Exploring the Impact of Childhood Adversity on Adolescent Executive Function: The Role of Pubertal Timing

by

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Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

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Abstract

Adverse childhood experiences (ACEs) have been consistently associated with negative impacts on individual's health and development including, but not limited to, changes in pubertal timing and the development of executive function; however, whether pubertal timing mediates the association between ACEs and executive functioning remains unknown. To address this gap, data was leveraged from a large-scale, nationally representative sample of American adolescents (Adolescent Brain Cognitive Development Study; *N* = 11,878, 52% male, 52.4% White, 13.4% Black, 24.0% Hispanic). Concurrent models assessed the integrity of adolescents' core executive function abilities via their performance on tasks of response inhibition, working memory, and cognitive flexibility (baseline assessment; 9-10 years), whereas prospective models examined adolescents' day-to-day executive functioning in life via parent ratings of their behavior (Time 5 follow-up assessment; 12-13 years). For females, but not males, earlier pubertal timing mediated pathways between greater ACE exposure and executive functions at both time points: at baseline, this was reflected in lower levels of performance on executive function tasks and at follow-up parent endorsement of executive function challenges in everyday living. These findings suggest there may be sex-specific pathways through which early adversity experiences impact subsequent development, with puberty emerging as a particularly important consideration for females vis-à-vis adolescent refinements in their capacity for cognitive self-regulation.
Keywords: executive function, adverse childhood experiences, puberty, adolescence, stress

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Literature Review

Adolescence as a developmental period is marked by profound physical, cognitive, and social change. During this time, early risk factors may exert effects on children's physical and cognitive maturation and shift the trajectory of their future development. This review will address research on the role of adversity in both physical and cognitive development. Additionally, it will address the role of puberty in associations between early adversity and later development. First, this review will discuss Adverse Childhood Experiences and their impacts across development. Second, it will explore the role of executive functions, their maturation across adolescence, and the role adversity plays in the development of these skills. Third, it will highlight puberty as a biological process that may be accelerated by environmental factors. Finally, it will establish linkages between physical and cognitive development as pathways by which adversity confers negative life outcomes.

Adverse Childhood Experiences

Adverse Childhood Experiences (ACEs) are negative experiences that occur early in life, such as abuse, neglect, and household dysfunction (Felitti et al., 1998). Exposure to experiences of childhood adversity are common, with the original ACE study finding reports of at least one ACE in over half of surveyed respondents (Felitti et al., 1998). Recent estimates place the prevalence of adverse experiences higher with only 22.4% of the population having experienced no ACEs (Madigan et al., 2023).

The assessment of ACEs first emerged in the medical field and was centered around predicting health risk with the original study finding associations between adverse events before the age of 18 and increased risk of death and disability, including but not limited to stroke and cardiovascular disease (Felitti et al., 1998). In the original ACE study, ten experiences were considered to be adverse, encompassing experiences of threat, neglect, and household instability such as experiences of physical abuse, food insecurity, and family member incarceration (Felitti et al., 1998). Findings also showed evidence of an ACE dose-response effect, with a greater number of ACEs conferring greater risk (Felitti et al., 1998). Adversity is thought to impact health through allostatic load, or the "wear and tear" on the body (McEwen & Stellar, 1993). Stressors force adaptation in the body, followed by a subsequent return to baseline. This is evidenced in biomarkers such as hormones (i.e. DHEA) and changes in heart rate and blood pressure. Chronic stressors tax the system as the body is forced to adapt repeatedly, leading to the "wear and tear". This wear and tear is further evidenced by associations between ACEs and increased risk of negative health outcomes, as established in multiple reviews and meta-analyses (Kalmakis & Chandler, 2015; Petruccelli et al., 2019).

While the impacts of early life adversity are evident throughout the life course, many of the deleterious effects of ACEs become manifest during adolescence (Amorim et al., 2023; Beal et al., 2019). Adolescence is a developmental transition between childhood and adulthood, typically operationalized as the period between 10 to 19 years (*Adolescent Health*, n.d.). Early adversity has been consistently linked to the development of psychopathology in the teen years with adversity in childhood predicting an earlier onset of depression in adolescence as compared to those without a history of adversity (Oldehinkel et al., 2014). Substance use, once again linked to history of adversity, also becomes prevalent during adolescence with the onset commonly occurring in the teenage years (Sung et al., 2004; Young et al., 2002). Consistent with these findings, a study of Portuguese thirteen-year olds found increased odds of depression and substance use in children whose early experiences where characterized by multiple adversities and household dysfunction (Amorim et al., 2023). Alongside substantive changes in physical, cognitive, and social-emotional development, adolescence is marked by increasing autonomy and independence—which typically results in youth receiving less scaffolding and support from the adults in their lives (Johnson et al., 2011). At the same time, there is a normative shift in social motivation, such that peers begin to take precedence over family (Jager et al., 2015). Additionally, adolescence marks a period of development associated with greater risk-taking behaviors (Romer, 2010). Adolescence thus represents a potential divergence from one's previous life trajectory whilst setting the stage for later life experience (Golsteyn et al., 2014; Raphael, 2013).

As previously stated, adolescence is also associated with the onset of many health concerns including substance use and the development of psychopathology (Cicchetti & Rogosch, 2002; Schulenberg et al., 2014). Given the many changes associated with adolescence as a developmental period, as well as the increased health risk, further understanding of pathways from adversity to negative health outcomes is imperative for improved equity in health and wellbeing (Luby et al., 2017). Links between ACEs and health outcomes occur via biological pathways such as blood pressure and other indicators of allostatic load, however, pathways such as cognitive indices of self-regulation have also been established (Finlay et al., 2022; Trossman et al., 2021, 2022). From this perspective, ACEs increase risk by impacting physiological wear and tear and by increasing the probability that an individual will engage in behaviors detrimental to their health such as substance abuse. Key to this association are executive functions, a set of skills necessary for self-regulation, which influence health risk and are impacted by adversity.

Executive Functions

Executive functions (EF) are a collection of higher-order cognitive abilities necessary for goal-directed behavior (Goldstein & Naglieri, 2014). EF emerges as a general resource during infancy, as reflected in gaze duration, with infants who showed shorter gaze durations being hypothesized to process information more effectively (Cuevas & Bell, 2014). Children who show this more efficient processing subsequently demonstrate better EF skills in early childhood (Cuevas & Bell, 2014). This top-down regulation of attention expands during the preschool years, where EF skills become increasingly differentiated into three separable though inter-related abilities (Carlson & Moses, 2001; Garon et al., 2008). These abilities enable young children to stop impulsive behaviors (inhibitory control; (Miyake & Friedman, 2012), hold important information in mind (working memory, Miyake & Friedman, 2012), and flexibly shift from one task to another (cognitive flexibility; Miyake & Friedman, 2012). Upon this foundation, more complex EF abilities such as planning and organization gradually develop (A. Diamond, 2013; Miyake et al., 2000).

