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THE ASSOCIATION BETWEEN COGNITIVE FLEXIBILITY, CHILDHOOD ADVERSITY EXPOSURE, AND INTERNALIZING AND EXTERNALIZING SYMPTOMS

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ABSTRACT

THE ASSOCIATION BETWEEN COGNITIVE FLEXIBILITY, CHILDHOOD ADVERSITY EXPOSURE, AND INTERNALIZING AND EXTERNALIZING SYMPTOMS

Emilie Paul

Adversity exposure in childhood is associated with greater risk for developing internalizing and externalizing problems throughout childhood and adolescence (Henry et al., 2021). Adversity exposure may confer risk by impacting executive function, including reduced cognitive flexibility, due to the neurobiological consequences of increased stress hormone exposure (Kavanaugh et al., 2017). Previous research has linked adversity exposure to reduced cognitive flexibility in youth (Kavanaugh et al., 2017). Lower cognitive flexibility is also associated with internalizing and externalizing problems including depression, anxiety, and disruptive mood dysregulation disorder in youth and adults (Patwardhan et al., 2021; Stange et al., 2017; Braenden et al., 2023). Although cognitive flexibility mediated the pathway from childhood adversity exposure to depression in college students (Huang et al., 2022), to date, no research has investigated this pathway with internalizing and externalizing problems during late childhood. Investigating this pathway in late childhood is important because of the developmental trajectory of cognitive flexibility and emergence of internalizing and externalizing symptoms. The present study addresses gaps in the literature by testing

whether cognitive flexibility mediates the association between adversity exposure and internalizing and externalizing problems in 9-11-year-old youth.

Analyses were conducted with data from the Adolescent Brain and Cognitive Development Study (ABCD), a longitudinal dataset of youth (study wave 1: 9-11 years old, study wave 2: 10-12 years old), sampled across the United States (n = 11,868). Adversity exposure was calculated as the sum of 31 items from youth and parent reported measures of abuse and neglect, domestic violence, household mental illness and substance use, and economic hardship. Cognitive flexibility was measured by the NIH toolbox Dimensional Card sort Task. Parent reported externalizing symptoms were measured by the Child Behavior Checklist (Achenbach, 1991). Youth reported internalizing symptoms were measured by The Brief Problem Monitoring Survey (Achenbach et al., 2011). As expected, greater adversity exposure was associated with lower cognitive flexibility, and higher internalizing and externalizing symptoms 1 year later. Cognitive flexibility mediated the association between adverse events and youth self-reported internalizing symptoms and parent reported externalizing symptoms.

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INTRODUCTION

In the United States, more than half of all children have experienced at least one adverse event, with 10% experiencing 3 or more adverse events before the age of 18 (McLaughlin et al., 2013; Sacks & Murphy, 2018). The impacts of childhood adversities are well documented, including an increased risk of developing internalizing symptoms and externalizing behavior problems in childhood, adolescence, and adulthood (Hoppen & Chalder, 2018). There has been much research into the biopsychosocial factors that link adversity to internalizing and externalizing problems across the lifespan (Sheffler et al., 2020, Mehta et al., 2023). The field of clinical neuroscience suggests that executive function, particularly cognitive flexibility, may play an important role in the development of internalizing and externalizing problems following adversity (e.g., Brooks et al., 2010, Rodrigues et al., 2019, Pauli-Pott et al., 2020). This role of cognitive flexibility has been primarily studied in young children and adults cross-sectionally, investigating cognitive flexibility as an outcome of adversity exposure or in relation to psychopathology in clinical samples with higher symptom severity. Consequently, much less is known about cognitive flexibility as a mechanism between adversity exposure and later internalizing and externalizing symptoms during late childhood and early adolescence in non-clinical samples. The present study addresses major gaps in the field by examining the longitudinal pathway through which adversity exposure impacts internalizing and externalizing symptoms via cognitive flexibility during late childhood in a large, diverse community sample of youth with a broad range of symptom presentation.

Cognitive Flexibility

Cognitive flexibility is a domain of executive function important in adjusting to the demands of the environment (Canas et al., 2006). Although definitions vary, cognitive flexibility is generally thought to encompass the ability to adapt thoughts and behaviors and is critical in the ability to switch thinking between different concepts (Ionescu, 2012). Dajani and Uddin (2015) describe cognitive flexibility as the ability to reconfigure one's response set to a new goal, which includes taking in and manipulating new information and inhibiting previous responses. For example, considering how to solve a problem, being given new information, and being able to adapt thinking to encompass this information relies on flexible thinking. Higher cognitive flexibility has been associated with positive outcomes such as trait resilience, psychological well-being, and coping with mood changing stimuli as to remain in a positive mood (Genet & Siemer, 2011; Hirt et al., 2008). Lower cognitive flexibility is associated with negative outcomes such as depression, obsessive thinking, and rumination, and has been proposed to be a transdiagnostic risk factor for psychopathology (Coenye et al., 2022).

Although impacts of cognitive flexibility on mental health symptoms have been explored, differing definitions of cognitive flexibility remain within the field. These differences arise partially due to overlap of constructs within executive function, as described by Morra et al.'s (2018) three-factor model that stipulates inhibition, updating, and shifting underlies executive functions. Set shifting, described by Bunge and Zelazo (2006) as a lower level of cognitive flexibility, is necessary for an individual to follow a set of rules, then shift to a new set of rules to complete a task. Task switching involves switching between different types of tasks and includes set shifting (Dajani & Uddin,

2015). Diamond (2013) proposed that cognitive flexibility is largely made up of set shifting and set shifting is a construct uniquely attributed to cognitive flexibility. Alternative definitions of cognitive flexibility include concepts such as awareness of alternative actions, willingness to adapt, and self-efficacy around flexible thinking (Morra et al., 2018; Dennis & Vander Wal, 2010).

While cognitive flexibility has been defined broadly in the field, the present study will conceptualize cognitive flexibility as the cognitive process necessary for set shifting and task switching because set shifting is uniquely attributed to cognitive flexibility (Dajani & Uddin, 2015). Currently, because there is no consensus for the definition of cognitive flexibility, there is variability in how it is measured. Neuropsychological tasks are commonly used but may not capture a wider conceptualization of cognitive flexibility as measured by self-report measures of behavior and cognition (e.g. Cognitive Flexibility Inventory) (Morra et al., 2018, Dennis & Vander Wal, 2010). The Dimensional Card Sort task is the most widely used measure of cognitive flexibility in children, and it explicitly tests set shifting ability, aligning with this study's conceptualization of cognitive flexibility (Zelazo, 2006).

Development of Cognitive Flexibility.

