are a number of factors which could have contributed to the present null findings. Firstly, the singular trajectory and lack of support for path models could indicate that CM has no shared quality nor unique subtype effects, and might instead amplify what is increasingly understood as a general genetic predisposition to non-specific mental illness (Caspi & Moffitt, 2018). Relatedly, it could indicate that non-morphological factors, such as genetics and environment, play a more profound role than previously thought. Another possible interpretation of these findings is that the hippocampus is a less important contributor to the development of psychopathology following CM than previously thought, and that hippocampal cognitions, processes, and skills are not a specific target for future interventions. However, it seems more plausible that the present null findings can be otherwise explained. For example, the mostly non-significant direct relationships between CM and mental health symptoms in the present study contradict decades of research; this finding alone suggests confounds and/or limitations of the present study. I posit that the null results of the current study can be explained by subfield nuances, timing of measurement, and study limitations.

Subfield Differences in Hippocampal Morphology

The hippocampus is a heterogeneous structure (Lavenex & Banta Lavenex, 2013; Poppenk et al., 2013), with recent research pointing to the hippocampal subfields being functionally connected to varying, specific brain networks (Alahmadi et al., 2023). The subfield differences may also vary according to the developmental stage of the individual. For example, Tamnes and colleagues (2014) found adolescents (age 14) and young adults, in contrast to children (ages 8 and 10-11), to selectively engage different regions of the hippocampus and parahippocampal gyrus when recollecting. This work aligns with research outlining the

functional reorganization of the hippocampus during adolescence, such that it becomes increasingly specialized for recollection (Ghetti et al., 2010; Keresztes et al., 2018). These findings are particularly relevant to the sample age of the present study. Other work examining the networks associated with CM has found physical abuse, but not sexual abuse, to be associated with the default mode network (i.e., a network involving the hippocampus) (Zhang et al., 2022). Notably, the work highlighted focuses exclusively on the functional interactions of brain networks, which was outside the scope of the present study. Still, the present findings could reflect a need for more nuanced examination of structural measures, as well as the involvement of additional, functional measures of the brain in subsequent models.

Timing of Measurements

It is possible that the timing of measurements in the present study confounded potential effects. Effects on brain development are highly influenced by the timing of measurement as well as the timing of exposure (Andersen, 2003). Some of the effects of CM on hippocampal development, in particular, were found to be delayed (Teicher & Samson, 2013). While the current study utilized longitudinal measurements, it is still possible that the amount of time between CM exposure, hippocampal scans, and mental health outcomes was not sufficient. With additional time points, models could be re-run to account for this lag in effects.

Further, results could indicate that CM timing – rather than or in addition to CM subtype – is an important future direction. That is, CM occurring in infancy is likely to differentially affect the developing individual when compared to CM occurring in childhood or adolescence. Indeed, extant work highlights both the subtype-specific impact of CM as well as the importance of developmental timing of CM exposure (Manly et al., 2001). This measurement timing may be problematic, as normative development incorporates (exposure-expectant) or compensates

(exposure-dependent) environmental stimuli as it occurs. For example, if physical abuse reliably predicts a decline in hippocampal growth 1-year after the experience, but measurements are only taken at age 11 years, the effect could go unnoticed amidst the normal variability in size. Similarly, if physical abuse predicts a rapid decline in growth if experienced before age 6, but has no effect if experienced after age 6, the effects could still go unnoticed at age 11 due to the normal variability in measurements. Indeed, it is possible that differing trajectories of CM subtypes were obscured by the use of CM as a time-invariable predictor in the current study. With CM, issues of developmental timing are further complicated by issues of frequency and chronicity of exposure. However, timing, frequency, and chronicity of CM exposure was not measured in the current study; instead, models could only account for whether or not CM had occurred at baseline.

Concerning the hippocampus, in particular, exploring CM as a time-variable predictor is an important future direction. Being involved in memory, the hippocampus is tasked with rapid, generic knowledge gain in early life, and throughout its maturation refines encoding processes to include detailed, long-lasting episodic memories (Keresztes et al., 2018). Therefore, it is reasonable to hypothesize that trauma, such as CM, occurring throughout childhood at different points in time could have different impacts on the hippocampus maturation process and would be recollected differently depending upon the developmental stage of the event; this, in turn, could negatively impact mental health. For example, emotional memories are durable (S. E. Williams et al., 2022), though memory retrieval can depend on the event's emotional valence as well as the individual's trauma exposure. Forest and Blanchette (2018) found that sexual abuse survivors had poorer episodic recall of neutral and general-emotional content compared to a control group, though trauma-related-emotional content recall was unimpaired. Further, a diminished ability to

selectively forget negative memories is associated with poorer mental health (Nørby, 2018). Thus, there is a need to assess whether or not, and how, these impairments in memory encoding and recollection are affected by timing of CM exposure. Longitudinal measurement and time-variable models of CM, relative to hippocampal morphology and mental health sequelae, should be tested.

Study Limitations

The present study, and in particular the dataset used, had considerable strengths. For example, ABCD is a developmental study by design. It contains large-scale, longitudinal data with a broad scope of measures, and is ideal for assessing individual differences in the developmental process (Saragosa-Harris et al., 2022) Still, results should be interpreted in light of several limitations, which could have contributed to the present null findings.

Firstly, CM measures within the ABCD dataset were limited to sole informant, singular method measures. Quantification of CM was restricted to binary yes/no, rather than incorporating explanatory dimensions of CM (e.g., duration, intensity, frequency). In addition to CM dimensions, it may be advantageous to explore the impact of relationship to the perpetrator. For example, abuse experienced from a family member may differentially impact the developing child when compared to abuse from a non-family member. Moreover, CM was reported by the parent, rather than the child. Methodological challenges plague the measurement of CM (Fallon et al., 2010; Laajasalo et al., 2023); while some research suggests both parent- and adolescent self-report of CM have good predictive validity (Tajima et al., 2004), other work points to the importance of utilizing child self-report measures, particularly when assessing the impact of CM on psychopathology symptomatology (Beasley et al., 2021). Indeed, Beasley and colleagues (2021) found child self-report of CM had 2–3 times higher reported prevalence than official

records (i.e., Child Protective Service reports). Likewise, youth's anxiety and depression symptoms were likely underestimated by the use of parent-report (Aebi et al., 2017; Hope et al., 1999). Future work should seek to leverage youth self-report of mental health symptomatology as well as CM experiences, which could also include important measures of timing, frequency, and chronicity of CM, as well as relationship to the perpetrator.

Secondly, CM subtypes were limited to examination of physical and sexual abuse. Measures of emotional abuse and neglect, which are the most prevalent CM subtypes, were missing from the ABCD study and, thus, the current analyses. As outlined in the introduction, abuse can be categorized as an act of commission (e.g., physical abuse, sexual abuse), which affects experience-dependent processes, or omission (e.g., forms of neglect), which affects experience-expectant processes. It is important to consider how these categorizations of abuse, in addition to comparing subtypes overall, would potentially impact the developing brain, especially given the varying outcomes associated with CM subtypes. For example, neglect is associated with internalizing issues and neurobiological changes, over and above other CM subtypes, while emotional abuse is most often an exacerbating factor (Warmingham et al., 2019, 2022). Similarly, Manly and colleagues (2001) found non-CM controls had less internalizing symptoms than children who experienced physical neglect, and less externalizing symptoms than children who experienced sexual abuse, physical abuse, or sexual and physical abuse. Together, these findings highlight the need to examine all CM subtypes, as well as diverse forms of subsequent psychopathology, in order to gain the most comprehensive view of the effects of CM. Future work could also explore how latent classes of CM, rather than subtypes alone, impact outcomes. For example, Warmingham and colleagues (2019) found differences in child behavioral and social-emotional outcomes according to three latent classes: chronic,

multi-subtype, only neglect in a single developmental period, and other single subtype in a single developmental period. Similar methods could be employed within a larger structural equation model to better understand neurodevelopmental trajectories following CM.

Thirdly, data missingness likely contributed to the present null findings. Issues with attrition are common in cohort designs, such as the ABCD study, and can greatly impact participant retention as well as depth and breadth of measures within the study (Saragosa-Harris et al., 2022). In the current study, just under 75% of data were missing in the structural MRI year 4 follow-up scans, and roughly 60% of data were missing across outcome measures. This considerable missingness, though estimated using FIML, is a major limitation of the current study which likely contributed to the non-significance effects. While FIML does take missingness into account and has been shown to produce reliable estimates even with a high degree of missingness (Grimm & Wagner, 2020; Lim & Cheung, 2022; Olinsky et al., 2003), these unbiased results assume data are missing at random (Madley-Dowd et al., 2019). When data are not missing at random, as in the present study, analyses risk being more biased and generalizability of findings are limited (Kang, 2013).

Fourthly, work utilizing the ABCD sample is limited in its generalizability (Saragosa-Harris et al., 2022), as the sample had unrepresentative, low prevalence of CM as well as anxiety, depression, and substance use. In part, these issues of prevalence could be attributed to the use of parent-report measures or cohort-design effects. Still, although designed to be nationally representative, ABCD site selection as well as research participation biases generally resulted in the sample underrepresenting rural families and overrepresenting families with higher income and greater levels of education (Compton et al., 2019). Smaller sample sizes are known to have greater variability, such that they may not be representative of the population of interest.

When considering the low prevalence of CM in the present sample, this likely undermined potential differences among latent growth curves such that potential differences among latent intercepts and slopes were underpowered, with overlapping error bars resulting in null findings. Indeed, national prevalence of physical abuse and sexual abuse, reported by 4,000 children 0-17 years old, are 18.1% and 0.2%[13], respectively (Finkelhor et al., 2015). In the current study, prevalence rates were 1.7% and 1.9%, respectively. Similarly, though mental health problems have been on the rise since the 2000s (Bor et al., 2014; Parodi et al., 2022) and were knowingly exacerbated by the COVID-19 pandemic (Jones et al., 2021), anxiety and depression scores – as well as substance use rates – in the current study were unexpectedly low, which could have contributed to the null findings of Aims 1 and 2.

Fifthly, the impact of the COVID-19 pandemic on data collection procedures in ABCD is noteworthy. Second-wave data collection was delayed due to the COVID-19 pandemic; in particular, in-person scans and biospecimen data were suspended while other data collection procedures were adapted to online formats (Saragosa-Harris et al., 2022). Second wave data collection was completed entirely virtually from March to August 2020, though a majority of assessments remained online well into the year 2022 (Saragosa-Harris et al., 2022). Thus, there is a significant level of missingness across year 2 and year 4 data, especially for measures which could not be completed in a virtual setting (e.g., MRI scans). It is also important to note that this attrition and related COVID-19 effects were likely to have differentially affected families with other concerns, such as health issues, financial worry, food insecurity, and other resource scarcity (Saragosa-Harris et al., 2022). This poses considerable issues for research aimed at understanding developmental trajectories of at-risk use, such as the current study; it is reasonable

[13] When reporting additional sexual offenses, such as statuatory sex, sexual assaults, and rape, prevalence was 21.7%.

to expect that future work with additional data and time points would evidence conflicting results to the current study. In particular, inclusion of additional time points would allow for the observation of linear, as well as non-linear, trajectories; this may aid in reflecting prior work which evidenced pre-pubertal gains and post-pubertal losses in hippocampal gray matter (Gogtay & Thompson, 2010). As such, it would be beneficial for the current models to be rerun following additional ABCD data releases. Additionally, future work should control for the potential historical effects in the ABCD study associated with COVID-19.