The traditional approach to EF assessment has entailed administering tests of relatively circumscribed skills directly to individuals, with inferences about skill integrity being made based on metrics of test performance (e.g., accuracy or reaction time; Goldstein & Naglieri, 2014). Examples of 'classic' EF tests include the Stroop test of inhibitory control, the Wisconsin Card Sort task for assessment of cognitive flexibility, and digit-span assessments of working memory (Conway et al., 2005; Grant & Berg, 1948; Stroop, 1935). Owing to concerns regarding the real-world relevance of these tests (i.e., lack of ecological validity), questionnaires were subsequently developed as an alternative form of EF assessment to putatively capture a broader suite of skills as applied in day-to-day life (Goldstein & Naglieri, 2014). Commonly used questionnaires solicit input from multiple informants, thereby providing information about EF application across several settings (e.g., self and close-other for adults, parent and teacher for children)—examples of which are the Behavior Rating Inventory of Executive Function (BRIEF) and the Barkley Deficits in Executive Functioning Skills (BDEFS) (Barkley, 2012; Davidson et al., 2016). Research examining associations between task-based and questionnaire assessments of EFs suggests that these measures examine different aspects of self-regulation (Friedman & Banich, 2019; Toplak et al., 2013). Task-based and reported EF assessments are poorly correlated, with differences in performance being thought to reflect the differences between optimal (i.e. standardized tasks) and typical performance (i.e. questionnaire reports) (Toplak et al., 2013). Additionally, questionnaires and tasks measure independent constructs. While measures such as the BRIEF include questions that reflect working memory skills that theoretically parallel those examined using working memory tasks, they also measure aspects of applied EF skills such as the organization of materials which do not have a task equivalent (Toplak et al., 2013). Despite evidence for the unique contribution of both measures, research examining associations between both task-based and reported EF skills is lacking with most studies considering only one method of EF assessment.

Adolescent Executive Functions

Adolescence has been identified as a sensitive period for EF development (Thompson & Steinbeis, 2020). Although the most dramatic improvements in core EF skills occur during early childhood, refinements continue into adolescence (Best & Miller, 2010). For example, working memory capacity and planning abilities continue to improve into young adulthood (Ferguson et al., 2021). This increase in EF abilities may be particularly important in helping adolescents meet increasing demands for independence associated with the transition from childhood to adulthood.

As children transition into adolescence, academic and social demands require greater EF skills (Crone et al., 2017). At the same time as these changing demands, the brain undergoes both structural and functional changes, particularly in the prefrontal cortex, the area that underlies EF abilities.

During adolescence, the brain undergoes massive reorganization—observed most dramatically in pre-frontal cortex, which is a hub in the neural circuity supporting EF (Dumontheil, 2016; Friedman & Robbins, 2022). Reorganization in the adolescent brain is characterized by the formation, strengthening, and elimination of synaptic connections, all of which allow the brain to optimize (i.e. increased efficiency in processing) and mature to adult-like functioning (Spear, 2013). MRI research examining structural changes in the adolescent brain has found increasing white matter tracts as the brain continues to refine connections (Paus, 2005). This myelination has also been linked to puberty, with greater myelination being linked to more advanced pubertal development (Corrigan et al., 2021). Changes in the adolescent brain are also associated with functional changes. In the context of cognitive control, fMRI research has found that older children show diminished activity in areas of the brain that underlie inhibitory skills (Luna et al., 2010). This suggests that as children age there is decreasing effort necessary to sustain these skills (Luna et al., 2010).

Structural and functional changes in the adolescent brain are also shaped by the influx of hormones associated with puberty (Blakemore, 2019). Gonadal hormones influence changes throughout the body, including the prefrontal cortex, potentially serving to signal the onset of synaptic pruning and the refinement of grey matter (Delevich et al., 2021). Much of the work establishing links between pubertal hormones and neural development has been conducted in animal models, though similar patterns have been observed in research with humans (Goddings et al., 2019). For example, in human research, higher levels of certain pubertal hormones (e.g., estradiol and DHEA) have been associated with more advanced physical development along with neural changes that reflect reorganization (e.g., decreases in gray matter density; Brouwer et al., 2015; Nguyen et al., 2013). These linkages suggest that rising pubertal hormone levels may initiate both physical and neurological maturation processes (see also; Blakemore, 2019; Herting et al., 2012).

Given the demands of adolescence and the accompanying physical and neurological changes, well-developed executive functioning skills may be increasingly important. In school, academic and social demands increase as children progress in development, all of which place greater demand on EF skills. Adolescents with better task-based EF skills showed better performance on tests of academic achievement, with specific skills relating to improvement in distinct subjects (e.g., great cognitive flexibility associated with better reading; Latzman et al., 2010). They are also required for successfully managing the increased independence expected high school, when the need for greater planning and organization coincides with less scaffolding from parents and teachers (Best et al., 2011). EF challenges are also linked to social difficulties and decreasing resilience across adolescence (Holmes et al., 2016; Martel et al., 2007). Adolescents with EF challenges show worse social functioning across adolescence, with endorsement of greater difficulty understanding and interpreting social cues which may be particularly detrimental in adolescence as friendships are prioritized and become key to well-being (Alsarrani et al., 2022; Ben-Asher et al., 2023).

EF skills are necessary for successful adaptation to changing demands in adolescence but are also implicated in risk. Adolescents with EF deficits are more likely to engage in impulsive, risky behaviors in laboratory tasks (Reynolds et al., 2019). These risky behaviors are associated, in turn, to substance use and risky sexual behavior (Reynolds et al., 2019). Individuals with EF challenges are more likely to engage in health-risk behaviors that increase illness and morbidity, including substance use (Gray-Burrows et al., 2019; Gustavson et al., 2017). Furthermore, EF deficits are considered to be a transdiagnostic risk factor for internalizing and externalizing psychopathology, such as depression, anxiety, and substance use disorder (Benca et al., 2017). EF skills are not just a risk factor for developing psychopathology but also may influence treatment for such challenges. For instance, lower EF skills have been found to reduce the efficacy of treatment for disorders such as PTSD and depression (Bardeen et al., 2022; Groves et al., 2018).

Adversity and Executive Functions

The development of EF is shaped by experiences early in life and are particularly susceptible to the impacts of adversity (Hostinar et al., 2012; Lund et al., 2022; Nikulina & Widom, 2013; Roos et al., 2016). One salient example is the Bucharest Early Intervention Project, in which children raised in institutional care were compared with children placed in foster care (Zeanah et al., 2017). Research examining the impacts of institutional rearing highlights the profound negative impacts on higher-order cognitive skills including EFs. In the institutional rearing environment, children received limited individualized attention from caregivers, and high staff turnover resulted in a lack of consistent attachment figures. In contrast, children raised in foster care received greater individual attention following placement. Further comparisons have also been drawn in the Bucharest Early Intervention Project between children who were previously institutionalized and then transitioned to foster care and a comparison sample of children who had never been placed in institutional care. When examining EF skills, children who had never been placed in institutional care showed the greatest cognitive skills as compared to children who had been institutionalized (Bos et al., 2009; Nelson et al., 2007). Some improvements in EF were observed in children placed in foster care but only in children who had been placed early in life (Bos et al., 2009; Nelson et al., 2007). These findings highlight the importance of the early rearing environment for cognitive development, as even intervention through foster care was unable to ameliorate the detrimental effects of institutional care.

EF deficits have also been demonstrated in children who have been maltreated, involved with protective services, and reared in poverty—suggesting that the impact of early adversity exposure on EF extends beyond instances of extreme deprivation (Hostinar et al., 2012; Lund et al., 2022; Nikulina & Widom, 2013; Roos et al., 2016). While deficits in EF linked to adversity can be found early in development, differences in cognitive development can magnify over time, as seen with IQ, where discrepancies between high and low SES children increase as children progress from elementary to high school (von Stumm & Plomin, 2015).

Adversities influence on EF abilities is thought to be particularly salient early in life. Insufficient environmental input is thought to negatively impact the developing brain, which requires appropriate stimulation for healthy development (McLaughlin et al., 2014). Animal models find that deficient early environments resulted in decreases in dendrites and brain volume (M. C. Diamond et al., 1966, 1975; Globus et al., 1973). Mirroring animal research, children raised in institutional environments have decreases in grey matter and cortical thickness which result in fewer connections and inefficient processing, hampering EF abilities (McLaughlin et al., 2014; Sheridan et al., 2012).

