# Not All Adversity is Created Equal: Differential Associations of Adversity Profiles with Adolescent Cognitive Control and Psychopathology

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## ABSTRACT

Adverse experiences have long-term consequences for biological, behavioral, and psychosocial adjustment. Adolescents may be particularly susceptible to these effects due to heightened sensitivity to environmental influences, the protracted development of the prefrontal cortex, and risk for psychopathology. We used a person-centered approach to characterize distinct profiles of adversity in early adolescence, and examined associations with later cognitive control and psychopathology. One hundred and sixty-seven adolescents (53% male) and their primary caregivers participated in a longitudinal study, with approximately one year in between each assessment. At Time 1 (*M*age = 14.07 years), we collected reports on seven indicators of adversity: socioeconomic disadvantage, abuse, neglect, household chaos, parent substance use, parent depression, and negative life events. At Times 2, 3, and 4, adolescents' behavioral performance and blood-oxygenation-level-dependent response during a cognitive control task were measured. Two years later, at Time 5, adolescents and their caregiver reported on adolescent internalizing and externalizing symptomatology. Using latent profile analysis, we identified three distinct adversity subgroups: a low risk group, a low socioeconomic status (SES)/high parent substance use (SU) group, and a high risk group. Adolescents in the low SES/high parent SU group had the lowest levels of behavioral cognitive control. Furthermore, the low SES/high parent SU group and the high risk group both had significantly higher levels of psychopathology relative to the low risk group. There were no significant group differences with respect to neural cognitive control, and neither neural nor behavioral cognitive control predicted psychopathology. A cumulative risk approach using a mean score of adversity produced a similar

general pattern of results, but obscured the unobserved heterogeneity in adverse experiences. These results highlight the utility of a person-centered approach to the characterization of adversity in adolescence and illustrate distinct developmental consequences for cognitive functioning and psychopathology. We expand upon prior empirical work by demonstrating that the co-occurrence of low SES and parent substance use may place adolescents at increased risk for deficits in behavioral cognitive control, which may be an important target for intervention and prevention efforts.

# Not All Adversity is Created Equal: Differential Associations of Adversity Profiles with Adolescent Cognitive Control and Psychopathology

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## GENERAL AUDIENCE ABSTRACT

During childhood and adolescence, most individuals are exposed to some form of adversity, such as abuse, neglect, poverty, or parent mental illness. These factors can have long-term effects on brain functioning and mental health. Adolescents may be especially affected by adversity because their brain is in an important stage of development and they are also more sensitive to social and environmental influences. The purpose of this study was to better understand if certain patterns of adversity experiences were associated with adolescents' self-regulation abilities and mental health outcomes. We recruited 167 adolescents and their primary caregivers from the community and asked them to report on adolescents' experiences of adversity at age 13-14. Specifically, we asked about socioeconomic status, abuse, neglect, household chaos, parent substance use, parent depression, and negative life events. Once each year for the next three years, adolescents completed a self-regulation task while they were in a Magnetic Resonance Imaging (MRI) machine. We examined their performance on the task as well as their brain activation. Two years later, at 18-19 years old, adolescents and their caregiver reported on the adolescent's mental health symptoms. Results indicated that there were three groups of adolescents with different combinations of adverse experiences: a low risk group, a low socioeconomic status (SES)/high parent substance use (SU) group, and a high risk group. Adolescents in the low SES/high parent SU group had the worst performance on the self-regulation task. Furthermore, both the low SES/high parent SU group and the high risk group had significantly higher mental health problems relative to the low risk group. There were group differences in terms of brain activation. Finally, neither performance nor brain activation during

self-regulation was associated with mental health problems. We also tested these associations by using an average score of adversity, rather than dividing participants into subgroups. When we compared these approaches, the results were generally similar, but the subgroup approach provided more specific information about what types of experiences put adolescents at higher risk for self-regulation and mental health problems. Thus, the subgroup approach may be useful for better understanding the nuanced consequences of adversity. Our findings further show that the co-occurrence of low SES and parent substance use may place adolescents at increased risk for deficits in self-regulation, which may be an important target for intervention and prevention efforts.

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# Chapter 1: Introduction

More than half (62%) of individuals in the United States experience at least one adverse event prior to age 18 (Merrick, Ford, Ports, & Guinn, 2018). While there is no consensus in the field as to what does and does not constitute an adverse experience, adversity can broadly be defined as "experiences which require significant adaptation by the developing child in terms of psychological, social and neurodevelopmental systems, and which are outside of the normal expected environment" (Lacey & Minnis, 2019, p. 2). Thus, adversity may include experiences such as poverty, abuse, neglect, parent psychopathology, and parent separation or divorce. These experiences during childhood and adolescence have profound implications for long-term physical and mental health (Felitti et al., 1998). Extensive work has demonstrated how experience of these events can create a cascading effect, with adversity affecting adjustment across multiple domains of functioning. Importantly, the nature of these effects likely depends on the developmental period in which they occur. For example, in adolescence, the protracted development of the prefrontal cortex (PFC) can leave individuals especially vulnerable to challenges in self-regulatory abilities such as cognitive control (Casey, Galvan, & Hare, 2005). This vulnerability may be compounded by stress effects that alter neurobiological structure and function. Furthermore, adolescence is the period of onset for many psychological disorders (Lee et al., 2014; Paus, Keshavan, & Giedd, 2008). Thus, it is imperative to elucidate the pathways through which adversity impinges on the development of cognitive control and psychopathology in adolescence.