Future Directions

In addition to those mentioned so far, several future directions should be noted.

Exploring Social Determinants of Health

While exploring the potential effects of social determinants of health were not plausible in the current study, they remain a notable concern. It was hypothesized that social determinants of health would moderate the association between CM and hippocampal morphology, such that known demographic risk factors would exacerbate the negative impact of CM on hippocampal development; future work should still seek to explore this question. It remains reasonable to posit that these variables (i.e., age, sex, race/ethnicity, parental educational attainment, and socioeconomic status/household income) would influence brain development.

Numerous studies have evidenced age-related differences in brain maturation processes (De Bellis, 2001; Tamnes et al., 2014, 2018), with adolescence being denoted as particularly important to the developing hippocampus (Keresztes et al., 2018; Kozareva et al., 2019). Additionally, sex-related differences in brain morphology are well-documented (Kaczkurkin et al., 2019; Yagi & Galea, 2019), though the patterns and prominence of those differences vary across developmental stages (Etchell et al., 2018). For example, while males tend to have a

greater total brain volume across development – and in adolescence specifically (De Bellis, 2001; Gennatas et al., 2017; Sowell et al., 2007) – females tend to develop quicker, such that they reach their peak brain volume earlier than males (De Bellis, 2001; Giedd et al., 1996, 1999; Lenroot et al., 2007). Moreover, sex-related differences in brain development, structure, and function are not always indicative of variance in behavior (Etchell et al., 2018). It is important that future work consider how age and sex factor into brain morphology, with specific attention given to how these factors differentially affect diverse brain structures and functions.

Moreover, lower levels of education (Bussy et al., 2021) and income (Tomasi & Volkow, 2021) negatively affect the developing brain. Research suggests differences in the quality and quantity of supportive/educational stimulation (Schneider et al., 2024; Tomasi & Volkow, 2021) and differences in income-related stressors (Schneider et al., 2024) as underlying causes of such effects. More work on how these variables impact the developing brain, and what exacerbating or protective factors exist, is warranted. To avoid oversimplification of these complex dynamics, research exploring these questions should also consider the role of culture and context (Schneider et al., 2024). Relatedly, recent work cautions against the oversimplified study of race/ethnicity-based differences in brain morphology. Indeed, Dumornay and colleagues (2023) found childhood adversity to attenuate race-related differences in gray matter volume, while Assari (2020) found the effect of family socioeconomic status to differentially affect Black and White families. Future work should exercise consideration of these important nuances when seeking to examine how social determinants of health (e.g., income, education, race/ethnicity) impact brain morphology and mental health sequelae.

Expanding the Current Study

Further, six additional future directions which expand upon the models in the current study were identified. First, the current model could be extended to include other brain structures that, like the hippocampus: undergo development during adolescence, have a particular vulnerability to stress, and are implicated in maltreatment and/or mental health etiology. These might include additional structures from the limbic system (e.g., amygdala) as well as the frontal cortex and other regulators of the HPA axis. Second, functional neuroimaging could be useful to incorporate in treatment efforts. Stratifying treatment by CM subtype has recently been suggested (Toth et al., 2020), and neuroscience researchers have found stratification based on biomarkers and circuitry to be effective (L. M. Williams & Hack, 2020). Integrating these fields, future work could incorporate a network-minded approach that would allow for even more multidisciplinary-informed treatment efforts. Third, the current model could include additional ages, outcomes, and frameworks. Carefully constructed, stage-salient models could use developmental traumatology (De Bellis, 2001) or the ecobiodevelopmental framework (Shonkoff et al., 2012) as the basis for additional projects. Fourth, manual segmentation procedures could be utilized to minimize under- and over-estimation associated with automated segmentation (Hagler et al., 2019). Fifth, variance within hippocampal volumes could be reduced by using normalization with respect to total intracranial volume, which would help to better define subtle differences and growth-related volumetric changes (Jalaluddin et al., 2013). Sixth, as ABCD was oversampled for siblings and twins (Saragosa-Harris et al., 2022), future studies could expand the current model to consider the potential role of family and genetics on developmental trajectories, and/or seek to account for site/batch effects across collection sites.

Hypothetical Relevance to Interventions

The current study sought to advance etiological understanding of adverse outcomes following CM and was intended to inform interventions and treatments. Null findings could be indicative of the hippocampus not being an appropriate target for interventions, and/or CM subtypes not needing nuanced treatment; however, the explanations and limitations outlined provide additional context. As such, I argue that more work is needed to establish whether or not interventions should or should not target cognitions, processes, and skills associated with specific neurocognitive structures to alleviate vulnerability to mental health sequelae, with respect to CM subtype.

Opinions on the integration of neuroscience and psychological intervention are mixed (Strege et al., 2021), and understanding how patterns of neural activity directly lead to changes in related behavioral symptoms is still an emerging field of research (Dutcher & Creswell, 2018). For example, behavioral interventions targeting the stress-response system may modulate the system by buffering reactivity responses, or by increasing top-down regulatory signals (Dutcher & Creswell, 2018). Indeed, some mindfulness meditation interventions buffer reactivity responses (Creswell & Lindsay, 2014; Lindsay et al., 2018), while others bolster functional connectivity of critical top-down regulatory networks (Creswell et al., 2016; Taren et al., 2017). It remains unclear, though, whether these differences are informed by previous experience (e.g., maltreatment), and how these system modifications translate into differential psychological and behavioral outcomes. Thus, research on predictors of brain structural and functional changes as well as how specific approaches target brain regions and processes is needed. Findings could then be integrated to critically inform interventions (De Raedt, 2020).

Indeed, in addition to helping with the selection and identification of optimal cognitive behavioral therapy procedures, interdisciplinary models can facilitate psychoeducation and inform procedural tailoring of therapeutic approaches (De Raedt, 2020; Kircanski et al., 2012; Strege et al., 2021). For example, hippocampal volume and mindfulness are positively associated (Baltruschat et al., 2021), specifically in adults with a history of CM (Joss & Teicher, 2021). As outlined prior, mindfulness interventions may buffer reactivity responses (Creswell & Lindsay, 2014; Lindsay et al., 2018) or bolster functional connectivity of critical top-down regulatory networks (Creswell et al., 2016; Taren et al., 2017). If trajectories of hippocampal volume differ according to CM subtype, it is possible that mindfulness interventions would be necessary for one subtype, but not another (e.g., if physical abuse was associated with lower volume, but sexual abuse was not, then mindfulness-based approaches are likely to more effectively target only physically abused individuals, while another intervention is more relevant for sexually abused individuals). Moreover, timing of interventions could be informed by this work; if physical abuse trajectories show a decline at age 14, but sexual abuse trajectories show a decline at age 18, critical knowledge on the optimal timing of interventions is gained. Additionally, these models can help to illuminate how certain trajectories inform behavioral outcomes. For example, interventions associated with buffered reactivity may have stronger effects on internalizing outcomes, whereas interventions associated with bolstered top-down regulatory networks could have stronger effects on externalizing outcomes. Then, given the known associations between CM subtypes and differential mental health sequelae, practitioners could select the intervention most suited to the experiences of their client and work towards preventing the most likely negative psychological outcomes. One educational review further details how integrative,

neuroscience-informed treatment planning can be implemented using research such as this (Ross et al., 2017).

Thus, additional research investigating neurodevelopmental correlates of CM, what system responses are occurring, and how these structural and functional changes pertain to associated stress-related outcomes (e.g., mental health), is necessary. Findings have the potential to critically inform the selection and developmental timing of interventions.

Conclusion

CM is a costly major public health issue necessitating research aimed at improving existing interventions. Elucidating the underlying mechanisms of this heterogeneity in CM sequelae is vital for improving treatment for victims of CM. Neurobiological data and trajectory mapping can be used to enhance CM interventions by identifying malleable targets. The hippocampus is an excellent focus of this approach. This study utilized longitudinal, multi-site, adolescent data and a novel statistical approach to investigate hippocampal development as a mechanism underlying mental health sequelae of CM. No differences in hippocampal trajectories for CM subtypes were found. Findings were likely influenced by variability in the data, as well as measurement and study limitations. Future work should seek to retest and expand upon these models, as findings have the potential to inform future treatment efforts by increasing knowledge of how maltreatment subtype and the hippocampus relate to the development of psychopathology.

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Tables and Figures

Table 1

Outline of the Time Each Measure was Distributed

Measure(s)	Description	Wave of Administration		
		Baseline	2-year FU	4-year FU
Demographics	Demographics	X		
KSADS-5 PTSD module	Maltreatment	X		
MRI	Hippocampus	X	X	X
CBCL	Anxiety & Depression			X
Lifetime Use Survey & PATH Intention to Use Scale	Substance Use			X

Table 2

Sample Descriptives Stratified by Maltreatment Subtype

	Total Sample (N = 11,466)	Physical Abuse (n = 199)	Sexual Abuse (n = 220)	Physical and Sexual Abuse (n = 35)	No Abuse (n = 11,082)
Age (years)					
Min.	8	9	8	9	8
Max.	11	11	10	10	11
Mean (*SD*)	9.48 (0.51)	9.50 (0.52)	9.50 (0.51)	9.43 (0.50)	9.48 (0.51)
Sex, *n* (%)					
Female	5,470 (47.7)	76 (38.2)	111 (50.5)	16 (45.7)	5299 (47.8)
Male	5,996 (52.3)	123 (61.8)	109 (49.5)	19 (54.3)	5783 (52.2)
Race/Ethnicity, *n* (%)					
White	8,525 (74.5)	142 (71.4)	173 (79.0)	28 (80.0)	8238 (74.5)
Black	2,424 (21.2)	77 (38.7)	55 (25.1)	12 (34.3)	2304 (20.8)
Hispanic	2,275 (20.1)	27 (14.0)	36 (16.4)	4 (11.8)	2216 (20.3)
Asian	787 (6.9)	11 (5.5)	12 (5.5)	2 (5.7)	766 (6.9)
Other	1,133 (9.9)	18 (9.0)	18 (8.2)	4 (11.4)	1101 (10.0)
Parent's Education, *n* (%)					
Less than high school diploma	742 (6.5)	9 (4.5)	10 (4.5)	1 (2.9)	724 (6.5)
High school diploma or GED	1,196 (10.4)	29 (14.6)	17 (7.7)	4 (11.4)	1154 (10.4)
Some college	3,341 (29.2)	79 (39.7)	100 (45.5)	12 (34.3)	3174 (28.7)
Bachelor's degree	3,244 (28.3)	53 (26.6)	55 (25.0)	13 (37.1)	3149 (28.5)
Postgraduate degree	2,926 (25.6)	29 (14.6)	38 (17.3)	5 (14.3)	2864 (25.9)
Annual Household Income, *n* (%)					
<$50,000	3,076 (29.3)	93 (50.8)	92 (44.9)	17 (50.0)	2908 (28.7)
$50,000-100,000	2,969 (28.3)	48 (26.2)	60 (29.3)	10 (29.4)	2871 (28.3)
>$100,000	4,455 (42.4)	42 (23.0)	53 (25.9)	7 (20.6)	4367 (43.0)