Less salient indicators of diminished environmental input, such as low socioeconomic status, have been shown to have negative effects on children's EF development (St. John et al., 2019; Vrantsidis et al., 2020). These underdeveloped EF skills are thought to reflect a myriad of factors, including limited language input and lower parental involvement associated with low SES families (McDorman et al., 2025; Romeo et al., 2022). In the context of EF, better language development is associated with greater EF skills and worse language development with weaker EF skills (Kuhn et al., 2014). Children from low-income families have been found to have weaker language processing abilities, as well as a more limited vocabulary (Fernald et al., 2013). This association has been found to be mediated by the parent-child relationship, with low SES caregivers often having less time and emotional resources to engage with young children, thus hampering their developing language skills (Pace et al., 2017). The caregiving environment also influences children's EF development outside of the impacts on language. Low socioeconomic status is also associated with decreased parental responsivity and lower sensitive parenting, which have been linked in turn to weaker EF skills (Bernier et al., 2012; Sarsour et al., 2011). While the environmental risk of low SES is not as severe as the documented impacts of severe deprivation (i.e. Romanian orphanages), the association between SES and EF skills highlight the importance of highly sensitive, enriching early environments in the development of EF skills.

Experiences of adversity that reflect experiences of threat (i.e. experiences of child abuse and sexual assault) are also linked with weaker EF skills (Sheridan & McLaughlin, 2014). The impacts of threat-based adversity has been thought to reflect changes in emotion processing which, in turn, disrupt EF skills (McCrory et al., 2011; Sheridan & McLaughlin, 2014). While experiences of threat and neglect often co-occur, experiences of threat such as childhood abuse are also a consistent predictor of lower EF skills (Kirke-Smith et al., 2012; Mezzacappa et al., 2001). Aversive experiences outside of those associated with extreme abuse show similar associations. In early childhood, harsh parenting behaviors such as those that reflect physical assault and psychological aggression are associated with worse EF skills (Lucassen et al., 2015).

A meta-analysis of the role of parenting behaviors on children's EF abilities finds a consistent, significant, negative association between harsh or negative parenting behaviors such as intrusiveness, negative affect, and hostility on children's EF skills (Valcan et al., 2018).

Ultimately, EF skills are shaped by the early environment. Experiences of both threat and deprivation are salient predictors of children's ability to engage in the higher order cognitive skills that are reflected in executive functioning abilities. These associations are consistent in both extreme and less salient indicators of childhood adversity. The impacts of early adversity extend beyond the brain, also shaping physical development such as pubertal timing.

Pubertal Timing

Puberty reflects the physical and sexual maturation that occurs during adolescence (Dorn & Biro, 2011). Hormonal changes signal the body to begin gonadal maturation, leading to breast development and the onset of menstruation in females, and voice change and facial hair growth in males (Dorn & Biro, 2011). Current estimates suggest that the onset of puberty begins between the ages of 8 and 13 in females and 9 and 14 in males (Breehl & Caban, 2025). While puberty is a process that occurs regardless of sex, most research has focused on these processes in females, with specific emphasis being placed on the age of menarche (age beginning menstruation) (Lacroix et al., 2025).

The average age of pubertal onset has been found to differ over time. Historical records find that for females, the age of menarche has shifted over time (Papadimitriou, 2016). In the 19^{th} and 20^{th} centuries, age of menarche has been observed to decrease from averages around age 15 to age 12, with this change being attributed primarily to better medical care and nutrition (Papadimitriou, 2016). Changes in the timing of menstruation have not been linear throughout history and have differed between cultures with prehistoric female being estimated to have undergone pubertal timing early (between 7 and 13), which was hypothesized to improve chances of reproduction (Papadimitriou, 2016).

Life History Theory has been leveraged to explain historical changes and heterogeneity in current estimates of pubertal timing (Belsky et al., 1991; Stumper et al., 2020). Per this theory, stress serves to signal scarcity to the body resulting in advanced pubertal timing so as to improve chances of passing along genetic information (Belsky et al., 1991). Consistent with this theory, environments historically characterized with increased scarcity recorded earlier ages of menarche (Papadimitriou, 2016). Conversely, records from the 18 and 1900s also emphasize the role of nutritional and health related factors for the onset of puberty, with menarche occurring later in development in environments characterized by health risk (Papadimitriou, 2016).

Pubertal processes are sensitive to environmental factors such as experiences of stress and adversity (Cousminer et al., 2016). Recent research on factors that hasten and stall the onset of puberty emphasizes the role of environmental factors in the timing of adolescents' physical development. Research on pubertal timing in elite athletes emphasizes the importance of sufficient physiological resources for the advent of menarche, with delays in puberty often occurring in female adolescents involved in rigorous physical training and dieting (Georgopoulos et al., 2010; Klentrou, 2006). This pattern is also found post puberty with amenorrhea occurring in athletes as well as females suffering from anorexia nervosa (Baker et al., 2014; Pauli & Berga, 2010). These findings suggest that without appropriate resources, the body is unable to begin or sustain the processes necessary for human reproduction.

At the same time, research has focused heavily on factors that result in advanced pubertal timing. Low socioeconomic status is a commonly examined, established predictor of advanced pubertal timing in Western samples (Deardorff et al., 2007; Sun et al., 2017). This association may be due in part to the role of body weight in pubertal timing, with adolescents with higher body mass index (BMI) undergoing puberty earlier (Gaml-Sørensen et al., 2025; Li et al., 2017). In North America, low socioeconomic status is often associated with higher BMI and decreased access to healthy food (Odoms-Young et al., 2024). These associations differ in developing nations where higher SES is linked to earlier pubertal timing which may reflect better access to resources (Anyanwu et al., 2016). Though associations between SES and BMI are well established in North American samples, links between SES and pubertal timing via BMI are not (Deardorff et al., 2014; Huang et al., 2020; Lee et al., 2000). Work by Deardorff and colleagues found no mediation via BMI in the association between SES and pubertal timing in females, instead highlighting the role of other adversity related stressors (Deardorff et al., 2014).

Adverse life experiences may reflect a key mechanism by which SES relates to advanced pubertal timing. Referring back to Life History Theory, experiences of stress and adversity may signal scarcity in the body, resulting in advanced reproductive maturation (Belsky et al., 1991). In females, adversity has been linked to advanced pubertal timing, with the inverse effect being observed in males (Suglia et al., 2020). Research examining genetic and cellular aging in females finds that females with a history of early adversity showed earlier menstruation as well as evidence of epigenetic aging (Hamlat et al., 2021, 2023). Despite these associations, a meta-analysis found weak evidence for the impact of adversity on pubertal timing, with effects being limited to specific adversities (Zhang et al., 2019). Specifically, associations were found between advanced pubertal timing and experiences of father absence, sexual abuse, and household dysfunction (Zhang et al., 2019).