Research suggests that cognitive flexibility begins to develop in early childhood, followed by a rapid period of development during middle childhood (Dajani & Uddin, 2015). Dick (2014) assessed the development of cognitive flexibility longitudinally in children from ages 6 to 10, and in college-aged adults using the Flexible Item Selection Task, a sorting task similar to the Dimensional Card Sort Task. Consistent with previous research finding that cognitive flexibility reaches relative maturity by age 12 (Anderson,

2002), children's task performance improved until age 10 and children aged 10 performed at near adult levels (Dick, 2014). The development of cognitive flexibility coincides with the development of brain structures known to be involved in cognitive flexibility, namely the prefrontal cortex (PFC) (Ezekiel et al., 2013). Maturation of the PFC and activation of the dorsolateral and ventrolateral PFC are important for cognitive flexibility, and mor`e specifically, task switching and set shifting (Buttelmann & Karbach, 2017; Dajani & Uddin, 2015; Kim et al., 2011). Thus, late childhood and early adolescence may be an important period to study cognitive flexibility as it has largely developed by then but remains plastic into young adulthood as the PFC continues to mature (Huizinga et al., 2006; Sharma et a., 2013).

Adversity Exposure and Cognitive Flexibility

Development of executive functions including cognitive flexibility can be impacted by adversity exposure during childhood and adolescence (Kavanaugh et al., 2017). In adolescents, total types of trauma exposure were positively associated with perseverative errors on the Wisconsin Card Sort Task (Berg, 1948), a neurocognitive measure of cognitive flexibility (Spann et al., 2012). Additionally, a meta-analysis of trauma-exposed youth found small to medium effect sizes for the association between childhood trauma and cognitive flexibility (Op Den Kelder et al., 2018). The effects of adversity exposure in childhood on cognitive flexibility may persist into adulthood as Kalia et al. (2021) found retrospective reporting of childhood adversity exposure predicted lower cognitive flexibility as measured by the Wisconsin Card Sort Task in college students (Mean age = 19.10 years) and adults (Mean age = 36.23 years).

The connection between adversity exposure and cognitive flexibility may be explained by the developmental traumatology model (De Bellis, 2001). This model stipulates the cognitive and behavioral consequences of early life stress result from changes in the stress response system, particularly the hypothalamic–pituitary–adrenal axis and its byproduct cortisol (De Bellis, 2001; Teicher et al., 2006). Early life stress has been associated with elevated and prolonged levels of circulating cortisol, catecholamines, and serotonin during development and related receptor changes into adulthood (De Bellis & Zisik, 2014). Prolonged exposure to elevated levels of these stress hormones and neurotransmitters during neurodevelopment may lead to delays in myelination, abnormal apoptosis, or inhibition of neurogenesis, especially in brain regions with a high-density of glucocorticoid receptors including the hippocampus and PFC (reviewed in De Bellis & Zisik, 2014; McKlveen et al., 2013; Kavanaugh et al., 2017). Evidence for the developmental traumatology model has been found in studies linking childhood stress and trauma exposure to reduced global volume of the PFC (Kavanaugh et al., 2017, Carrion & Wong, 2012, Hart & Rubia, 2012). Maturation of the PFC and activation of the dorsolateral and ventrolateral PFC have been linked to cognitive flexibility, and more specifically task switching and set shifting (Buttelmann & Karbach, 2017; Dajani & Uddin, 2012; Kim et al., 2012). Thus, changes in maturation of the PFC following childhood stress may explain the connection between adversity exposure and cognitive flexibility found in children and adults.

Internalizing and Externalizing Outcomes related to Cognitive Flexibility.

Puberty, which typically begins at ages 8 to 13 in girls and 9 to 14 in boys (Khan, 2019), marks a significant increase in internalizing disorders, with rates of internalizing

disorders increasing 1.5 times by ages 13 to 15 (Costello et al., 2011). The trajectory of externalizing disorders is more varied; however, adolescence marks the beginning of externalizing behaviors for a subset of youth (Moffitt, 2006). The developmental trajectory of internalizing disorders and externalizing behaviors may coincide with atypical development of the PFC (Casey et al., 2008). For example, Mincic (2015) and Yang and Raine (2016) both found that reduced volume of the PFC was associated with the emergence of mental health problems in adolescence. Thus, late childhood and early adolescence is a particularly important time to understand the effect of cognitive flexibility on internalizing symptoms and externalizing behaviors.

Internalizing and Cognitive Flexibility. Deficits in cognitive flexibility have been linked to multiple internalizing disorders including anxiety, depression, post-traumatic stress, obsessive-compulsive, and eating disorders across the lifespan (Evans et al., 2016; Han et al., 2015; Stange et al., 2017; Wang et a., 2021; Gruner & Pittenger, 2017; Ben-Zion et al., 2018). In a study of 12,462 young children, cognitive flexibility during kindergarten and end of first grade (Mean age = 5.51 years) was directly associated with lower internalizing problems at the next assessment (subsequent fall or spring) (Patwardhan et al., 2021). Brook et al. (2010) found depressed youth scored significantly lower on the cognitive flexibility domain of a neuropsychological battery in a cross-sectional study of 50 children and adolescents (ages 9-17), though the effect size was small. Similarly, anxiety disorders in children and adolescents have been linked cross-sectionally to poorer planning ability compared to a healthy control group on a task involving cognitive flexibility, working memory, and attentional control (Rodrigues et al., 2019). Additionally, a meta-analysis of 147 cross-sectional studies that included

samples of children, adolescents, and adults found strong evidence for a negative association between cognitive flexibility and depression (Stange et al., 2017), however, longitudinal research has been limited. Cross-sectional studies of cognitive flexibility in late childhood and adolescence generally find lower cognitive flexibility in disorder specific populations. However, there is a dearth of research on cognitive flexibility and internalizing symptoms in non-clinical samples.

There is some evidence for a longitudinal association between cognitive flexibility and internalizing symptoms in late childhood and adolescence. For example, Evans et al. (2016) and Han et al. (2015) found that youth who demonstrated high levels of cognitive flexibility, as measured by the Wisconsin Card Sort task, had lower depressive symptoms at ages 9 to 16 and at a 4-month follow-up, and lower anxiety symptoms at a 2-year follow-up. However, these studies included racially and ethnically homogenous samples of adolescents or did not report demographic characteristics of their samples. Relatedly, most studies on cognitive flexibility and internalizing disorders used clinical samples. As a result, it is unclear whether the negative associations between cognitive flexibility and internalizing generalize to youth experiencing subclinical levels of internalizing symptoms, children with clinical levels of symptoms who have not been assessed in a clinical setting, and diverse samples of adolescents who may experience additional stress related to discrimination.

Externalizing and Cognitive Flexibility.

Surprisingly, few studies have examined the link between cognitive flexibility and externalizing behavior in late childhood despite late childhood being a critical period of development for cognitive flexibility (Braenden et al., 2023). In a study of children ages

6 to 12 years old, children with disruptive mood dysregulation disorder had clinically elevated problems with cognitive flexibility compared to age norm scores when cognitive flexibility was measured by parent report (Braenden et al., 2023). However, this association was not significant when cognitive flexibility was measured by a neurocognitive test. Parents may have been primed to report cognitive flexibility difficulties due to the topic of the study, making parent reports either a more biased or sensitive measure of cognitive flexibility relative to neurocognitive testing. There may also be possible differences in how cognitive flexibility was defined by parent reported measures and neurocognitive testing. Braenden et al. (2023) also found that lower levels of cognitive flexibility were related to irritability, a symptom of both internalizing and externalizing behaviors. Regulation of anger has been shown to be inversely related to externalizing in children (Zeman et al., 2002) and lower levels of cognitive flexibility predicted greater aggression, anger, and lower interpersonal problem-solving following induction of angry rumination, indicating lower regulation of anger (Yazici & Mergen, 2022; Ozdogen et al., 2021; Finnigan, 2006).