Table 3

Measure Descriptives

	N	Min.	Max.	*M*	*SD*	Skewness		Kurtosis	
						Stat.	*SE*	Stat.	*SE*
*PA	11466	0	1	0.02	0.13	7.39	0.02	52.66	0.05
*SA	11466	0	1	0.02	0.14	7.01	0.02	47.16	0.05
*PA+SA	11466	0	1	0.00	0.06	18.02	0.02	322.74	0.05
LdenB	11393	52.59	83.52	73.45	3.37	-0.58	0.02	-0.81	0.05
Lden2	7862	58.75	90.83	73.60	3.37	-0.52	0.03	-0.83	0.06
Lden4	2924	64.42	82.40	73.54	3.41	-0.46	0.05	-1.07	0.09
RdenB	11393	60.88	85.33	73.50	3.59	-0.58	0.02	-1.08	0.05
Rden2	7862	62.17	95.24	73.71	3.50	-0.51	0.03	-0.87	0.06
Rden4	2925	63.24	94.37	73.66	3.59	-0.41	0.05	-0.80	0.09
LvolB	11392	1853.20	5913.10	4017.28	403.07	0.18	0.02	0.33	0.05
Lvol2	7861	1578.10	5973.40	4080.54	409.71	0.15	0.03	0.51	0.06
Lvol4	2924	2701.90	5650.10	4132.75	423.96	0.23	0.05	0.05	0.09
RvolB	11393	1460.70	6695.30	4142.49	421.02	0.22	0.02	0.59	0.05
Rvol2	7862	2724.50	6207.80	4215.56	424.39	0.22	0.03	0.25	0.06
Rvol4	2925	2895.70	5856.00	4268.92	442.72	0.24	0.05	0.07	0.09
Anxiety	4500	50.00	91.00	53.12	5.85	2.44	0.04	6.26	0.07
Depression	4500	50.00	100.00	53.82	6.10	2.34	0.04	6.78	0.07
SU	11466	0	2	0.16	0.53	3.05	0.02	7.48	0.05

Note. PA = physical abuse, SA = sexual abuse, PA+SA = both physical and sexual abuse, SU = substance use. Hippocampus measures represented as: L = left, R = right, den = density, vol = volume, B = baseline, 2 = 2-year follow-up, 4 = 4-year follow-up. * indicates the variable is binary and skew/kurtosis is expected.

Table 4

Bivariate Correlations Among Key Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Physical Abuse	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2. Sexual Abuse	.152***	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3. Physical and Sexual Abuse	.416***	.396***	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
4. Baseline Left Density	.019*	.002	.010	1	-	-	-	-	-	-	-	-	-	-	-	-	-
5. 2-year Left Density	.019	.010	.020	.914***	1	-	-	-	-	-	-	-	-	-	-	-	-
6. 4-year Left Density	.010	.025	.007	.877***	.900***	1	-	-	-	-	-	-	-	-	-	-	-
7. Baseline Right Density	.019*	.001	.011	.957***	.909***	.880***	1	-	-	-	-	-	-	-	-	-	-
8. 2-year Right Density	.019	.009	.018	.896***	.955***	.885***	.927***	1	-	-	-	-	-	-	-	-	-
9. 4-year Right Density	.017	.032	.009	.867***	.889***	.957***	.898***	.911***	1	-	-	-	-	-	-	-	-
10. Baseline Left Volume	-.014	-.032***	-.016	.105***	.098***	.115***	.110***	.098***	.119***	1	-	-	-	-	-	-	-
11. 2-year Left Volume	-.015	-.019	-.009	.114***	.126***	.139***	.123***	.124***	.144***	.939***	1	-	-	-	-	-	-
12. 4-year Left Volume	-.028	-.005	-.002	.120***	.126***	.152***	.134***	.134***	.148***	.936***	.940***	1	-	-	-	-	-
13. Baseline Right Volume	-.008	-.025**	-.010	.140***	.137***	.164***	.129***	.117***	.146***	.858***	.845***	.851***	1	-	-	-	-
14. 2-year Right Volume	-.013	-.023*	-.001	.145***	.157***	.178***	.134***	.139***	.161***	.841***	.856***	.854***	.954**	1	-	-	-
15. 4-year Right Volume	-.031	-.021	-.005	.161***	.159***	.189***	.150***	.146***	.170***	.847***	.853***	.879***	.945***	.951***	1	-	-
16. Anxiety	.025	.063**	-.010	-.005	.007	-.009	-.001	.003	-.002	-.010	-.012	-.010	.001	-.003	-.007	1	-
17. Depression	-.004	.036*	-.021	-.030*	-.018	-.043*	-.033*	-.019	-.042*	-.022	-.040*	-.038*	-.028	-.032*	-.036	.602***	1
18. Substance Use	-.008	.010	-.002	-.049***	-.055***	-.024	-.049***	-.054***	-.026	.012	-.006	-.022	.017	.004	-.011	.054***	.051***

Note. * Correlation is significant at the 0.05 level (2-tailed). ** Correlation is significant at the 0.01 level (2-tailed). *** Correlation is significant at the 0.001 level (2-tailed).

Table 5

Covariate Coefficients (r, t, F or χ2)

	Hippocampal Morphology		Outcomes		
	Left Density	Right Density	Anxiety	Depression	Substance Use
Child age	¶ -0.030	¶ -0.010	¶ -0.020	¶ -0.004	§ 54.579***
Child sex	‡ 2.373*	‡ 1.863	‡ -4.871***	‡ -2.684**	† 16.541***
Child race/ethnicity					
White	‡ 1.044	‡ 1.887	‡ -6.828***	‡ -3.471***	† 58.707***
Black	‡ 1.522	‡ 1.183	‡ 4.433***	‡ 1.393	† 49.866***
Hispanic	‡ -4.011***	‡ -3.903***	‡ 1.304	‡ 0.271	† 17.291***
Asian	‡ -1.264	‡ 0.622***	‡ -0.928	‡ -0.712	† 0.224
Other	‡ -1.913	‡ -2.450*	‡ -0.639	‡ -0.850	† 4.487
Parent education	§ 1.709	§ 2.156	§ 1.807	§ 0.623	† 68.403***
Household income	§ 4.818**	§ 6.873***	§ 0.295	§ 4.053*	† 46.681***

Note. Sex 0=male, 1=female. Race/ethnicity 0=not endorsed, 1=endorsed. † Cross-tabs with Pearson chi-square tests for two categorical variables. ‡ Independent samples t-test for one categorical (two groups) and one continuous variable. § One-way ANOVA for one categorical (3+ groups) and one continuous variable. ¶ Pearson correlation for two continuous variables. * Significant at $p < 0.05$. ** Significant at $p < 0.01$. *** Significant at $p < 0.001$.

Table 6

Model Fit Statistics Indicative of Good Model Fit

Fit Statistic	Value
$\chi 2$	Small value, non-significant *p* value
RMSEA	≤ .06
SRMR	≤ .08
TLI	≥ .95
CFI	≥ .95
AIC	< competing model
BIC	< competing model

Table 7

Summary of Study Results

Hypothesis	Models Tested	Conclusion
1a: Hippocampal volume and gray matter density will increase over time (i.e., with age) for all adolescents.	1-9	Partially supported; left and right density increases over time (LGCMs 8-9)
1b: All CM subtypes, compared to a non-CM control group, will exhibit less hippocampus development (i.e., less increase over time in hippocampal volume and gray matter density). Additionally, although there is not enough previous research to make slope predictions for each CM subtype, it is hypothesized that each CM subtype will have a distinct slope.	10-11	Not supported
1c: An interaction of time and CM on brain development will exist, such that unique trajectories of hippocampal structural development will emerge for each of the CM subtypes as well as the non-CM control group.	10-11	Not supported
2a: Incidence of CM at baseline will be associated with greater anxiety, depression, and substance use at follow-up waves.	12-17	Partially supported; CM predicts anxiety in left density models (LGCM 12)
2b: Hippocampal morphology will mediate the direct relationship between CM and mental health outcomes, such that less developed hippocampal trajectories will be associated with higher rates of anxiety, depression, and substance use.	12-17	Not supported
3a: Social determinants of health will moderate the association between CM and hippocampal morphology, such that known demographic risk factors (i.e., younger, female, racial/ethnic minority, lower parental educational attainment, and lower household income), will exacerbate the negative impact of CM on hippocampal development.	Analyses not conducted due to lack of support for hypothesis 2b	

Table 8

Model Fit Statistics for Latent Growth Curve Models 1-11 (Aim 1)

Model Fit Statistics	χ^2	*df*	*p*	*RMSEA (90% CI)*	*SRMR*	*TLI*	*CFI*	*AIC*	*BIC*
Unconditional Models									
Volume + density									
1. Whole	275,045.848	73	0.000	0.573 (0.571, 0.575)	24.132	-0.704	0.000	1,026,287.872	1,026,412.774
2. Left	125,138.624	16	0.000	0.826 (0.822, 0.830)	31.425	-1.731	0.000	528,646.650	528,727.468
3. Right	127,564.278	16	0.000	0.834 (0.830, 0.838)	30.999	-1.524	0.000	530,877.016	530,957.835
Volume									
4. Whole	17,108.646	16	0.000	0.305 (0.301, 0.309)	0.089	0.760	0.744	611,260.358	611,341.177
5. Left	8,220.515	1	0.000	0.847 (0.831, 0.862)	1.097	-0.053	0.649	314,395.372	314,454.149
6. Right	13,990.600	1	0.000	1.105 (1.089, 1.120)	0.273	-0.609	0.464	319,330.873	319,389.650
Density									
7. Whole	4,946.763	16	0.000	0.164 (0.160, 0.168)	0.042	0.916	0.910	163,123.215	163,204.033
8. Left	2.027	1	0.155	0.009 (0.000, 0.029)	0.003	1.000	1.000	97,808.315	97,867.093
9. Right	6.770	1	0.009	0.022 (0.009, 0.040)	0.005	0.999	1.000	98,551.149	98,609.926
Conditional Models									
10. Left density	3.121	4	0.538	0.000 (0.000, 0.013)	0.002	1.000	1.000	97,815.204	97,918.064
11. Right density	7.390	4	0.117	0.009 (0.000, 0.018)	0.003	0.999	1.000	98,557.183	98,660.043

Table 9

Variance and Covariances for Unconditional Latent Growth Curve Models 8 and 9

Model Parameters	β	*B*	*SE*	*Z*	*p*
Intercepts					
8. Left density					
Latent intercept (i)	**22.331**	**73.454**	**0.031**	**2336.985**	**0.000**
Latent slope (s)	**0.254**	**0.153**	**0.012**	**12.804**	**0.000**
9. Right density					
Latent intercept (i)	**21.199**	**73.519**	**0.033**	**2195.255**	**0.000**
Latent slope (s)	**0.579**	**0.212**	**0.012**	**18.187**	**0.000**
Variances					
8. Left density					
Latent intercept (i)	**1.000**	**10.820**	**0.184**	**58.914**	**0.000**
Latent slope (s)	**1.000**	**0.364**	**0.061**	**5.963**	**0.000**
9. Right density					
Latent intercept (i)	**1.000**	**12.028**	**0.199**	**60.457**	**0.000**
Latent slope (s)	**1.000**	**0.134**	**0.063**	**2.119**	**0.034**
Covariances (i ~~ s)					
8. Left density	**-0.232**	**-0.461**	**0.090**	**-5.093**	**0.000**
9. Right density	**-0.322**	**-0.408**	**0.094**	**-4.341**	**0.000**

Note. i = intercept, s = slope. **Bold** text indicates statistical significance.