Pubertal Timing and Executive Functioning

Little is known about the role of pubertal timing in executive functioning development. Current research shows mixed results, with one study showing that earlier pubertal timing was associated with worse executive functioning when pubertal timing was examined as a mediator in the relation between SES and EF (Stumper et al., 2020). In this study, adolescents were recruited from a community sample and assessed at age 13, 14 and 15. Pubertal timing at the first assessment was then examined as a mediator between SES and concurrent and prospective EF and attention abilities as measured by the WISC-IV digit span and TEA-Ch attention tasks respectively. Findings suggest that low SES was linked to more advanced pubertal timing and negatively impacted both EF skills and complex measures of attention. Conversely, research has also found that earlier pubertal timing was associated with more rapid development of attention in adolescence as measured by the Child Behavior Checklist (Chaku & Hoyt, 2019). However, the conclusions of these studies are limited in their examination of EF, as neither study considered the full spectrum of core EF skills, instead examining working memory and reports of attention (Chaku & Hoyt, 2019; Miyake et al., 2000; Stumper et al., 2020). Additionally, these studies examined sex as a moderator in the association between pubertal timing and EF, potentially drawing a false equivalency between pubertal processes in males and females. Specifically, puberty differs between males and females, both in terms of timing and the impacts of maturation (Dorn & Biro, 2011). Furthermore assessments of pubertal timing use different questions to examine pubertal development stage in males and females (i.e. menarche questions only for females; (Petersen et al., 1988)

Despite limited research examining the role of pubertal timing on EF directly, puberty has established impacts on the prefrontal cortex, the area of the brain underlying these higher order cognitive abilities (Brouwer et al., 2015; Nguyen et al., 2013). These associations are particularly prominent in neuronal pruning with more advanced pubertal timing being linked to earlier sculpting of prefrontal cortical areas (Brouwer et al., 2015; Nguyen et al., 2013). Alterations in the timing of puberty may subsequently disrupt the development of the prefrontal cortex, thus impacting the development of these higher order cognitive skills.

Current Study

Early adversity experiences have been consistently linked to both advanced pubertal timing and deficits in executive functioning skills (Colich & McLaughlin, 2022; Johnson et al., 2021). As previously stated, little work has examined associations between pubertal timing and EF skills and existing studies have been limited in their assessment of EF (Chaku & Hoyt, 2019; Stumper et al., 2020). Furthermore, no study has examined pubertal timing as a potential mediator of the adversity-EF association. Additionally, existing work has considered biological sex as a moderator of such associations which may falsely equate puberty between males and females. Further understanding associations between adversity, pubertal timing, and executive functioning skills may elucidate associations between adversity and pubertal timing with the development of psychopathology. Given that both pubertal timing and EF deficits reflect a transdiagnostic risk factor for the development of psychopathology, understanding influences of both adversity and pubertal timing on EF skills may allow for improved understanding of the mechanisms by which adversity yields negative health outcomes (Ho et al., 2024a; Zelazo, 2020).

Ultimately, this thesis seeks to build on existing research by examining pubertal timing as a mediator between adverse childhood experiences and executive functioning. Analyses will examine the role of pubertal timing as a mediator between ACEs and EF skills and will consider

EF skills more thoroughly by examining task-based measures that reflect core EF skills (i.e. inhibitory control, working memory, and cognitive flexibility) and parent reported EF skills. Understanding these associations will provide a foundation that allows for further examination of the mechanisms by which adversity is linked to negative life outcomes, particularly the development of psychopathology. Additionally, this thesis will also examine these associations in both males and females, adding to the limited literature surrounding the impacts of puberty in males.

Introduction

Adverse childhood experiences (ACEs) are stressful life events that occur during development—such as experiences of abuse and/or neglect, witnessing familial violence, or having a parent or caretaker with severe mental illness (Felitti et al., 1998). The original ACE study by Felitti and colleagues (1998) demonstrated that childhood adversities are common and harmful: compared with those who endorsed low levels of early adversity exposure, individuals with 4 or more ACEs were significantly more likely to engage in health-risk behaviors (e.g., smoking) and suffer from chronic illnesses (e.g., heart disease) that hasten mortality. In the interim period of nearly 30 years, systematic reviews and meta-analyses have further illuminated the negative and longstanding consequences of ACE exposure for multiple facets of adult function, including psychological adjustment (Abate et al., 2024). With associations now well-established, an important direction for ACE research is identifying mechanisms that account for the deleterious influence of early adversity experiences on prevalent, impairing, and socially burdensome adult outcomes like mental illness (Gladieux et al., 2023).

The developmental sequela of ACEs are evident before youth reach adulthood. In neurocognitive development, for example, some of the largest effects linked to early adversity exposure have been documented in prefrontal brain regions that support executive functions (Cooke et al., 2023; Friedman & Robbins, 2022; Sheridan & McLaughlin, 2014). Executive functions (EFs) are self-regulatory cognitive abilities used to work toward and attain goals, examples of which include keeping goal-relevant information in mind, stopping prepotent but goal-irrelevant behaviors, and flexibly shifting between mindsets and/or activities in the service of goal attainment (Miyake et al., 2000). EFs are also consistently linked with academic success and social skills while also serving as a protective factor against the development of psychopathology (Holmes et al., 2016; Latzman et al., 2010; Lynch et al., 2021). Given the importance of EF skills on overall well-being, it is essential that researchers understand how these skills develop. EF skills emerge early in childhood and improve rapidly, particularly between the ages of 2 and 5 (Carlson & Moses, 2001; Garon et al., 2008). Research on EF development has focused primarily on the preschool years despite the fact that these skills continue to mature beyond the early and middle childhood years (Best & Miller, 2010; Carlson, 2005; Garon et al., 2008). Adolescence as a developmental period has been identified as a sensitive period for the development of EF skills but remains understudied (Thompson & Steinbeis, 2020). Understanding EF skills in adolescence is imperative, not only to understand the full scope of development for these important life skills, but also due to the nature of adolescence, a time period of increased demands and emerging substance use, risky behaviors, and mental illness; all common associations of poor EF skills (Blakemore, 2019; Feldstein & and Miller, 2006; Paus et al., 2008; Powers & and Casey, 2015).

Experiences of early adversity have well documented negative impacts on the development of EF, as demonstrated on tasks of relatively circumscribed EF abilities and on rating scales of broader EF behaviours in everyday life (D. Johnson et al., 2021; Lund et al., 2020, 2022). Both extreme forms of deprivation as seen in studies of Romanian orphans growing up in crowded, understaffed conditions as well less salient forms of adversity such as lower socioeconomic status have been found to negatively impact EF skills (Bos et al., 2009; Roos et al., 2016; Sheridan et al., 2012).

Early adversity exposure also influences physical maturation, such as the age at which youth enter puberty with greater adversity being associated with more advanced pubertal development at earlier ages (Hamlat et al., 2022; Ho et al., 2024a; Suglia et al., 2020). Puberty is triggered by sexually dimorphic hormones that drive the development of secondary sex characteristics, with onset typically occurring earlier in females than males (Dorn & Biro, 2011). Influences on pubertal timing and tempo (more advanced pubertal timing being linked to protracted development) include common and sex-specific genetic effects, epigenetic mechanisms, and environmental factors (Cousminer et al., 2016). Regarding the latter, pubertal timing has been identified as a physiological marker of stress-induced "wear and tear" on the body that is evident by adolescence (Whelan et al., 2021). Evidence across studies hints that early adversity experiences hasten the onset of puberty in females, which in turn increases their risk of self-regulatory challenges and coincident mental health concerns in adolescence (Ho et al., 2024a; Stumper et al., 2020). These effects remain unclear in males (Suglia et al., 2020).

Pubertal timing itself also influences neural development with the influx of sex hormones signaling the onset of maturational changes in the brain (Blakemore, 2019). More advanced pubertal development has been linked with greater neural pruning and myelination which reflects a more advanced stage of neural development necessary for mature information processing (Corrigan et al., 2021; Luna et al., 2010). These changes are particularly evident in the prefrontal-cortex which as previously stated, underlies EF abilities (Delevich et al., 2021). Limited research has examined associations between pubertal timing and EF skills with existing studies showing conflicting results and limited methods of EF assessment (Chaku & Hoyt, 2019; Stumper et al., 2020). Given the impacts of pubertal timing on neurological structures underlying EF abilities, understanding how changes in the timing of physical development may yield changes in EF is essential. To our knowledge, no studies have examined whether the association of ACEs and EF is mediated by pubertal timing and, if so, whether patterns differ based on sex.

Furthermore, given that the impacts of adversity on pubertal timing appear to be most prominent in females, understanding sex differences in potential associations is necessary.