Previous research on the relation of externalizing symptoms and cognitive flexibility in younger children has yielded mixed results. Research on children ages 4 to 6 found no association between parent and teacher reports of cognitive flexibility and externalizing behavior (Patwardhan et al., 2021; Romero-Lopez et al., 2017). In contrast, a meta-analysis of 5 studies found a small but significant effect for lower cognitive flexibility predicting greater externalizing behavior problems in preschool-aged children (Schoemaker et al., 2013). The effects for executive functioning more broadly on externalizing were stronger in older preschoolers compared to younger preschoolers,

which may be evidence that the small effects and null findings observed in some studies are due to measurement in early childhood. The tenuous association between cognitive flexibility and externalizing behavior in early childhood is not surprising in the context of development, as increases in cognitive flexibility occur most in middle to late childhood (Dick, 2014). Research on adults has provided the most consistent evidence for a link between cognitive flexibility deficits and externalizing disorders, demonstrating developmental changes from early childhood (Morgan & Lilienfield, 2000; Willcut et al., 2005).

Evidence from Intervention Studies. Treatment outcome literature for interventions focused on cognitive flexibility provides the strongest evidence for the role of cognitive flexibility in the etiology of externalizing disorders. Indeed, many psychotherapies for internalizing and externalizing symptoms, either explicitly or inadvertently, target cognitive flexibility. Neurocognitive interventions, cognitive-behavioral therapy, and acceptance and commitment therapy have all shown some evidence of increasing cognitive flexibility and improving psychological outcomes (Ben-Zion et al., 2018; Nagata et al., 2018; Lackner et al., 2022). Interventions targeting cognitive flexibility in preschoolers have been shown to reduce ADHD symptoms and externalizing behaviors, suggesting cognitive flexibility and externalizing behaviors are related (Pauli-Pott et al., 2020). In a meta-analysis of 12 studies that tested cognitive flexibility intervention in early childhood (3-6 years old) on externalizing, a moderate effect size was found. The four well-controlled studies that included an active control group and either neurocognitive assessment or blind rating following intervention, found large main effects (Pauli-Pott et al., 2020). Overall, there is evidence in both young

children and adults that intervention targeting cognitive flexibility can improve mental health outcomes, however this has not been studied in late childhood and early adolescence, a developmental period following rapid development of cognitive flexibility.

Cognitive Flexibility as a Mediator

Research on children, adolescents, and adults points to cognitive flexibility as a possible mechanism that explains the link between childhood adversity and internalizing and externalizing behaviors (Op Den Kelder et al., 2018; Stange et al., 2017; Schoemaker et al., 2013). Although no studies have examined cognitive flexibility as a mediator of adverse experiences and internalizing and externalizing symptoms in late childhood and early adolescence, research on college students suggest cognitive flexibility mediates the association between childhood trauma and depression. Specifically, Huang et al. (2022) found that greater trauma exposure and stressful life events were associated with lower levels of cognitive flexibility, which in turn predicted greater levels of depression in a large, cross-sectional study of first-year college students (Mean age of 18.2 years). Given the importance of late childhood and early adolescence for the development of cognitive flexibility and internalizing and externalizing symptoms, it will be important to examine cognitive flexibility as a mediator during this developmental period.

Current Study

Previous studies found that greater exposure to adversity was associated with lower levels of cognitive flexibility and in turn, greater internalizing and externalizing symptoms. However, the literature is primarily limited to young children and adult samples, even though late childhood is a sensitive period of development for cognitive

flexibility and internalizing and externalizing (Dajani & Uddin, 2015). Thus, the present study tested whether cognitive flexibility mediated the association between childhood adversity exposure (ages 9-11 years) and internalizing and externalizing symptoms one year later (ages 10-12 years old) using data from a nationally representative sample of 11,880 youth. Cognitive flexibility was expected to mediate the relation between adversity exposure and internalizing and externalizing symptoms, such that adversity exposure would be associated with lower cognitive flexibility, which in turn would predict greater internalizing and externalizing symptoms.

METHODS

Procedures

Data for this study were drawn from the Adolescent Brain and Cognitive Development (ABCD) Study™, a longitudinal study of brain development and child health that collected a broad range of measures on childhood experiences, behaviors, brain imaging, hormones, and the child's environment (Garavan et al., 2018). Participants were recruited across 17 states from geographic areas near 21 ABCD Study™ research sites in the United States, aimed to reflect socio-demographic factors of the United States population such as race, ethnicity, and socioeconomic status (Garavan et al., 2018). Of note, this is a nationally representative, non-clinical sample that has been described as "generally high functioning" in terms of mental health (Karcher & Barch, 2021). Participants were generally approached for participation in public schools (>90%) stratified based on socioeconomic status, gender, racial and ethnic composition, and location. Fewer than 10% of participants were recruited from other avenues such as community events, non-targeted schools, and referrals. Inclusion criteria included youth between the ages of 9 and 10 years old at the first timepoint of data collection and residing within 50 miles of an ABCD Study™ research site. Data collection is ongoing and occurs annually in person at ABCD Study™ research sites, starting at age 9 to 10 and will continue until participants are 18 to 19 years old. This study is currently in its fifth year of data collection and a detailed summary of the protocol and current findings is described in Karcher and Barch (2021).

Data from study waves 1 and 2 were included in the present study and only a subset of collected measures were included in analyses. Adversity exposure, cognitive

flexibility, demographic information, and pubertal stage were assessed at study wave 1. Previous literature has indicated youth may be better reporters of internalizing symptoms and parents of externalizing symptoms, therefore externalizing was assessed by parent report at study waves 1 and 2 and internalizing was assessed by the youth reported at study wave 2 only (Miller et al., 2014; Salbach-Andrae et al., 2009).

Participants

Data from the first (n = 11,880, *Mean age* = 9.92 years, SD = 0.63) and second (n = 11,197, *Mean age* = 10.92 years, SD = 0.63) study waves were included in analyses. At the first study wave, the sample identified as 21% Hispanic or Latino, 74% White or Caucasian, 21% Black or African American, 6% Asian American, 3% American Indian or Native American, <1% Hawaiian, <1% Guamanian, <1% Samoan, and 6% Other. Twelve percent of the sample identified as multiple races. Caregivers reported gender of the children as 52% male, 48% female, <1% trans male, <1% trans female, <1% Gender Queer, and <1% Other. Children identifying as genderqueer and other were excluded due to small sample size (n = 6). The most common total household income in the past 12 months was $100,000 through $199,999 (30.5%) and ranged from less than $5,000 (3.8%) to $200,000 and greater (11.5%).