Table 10

Parameter Estimates from Conditional Latent Growth Curve Model using Left-Brain Morphology (LGCM 10)

Model Parameters		β	*B*	*SE*	*Z*	*p*
Intercepts						
Density$_i$		**22.367**	**73.445**	**0.032**	**2297.960**	**0.000**
Density$_s$		**0.271**	**0.157**	**0.012**	**12.673**	**0.000**
Variances						
Density$_i$		**1.000**	**10.813**	**0.184**	**58.904**	**0.000**
Density$_s$		**1.000**	**0.363**	**0.061**	**5.940**	**0.000**
Covariances						
Density$_i$	**Density$_s$**	**-0.222**	**-0.424**	**0.095**	**-4.481**	**0.000**
Regressions						
PA	□ Density$_i$	0.018	0.462	0.265	1.745	0.081
SA		-0.001	-0.022	0.250	-0.089	0.929
PA+SA		0.003	0.176	0.674	0.262	0.794
PA	□ Density$_s$	0.009	0.039	0.101	0.382	0.703
SA		-0.011	-0.049	0.097	-0.506	0.613
PA+SA		-0.008	-0.089	0.255	-0.349	0.727

Note. $_i$ = intercept, $_s$ = slope, PA = physical abuse, SA = sexual abuse, PA+SA = both physical and sexual abuse. **Bold** text indicates statistical significance.

Table 11

Parameter Estimates from Conditional Latent Growth Curve Model using Right-Brain Morphology (LGCM 11)

Model Parameters		β	*B*	*SE*	*Z*	*p*
Intercepts						
Density$_i$		**21.196**	**73.511**	**0.034**	**2158.430**	**0.000**
Density$_s$		**0.577**	**0.211**	**0.012**	**17.807**	**0.000**
Variances						
Density$_i$		**1.000**	**12.022**	**0.199**	**60.451**	**0.000**
Density$_s$		**0.999**	**0.134**	**0.063**	**2.114**	**0.035**
Covariances						
Density$_i$	**Density$_s$**	**-0.322**	**-0.408**	**0.094**	**-4.338**	**0.000**
Regressions						
PA	□ Density$_i$	0.018	0.489	0.282	1.734	0.083
SA		-0.003	-0.087	0.266	-0.329	0.742
PA+SA		0.005	0.335	0.718	0.466	0.641
PA	□ Density$_s$	0.024	0.067	0.098	0.681	0.496
SA		0.009	0.024	0.094	0.258	0.797
PA+SA		-0.032	-0.211	0.248	-0.849	0.396

Note. $_i$ = intercept, $_s$ = slope, PA = physical abuse, SA = sexual abuse, PA+SA = both physical and sexual abuse. **Bold** text indicates statistical significance.

Table 12

Model Fit Statistics for Latent Growth Curve Models 12-17 (Aim 2)

Model Fit Statistics	χ^2	*df*	*p*	*RMSEA (90% CI)*	*SRMR*	*TLI*	*CFI*	*AIC*	*BIC*
Mediation Models									
Left density									
12. □Anxiety	684.269	7	0.000	0.092 (0.086, 0.098)	0.121	0.909	0.965	127,148.115	127,287.710
13. □Depression	38.365	7	0.000	0.020 (0.014, 0.026)	0.011	0.996	0.998	126,886.448	127,026.044
14. □SU	5.949	6	0.429	0.000 (0.000, 0.026)	0.008	1.000	1.000	-	-
Right density									
15. □Anxiety	41.380	7	0.000	0.021 (0.015, 0.027)	0.015	0.996	0.998	127,244.920	127,384.516
16. □Depression	42.717	7	0.000	0.021 (0.015, 0.027)	0.015	0.996	0.998	127,628.139	127,767.734
17. □SU	4.822	6	0.567	0.000 (0.000, 0.023)	0.006	1.001	1.000	-	-

Note. SU = substance use. FIML could not be used for models with an endogenous categorical variable (LGCM 14 and 17); instead, listwise deletion was implemented. Scaled statistics are provided. LGCM 14 *n* = 2,536. LGCM 17 *n* = 2,537.

Table 13

Parameter Estimates from Mediational Models 12-14 using Left-Brain Morphology

Model Parameters			β	*B*	*SE*	*Z*	*p*
Path *a* (Anxiety Outcome)							
12.	PA	□ $Density_i$	0.019	0.468	0.263	1.778	0.075
	SA		-0.001	-0.019	0.248	-0.077	0.939
	PA+SA		0.003	0.181	0.670	0.271	0.787
	PA	□ $Density_s$	0.019	0.044	0.102	0.431	0.667
	SA		-0.025	-0.055	0.098	-0.561	0.575
	PA+SA		-0.015	-0.085	0.256	-0.331	0.740
Path *a* (Depression Outcome)							
13.	PA	□ $Density_i$	0.019	0.478	0.265	1.803	0.071
	SA		-0.001	-0.012	0.250	-0.049	0.961
	PA+SA		0.003	0.152	0.675	0.226	0.821
	PA	□ $Density_s$	0.751	0.026	0.100	0.260	0.795
	SA		-1.942	-0.064	0.096	-0.668	0.504
	PA+SA		-0.333	-0.027	0.252	-0.109	0.914
Path *a* (Substance Use Outcome)							
14.	PA	□ $Density_i$	0.012	0.313	0.797	0.392	0.695
	SA		0.029	0.658	0.589	1.117	0.264
	PA+SA		0.000	-0.013	1.493	-0.009	0.993
	PA	□ $Density_s$	0.047	0.046	0.134	0.342	0.733
	SA		-0.040	-0.035	0.095	-0.369	0.712
	PA+SA		-0.132	-0.248	0.236	-1.050	0.294
Path *b*							
12.	**$Density_i$**	**□ Anxiety**	**0.348**	**0.727**	**0.002**	**291.69**	**0.000**
	$Density_s$		-0.078	-1.711	1.033	-1.656	0.098
13.	$Density_i$	□ Depression	-0.028	-0.054	0.029	-1.848	0.065
	$Density_s$		**0.275**	**370.342**	**31.679**	**11.691**	**0.000**
14.	$Density_i$	□ Substance Use	-0.050	-0.016	0.009	-1.724	0.085
	$Density_s$		0.061	0.497	4.282	0.116	0.908
Path *c'* (Direct Effect)							
12.	**PA**	**□ Anxiety**	**0.034**	**1.741**	**0.806**	**2.160**	**0.031**
	SA		**0.066**	**3.225**	**0.783**	**4.120**	**0.000**
	PA+SA		**-0.057**	**-6.889**	**2.116**	**-3.256**	**0.001**
13.	PA	□ Depression	-0.200	-9.344	37.201	-0.251	0.802
	SA		0.588	26.134	35.423	0.738	0.461
	PA+SA		0.047	5.166	93.352	0.055	0.956
14.	PA	□ Substance Use	-0.055	-0.434	0.394	-1.102	0.271
	SA		0.044	0.314	0.264	1.187	0.235
	PA+SA		0.004	0.067	1.225	0.055	0.956
Path *a-b* (Indirect Effects)							
12.	PA - $Density_i$	□ Anxiety	0.007	0.340	0.191	1.778	0.075

	SA - Density$_i$		0.000	-0.014	0.180	-0.077	0.939
	PA+SA - Density$_i$		0.001	0.132	0.487	0.271	0.787
	PA - Density$_s$		-0.001	-0.075	0.180	-0.417	0.677
	SA - Density$_s$		0.002	0.094	0.176	0.533	0.594
	PA+SA - Density$_s$		0.001	0.145	0.447	0.325	0.745
13.	PA - Density$_i$	□ Depression	-0.001	-0.026	0.020	-1.290	0.197
	SA - Density$_i$		0.000	0.001	0.013	0.049	0.961
	PA+SA - Density$_i$		0.000	-0.008	0.036	-0.224	0.823
	PA - Density$_s$		0.206	9.641	37.187	0.259	0.795
	SA - Density$_s$		-0.534	-23.748	35.408	-0.671	0.502
	PA+SA - Density$_s$		-0.092	-10.123	93.312	-0.108	0.914
14.	PA - Density$_i$	□ Substance Use	-0.001	-0.005	0.013	-0.382	0.702
	SA - Density$_i$		-0.001	-0.010	0.011	-0.936	0.349
	PA+SA - Density$_i$		0.000	0.000	0.023	0.009	0.993
	PA - Density$_s$		0.003	0.023	0.203	0.112	0.911
	SA - Density$_s$		-0.002	-0.017	0.160	-0.109	0.913
	PA+SA - Density$_s$		-0.008	-0.123	1.066	-0.116	0.908
Path *c* (Total Effect)							
12.	Anxiety Outcome		0.053	-1.300	1.784	-0.729	0.466
13.	Depression Outcome		0.015	-2.307	1.691	-1.364	0.173
14.	Substance Use Outcome		-0.016	-0.186	0.474	-0.392	0.695

Note. $_i$ = intercept, $_s$ = slope, PA = physical abuse, SA = sexual abuse, PA+SA = both physical and sexual abuse. **Bold** text indicates statistical significance.