Current Study

To begin addressing this knowledge gap, we present an analysis of large-scale longitudinal data collected and maintained by the Adolescent Brain Cognitive Development (ABCD) Study. There are several reasons why we focused our investigation on this transitional stage of development. First, during adolescence there is a cultural expectation that youth will begin using their EF abilities more autonomously rather than relying primarily on parents and teachers (e.g., Soenens et al., 2017). Second, although EF abilities approach a developmental plateau during the teen years, individual differences in maturation rates mean that some adolescents reach adult-levels of EF at younger or older ages than others (Best et al., 2009). Lastly, refinements in adolescent EF are supported by increasingly specialized brain networks in which prefrontal regions play a particularly influential role (Fiske & Holmboe, 2019; Friedman & Robbins, 2022). This adolescent 'sculpting' of prefrontal brain areas is hypothesized to be influenced by gonadal hormones that become elevated with the onset of puberty (i.e., estrogen, progesterone, testosterone; Delevich et al., 2021). We hypothesized that greater ACE exposure would be associated with worse EF and that these associations would be mediated by earlier onset of puberty. We further hypothesized that early pubertal timing would have a more deleterious impact on the EFs of adolescent females compared with their male counterparts.

Methods

Participants and Procedure

Data were drawn from the large-scale longitudinal study of Adolescent Brain and Cognitive Development (ABCD). Participants were recruited across 21 research sites in the United States. 11,878 youth ages 9 to 10 years were recruited via probability sampling of U.S. elementary schools to approximate the racial and ethnic composition of youth in the U.S. population (52.4% White, 13.4% Black, 24.0% Hispanic; Garavan et al., 2018). Our study included 11,865 participants (Male = 6188, Female = 5677) who had valid data on at least one measure that was the focus of our investigation at baseline and the three-year follow-up (2016-2021; data release 5.1). Because we analyzed male and female data separately, our sample did not include participants identified as intersex at birth ($n = 3$). Children attended follow-up visits annually with additional phone call check-ins every six months (see Garavan et al., 2018 for more details). This study leveraged data from the baseline assessment (age 9-10, mean age 9.96 years) as well as follow-ups 1-3 with children aged 10-11 at follow-up 1 (mean age 10.96 years), 11-12 at follow-up 2 (mean age 12.07 years), and 12-13 at follow-up 3 (mean age 12.96). Cross-sectional analyses of task-based EF leveraged data from the baseline assessment. Longitudinal analyses utilized ACE scores from the baseline assessment to predict parent-reported EF challenges follow-up 3. Parent reports of youth's pubertal status from the baseline assessment through to follow-up 3 were used to characterize the intercept (timing) and slope (tempo) of pubertal timing for the follow-up analysis.

Ethics approval for our study was received by the Human Research Ethics Board at the University of Waterloo. The ABCD study was originally reviewed by a centralized institutional review board at the University of California, San Diego and all study sites received approval from their own institutional review boards. Study participants provided written assent and caregivers provided written informed consent. Parents and children completed an extensive test battery including questionnaire, biomarker, and neuroimaging assessment. Additional procedural information about the ABCD study is described by Garavan et al. (2018).

Measures

Family Income

Parents reported on total combined gross annual household income on a scale of 1-10 (1 = < $5,000, 2 = $5,000–$12,000, 3 = $12,000–$16,000, 4 = $16,000–$25,000, 5 = $25,000–$35,000, 6 = $35,000–$50,000, 7 = $50,000–$75,000, 8 = $75,000–$100,000, 9 = $100,000–$200,000, and 10 = > $200,000). Participants' median household income was between $75,000–$100,000.

Adverse Childhood Experiences

Information available in the ABCD dataset covers 9 of 10 domains listed in the original ACE study (Felitti et al., 1998), including physical abuse, sexual abuse, household violence, parental alcohol abuse, parental mental health issues, parent marital status (divorced/separated), emotional neglect, physical neglect, and household criminal justice system involvement (Appendix A). Emotional abuse is one domain that is not covered in the ABCD dataset. Following the example of Nagata et al., 2023, ACE scores were calculated by summing child and parent responses to questions asked at baseline, coded in a binary manner as present (1) or absent (0), and then summed to reflect scores of 0, 1, 2, 3, and 4 or more (see also Raney et al., 2022; Testa et al., 2022). ACEs were coded as present if either parent or child endorsed the experience.

Executive Functions at Baseline

Three subtests from the NIH Toolbox assessed core EF abilities at baseline. All tasks were administered on an iPad. NIH Toolbox EF tasks were validated for developmental sensitivity, reliability, and convergent validity for individuals age 3-85 years old (Tulsky et al., 2014; Zelazo et al., 2013).

In the List Sorting Task (working memory), youth were presented with a series of stimuli that appeared one at a time. Stimuli were pictures of familiar items that were also read aloud (e.g., youth saw a picture of an elephant and heard the word ‘elephant’). After the pictures and labels were presented, the youth had to recall the animals they had seen in order from smallest to largest. Sequences began with 2 items and increased in length by 1 until youth responded incorrectly to two trials at a given length. Total correct responses were recorded (max = 28). This task has been assessed for both convergent and discriminant validity and shows significant correlations with existing common working memory tasks (i.e. Letter-Number Sequencing) but not tasks of receptive vocabulary (Tulsky et al., 2014).

For the Flanker Task (response inhibition), youth were presented with a centrally arranged row of five fish and were instructed to make a keypress response based on the direction in which the middle fish was ‘swimming’ (e.g., left arrow for leftward facing fish). Later trials involved arrow stimuli in lieu of fish. On congruent trials, all fish/arrows pointed in the same direction. On incongruent trials, the central fish/arrow pointed in the opposite direction as the flanking fish/arrows. After practice with feedback, youth completed 20 test trials with fish stimuli and, if at least 90% accurate, completed 20 additional test trials with arrows. Congruent and incongruent trials were randomly intermixed. Total correct responses were recorded (max = 40). Age-corrected standard scores were calculated using both accuracy and reaction time.

On the Dimensional Change Card Sort Task (cognitive flexibility), youth were instructed to match bivalent pictures to a target based on a verbal prompt to sort the pictures by either color or shape. For example, if shown an image of a red rabbit, red boat, and white rabbit and asked to match by color they would select the red rabbit and red boat. If asked to match by shape they would select the red and white rabbits. Following practice with feedback, youth completed 5 test trials sorting by shape, 5 test trails sorting by color, and then 30 test trials in which shape and color sorting were intermixed. Total correct responses were recorded (max = 40).

Parent Reported Executive Dysfunction at Follow-up

Because task performance was not available at the three-year follow-up, we assessed youths' day-to-day EF challenges as assessed via parent report on the Barkley Deficits in Executive Function Scale-Short Form (BDEFS; Barkley, 2012). The BDEFS assesses multiple areas of EF challenge manifested in youths' behaviors in everyday life, including problems with time management, organization, problem solving, self-restraint, motivation, and regulation of emotion. The scale consists of 14 items rated on a 4-point scale ranging from 1 (*Never or rarely*) to 4 (*Very often*). Responses were summed into a total score, with higher values reflecting more EF challenge (max = 56, α = .98).

Pubertal Timing

Parents reported on their children's pubertal development at baseline (ages 9-10) and at follow-ups 1-3 using the Pubertal Development Scale (PDS; Petersen et al., 1988). This scale was designed to assess pubertal development in cis-gender adolescents. Parent report was chosen over adolescent report because it is more reliable in early pubertal development (Petersen et al., 1988). The PDS includes 3 items rated on a 4-point scale ranging from 1 (*has not yet begun*) to 4 (*seems complete*) that assesses general pubertal changes (e.g., growth, skin changes, and hair changes) as well as changes that are more specific to females (breast development and menarche) or males (voice changes and growth of facial hair). Scores were aggregated (3 = prepuberty, 4-5 = early puberty, 6-8 = mid puberty, 9-11= late puberty, 12 = post puberty) to create a timing category (1 = prepuberty, 2 = early puberty, 3 = mid puberty, 4 = late puberty, 5 = post puberty; Petersen et al., 1988).