Measures

Adversity Exposure

Total exposure to adverse events included 31 items pertaining to child and parent-report measures of child abuse and neglect, domestic violence exposure, caregiver mental illness, caregiver substance use, and economic hardship. The 31 items were selected from 5 measures based on previous research with this sample that characterized exposure to

adverse events, and found adverse events exposure was related to increased odds of binge eating disorder, as well as problematic screen use (Hoffman et al., 2019; Chu et al., 2022; Raney et al., 2023). Consistent with Hoffman et al. (2019), the 31 items capturing exposure to adverse events during childhood were summed, with higher scores reflecting greater exposure to different forms of adversity (Table 1).

Parents reported if *child physical abuse* (3 items)*, sexual abuse* (2 items)*, peer sexual assault* (1 item), and *grief* (1 item) occurred (1 = yes or 0 = no) in the youth's lifetime on the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) PTSD Module – parent report (Kaufman et al., 1997). *Child emotional and physical neglect* was measured by the ABCD Parental Monitoring Survey – child self-report and the Children's Report of Parental Behavior (Schafer, 1965). The Parental Monitoring Survey included 3 items (e.g. "*How often do your parents/guardians know where you are?*"), rated on a five-point scale ranging from "Never" to "Always or Almost Always". Responses of "Never" and "Almost Never" were coded as yes (1) of *neglect*. The Child Relationship Behavior Inventory included two items (e.g. "*Parent believes in showing love to me*") rated on a three-point scale ranging from "Not like him/her" to "A lot like him/her". Responses of "Not like him/her" were coded as yes (1) of *neglect*.

Witnessing domestic violence exposure (3 items) was measured with the Family Environment Scale – child self-report and parent report (Moos. & Moos,1994), and the K-SADS PTSD Module – Parent report (Kaufman et al.,1997). Two items on the Family Environment Scale with true or false response options were evaluated. Because the two items are identical on child and parent report, an endorsement of each item from either parent or child was coded as yes (1) to an item. One item assessing domestic violence in

the household from the K-SADS PTSD Module was included; "*Witness the grown ups in the home push, shove, or hit one another.*"

Household substance use (1 item) and *criminal household member* (1 item) were measured by the Family History Assessment – parent report. One item asked about issues due to substance use of blood relatives. If the relative was the child's mother or father, this item was coded as yes (1). A second item asked whether the child has any blood relatives who get in trouble with police or the law and was coded as yes (1) or no (0).

Household mental illness (5 items), *economic hardship* (7 items), and *divorce of parents* (1 item) were measured by the Demographic and Family History Assessment-parent report. Household mental illness included questions assessing if either parent had problems related to anxiety, depression, mania, paranoia, "nervous breakdown", and suicide attempt. Economic hardship was measured by asking if in the past 12 months, the family had difficulty affording necessities such as mortgage/rent, utilities, and medical care (1 = yes, 0 = no).

Cognitive Flexibility

Cognitive flexibility was measured using the Dimensional Change Card Sort task, as part of the National Institute of Health's Toolbox Cognition Measures (Gershon et al., 2013). The Dimensional Change Card Sort task, developed by Zelazo and colleagues (2006), has been widely used to assess cognitive flexibility and executive function in children (Zelazo, 2006; Beck et al., 2011). The Dimensional Change Card Sort task and its more difficult version have shown excellent test-retest reliability in past studies (ICCs = .90 to .94; Beck et al., 2011). During the Dimensional Change Card Sort task, youth were asked to sort cards by one parameter (e.g., by shape), and then switch to sorting

cards by another parameter (e.g., color) for 38 trials. This task assesses flexible rule use, switching, and inhibiting involved in cognitive flexibility. For this analysis, a tabulated age-corrected t-score published by the ABCD study was used with higher t scores reflecting higher cognitive flexibility.

Externalizing Symptom Severity

Externalizing symptoms were measured using the Child Behavior Checklist (CBCL) (Achenbach, 1991). The CBCL is a parent-report measure that assesses the behaviors and emotional problems of children. The measure consists of 113 items rated on a 3-point Likert scale of *absent, occurs sometimes,* or *occurs often*. The CBCL has been widely used to assess emotional and behavioral problems in children by parent report, with strong psychometric properties of its subscales (Achenbach & Rescorla, 2001). Externalizing symptom subscale (study sample $\omega = 0.92$) is the sum of aggression (18 items) and rule-breaking (17 items) subscales.

Internalizing Symptom Severity

Internalizing symptoms were measured using The Brief Problem Monitoring Survey. The Brief Problem Monitoring Survey (BPM) is a youth-report measure that assesses the behaviors and emotional problems of children (Achenbach et al., 2011). The measure consists of 19 items rated on a 3-point Likert scale of *not true, somewhat true,* or *very true*. The BPM draws items directly from the CBCL and has strong psychometric properties of its subscales (Achenbach & Rescorla, 2001). Internalizing symptoms subscale (study sample $\omega = 0.72$) is a sum of 6 items (Achenbach et al., 2011).

Covariates

Parents reported on their child's age in months, gender, race and ethnicity, and household income at the first study wave. Child's gender was reported as either male, female, trans male, trans female, genderqueer, or other, and gender was recoded as boys (including trans boys) and girls (including trans girls). Parents could select from over 16 racial categories (Table 2), which were collapsed into five groups (White or Caucasian, Black or African American, Asian American, Native American or American Indian and multiple or other race) due to small sample size (less than 3%) of many of the categories. Race categories were dummy coded with White or Caucasian as the reference group due it being the largest group in this sample. Race was included as a covariate to account for minority stress and racial-based trauma, which was not adequately captured within the Adverse Events variable. Household income was measured by parent report as the total yearly household income.

Pubertal stage was assessed using parent report of the Tanner Staging (Marshall & Tanner, 1969, 1970). Tanner Stage is an objective classification of 5 stages from pre-pubertal to post-pubertal based on physical characteristics of puberty including pubic hair, breast development, and male external genitalia. Parents were asked to look at 5 drawings of pubic hair and female breast development or male external genitalia and choose the image that most closely resembled their child. Previous studies have found self-reported and parental reported Tanner Stage are highly correlated and therefore, only parent report was included in this analysis to limit missing data (Dorn et al., 1990). An average of the pubic hair stage and female breast or male external genitalia development stage was used to assign a puberty stage ranging from prepubescent (1) to postpubescent

(5). Tanner staging was included as a covariate to account for puberty-related differences in symptom presentation.