Table 14

Parameter Estimates from Mediational Models 15-17 using Right-Brain Morphology

Model Parameters			β	*B*	*SE*	*Z*	*p*
Path *a* (Anxiety Outcome)							
15.	PA	□ $Density_i$	0.019	0.493	0.280	1.763	0.078
	SA		-0.003	-0.087	0.264	-0.328	0.743
	PA+SA		0.005	0.326	0.712	0.457	0.647
	PA	□ $Density_s$	0.668	0.069	0.099	0.695	0.487
	SA		0.132	0.013	0.095	0.136	0.892
	PA+SA		-0.820	-0.201	0.251	-0.801	0.423
Path *a* (Depression Outcome)							
16.	PA	□ $Density_i$	0.019	0.493	0.280	1.764	0.078
	SA		-0.003	-0.087	0.264	-0.328	0.743
	PA+SA		0.005	0.326	0.712	0.457	0.648
	PA	□ $Density_s$	0.548	0.069	0.099	0.692	0.489
	SA		0.108	0.013	0.095	0.135	0.893
	PA+SA		-0.675	-0.201	0.251	-0.800	0.424
Path *a* (Substance Use Outcome)							
17.	PA	□ $Density_i$	0.018	0.490	0.841	0.582	0.560
	SA		0.027	0.672	0.628	1.070	0.285
	PA+SA		-0.004	-0.224	1.536	-0.146	0.884
	PA	□ $Density_s$	NA	0.011	0.146	0.072	0.942
	SA		NA	0.031	0.101	0.312	0.755
	PA+SA		NA	-0.220	0.244	-0.903	0.366
Path *b*							
15.	$Density_i$	□ Anxiety	-0.001	-0.002	0.026	-0.066	0.947
	$Density_s$		**0.573**	**248.099**	**16.415**	**15.114**	**0.000**
16.	**$Density_i$**	**□ Depression**	**-0.030**	**-0.053**	**0.027**	**-1.975**	**0.048**
	$Density_s$		**0.723**	**269.113**	**17.613**	**15.279**	**0.000**
17.	$Density_i$	□ Substance Use	-0.041	-0.012	0.008	-1.429	0.153
	$Density_s$		NA	0.054	0.135	0.400	0.689
Path *c'* (Direct Effect)							
15.	PA	□ Anxiety	-0.347	-15.551	24.812	-0.627	0.531
	SA		0.006	0.241	23.699	0.010	0.992
	PA+SA		0.413	43.780	62.494	0.701	0.484
16.	PA	□ Depression	-0.390	-18.217	26.909	-0.677	0.498
	SA		-0.024	-1.077	25.707	-0.042	0.967
	PA+SA		0.444	49.053	67.782	0.724	0.469
17.	PA	□ Substance Use	-0.052	-0.411	0.331	-1.239	0.215
	SA		0.041	0.293	0.209	1.399	0.162
	PA+SA		-0.003	-0.047	0.616	-0.076	0.940
Path *a-b* (Indirect Effects)							
15.	PA - $Density_i$	□ Anxiety	0.000	-0.001	0.013	-0.066	0.947

	SA - Density$_i$		0.000	0.000	0.002	0.065	0.948
	PA+SA - Density$_i$		0.000	-0.001	0.008	-0.065	0.948
	PA - Density$_s$		0.383	17.145	24.803	0.691	0.489
	SA - Density$_s$		0.075	3.216	23.690	0.136	0.892
	PA+SA - Density$_s$		-0.470	-49.871	62.469	-0.798	0.425
16.	PA - Density$_i$	□ Depression	-0.001	-0.026	0.020	-1.316	0.188
	SA - Density$_i$		0.000	0.005	0.014	0.324	0.746
	PA+SA - Density$_i$		0.000	-0.017	0.039	-0.445	0.656
	PA - Density$_s$		0.396	18.514	26.899	0.688	0.491
	SA - Density$_s$		0.078	3.461	25.697	0.135	0.893
	PA+SA - Density$_s$		-0.488	-53.989	67.754	-0.797	0.426
17.	PA - Density$_i$	□ Substance Use	-0.001	-0.006	0.011	-0.539	0.590
	SA - Density$_i$		-0.001	-0.008	0.009	-0.854	0.393
	PA+SA - Density$_i$		0.000	0.003	0.018	0.145	0.884
	PA - Density$_s$		Inf	0.001	0.008	0.071	0.943
	SA - Density$_s$		Inf	0.002	0.007	0.238	0.812
	PA+SA - Density$_s$		Inf	-0.012	0.032	-0.369	0.712
Path *c* (Total Effect)							
15.	Anxiety Outcome		0.059	-1.041	1.621	-0.642	0.521
16.	Depression Outcome		0.015	-2.293	1.691	-1.356	0.175
17.	Substance Use Outcome		Inf	-0.186	0.474	-0.392	0.695

Note. $_i$ = intercept, $_s$ = slope, PA = physical abuse, SA = sexual abuse, PA+SA = both physical and sexual abuse. **Bold** text indicates statistical significance.

Figure 1

Conceptual Model of Study Aims

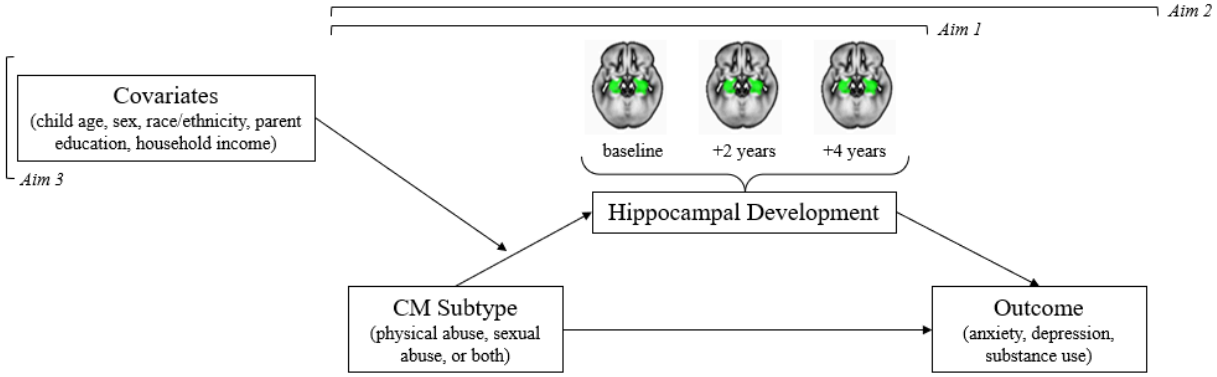


Figure 2

Statistical Model of Unconditional Latent Growth Curve Models (Aim 1, hypothesis 1a)

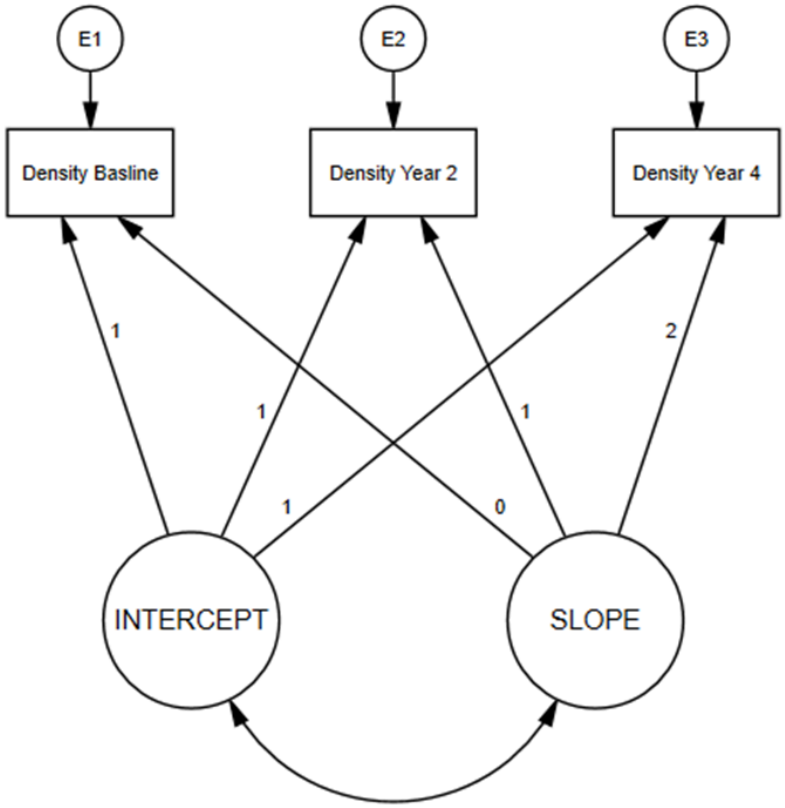


Figure 3

Statistical Model of Conditional Latent Growth Curve Models (Aim 1, hypothesis 1b and 1c)

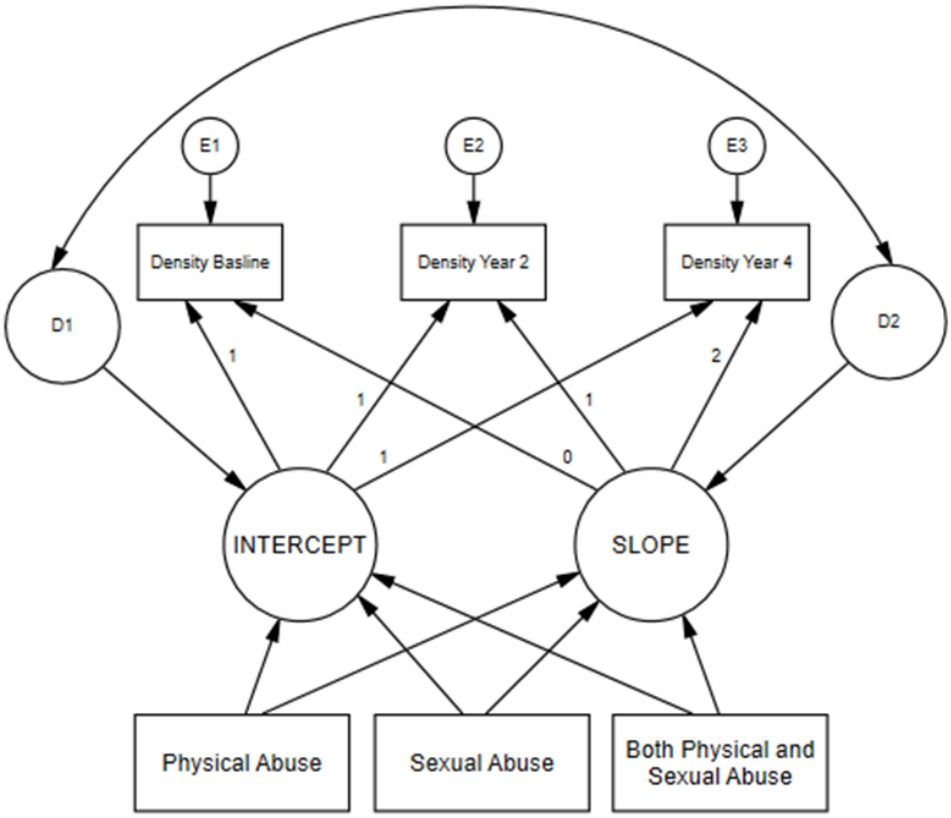


Figure 4

Statistical Model of Mediational Models (Aim 2)