## Theoretical and Methodological Models of Adversity

A number of different theoretical models and methodological approaches have been adopted to better understand the consequences of adversity. The most straightforward methodological approach is to consider one specific type of adversity in the prediction of

adjustment outcomes. For example, a large body of research has tested the effect of maltreatment, independent of other adversities, on psychopathology outcomes in adolescence (see Trickett, Negriff, Ji, & Peckins, 2011 for a review). Testing the effects of adversity in this way allows for specific conclusions regarding the association between a given risk factor and a particular outcome. At the same time, these models are limited in that they do not account for the high co-occurrence of adversities. Indeed, the majority of individuals who report experiencing any given adversity have also been exposed to at least one other adversity (Felitti et al., 1998; McLaughlin et al., 2012a).

To address some of these conceptual limitations, the *cumulative risk model* (Rutter, 1983) has commonly been used to capture the summation of adverse experiences across different domains, including both proximal (e.g., harsh parenting) and distal (e.g., socioeconomic) risk factors. This model is most commonly tested by dichotomizing each risk factor based on whether or not an individual was exposed to that risk, and the values are then summed across the risk categories (Rutter, 1983). Cumulative risk is an additive model, such that each additional risk that an individual is exposed to incrementally increases the likelihood of maladjustment. For example, the number of risk factors experienced in childhood, regardless of the type of risk, has been associated with heightened externalizing symptomatology in middle childhood (Deater-Deckard, Dodge, Bates, & Pettit, 1998). Other multiple-risk metrics have increasingly been implemented through a variety of methodological approaches including summary scores and factor analysis (Evans, Li, & Whipple, 2013). The cumulative risk approach has recently gained traction in clinical practice through Adverse Childhood Experiences (ACEs) initiatives which promote trauma-informed care for children and adolescents who have been exposed to adversity. Although ACEs-informed care provides important insights into risk factors that may underlie psychopathology phenotypes, there are inherent limitations in reducing the complexity of

adversity to a single score (see Lacey & Minnis, 2019 for a review). Importantly, if qualitatively different adverse experiences have different pathways to psychopathology outcomes, then a more nuanced understanding of which experiences impact which pathways to maladjustment will promote increasingly precise and targeted intervention efforts for vulnerable youth.

While cumulative risk models have remained the prevailing approach for some time, more recent models have sought to address the unique features of different adverse experiences. One such model, the *dimensional model of adversity and psychopathology (DMAP)*, posits that two core dimensions underlie most adverse experiences: threat and deprivation (McLaughlin & Sheridan, 2016; Sheridan & McLaughlin, 2014). The threat dimension includes experiences that impose direct harm on the child's physical health or well-being. For example, physical abuse, sexual abuse, and witnessing domestic violence are all experiences that are high on the dimension of threat. Alternatively, some experiences are more accurately characterized by deprivation, or absence of sensory input or fulfillment of needs, such as neglect and poverty. Empirical tests of the DMAP have identified differential outcomes associated with threat versus deprivation. For example, there is evidence for distinct developmental consequences of threat (exposure to violence) versus deprivation (poverty) in adolescence, such that heightened threat was associated with worse emotion regulation whereas deprivation was associated with worse cognitive control (Lambert, King, Monahan, & McLaughlin, 2017). Notably, when compared with a cumulative risk approach (i.e., summed total number of adversities), these patterns of results were obscured. In this way, dimensional models such as the DMAP offer increased specificity in identifying the processes whereby adverse experiences impinge on adaptive adolescent development. At the same time, DMAP reduces adverse experiences into two broad domains, and cannot capture effects of co-occurring threat and deprivation experiences within the same person.

Both cumulative stress and DMAP approaches are *variable-centered*, in that they describe associations between study variables and assume that these associations apply uniformly to all individuals (Laursen & Hoff, 2006). For example, a cumulative risk model would posit that the effect of adversity on adjustment holds similarly across the entire population. However, the effects of adverse experiences are far from homogenous, and the experiences themselves vary widely in type, severity, timing, and chronicity. *Person-centered* approaches are able to capture unobserved heterogeneity by categorizing individuals into subpopulations based on shared attributes (Muthen & Muthen, 2000). Latent class analysis (LCA) and latent profile analysis (LPA) are two such person-centered approaches which identify qualitatively different groups on a set of indicators, and account for the interrelations among the indicators. LCA identifies these groups based on categorical indicators, whereas LPA uses continuous indicators. In this way, specific constellations of adverse experiences can be characterized and can provide insight into how *typologies* of adverse experiences contribute to child and adolescent adjustment outcomes.