Analytic Plan

Preliminary Analyses

Variables were assessed for missing values, outliers, and normality. Due to positive skew of the data, exposure to adverse events sum scores were winsorized to 3 SD above the Mean (n = 228), with final scores ranging from 0 to 10 adverse events (Table 3). Children who experienced 10 adverse events (n = 129) and children who experienced 11 or more adverse events (n = 228) did not significantly differ on cognitive flexibility, parent reported externalizing symptom severity at study waves 1 and 2, and youth reported internalizing symptom severity at study wave 2 (*p*s .13 to .67; Table 4). Less than 2% of data was missing for cognitive flexibility at study wave 1. About 6% of data was missing at study wave 2 for internalizing and externalizing symptom severity. Missing data was handled using Full Information Maximum Likelihood estimation (Enders, 2013). Differences between participants with complete and missing data for study waves 1 and 2 were assessed by chi square test for study wave 1 demographic characteristics (race, gender, household income) and independent sample t-tests for adverse events, internalizing, and externalizing (Appendix I). Youth with missing data had significantly higher adversity exposure, lower cognitive flexibility, and higher parent reported externalizing symptoms at study wave one; however, effect sizes were small. Bivariate correlations and ANOVAs were conducted to explore the relation of the key study variables (adversity exposure, cognitive flexibility, internalizing symptoms, externalizing symptoms) and covariates (gender, race, puberty status, and age) (Table 6).

Outcome Analyses

Models testing for an indirect effect from adversity exposure (wave 1) to youth-reported internalizing and parent-reported externalizing symptoms (wave 2) via cognitive flexibility (wave 1) were performed using JASP software (JASP, 2023) (Figure 1 & 2). Models were run separately for internalizing and externalizing symptoms and children's age (in months), gender, race, household income, and pubertal stage were included as covariates. Model 2 was run with and without controlling for parent reports of externalizing symptoms at study wave 1 due to the strong correlation with externalizing ($r = .74$, $p < .001$) at study wave 2. A sensitivity analysis was conducted to determine whether the results were sensitive to the winsorization of adversity exposure. Models 1 and 2 were run again including the non-winsorized adversity exposure variable.

RESULTS

Results from a bivariate correlation analysis are presented in Table 5. As expected, greater exposure to adversity was associated with lower levels of cognitive flexibility and higher levels of internalizing and externalizing symptoms at study waves 1 and 2. Lower levels of cognitive flexibility were associated with higher levels of internalizing symptoms at study wave 2. Lower levels of cognitive flexibility also were associated with higher levels of externalizing symptoms at study wave 1 and study wave 2.

Internalizing Mediation Models

In Model 1, mediation analysis was performed to test for an indirect effect from adverse events at wave 1 to internalizing symptoms at wave 2 via cognitive flexibility at wave 1 (Figure 1, Table 7). The total effect of the model was significant. A greater number of adverse events were associated with lower levels of cognitive flexibility and greater youth reported internalizing symptoms one year later (Model 1a). Greater cognitive flexibility was associated with lower levels of youth-reported internalizing symptoms and cognitive flexibility mediated the association between adverse events and youth self-reported internalizing symptoms (Model 1a). Findings remained substantially the same when including non-winsorized total adverse events as a predictor in the model of youth reported internalizing symptoms (Model 1b, Appendix. II).

Externalizing Mediation Models

Model 2 tested whether adversity exposure at wave 1 was associated with externalizing symptoms at wave 2 via cognitive flexibility at wave 1 (Figure 2, Table 8). The total effect of Model 2 was significant. A greater number of total adverse events

were associated with lower levels of cognitive flexibility and greater parent reported externalizing symptoms. Greater levels of cognitive flexibility were also associated with lower levels of parent reported externalizing symptoms. The indirect effect was significant for the model, suggesting cognitive flexibility partially mediates the relation between adversity exposure and externalizing behaviors (Model 2a, Table 8). Findings remained the same when non-winsorized number of total adverse events was included as a predictor in the models (Model 2b, Appendix II). When parent reported externalizing symptoms at study wave 1 was controlled for, the indirect effect of parent reported externalizing was no longer significant (Model 2c, Appendix II).

DISCUSSION

The present study investigated the mediating effect of cognitive flexibility on the association between adverse events and internalizing and externalizing symptoms during late childhood and early adolescence, an understudied yet critical period for neurodevelopment. As expected, greater exposure to adversity was related to lower levels of cognitive flexibility, as well as higher internalizing and externalizing symptoms measured one year later, however effect sizes were small. Cognitive flexibility mediated the association between adversity exposure and both internalizing and externalizing symptoms. This study's results add to the growing literature indicating cognitive flexibility as one mechanism between early adversity and later mental health symptoms using data from a large, diverse sample of children and adolescents that is powered to detect small effects.

In separate studies, previous research has linked greater adversity exposure to lower levels of cognitive flexibility and greater internalizing symptoms across disorders in children, adolescents, and adults (Stange et al., 2017; Lee & Orsillio, 2014; Gruner & Pittenger, 2017; Tchanturia et al., 2004; Miles et al., 2023; Byrne et al., 2021; Ben-Zion et al., 2018). Thus, it is not surprising that in the present study, adversity exposure and lower cognitive flexibility both predicted greater internalizing symptoms across one year during late childhood and early adolescence. Although few studies have longitudinally examined the mediating effect of cognitive flexibility on adversity exposure and internalizing symptoms during late childhood, Huang et al. (2022) found greater trauma exposure and stressful life events were associated with lower levels of cognitive flexibility, which in turn predicted greater levels of depression in college students in a

cross-sectional study. Our findings build on research by Huang et al. (2022) by demonstrating that the mediating effect of cognitive flexibility on internalizing symptoms following adversity exposure may begin to appear in late childhood and persist across adolescence and into adulthood.

Previous studies on adversity exposure, cognitive flexibility, and externalizing symptoms have yielded inconsistent results that may be related to age or developmental differences between samples. Research on adults consistently implicates deficits in cognitive flexibility in the development of externalizing behaviors including ADHD, antisocial behaviors, and aggression (Morgan and Lilienfield, 2000; Willcut et al., 2005; Zeman et al., 2002). Research on young children is less consistent, with some studies finding no association between cognitive flexibility and externalizing and others finding a small effect (Schoemaker et al., 2013). This may be due, at least in part, to measuring cognitive flexibility earlier in development (Anderson, 2002; Dick, 2014) or differences in early childhood versus adolescent onset of externalizing problems (Moffit, 2006). The associations we found related to cognitive flexibility (measured between 9 and 11 years old) were consistent with associations seen between cognitive flexibility and disruptive mood disorder in children aged 6 to 12 (Braenden et al., 2023). Although no studies have examined the mediational pathway from adversity exposure to externalizing via cognitive flexibility, our results are supported by research linking cognitive flexibility to both adversity exposure and externalizing behavior.