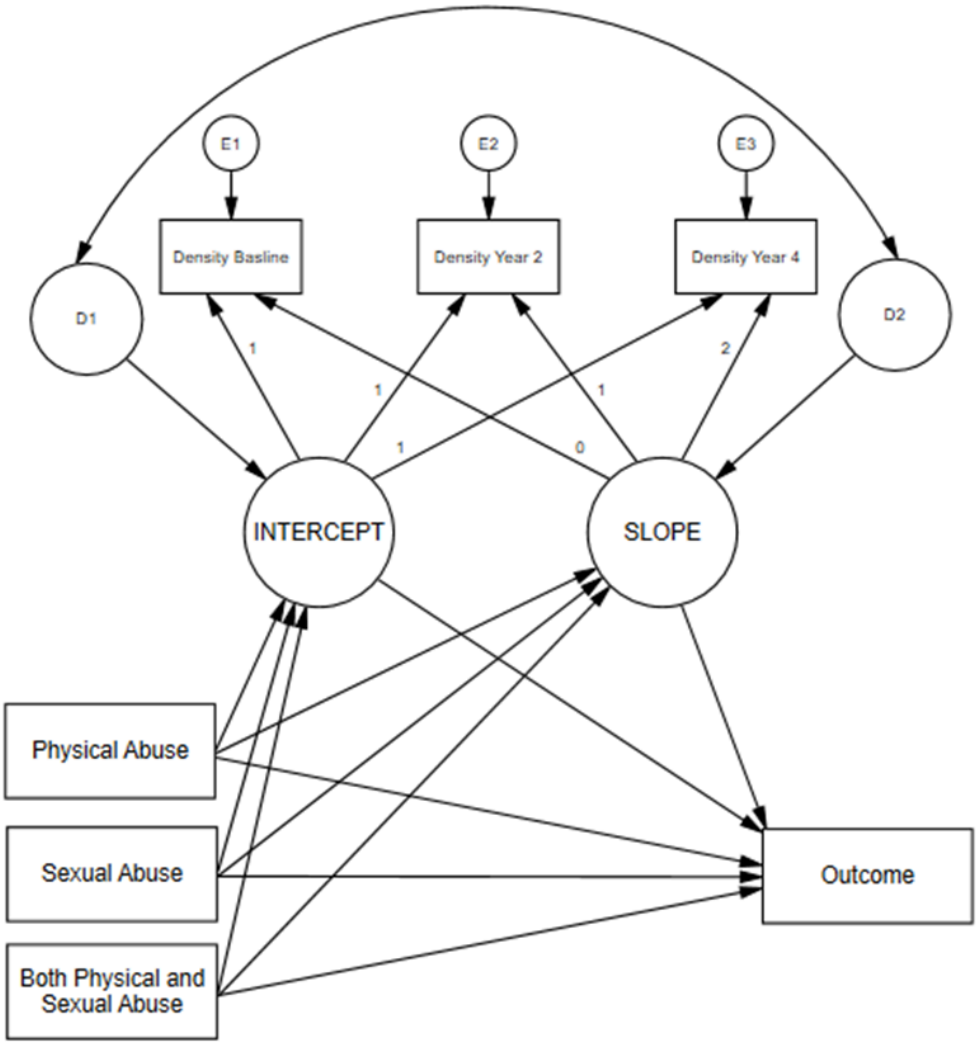


Figure 5

Statistical Model of Moderated Mediational Models (Aim 3)

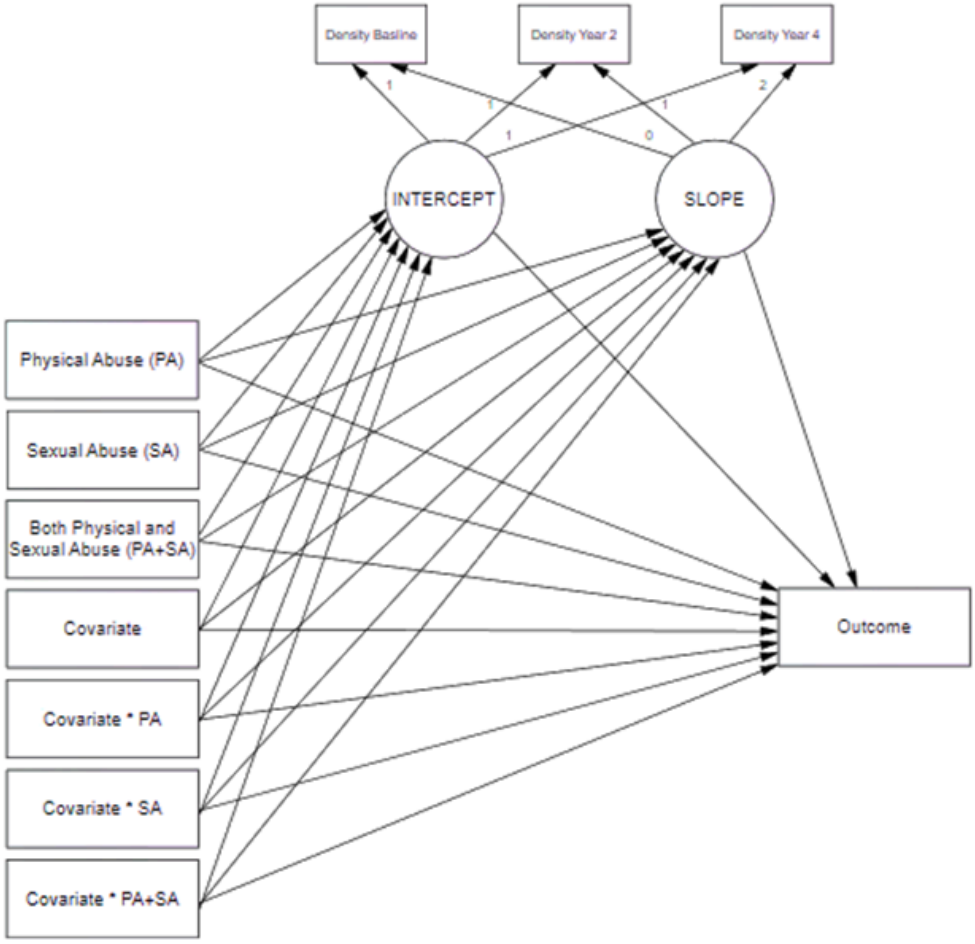
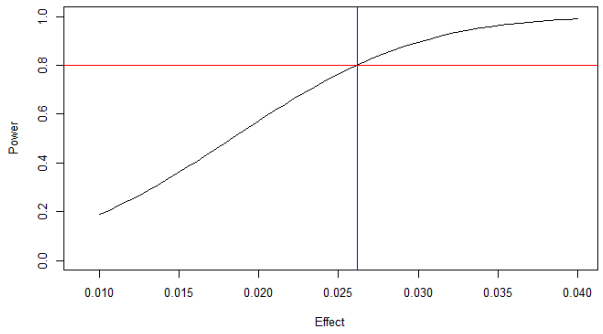


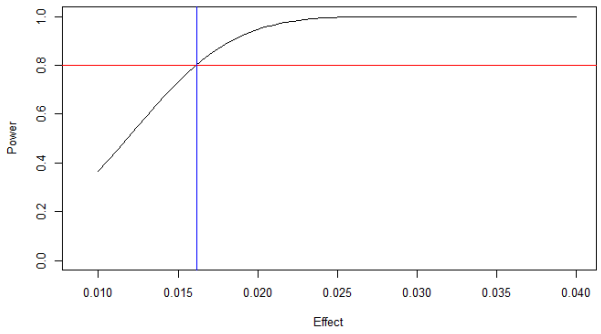
Figure 6

Power to Detect Root Mean Square Error of Approximation (RMSEA) Effects with N = 11,466

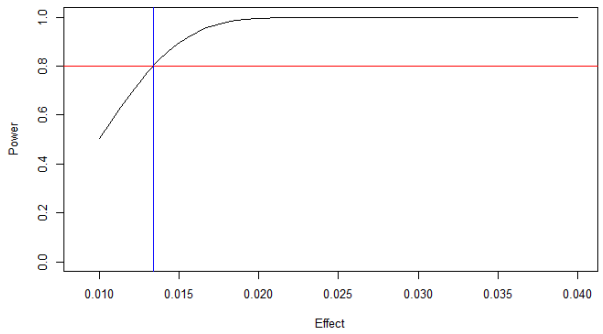
(a) Unconditional Models



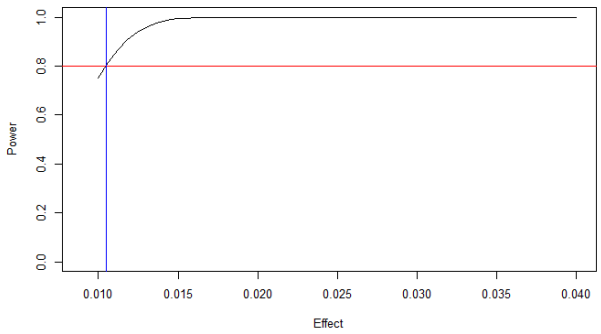
(b) Conditional Models



(c) Mediation Models



(d) Moderated Mediation Models



Appendices

Appendix A

Kiddie Schedule for Affective Disorders and Schizophrenia for DSM-5 (KSADS-5)

Post-Traumatic Stress Disorder (PTSD) Module

(Kaufman et al., 2017)

Relevant items for physical abuse in *italics*. Relevant items for sexual abuse <u>underlined</u>.

Items

1. A car accident in which your child or another person in the car was hurt bad enough to require medical attention
2. Another significant accident for which your child needed specialized and intensive medical treatment
3. Witnessed or caught in a fire that caused significant property damage or personal injury
4. Witnessed or caught in a natural disaster that caused significant property damage or personal injury
5. Witnessed or present during an act of terrorism (e.g., Boston marathon bombing)
6. Witnessed death or mass destruction in a war zone
7. Witnessed someone shot or stabbed in the community
8. *Shot, stabbed, or beaten brutally by a non-family member*
9. *Shot, stabbed, or beaten brutally by a grown up in the home*
10. *Beaten to the point of having bruises by a grown up in the home*
11. *A non-family member threatened to kill your child*
12. *A family member threatened to kill your child*
13. Witness the grownups in the home push, shove or hit one another
14. <u>A grown up in the home touched your child in their privates, had your child touch their privates, or did other sexual things to your child</u>
15. <u>An adult outside your family touched your child in their privates, had your child touch their privates or did other sexual things to your child</u>
16. <u>A peer forced your child to do something sexually</u>
17. Learned about the sudden unexpected death of a loved one

Coding

0 = No; 1 = Yes

Appendix B

Child Behavior Checklist (CBCL)

Anxiety and Depression Syndrome Scales

(Achenbach, 2011; Achenbach & Ruffle, 2000)

Anxiety Items

1. Cries a lot
2. Fears certain animals, situations, or places, other than school
3. Fears going to school
4. Fears they might think or do something bad
5. Feels they have to be perfect
6. Feels or complains that no one loves them
7. Feels worthless or inferior
8. Nervous, highstrung, or tense
9. Too fearful or anxious
10. Feels too guilty
11. Self-conscious or easily embarrassed
12. Talks about killing self
13. Worries

Depression Items

1. There is very little they enjoy
2. Would rather be alone than with others
3. Refuses to talk
4. Secretive, keeps things to self
5. Too shy or timid
6. Underactive, slow moving, or lacks energy
7. Unhappy, sad, or depressed
8. Withdrawn, doesn't get involved with others

Coding

0 = Not True; 1 = Somewhat/Sometimes True; 2 = Very/Often True

Appendix C

Lifetime Use Interview

(Lisdahl & Price, 2012)

Example walkthrough of the Lifetime Use Interview below. The item used in the current study is denoted with *italics*.