Analytic Approach

Measures of adversity, pubertal timing, EF, and demographic data were initially inspected for missingness (% missing: age = .09%, sex = .03%, family income = 8.57%, inhibitory control = 1.29%, working memory = 1.65%, cognitive flexibility = 1.28%, pubertal timing = 3.95%, reported EF challenges = 15.92%). Missing data were handled using Full Information Maximum Likelihood (FIML) in analyses with Mplus (see below). Within male and female groups, data were approximately normally distributed and no univariate or multivariate outliers were identified. We conducted separate sex-based analyses rather than treating sex as a moderator, given evidence that pubertal timing is a qualitatively different construct for females and males and hence should be examined in these groups separately (Marceau et al., 2011).

Next, baseline EF was modeled as a latent factor using age-corrected accuracy scores from the 3 EF tasks. Latent variable approaches to the assessment of EF skills is recommended to improve reliability and limit task-impurity (Friedman & Banich, 2019). As the model was just-identified, we examined the significance of individual factor loadings to adjudicate whether to include a latent factor of EF in our analyses. The decision to use latent factor loadings was supported by significant standardized factor loadings for inhibitory control ($\lambda = 0.64$, $p < .001$), working memory ($\lambda = 0.39$, $p < .001$), and cognitive flexibility ($\lambda = 0.68$, $p < .001$).

We then created two mediation models. Our first mediation model focused on cross sectional baseline data, when youth were between 9 and 10 years of age. This model examined the direct path of ACEs to youths' EF task performance with an indirect path of pubertal timing. Baseline age and family income were treated as covariates of ACEs and pubertal timing and family income were treated as a covariate of youths' EF. Our second model focused on follow-up data, when youth were between 12 and 13 years of age. This model examined the direct path of baseline ACEs to parent ratings of youths' parent reported EF challenges at follow-up, with an indirect path consisting of pubertal intercept (pubertal level at baseline) and pubertal slope (rate of pubertal change between baseline and follow-up, including assessment of puberty during intermediate years). Analyses were conducted in Mplus (Version 8.11) using 10,000 Bootstrapped confidence intervals. Descriptive statistics and bivariate correlations were calculated using R version 4.1.2.

Results

Table 1 presents descriptive statistics for participants. Bivariate correlations amongst key study variables are presented in Table 2.

Cross Sectional Models

Cross sectional mediation models are presented in Table 3. For females, adverse childhood experiences were not directly associated with EF task performance. However, greater ACE exposure significantly predicted earlier pubertal onset (β = .04, p = .003, 95% *CI* = .014, .051) which in turn was significantly associated with worse EF task performance (β = -.050, p = .004, 95% *CI* = -.745, -.203). Further, pubertal timing significantly mediated the relation between ACEs and EF (β = -.002, p = 0.045, 95% *CI* = -.004, -.001). For males, neither the direct effect of ACEs on pubertal timing nor the direct effect of ACEs on EF task performance were significant. Like females, however, earlier pubertal timing was significantly associated with worse EF task performance (β = -.067, p < .001, 95% *CI* = -.096, - .037). There was no evidence of statistical mediation in the male model.

Unconditional Latent Growth Model

As was expected, pubertal timing varied across youth and increased over time for both females (intercept: estimate = 0.65, p < .001; slope: estimate = 0.05, p < .001) and males (intercept: estimate = 0.25, p < .001; slope: estimate = 0.07, p < .001).

Longitudinal Models

Follow-up models are presented in Table 4**.** For females, there were significant direct effects of adverse childhood experiences on pubertal intercept (β = .05, p = .001, 95% *CI* = .028, .077) and pubertal slope (β = -0.05, p = 0.007, 95% *CI* = -.082, -.020) showing that female youth with more ACEs entered puberty at earlier ages and showed more protracted pubertal development. There also were significant direct effects of ACEs and pubertal intercept on parent reported executive dysfunction such that a greater number of ACEs and more advanced pubertal development at baseline (intercept) were associated with greater parent reported executive dysfunction (ACEs: $\beta = .20$, $p < .001$, 95% $CI = .171, .223, 0.223$; pubertal intercept: $\beta = .06$, $p = .004$, 95% $CI = .025, .094$). The association between ACEs and parent EF ratings was significantly mediated by pubertal intercept ($\beta = .00$, $p = .030$, 95% $CI = .001, .006$), but not slope ($\beta = -.00$, $p = .243$, 95% $CI = -.005, .000$) showing that ACEs were associated with more advanced pubertal development at baseline (intercept) and subsequent greater parent reported EF challenges for females. For males, there was a significant direct effect of ACEs on parent reported EF challenges ($\beta = .21$, $p < .001$, 95% $CI = .187, .236$). However, no other direct effects were significant nor was there an indication of statistical mediation.

Discussion

This study aimed to examine the association between childhood adversity exposure and EF and reported EF challenges via pubertal timing. Consistent with expectations, our pattern of results varied for females and males. In females, pubertal onset mediated concurrent and prospective associations between ACEs and EFs such that female youth with a greater number of adverse experiences showed worse EF skills at baseline and greater reported EF challenges follow-up, with this association linked to earlier pubertal onset. This finding was observed when modeling core executive abilities using task performance at ages 9 to 10 and when using parent report of broad executive dysfunction at ages 12 to 13 suggesting that the influence of adversity via pubertal timing impacts core functional EF skills measured in optimal conditions as well as the application of these skills in day-to-day life. In males, an association between ACEs and EFs was discernable only at follow-up assessments of EF challenges in daily life and was not mediated by puberty. Whilst the experience of childhood adversity has potential to disrupt myriad facets of development irrespective of one's sex (e.g., Künzi et al., 2024), our findings suggest that ACE exposure has a particularly deleterious effect on the EF development of females by hastening the age at which they enter puberty.

It has been theorized that stressful rearing environments may be interpreted as indicators of scarcity and instability by the body, resulting in earlier pubertal maturation to increase the likelihood of reproductive success (Belsky et al., 1991). Advanced pubertal timing has also been conceptualized as indicative of greater allostatic load which reflects chronic "wear and tear" on the body associated with repeated adaptation to stressors (Joos et al., 2018; McEwen & Stellar, 1993). Consistent with this theory, two recent investigations have shown that early adversity experiences are linked to earlier pubertal timing and accelerated epigenetic age—with the caveat that these investigations were restricted to females (Hamlat et al., 2021, 2023). Somewhat more tempered conclusions have emerged from meta-analytic reviews of studies with female participants. For example, Zhang et al., (2019) found no significant association between total ACE scores and pubertal timing, though small to medium effects emerged when specific adversities were examined (e.g., sexual abuse). More recently, Ding & Lei, (2024) reported an overall moderate effect of total ACEs on a broad range of female reproductive events but cautioned against over-interpretation of the result given considerable heterogeneity across studies. Fewer investigations have included males and findings have been mixed, with evidence of precocious, delayed, and no change in pubertal maturation following early adversity experiences (Gur et al., 2019; Ho et al., 2024b; Suglia et al., 2020).