Cognitive flexibility mediated the association between adversity exposure and both internalizing and externalizing symptoms in youth, which may provide evidence for a common factor of psychopathology (Caspi & Moffit, 2018). Cognitive flexibility may

also be impacting a specific underlying processes that contribute to both internalizing and externalizing symptoms. For example, rumination involves thinking and behaving in a way that focuses on one's own current distressed state and is a transdiagnostic factor for both internalizing and externalizing disorders in adolescents (Garnefski et al., 2005). Rumination may be related to cognitive flexibility as people who ruminate can have difficulty integrating new information into their mental sets (set shifting) (Davis & Nolen-Hoeksema, 2000). Indeed, previous research has demonstrated an association between rumination and lower cognitive flexibility (Davis & Nolen-Hoeksema, 2000). Additionally, resilience has been described as flexibility in adapting to change, a process that is necessary for cognitive flexibility (Genet & Siemer, 2011). In fact, multiple studies have found positive associations between cognitive flexibility and resilience in adults (Parsons et al., 2016; Southwick et. al., 2005). The adaptability to change may reduce the likelihood of experiencing negative sequalae following adverse events, such as internalizing and externalizing symptoms (Genet & Siemer, 2011). Future studies should investigate rumination and resilience as transdiagnostic mechanisms that may explain the mediating effect of cognitive flexibility on adversity exposure and internalizing and externalizing problems in children and adolescents.

Clinical Implications

Optimistically, cognitive flexibility remains somewhat plastic through the lifespan (Van de Ven, 2017; Masley et. al., 2009; Zou et al., 2020), indicating it as an intervention target. Previous research has shown the efficacy of cognitive flexibility training on reducing deficits in cognitive flexibility seen after trauma exposure in adults, which in turn was related to better treatment outcomes for PTSD (Ben-Zion et al., 2018).

Cognitive flexibility's impact on treatment outcomes points to the benefit of conducting thorough assessment of possible neurocognitive deficits in the context of trauma intervention and addressing cognitive flexibility in therapy for adversity related psychopathology. Because lower cognitive flexibility was shown to be associated with even subclinical internalizing and externalizing symptoms in youth, our findings highlight the need for screening of adversity exposure and mental health symptoms in youth beyond clinical settings. Additionally, these findings could indicate cognitive flexibility as a target as both a preventative intervention following exposure to adversity in youth, and for children who display subthreshold internalizing and externalizing symptoms. Late childhood and adolescence often include school transitions and experiences which may be facilitated by cognitive flexibility, indicating the importance of assessment and intervention at this developmental stage.

Limitations and Future Directions

Using a nationally representative sample provided many benefits; however, the present study had several limitations. First, previous literature has found differences in cognitive flexibility between maltreatment types in adolescents, particularly physical and sexual abuse (Kavanaugh et al., 2013), suggesting adversity type may be a moderator. However, the present sample experienced relatively low rates of adversity-exposure, which prohibited the assessment of differences in outcomes by adversity type. Second, the present study included a single measure of cognitive flexibility that only captured children's set shifting and task switching ability. Broader definitions of cognitive flexibility captured by behavioral observation such as perceived ability to cope and adapt were not measured. Future studies should include multiple measures of cognitive

flexibility to determine whether the association between adversity exposure, cognitive flexibility, and internalizing and externalizing problems are specific to set shifting and task switching or extend to parents reports of adaptation and coping components of cognitive flexibility. Third, cognitive flexibility was measured at a single study wave at the same time as adversity exposure, which precluded the examination of how exposure to adversity impacts development of cognitive flexibility across adolescence. Future studies should examine cognitive flexibility development beyond one year to determine if these findings persist into adulthood, as has been found in cross-sectional studies of adults (Morgan & Lilienfield, 2000; Willcut et al., 2005; Stange et al., 2017).

Conclusion

The present study demonstrated that cognitive flexibility is a cognitive mechanism through which adversity exposure impacts the development of internalizing and externalizing symptoms using a large, diverse, nationally representative sample of youth. Results from the present study extend the generalizability of these results beyond clinical samples to youth with relatively low rates of adversity exposure and subclinical internalizing and externalizing symptoms. Additionally, including a diverse sample of youth from various geographic, socioeconomic, and racial and ethnic backgrounds strengthens the generalizability of our results, highlighting the significance of cognitive flexibility in the connection between adversity exposure and mental health concerns. Clinical implications of this research include targeting cognitive flexibility deficits in youth who have experienced adversity to reduce development of later mental health problems. Future studies should include multiple measures of cognitive flexibility at

multiple timepoints, as well as assessing cognitive flexibility as a mediator across adolescence as development continues.

Table 1. *Adversity Exposure Items.*

Type	*Measure*	*Item*	*Response*
Child physical abuse	KSADS	Shot, stabbed, or beaten brutally by a grown up in the home	Yes =1 or No =0
	KSADS	Beaten to the point of having bruises by a grown up in the home	Yes =1 or No =0
	KSADS	A family member threatened to kill your child	Yes =1 or No =0
Child sexual abuse	KSADS	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	Yes =1 or No =0
	KSADS	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	Yes =1 or No =0
Peer sexual assault	KSADS	A peer forced your child to do something sexually	Yes =1 or No =0
Grief	KSADS	Learned about the sudden unexpected death of a loved one	Yes =1 or No =0
Child emotional and physical neglect	PMQ	How often do your parents/guardians know where you are?	Never, Almost never = 1, Sometimes, Often, Always or Almost Always = 0
	PMQ	How often do your parents know who you are with when you are not at school and away from home?	Never/Almost never = 1 Sometimes, Often, Always or Almost Always = 0
	PMQ	If you are at home when your parents or guardians are not, how often do you know how to get in touch with them?	Never/Almost never = 1 Sometimes, Often, Always or Almost Always = 0
	PMQ	How often do your parents/guardians know where you are?	Never/Almost never = 1 Sometimes, Often, Always or Almost Always = 0
	KSADS	Witness the grownups in the home push, shove or hit one another	Yes =1 or No =0
	FES	Family members sometimes hit each other	Yes =1 or No =0
	FES	Family members sometimes get so angry they throw things	Yes =1 or No =0
Household Substance Use	FHA	Has any blood relativeof your child ever had any problems due to alcohol such as: marital separation or divorce, laid off or fired from work, arrests or DUIs; alcohol harmed their health; in an alcohol treatment program; suspended or expelled from school 2 or more times; isolated self from family, caused arguments or were drunk a lot?	Mother or Father Yes =1 or No =0

Criminal Household Member	FHA	Has any blood relative of your child been the kind of person who never holds a job for long, or gets into fights, or gets into trouble with the police from time to time, or had any trouble with the law as a child or an adult?	Mother or Father Yes =1 or No =0
Household Mental Illness	FHA	Has ANY blood relative of your child ever attempted or committed suicide?	Mother or Father Yes =1 or No =0
	FHA	Has any blood relative of your child ever suffered from depression?	Mother or Father Yes =1 or No =0
	FHA	Has any blood relative of your child ever suffered from mania?	Mother or Father Yes =1 or No =0
	FHA	Has any blood relative of your child ever suffered from paranoia?	Mother or Father Yes =1 or No =0
	FHA	Has any blood relative of your child ever suffered from nerves/nervous breakdown?	Mother or Father Yes =1 or No =0
Economic Hardship	Demographics	In the past 12 months, needed food but couldn't afford to buy it or couldn't afford to go out to get it?	Yes =1 or No =0
	Demographics	In the past 12 months, were without telephone service because you could not afford it?	Yes =1 or No =0
	Demographics	In the past 12 months, didn't pay the full amount of the rent or mortgage because you could not afford it?	Yes =1 or No =0
	Demographics	In the past 12 months, were evicted from your home for not paying the rent or mortgage?	Yes =1 or No =0
	Demographics	In the past 12 months, had services turned off by the gas or electric company, or the oil company wouldn't deliver oil because payments were not made?	Yes =1 or No =0
	Demographics	In the past 12 months, had someone who needed to see a doctor or go to the hospital but didn't go because you could not afford it?	Yes =1 or No =0
	Demographics	In the past 12 months, had someone who needed a dentist but couldn't go because you could not afford it?	Yes =1 or No =0
Divorce of parents	FHA	Divorced/Separated	Yes =1 or No =0

Note. KSADS = Kiddie Schedule for Affective Disorders and Schizophrenia; PMQ = Parental Monitoring Questionnaire; FES = Family Environment Scale; FHA = Family History Assessment.