Items	**Coding**
Have you heard of ___ ?	0 = No; 1 = Yes
I want to start by asking if you have EVER TRIED any of the following drugs in your life. Have you ever tried_____at any time in your life?	*0 = No; 1 = Yes*
How many total times have you ___ ?	(quantity)
How old were you the first time you had a ___ ?	(years)
Did you continue to ___ after the first ___ ?	0 = No; 1 = Yes
Do you use ___ regularly?	0 = No; 1 = Yes
When did you start using ___ regularly?	0 = No; 1 = Yes

Please see https://nda.nih.gov/data_structure.html?short_name=abcd_ysu02 for a full list of substances assessed. Substances measured in the current study include:

1. A sip of alcohol such as beer, wine or liquor (rum, vodka, gin, whiskey)
2. A full drink of beer, wine or liquor (rum, vodka, gin, whiskey)
3. A puff from a tobacco or electronic cigarette, or vape pens, or e-hookah
4. Tobacco cigarette - more than just a puff
5. Electronic cigarettes, vape pens, or e-hookah - more than just a puff
6. Smokeless tobacco, "chew" or snus
7. Cigars, including traditional cigars, little cigars, or cigarillos
8. Hookah
9. Pipes
10. Nicotine replacement, such as patches, gums, nasal sprays, inhalers, and lozenges
11. A puff or eaten any marijuana, also called pot, grass, weed or ganja
12. Smoked marijuana, also called pot, grass, weed, ganja - more than just a puff
13. Blunts, when you combine tobacco and marijuana in joints
14. Vaped marijuana
15. Marijuana that you eat, such as pot cookies, gummy bears, brownies

16. Marijuana oils or concentrates, such as "710"; hash oil; BHO/butane hash oil/dabs/shatter/budder/honey oil; Co2 oil/vaporizer pen; Rick Simpson Oil/RSO/phoenix tears
17. Marijuana oils or concentrates, such as "710"; hash oil; BHO/butane hash oil/dabs/shatter/budder/honey oil; Co2 oil/vaporizer pen; Rick Simpson Oil/RSO/phoenix tears
18. Marijuana infused alcohol drinks
19. Concentrated marijuana tinctures
20. Fake marijuana or synthetics such as K2 and spice
21. Cannabidiol (CBD)
22. Stimulant drugs such as cocaine, crack cocaine
23. Cathinones such as Bath salts, drone, M-cat, MDVP or meph
24. Methamphetamine, meth, or crystal meth
25. Ecstasy, molly, or MDMA
26. Ketamine or special K
27. The depressant drug GHB, liquid G, or Georgia home boy
28. Heroin, opium, junk, smack, or dope
29. Hallucinogen drugs that cause people to see or experience things that are not real, such as LSD or acid, PCP or angel dust, peyote, mescaline, DMT, AMT, Foxy
30. Hallucinogen drug: magic mushrooms or shrooms
31. Hallucinogen drug: salvia
32. Steroids such as arnolds, pumpers, or roids
33. Bittamugen or byphoditin
34. Liquids, sprays, and gases (this includes substances like poppers, correction fluid, gasoline, glue, shoe polish, spray paints, or nitrous oxide of 'whippits')
35. Stimulant drugs such as amphetamine, Ritalin, Adderall, ephedrine in any way a doctor did not direct you to use them
36. Prescription anxiolytics, tranquilizers, or sedatives in any way a doctor did not direct you to use them, such as Xanax, Ativan, Valium, Rohypnol, or sleeping pills
37. Prescription pain relievers such as Vicodin, Lortab, Norco, Hydrocodone, Oxycontin or Percocet that you used in any way a doctor did not direct you to use them (this does not include over-the-counter, pain relievers such as aspirin, Tylenol, Advil)
38. Over the counter cough or cold medicine or DXM

Appendix D

PATH Intention to Use Scale

(Hyland et al., 2017)

Items

1. Have you ever been curious about using a tobacco product such as cigarettes, e-cigarettes, hookah, or cigars?
2. Have you ever been curious about drinking alcohol?
3. Have you ever been curious about trying marijuana?
4. Do you think you will try a tobacco product soon?
5. Do you think you will try alcohol soon?
6. Do you think you will try marijuana soon?
7. If one of your best friends were to offer you a tobacco product, would you try it?
8. If one of your best friends were to offer you alcohol, would you try it?
9. If one of your best friends were to offer you marijuana, would you try it?

Coding

1 = Definitely yes; 2 = Probably yes; 3 = Probably not; 4 = Definitely not; 5 = Don't know; 6 = Refused to answer