Although males had significantly more ACEs than females, ACE exposure only predicted EF at follow-up (not baseline) and this effect was not mediated by pubertal timing. For males, the impact of adversity on pubertal timing may emerge later than in females because males typically complete pubertal development later in adolescence (Brix et al., 2019). This difference in pubertal timing is reflected in our sample. At baseline, most males (74%) were rated at a prepubescent level whereas most females (47%) were rated a mid-pubertal level. By the third follow-up, males were on average early or mid-puberty whereas females were on average in mid to late puberty. Given that puberty is completed later for males (Brix et al., 2019), it is possible that the impact of adversity of ACEs on EF via pubertal timing is also protracted. Accordingly, research will need to extend age ranges beyond the early adolescent years to find evidence of this pattern in males, should it exist.

Very little research has examined the implications of pubertal development for EF. In our study, adolescents who entered puberty at younger ages had lower scores on a latent construct of

EF that was derived from their baseline performance on well-validated tasks assessing 'core' executive abilities (i.e., working memory, response inhibition, and mental flexibility; Gershon et al., 2013). Similarly, a longitudinal investigation of 639 adolescents ages 12 to 13 years found that earlier pubertal onset was associated with worse performance on tasks of working memory and attention—though only the latter effect remained significant when youth were reassessed one year later (Stumper et al., 2020). This null result may reflect transitory effects of puberty on attention. Puberty's role in EF may be attributed to changes in brain structure that coincide with the advent of puberty, particularly the restructuring of white matter tracks that have been linked to EF abilities (Cristofori et al., 2015; Herting et al., 2012; Ladouceur et al., 2012; Pfeifer & Allen, 2021). Additionally, advanced pubertal timing has been conceptualized as indicative of allostatic load, or the biological embedding of adversity, which connects back to existing research on the role of adversity in EF development (Hostinar et al., 2012; Joos et al., 2018; Roos et al., 2016). In this context, pubertal timing may be a symptom of the impact of adversity on development. Finally, advanced pubertal timing may reflect a psychosocial stressor, particularly for females (Blumenthal et al., 2009; Conley & Rudolph, 2009; Deardorff et al., 2007; Sontag et al., 2011).

At follow-up, we found that the association between pubertal timing and EF remained significant only for females. Specifically, females who entered puberty at earlier ages were subsequently rated by their parents as showing more evidence of executive dysfunction in their everyday lives. In contrast to our result, a longitudinal investigation of 1099 adolescents ages 9 to 15 found that earlier onset of puberty predicted greater improvement in parent ratings of attention problems for both females and males over time (Chaku & Hoyt, 2019). Importantly, however, attention problems were taken from the Child Behavior Checklist and reflect behavioral symptoms of ADHD (i.e., inattention, hyperactivity, impulsivity)—which is often correlated but not synonymous with challenges in EF (McAuley et al., 2010). By examining the influence of adversity and pubertal timing on both parent-reported EF challenges and task assessed EF skills, we are able to consider the impact of these factors on a more comprehensive assessment of self-regulatory skills (Friedman & Banich, 2019; Toplak et al., 2013).

Limitations and Future Directions

Our results were based on longitudinal data from a large sample of nationally representative youth, which enables us to report upon the temporal unfolding of events following ACE exposure in a manner that may be more likely to generalize to the broader population of US adolescents (vs. smaller clinical or high-risk samples). Though the size of these effects is small, they may shed light on unique sex specific pathways by which adversity impacts individuals. Despite a similarly large sample size as well as significantly greater ACE endorsement (see Table 2), no association between ACEs, pubertal timing, and EF skills or day-to-day EF challenges were found in male youth. Furthermore, research has suggested that the influence of adversity on pubertal timing may be limited to experiences of specific adversities (Zhang et al., 2019). As ACEs reflect a relatively superficial assessment of adversity experiences, more nuanced analysis of adversity type may be necessary to fully examine the impacts of adversity on EF via pubertal timing (Holden et al., 2020).

Additionally, there are several methodological issues we were not able to address that should be considered in future work. First, because the ABCD study did not include an ACE questionnaire, our ACE scores were reconstructed from items pulled from youth or parent-report. Parents may lack full knowledge of their children's experiences, thereby introducing a potential source of error into our measurement of ACEs. Relatedly, our ACE scores reflect a sum of adversity experiences but there is evidence that EF development is more vulnerable to experiences of deprivation compared with experiences of threat (D. Johnson et al., 2021; Sheridan & McLaughlin, 2014). Although we endeavored to align our investigation with the broader ACE literature, it may be informative to incorporate dimensional assessments of adversity through the creation of latent constructs using data in the ABCD study (e.g., Oshri et al., 2024). Second, although our examination of EF using task performance and parent ratings of executive functioning challenges provides greater coverage of the construct than either used in isolation, our models would be bolstered by having both forms of EF assessment available at both time points (i.e., baseline and follow-up) (Toplak et al., 2008). Parent reports of EF were not collected at baseline and COVID-related disruptions precluded administration of EF tasks to youth at the 2-year follow-up. Task-based EF data for the 4-year follow-up is not yet currently available but will be upon release 6 (Haist & Jernigan, n.d.). Additional timepoints for measurement of both task-based EF skills and reported EF challenges would allow for examination of whether the observed effects are a transient byproduct of puberty or reflect a continuous or growing impact of adversity. Lastly, we acknowledge that nearly all of our sample was comprised of cis-gender youth. As such, additional stress related to the experience of non-cis-gender youth was not considered in our analyses. Moreover, our analyses could not disentangle effects of sex and gender. In our investigation, we were not able to dig deeper into female-specific effects of adversity on EF via pubertal timing to probe whether female socialization and/or additional stress related to the female pubertal development (e.g., menstruation and sexualization) may have accounted for or compounded the impact of childhood adversity.

Finally, executive functioning skills are largely heritable and associated with many of the life stressors found in ACE scores (Freis et al., 2022). Thus, we cannot discount underlying genetic contributions by which difficulties in EF may be inherited. The impacts of parents own EF may be evident in their children's skills but also play a role in shaping risk in the environment (Scarr & McCartney, 1983).

Conclusion

Our findings highlight the impact of adverse life experiences on pubertal timing and EF development for female adolescents. The adolescent developmental period coincides with greater engagement in health-risk behaviors and an increase in mental health concerns (Ho et al., 2024b; Snyder, 2013; Zelazo, 2020). Thus, the relation between adversity, pubertal timing, and executive function may reflect a mechanism by which adverse childhood experiences and accelerated pubertal timing impact mental health risk. This may be particularly important during adolescence when physical maturation and neurological maturation coincide. Ultimately, this study provides insights into sex-specific biological pathways from adversity to executive functioning which builds the foundation for better understanding the development of psychopathology.

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Appendices

Table 1

Descriptive Statistics for Males and Females

Variable	Male			Female			*t*-test
Baseline	*N*	*Mean*	*SD*	*N*	*Mean*	*SD*	
Age	6183	9.49	.51	5671	9.47	.51	2.31*
Family Income	5642	7.23	2.42	5207	7.21	2.42	.45
ACEs	6188	1.33	1.14	5677	1.24	1.14	4.18***
Pubertal Level	5935	1.36	.61	5459	2.18	.91	-55.74***
Response Inhibition	6106	95.81	14.03	5606	95.00	13.27	3.20**
Working Memory	6085	101.00	14.96	5584	100.06	14.58	3.46***
Cognitive Flexibility	6106	96.09	15.30	5607	97.41	14.99	-4.73***
Follow Up							
Pubertal Level 1-Year Follow-up	5579	1.55	.72	5083	2.67	.90	-70.53***
Pubertal Level 2-Year Follow-up	5428	2.01	.91	4854	3.28	.80	-75.16***
Pubertal Level 3-Year Follow-up	5042	2.54	.98	4446	3.71	.67	-69.08***
Parent Reported Executive Dysfunction	5254	24.34	8.72	4722	21.98	7.89	14.20***

Note. ACE = adverse childhood experiences. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2