Table 2. *Sample Demographics at Study Wave 1*

Variable	*M* (*SD*)	*Frequency* (%)
Age	9.92 (0.63)	
Gender		
Male		5667 (47.70)
Female		6188 (52.01)
Race & Ethnicity		
Black		2415 (20.33)
Asian		712 (5.99)
Native American/Pacific Islander		390 (3.28)
Caucasian		8419 (70.87)
Multiracial		1414 (11.90)
Other		837 (7.05)
Yearly Household Income		
Less than $5,000		417 (3.51)
$5,000 - $11,999		421 (3.55)
$12,000 - $15,999		273 (2.30)
$16,000 - $24,999		524 (4.41)
$25,000 - $34,999		654 (5.51)
$35,000 - $49,999		934 (7.86)
$50,000 through $74,999		1499 (12.61)
$75,000 through $99,999		1572 (13.24)
$100,000 through $199,999		3314 (27.90)
$200,000 and greater		1250 (10.525)

Table 3. *Frequency Statistics for Adversity Exposure*

# Exposure Items Endorsed	Frequency of Endorsement	% Sample Endorsement
0	2599	21.9%
1	2725	22.9%
2	2099	17.7%
3	1421	12.0%
4	844	7.1%
5	602	5.1%
6	455	3.8%
7	322	2.7%
8	280	2.4%
9	166	1.4%
10	129	1.1%
11 and above	228	1.9%

Table 4. *Results of T-tests comparing youth with 10 adverse events and youth with greater than 10 adverse events*

Study Variable	10 events (n=129)		>10 events (n=228)				
	M	SD	M	SD	t	p	Cohen's d
1. Cognitive Flexibility	91.7	12.7	92.4	14.5	-0.43	0.67	-0.048
2. PR Externalizing (Wave 1)	55.9	12.6	56.9	11.8	-0.75	0.45	-0.083
3. PR Externalizing (Wave 2)	53.0	13.6	55.0	12.5	-1.30	0.20	-0.149
4. YR Internalizing (Wave 2)	54.4	6.4	55.6	6.4	-1.50	0.13	-0.182

Table 5. *Descriptive Statistics and Correlations of Study Variables*

Event	M	SD	1	2	3	4	5	6	7
1. Adverse Events	2.52	2.54	-						
2. Cognitive Flexibility	96.71	15.16	-.13**	-					
3. PR Externalizing (Wave 1)	45.72	10.34	.35**	-.08**	-				
4. PR Externalizing (Wave 2)	45.20	10.13	.32**	-.07**	.74**	-			
5. YR Internalizing (Wave 2)	53.34	5.27	.15**	-.07**	.12**	.14**	-		
6. Age	9.91	6.25	-.03**	.04**	-.03**	-.02*	-.05*	-	
7. Gender			-.02	.04**	-.08**	-.06**	-.13**	-.02**	-

Note. ** $p < .001$, * $p < .05$. PR = parent reported, YR = youth reported

Table 6. *ANOVAs with Race and Ethnicity Groups*

Grouping Variable	Variable	Mean (SD)	F or t	*η2*	df
Adversity Exposure			176.67**	.056	4, 11861
	1.Caucasian	2.17 (2.33)	2. -23.31** 3. -6.29** 4. 6.99** 5. -11.74**		
	2. Black or African American	3.65 (2.80)	3. -1.54 4. 15.52** 5. 10.08**		
	3. Native American or American Indian	4.15 (2.69)	4. 8.84** 5. 4.01**		
	4. Asian American	1.04 (1.47)	5. 5.30**		
	5. Multiple or Other	2.87 (2.70)			
Cognitive Flexibility			93.29**	.031	4, 11709
	1.Caucasian	98.12 (15.04)	2. 18.48** 3. 2.55 4. -3.68 5. 4.88**		
	2. Black or African American	90.92 (13.75)	3. -1.21 4. -10.55** 5. -11.42**		
	3. Native American or American Indian	93.26 (13.96)	4. -3.98** 5.1.60		
	4. Asian American	101.71 (17.19)	5. 5.30**		
	5. Multiple or Other	96.34 (15.28)			
PR Internalizing (wave 1)			28.70**	.010	4, 11192
	1.Caucasian	48.92 (10.52)	2. 8.39** 3. 1.52 4. 5.36** 5. -2.38		
	2. Black or African American	46.51 (10.56)	3. -.20 4. 1.85 5. -8.71**		
	3. Native American or American Indian	46.79 (11.02)	4. 1.06 5. -1.95		
	4. Asian American	45.13 (9.66)	5. -6.02**		
	5. Multiple or Other	49.55 (10.79)			
PR Externalizing (wave 1)			17.76**	.006	4, 11192
	1.Caucasian	98.12 (15.04)	2. -4.34** 3. -.24 4. 6.04** 5. -3.74*		
	2. Black or African American	90.92 (13.75)	3. 0.64 4. 7.43** 5. 0.72		
	3. Native American or American Indian	93.26 (13.96)	4. 2.95* 5.-.46		
	4. Asian American	101.71 (17.19)	5. -7.17**		
	5. Multiple or Other	96.34 (15.28)			

**p value <.001, *p value <.05

Table 7. *Mediation Results of Model 1a using winsorized Total Adverse Events.*

Model 1a. Paths and Effects with Youth Reported Internalizing

					95% Confidence Interval	
Effect		Estimate	SE	p	Lower	Upper
Direct	Total Adverse Events → YR Internalizing	.140	.010	<.001	.120	.159
Direct	Total Adverse Events → CF	-.09	.056	<.001	-.616	-.395
Direct	CF → YR Internalizing	-.058	.003	<.001	-.027	-.014
Indirect	Total Adverse Events → CF→ YR Internalizing	.004	9.464^{-4}	<.001	.002	.006