Appendix E

R Code for Main Analyses

```
##############################################################################
##unconditional growth##

unco.full.VolDen <- 'i =~ 1*LdenB + 1*Lden2 + 1*Lden4 + 1*RdenB + 1*Rden2 + 1*Rden4
                     + 1*LvolB + 1*Lvol2 + 1*Lvol4 + 1*RvolB + 1*Rvol2 + 1*Rvol4
                   s =~ 0*LdenB + 1*Lden2 + 2*Lden4 + 0*RdenB + 1*Rden2 + 2*Rden4
                     + 0*LvolB + 1*Lvol2 + 2*Lvol4 + 0*RvolB + 1*Rvol2 + 2*Rvol4'
fit.unco.full.VolDen <- growth(unco.full.VolDen, data=dset, missing = "FIML")
summary(fit.unco.full.VolDen, fit.measures = T, standardized = T)

unco.L.VolDen <- 'i =~ 1*LdenB + 1*Lden2 + 1*Lden4 + 1*LvolB + 1*Lvol2 + 1*Lvol4
                  s =~ 0*LdenB + 1*Lden2 + 2*Lden4 + 0*LvolB + 1*Lvol2 + 2*Lvol4'
fit.unco.L.VolDen <- growth(unco.L.VolDen, data=dset, missing = "FIML")
summary(fit.unco.L.VolDen, fit.measures = T, standardized = T)

unco.R.VolDen <- 'i =~ 1*RdenB + 1*Rden2 + 1*Rden4 + 1*RvolB + 1*Rvol2 + 1*Rvol4
                  s =~ 0*RdenB + 1*Rden2 + 2*Rden4 + 0*RvolB + 1*Rvol2 + 2*Rvol4'
fit.unco.R.VolDen <- growth(unco.R.VolDen, data=dset, missing = "FIML")
summary(fit.unco.R.VolDen, fit.measures = T, standardized = T)

unco.fullVol <- 'i =~ 1*LvolB + 1*Lvol2 + 1*Lvol4 + 1*RvolB + 1*Rvol2 + 1*Rvol4
                 s =~ 0*LvolB + 1*Lvol2 + 2*Lvol4 + 0*RvolB + 1*Rvol2 + 2*Rvol4'
fit.unco.fullVol <- growth(unco.fullVol, data=dset, missing = "FIML")
summary(fit.unco.fullVol, fit.measures = T, standardized = T)

unco.Lvol <- 'i =~ 1*LvolB + 1*Lvol2 + 1*Lvol4
              s =~ 0*LvolB + 1*Lvol2 + 2*Lvol4'
fit.unco.Lvol <- growth(unco.Lvol, data=dset, missing = "FIML")
summary(fit.unco.Lvol, fit.measures = T, standardized = T)

unco.Rvol <- 'i =~ 1*RvolB + 1*Rvol2 + 1*Rvol4
              s =~ 0*RvolB + 1*Rvol2 + 2*Rvol4'
fit.unco.Rvol <- growth(unco.Rvol, data=dset, missing = "FIML")
summary(fit.unco.Rvol, fit.measures = T, standardized = T)

unco.fullDen <- 'i =~ 1*LdenB + 1*Lden2 + 1*Lden4 + 1*RdenB + 1*Rden2 + 1*Rden4
                 s =~ 0*LdenB + 1*Lden2 + 2*Lden4 + 0*RdenB + 1*Rden2 + 2*Rden4'
fit.unco.fullDen <- growth(unco.fullDen, data=dset, missing = "FIML")
summary(fit.unco.fullDen, fit.measures = T, standardized = T)

unco.Lden <- 'i =~ 1*LdenB + 1*Lden2 + 1*Lden4
              s =~ 0*LdenB + 1*Lden2 + 2*Lden4'
```

```
fit.unco.Lden <- growth(unco.Lden, data=dset, missing = "FIML")
summary(fit.unco.Lden, fit.measures = T, standardized = T)

unco.Rden <- 'i =~ 1*RdenB + 1*Rden2 + 1*Rden4
              s =~ 0*RdenB + 1*Rden2 + 2*Rden4'
fit.unco.Rden <- growth(unco.Rden, data=dset, missing = "FIML")
summary(fit.unco.Rden, fit.measures = T, standardized = T)

############################################################################
##conditional growth##

## conditional growth model for L density
co.model.LDen <- 'i =~ 1*LdenB + 1*Lden2 + 1*Lden4
            s =~ 0*LdenB + 1*Lden2 + 2*Lden4
            i ~ PAbinary2 + SAbinary + AbuseBo2
            s ~ PAbinary2 + SAbinary + AbuseBo2'
fit.co.LDen <- growth(co.model.LDen, data=dset, missing = "FIML")
summary(fit.co.LDen, fit.measures = T, standardized = T)

## conditional growth model for R density
co.model.RDen <- 'i =~ 1*RdenB + 1*Rden2 + 1*Rden4
            s =~ 0*RdenB + 1*Rden2 + 2*Rden4
            i ~ PAbinary2 + SAbinary + AbuseBo2
            s ~ PAbinary2 + SAbinary + AbuseBo2'
fit.co.RDen <- growth(co.model.RDen, data=dset, missing = "FIML")
summary(fit.co.RDen, fit.measures = T, standardized = T)

############################################################################
##mediation##

## mediation for L density, anxiety outcome
mediation.model.LDenAnx <- '#latent intercept
                    i =~ 1*LdenB + 1*Lden2 + 1*Lden4
            #latent slope
                s =~ 0*LdenB + 1*Lden2 + 2*Lden4
            #three direct effects
                Anx4t ~ c1*PAbinary2
                Anx4t ~ c2*SAbinary
                Anx4t ~ c3*AbuseBo2
            #six a paths and two b paths
                i ~ a1*PAbinary2
                i ~ a2*SAbinary
                i ~ a3*AbuseBo2
                s ~ a4*PAbinary2
                s ~ a5*SAbinary
                s ~ a6*AbuseBo2
```

```
            Anx4t ~ b1*i
            Anx4t ~ b2*s
        #six indirect effects (a*b)
            a1b1 := a1*b1
            a2b1 := a2*b1
            a3b1 := a3*b1
            a4b2 := a4*b2
            a5b2 := a5*b2
            a6b2 := a6*b2
        #one total effect
            total := c1 +c2 + c3 + a1b1 + a2b1 + a3b1 + a4b2 + a5b2 + a6b2'
fit.med.LDenAnx <- growth(mediation.model.LDenAnx, data=dset, missing = "FIML")
summary(fit.med.LDenAnx, fit.measures = T, standardized = T)

## mediation for L density, depression outcome
mediation.model.LDenDep <- '#latent intercept
                i =~ 1*LdenB + 1*Lden2 + 1*Lden4
        #latent slope
            s =~ 0*LdenB + 1*Lden2 + 2*Lden4
        #three direct effects
            Dep4t ~ c1*PAbinary2
            Dep4t ~ c2*SAbinary
            Dep4t ~ c3*AbuseBo2
        #six a paths and two b paths
            i ~ a1*PAbinary2
            i ~ a2*SAbinary
            i ~ a3*AbuseBo2
            s ~ a4*PAbinary2
            s ~ a5*SAbinary
            s ~ a6*AbuseBo2
            Dep4t ~ b1*i
            Dep4t ~ b2*s
        #six indirect effects (a*b)
            a1b1 := a1*b1
            a2b1 := a2*b1
            a3b1 := a3*b1
            a4b2 := a4*b2
            a5b2 := a5*b2
            a6b2 := a6*b2
        #one total effect
            total := c1 +c2 + c3 + a1b1 + a2b1 + a3b1 + a4b2 + a5b2 + a6b2'
fit.med.LDenDep <- growth(mediation.model.LDenDep, data=dset, missing = "FIML")
summary(fit.med.LDenDep, fit.measures = T, standardized = T)

## mediation for R density, anxiety outcome
mediation.model.RDenAnx <- '#latent intercept
```

```
                i =~ 1*RdenB + 1*Rden2 + 1*Rden4
        #latent slope
            s =~ 0*RdenB + 1*Rden2 + 2*Rden4
        #three direct effects
            Anx4t ~ c1*PAbinary2
            Anx4t ~ c2*SAbinary
            Anx4t ~ c3*AbuseBo2
        #six a paths and two b paths
            i ~ a1*PAbinary2
            i ~ a2*SAbinary
            i ~ a3*AbuseBo2
            s ~ a4*PAbinary2
            s ~ a5*SAbinary
            s ~ a6*AbuseBo2
            Anx4t ~ b1*i
            Anx4t ~ b2*s
        #six indirect effects (a*b)
            a1b1 := a1*b1
            a2b1 := a2*b1
            a3b1 := a3*b1
            a4b2 := a4*b2
            a5b2 := a5*b2
            a6b2 := a6*b2
        #one total effect
            total := c1 +c2 + c3 + a1b1 + a2b1 + a3b1 + a4b2 + a5b2 + a6b2'
fit.med.RDenAnx <- growth(mediation.model.RDenAnx, data=dset, missing = "FIML")
summary(fit.med.RDenAnx, fit.measures = T, standardized = T)

## mediation for R density, depression outcome
mediation.model.RDenDep <- '#latent intercept
                i =~ 1*RdenB + 1*Rden2 + 1*Rden4
        #latent slope
            s =~ 0*RdenB + 1*Rden2 + 2*Rden4
        #three direct effects
            Dep4t ~ c1*PAbinary2
            Dep4t ~ c2*SAbinary
            Dep4t ~ c3*AbuseBo2
        #six a paths and two b paths
            i ~ a1*PAbinary2
            i ~ a2*SAbinary
            i ~ a3*AbuseBo2
            s ~ a4*PAbinary2
            s ~ a5*SAbinary
            s ~ a6*AbuseBo2
            Dep4t ~ b1*i
            Dep4t ~ b2*s
```

```
        #six indirect effects (a*b)
            a1b1 := a1*b1
            a2b1 := a2*b1
            a3b1 := a3*b1
            a4b2 := a4*b2
            a5b2 := a5*b2
            a6b2 := a6*b2
        #one total effect
            total := c1 +c2 + c3 + a1b1 + a2b1 + a3b1 + a4b2 + a5b2 + a6b2'
fit.med.RDenDep <- growth(mediation.model.RDenDep, data=dset, missing = "FIML")
summary(fit.med.RDenDep, fit.measures = T, standardized = T)

##############################################################################
##mediation with categorical ordinal outcome##

# SUfinal is an ENDOGENOUS (dependent) categorical (ordinal) variable. Thus, it is necessary
# to tell lavaan to deal with it as such using the ordered= argument. When the ordered= argument
# is used, lavaan will automatically switch to the WLSMV estimator: it will use diagonally
# weighted least squares (DWLS) to estimate the model parameters, but it will use the full
# weight matrix to compute robust standard errors, and a mean- and variance-adjusted test
# statistic. However, FIML is not currently supported when using the ordered= argument.
# Instead, the default method in lavaan's SEM syntax, listwise deletion, will be used.

## mediation for L density, substance use outcome
mediation.model.LDenSU <- '#latent intercept
                i =~ 1*LdenB + 1*Lden2 + 1*Lden4
        #latent slope
            s =~ 0*LdenB + 1*Lden2 + 2*Lden4
        #three direct effects
            SUfinal ~ c1*PAbinary2
            SUfinal ~ c2*SAbinary
            SUfinal ~ c3*AbuseBo2
        #six a paths and two b paths
            i ~ a1*PAbinary2
            i ~ a2*SAbinary
            i ~ a3*AbuseBo2
            s ~ a4*PAbinary2
            s ~ a5*SAbinary
            s ~ a6*AbuseBo2
            SUfinal ~ b1*i
            SUfinal ~ b2*s
        #six indirect effects (a*b)
            a1b1 := a1*b1
            a2b1 := a2*b1
            a3b1 := a3*b1
            a4b2 := a4*b2
```

```
            a5b2 := a5*b2
            a6b2 := a6*b2
        #one total effect
            total := c1 +c2 + c3 + a1b1 + a2b1 + a3b1 + a4b2 + a5b2 + a6b2'
fit.med.LDenSU <- growth(mediation.model.LDenSU, data=dset, ordered = "SUfinal")
summary(fit.med.LDenSU, fit.measures = T, standardized = T)

## mediation for R density, substance use outcome
mediation.model.RDenSU <- '#latent intercept
                i =~ 1*RdenB + 1*Rden2 + 1*Rden4
        #latent slope
            s =~ 0*RdenB + 1*Rden2 + 2*Rden4
        #three direct effects
            SUfinal ~ c1*PAbinary2
            SUfinal ~ c2*SAbinary
            SUfinal ~ c3*AbuseBo2
        #six a paths and two b paths
            i ~ a1*PAbinary2
            i ~ a2*SAbinary
            i ~ a3*AbuseBo2
            s ~ a4*PAbinary2
            s ~ a5*SAbinary
            s ~ a6*AbuseBo2
            SUfinal ~ b1*i
            SUfinal ~ b2*s
        #six indirect effects (a*b)
            a1b1 := a1*b1
            a2b1 := a2*b1
            a3b1 := a3*b1
            a4b2 := a4*b2
            a5b2 := a5*b2
            a6b2 := a6*b2
        #one total effect
            total := c1 +c2 + c3 + a1b1 + a2b1 + a3b1 + a4b2 + a5b2 + a6b2'
fit.med.RDenSU <- growth(mediation.model.RDenSU, data=dset, ordered = "SUfinal")
summary(fit.med.RDenSU, fit.measures = T, standardized = T)

##############################################################################
##mediation with moderation SAMPLE CODE##

## moderated mediation for L density, Anxiety outcome + covariate age
mediation.model.LdenAnxAge <- '#latent intercept
                i =~ 1*LdenB + 1*Lden2 + 1*Lden4
        #latent slope
            s =~ 0*LdenB + 1*Lden2 + 2*Lden4
        #three direct effects
```

```
            Anx4t ~ c1*PAbinary2
            Anx4t ~ c2*SAbinary
            Anx4t ~ c3*AbuseBo2
        #fourteen a paths and two b paths
            i ~ a1*PAbinary2
            i ~ a2*SAbinary
            i ~ a3*AbuseBo2
            s ~ a4*PAbinary2
            s ~ a5*SAbinary
            s ~ a6*AbuseBo2
            i ~ a7*Age
            i ~ a8*AgePA
            i ~ a9*AgeSA
            i ~ a10*AgeBo
            s ~ a11*Age
            s ~ a12*AgePA
            s ~ a13*AgeSA
            s ~ a14*AgeBo
            Anx4t ~ b1*i
            Anx4t ~ b2*s
        #fourteen indirect effects (a*b)
            PAi := a1*b1
            SAi := a2*b1
            Boi := a3*b1
            PAs := a4*b2
            SAs := a5*b2
            Bos := a6*b2
            Agei := a7*b1
            AgePAi := a8*b1
            AgeSAi := a9*b1
            AgeBoi := a10*b1
            Ages := a11*b2
            AgePAs := a12*b2
            AgeSAs := a13*b2
            AgeBos := a14*b2
        #one total effect
            total := c1 + c2 + c3 + PAi + SAi + Boi + PAs + SAs + Bos
                + Agei + AgePAi + AgeSAi + AgeBoi + Ages + AgePAs
                + AgeSAs + AgeBos'
fit.med.LdenAnxAge <- growth(mediation.model.LdenAnxAge, data=dset, missing = "FIML")
summary(fit.med.LdenAnxAge, fit.measures = T, standardized = T)
```

Vita

Casey Burton Corso earned her Bachelor of Arts in Psychology and Religious Studies from the University of Virginia in 2019 and went on to obtain a Master of Education in Applied Developmental Psychology from the University of Virginia's Curry School of Education in 2020. She received a Master of Science in Psychology from Virginia Commonwealth University in 2022 and is currently a doctoral candidate there. Her research focuses on multi- and inter-generational trends, social-emotional development, and substance use prevention in the context of adverse childhood experiences (ACEs), especially child maltreatment and abuse. Casey's interdisciplinary work reflects her nuanced understanding of how trauma can impact both the individual and family.

Throughout her doctoral studies, Casey has demonstrated her expertise in a variety of teaching and mentorship roles. She has also consistently contributed to the field via conference presentations and scholarly publications. Further, she boasts numerous honors and awards, such as the 2023 American Psychological Association Division 43's family-based student research award. Also in 2023, Casey was awarded the National Partnership to End Interpersonal Violence Across the Lifespan (NPEIV) Founders Award for her research which best exemplified the goals of NPEIV to reduce, eliminate, or prevent violence through research, practice, policy, or advocacy. Casey's scientific rigor and commitment to advancing diversity in health-related research was further recognized in 2024 by the National Institute of Mental Health (NIMH), such that she was awarded a perfect score on her proposal for the prestigious Ruth L. Kirschstein National Research Service Award for Individual Predoctoral Fellowships. This funding (F31MH134546) supported Casey's dissertation research in developmental cognitive neuroscience and child maltreatment.