Bivariate Correlations for Males (Below Diagonal) and Females (Above Diagonal)

Variable	1.	2.	3.	4.	5.	6.	7.	8.
1. Age	--	.05**	-.05**	.26**	-.02	.02	-.00	-.01
2. Income	.03**	--	-.23**	-.24**	.18**	.31**	.20**	-.02
3. ACE	-.05**	-.25**	--	.09**	-.04*	-.09**	-.08**	.19**
4. Pubertal Development	.10**	-.24**	.05**	--	-.07**	-.12**	-.08**	.04**
5. Inhibitory Control	-.03*	.15**	-.03*	-.05**	--	.24**	.45**	-.06**
6. Working Memory	.03*	.28**	-.07**	-.11**	.25**	--	.25**	-.07**
7. Cognitive Flexibility	.01	.19**	-.07**	-.09**	.42**	.28**	--	-.09**
8. Parent Reported Executive Dysfunction	.00	-.01	.20**	-.03	-.05**	-.08**	-.06**	--

Note. ACE = adverse childhood experiences. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 3

Coefficients from mediation analyses of ACEs on latent EF through pubertal timing for males and females at baseline (age 9-10)

	Males			Females		
	b(SE)	*β*	95% CI (LL, UL)	*b(SE)*	*β*	95% CI (LL, UL)
Total effect	-.18 (.13)	-.02	-.052, .004	-.20 (.13)	-.03	-.056, .002
Direct effect	-.17 (.12)	-.02	-.051, .004	-.19 (.13)	-.03	-.054, .004
EF on						
ACEs	-.17 (.12)	-.02	-.051, .004	-.19 (.13)	-.03	-.054, .004
Pubertal Timing	**-.93 (.25)**	**-.07**	**-.096, -.037**	**-.47 (.16)**	**-.05**	**-.080, -.022**
Income	**1.05 (.07)**	**.31**	**.274, .337**	**1.16 (.07)**	**.33**	**.300, .366**
Pubertal Timing on						
ACEs	.00 (.01)	.01	-.016, .031	**.03 (.01)**	**.04**	**.018, .063**
Age	**.13 (.02)**	**.11**	**.089, .132**	**.51 (.02)**	**.28**	**.259, .301**
Income	**-.06 (.00)**	**-.24**	**-.270, -.218**	**-.10 (.01)**	**-.25**	**-.274, -.230**

	Males: Unstandardized		Males: Completely Standardized		Females: Unstandardized		Females: Completely Standardized	
	b (SE)	95% CI (LL, UL)	*β (SE)*	95% CI (LL, UL)	*b (SE)*	95% CI (LL, UL)	*β (SE)*	95% CI (LL, UL)
Indirect effect via pubertal timing	-.00 (.00)	-.016, .007	-.00 (.00)	-.002, .001	**-.02 (.01)**	**-.031, -.005**	**-.00 (.00)**	**-.004, -.001**

Note. ACE = adverse childhood experiences. Significant effects are bolded.

Table 4

Coefficients from mediation analyses of ACEs on parent-rated EF for males and females at follow-up (age 12-13)

	Males			Females		
	b(SE)	*β*	95% CI (LL, UL)	*b(SE)*	*β*	95% CI (LL, UL)
Total effect	**1.62 (.12)**	**.21**	**.187, .236**	**1.38 (.11)**	**.20**	**.172, .224**
Direct effect	**1.62 (.12)**	**.21**	**.187, .236**	**1.37 (.11)**	**.20**	**.171, .223**
EF on						
ACEs	**1.62 (.12)**	**.21**	**.187, .236**	**1.37 (.11)**	**.20**	**.171, .223**
Income	**.12 (.06)**	**.03**	**.005, .062**	**.12 (.06)**	**.04**	**.007, .064**
Pubertal intercept	-.46 (.33)	-.03	-.057, .004	**.59 (.20)**	**.06**	**.025, .094**
Pubertal slope	-.28 (.59)	-.01	-.037, .020	1.21 (.87)	.04	-.006, .077
Pubertal intercept on						
ACEs	.00 (.01)	.01	-.018, .037	**.04 (.01)**	**.05**	**.028, .077**
Age	**.13 (.02)**	**.14**	**.114, .164**	**.54 (.02)**	**.34**	**.314, .359**
Income	**-.06 (.00)**	**-.32**	**-.346, -.283**	**-.09 (.01)**	**-.28**	**-.300, -.252**
Pubertal slope on						
ACEs	.00 (.00)	.01	-.018, .042	**.01 (.00)**	**-.05**	**-.082, -.020**
Age	**.17 (.01)**	**.33**	**.340, .361**	**.06 (.01)**	**-.12**	**-.152, -.093**
Income	**.01 (.00)**	**.05**	**.018, .084**	**.02 (.00)**	**.17**	**.140, .207**

	Unstandardized		Completely Standardized		Unstandardized		Completely Standardized	
Indirect effect via…	*b (SE)*	95% CI (LL, UL)	*β (SE)*	95% CI (LL, UL)	*b (SE)*	95% CI (LL, UL)	*β (SE)*	95% CI (LL, UL)
Pubertal intercept	-.00 (.00)	-.013, .002	.00 (.00)	-.002, .000	**.02 (.01)**	**.009, .043**	**.00 (.00)**	**.001, .006**
Pubertal slope	-.00 (.00)	-.010, .002	.00 (.00)	-.001, .000	-.01 (.01)	-.036, .000	-.00 (.00)	-.005, .000

Note. ACE = adverse childhood experiences. Significant effects are bolded.

Figure 1

Baseline Mediation Model Examining the Influence of ACEs on Youths' Latent EF Performance via Pubertal Timing

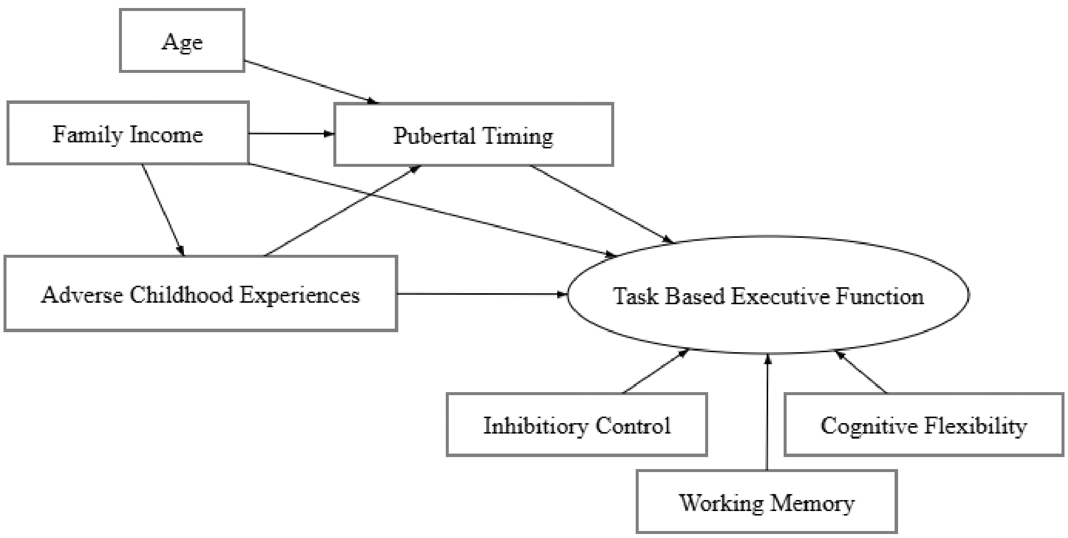

Figure 2

Follow-up Mediation Model Examining the Influence of ACEs on Parent Ratings of Youths' EF Challenges via Pubertal Development