Note. CF = Cognitive Flexibility, YR = Youth reported

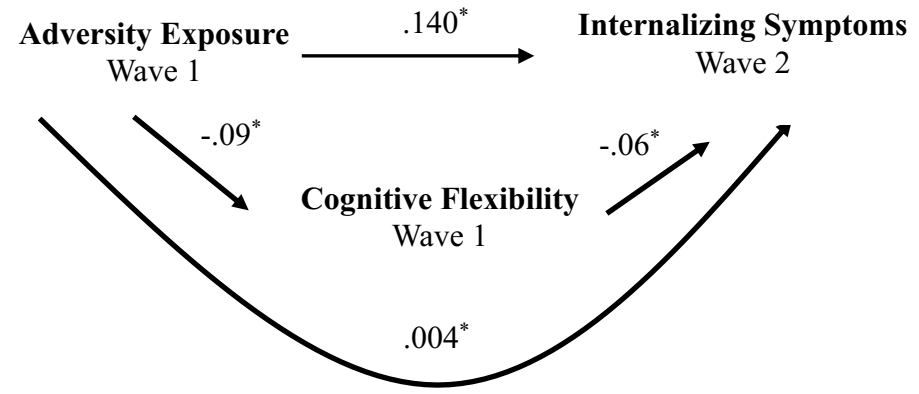
Table 8. *Mediation Results of Model 2a using winsorized Total Adverse Events.*

Model 2a. Paths and Effects with Parent Reported Externalizing

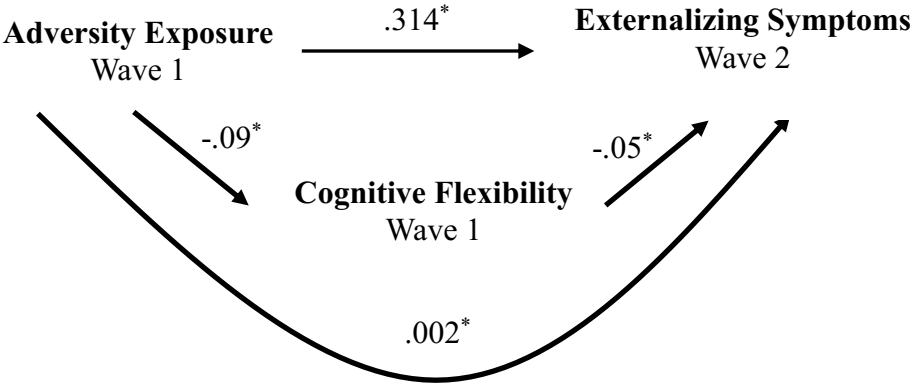
					95% Confidence Interval	
Effect		Estimate	SE	p	Lower	Upper
Direct	Total Adverse Events → PR Externalizing	.314	.009	<.001	.296	.333
Direct	Total Adverse Events → CF	-.09	.056	<.001	-.616	-.395
Direct	CF → PR Externalizing	-.054	.006	<.001	-.048	.023
Indirect	Total Adverse Events → CF→ PR Externalizing	.002	$8.237e^{-4}$	.003	$8.68e^{-4}$	.004

Figure 1. *Mediation Models*

Model 1a. *Mediating effect of cognitive flexibility on the pathway of adversity exposure and Internalizing symptoms*



Model 2a. *Mediating effect of cognitive flexibility on the pathway of adversity exposure and Externalizing symptoms*



Note. Adverse Events measured as a sum of 31 items assessing youth's adversity exposure at study wave 1. Cognitive flexibility measured by an age corrected t score of the Dimensional Card Sort Task at study wave 1. Internalizing Symptoms measured one year later by the Behavior Monitoring Scale (youth report) at study wave 2. Externalizing Symptoms measured one year later by the Child Behavior Checklist (parent report) at study wave 2. *p value < .05.

Appendix I. *Results of T-tests and Chi squared comparing youth with complete data and youth with incomplete data.*

Study Variable	Complete		Missing Time 2		t	p	Cohen's d
	M	SD	M	SD			
1. Cognitive Flexibility	96.9	15.1	93.7	15.1	5.14	<.001	.209
2. Adverse Events	2.5	2.5	3.1	2.7	-6.17	<.001	-.248
3. PR Internalizing (Wave 1)	48.4	10.6	48.4	11.2	0.21	.84	.001
4. PR Externalizing (Wave 1)	45.7	10.3	46.8	11.1	-2.78	.006	-.248

Study Variable	Complete		Missing Time 2			
	N	%	N	%	x^2	p
Gender					1.62	.203
Girl	5342	47.7	325	5.7		
Boy	5866	52.3	322	5.2		
Race					151.61	<.001
White or Caucasian	7234	64.5	279	42.9		
Black or African American	1671	14.9	197	30.3		
Asian American	231	5.7	12	1.83		
Native American or American Indian	57	0.51	5	0.77		
Multiple races or Other	2026	18.06	158	24.27		

Appendix II. *Sensitivity Analyses (Models 1 and 2c, Models 1 and 2d)*

Model 1b. Paths and Effects with Nonwinsorized Total Adverse Events for Internalizing

					95% Confidence Interval	
Effect		Estimate	SE	p	Upper	Lower
Direct	Total Adverse Events → CF	-.082	.053	<.001	-.566	-.358
Direct	Total Adverse Events → YR Internalizing	.137	.010	<.001	.117	.157
Direct	CF → YR Internalizing	-.058	.003	<.001	-.027	-.014
Indirect	Total Adverse Events → CF→ YR Internalizing	.004	9.266^{-4}	<.001	.002	.006

Model 2b. Paths and Effects with Nonwinsorized Total Adverse Events for Externalizing

					95% Confidence Interval	
Effect		Estimate	SE	p	Upper	Lower
Direct	Total Adverse Events → PR Externalizing	.312	.009	<.001	.294	.331
Direct	Total Adverse Events → CF	-.082	.053	<.001	-.566	-.358
Direct	CF → PR Externalizing	-.054	.006	<.001	-.048	.023
Indirect	Total Adverse Events → CF→ PR Externalizing	0.002	$8.046e^{-4}$	.002	$8.923e^{-4}$	.004

Model 1c. Paths and Effects with Controlling for Wave 1 Parent Reported Internalizing

					95% Confidence Interval	
Effect		Estimate	SE	p	Upper	Lower
Direct	Total Adverse Events → PR Internalizing	.050	.008	<.001	.035	.065
Direct	Total Adverse Events → CF	-.082	.059	<.001	-.604	-.373
Direct	CF → PR Internalizing	.007	.005	.300	-.005	.0015
Indirect	Total Adverse Events → CF→ PR Internalizing	$-8.890e^{-4}$	$5.932e^{-4}$	.134	-.002	.001

Model 2c. Paths and Effects with Controlling for Wave 1 Parent Reported Externalizing

					95% Confidence Interval	
Effect		Estimate	SE	p	Upper	Lower
Direct	Total Adverse Events → PR Externalizing	.066	.007	<.001	.052	.080
Direct	Total Adverse Events → CF	-.073	.060	<.001	-.551	-.316
Direct	CF → PR Externalizing	-.010	.004	.121	-.015	.002
Indirect	Total Adverse Events → CF→ PR Externalizing	$4.631e^{-4}$	$4.798e^{-4}$	.334	$-4.772e^{-4}$	.001

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