While traditional variable-centered techniques have been the prevailing approach in the adversity literature, initial efforts to identify unique profiles of adverse experiences using a person-centered approach have yielded novel insights. For example, Hagan, Sulik, and Lieberman (2015) used LCA to categorize children ages 3-6 based on parent-reported exposure to traumatic events. The results identified three unique classes: severe exposure, witness to violence, and moderate exposure. Importantly, these groups were significantly different from each other on psychopathology outcomes, such that the severe exposure group demonstrated significantly higher internalizing and post-traumatic stress symptoms compared to the other two groups. Using the same assessment of trauma exposure with early adolescents, King, Humphreys, Camacho, and Gotlib (2019) identified a three-class solution (direct victimization,

family instability, and typical/low) which differentially predicted brain volume in stress-sensitive regions. Specifically, the class that was characterized by direct victimization (i.e., high levels of abuse and neglect) had significantly smaller hippocampal volumes than the typical/low class. These results suggest that adverse experiences defined by abuse and neglect are particularly relevant for hippocampal development. Importantly, in both of these studies, individual differences in the outcome variables were not explained by a variable-centered approach following the cumulative risk model (i.e., average or sum of number of events experienced), pointing to the value of more specific or dimensional approaches to the modeling of adversity. To our knowledge, King et al. (2019) is the only study that has attempted to link profiles of adversity to brain development. Their cross-sectional findings regarding differential effects of adverse experiences on brain structure warrant further investigation to see if these patterns similarly extend to differences in brain function over time, especially in other regions that are vulnerable to stress effects, such as the prefrontal cortex.

## Adversity and Adolescent Psychopathology

Adverse experiences are a common feature underlying many different psychopathology phenotypes. Here, we consider adverse experiences that may be particularly germane to the developmental period of adolescence, including socioeconomic disadvantage, maltreatment, home environment, parent mental health, and negative life events.

### *Socioeconomic Disadvantage*

Socioeconomic status (SES) is best understood as the overall social standing of a family and typically includes factors such as parent education, income, and occupation (McLoyd, 1998). Most prior work has focused on the consequences of low SES in early childhood, but there are emerging findings that SES affects adolescent adjustment as well. For example, meta-analytic evidence demonstrates that SES (i.e., family income, education, and occupational prestige) is

consistently associated with antisocial behavior in adolescence (Piotrowska, Stride, Croft, & Rowe, 2015). In addition to objective indicators of SES, subjective social standing is also consistently associated with adolescent mental health outcomes. For example, adolescents who rank themselves lower on social hierarchies relative to their peers have higher odds of anxiety disorders, mood disorders, disruptive behaviors, and substance use disorders (McLaughlin, Costello, Leblanc, Sampson, & Kessler, 2012b). These findings are corroborated by a meta-analysis demonstrating that across studies, subjective SES is associated with adolescent mental health outcomes such as depression, anxiety, and psychological well-being (Quon & McGrath, 2014).

### *Maltreatment*

Maltreatment, including physical abuse, sexual abuse, and neglect, is a clearly established risk factor for adolescent psychopathology (Cicchetti & Toth, 2016). Longitudinal research indicates that maltreatment during childhood is associated with a host of adjustment outcomes in adolescence, including externalizing problems such as delinquency and substance use (see Trickett et al., 2011 for a review) and internalizing problems such as depression (Brown, Cohen, Johnson, & Smailes, 1999). However, the manifestation of these symptoms may depend on developmental timing. For example, Thornberry, Ireland, and Smith (2001) found that maltreatment occurring during adolescence was more strongly associated with adolescent psychopathology, relative to maltreatment occurring only during childhood. Thus, maltreatment occurring within the developmental period of adolescence may be a particularly salient risk factor.

### *Household Chaos*

Household chaos is characterized by noise, crowding, and lack of structure or routine in a home (Wachs & Evans, 2010). Chaos may interfere with adaptive development across a number

of domains (see Marsh, Dobson, & Madison, 2020 for a review), and has demonstrated associations with children's psychological distress (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005) and conduct problems (Deater-Deckard et al., 2009). While most research on household chaos has focused on children, there is initial evidence that higher household chaos is associated with heightened adolescent health risk behaviors (Chatterjee, Gillman, & Wong, 2015) and depression (Tucker, Sharp, Van Gundy, & Rebellon, 2018). Furthermore, household chaos is a potential risk factor for adolescent executive functioning development (Brieant, Holmes, Deater-Deckard, King-Casas, & Kim-Spoon, 2017) which may cascade into later psychopathology.

***Parent Mental Health***

Parent mental health, including both substance use and depression, has also been identified as a risk factor for adolescent development of psychopathology. For example, maternal substance use is associated with major depressive disorder in adolescence (Ohannessian et al., 2004) and parent substance use disorders contribute to increases in substance use across adolescence (Walden, Iacono, & McGue, 2007). Parent depression is also a well-established risk factor for adolescent affective problems, and maternal depression is predictive of both higher levels and longitudinal increases in adolescent depressive symptoms (Garber & Cole, 2010). Together, the co-occurrence of substance use and other mental health problems can exacerbate effects on adolescent psychopathology (Sellers et al., 2013). These effects may be indirect, such that parent mental illness affects adolescent psychopathology through its influence on parenting behaviors such as parental monitoring (Van Loon, Van de Ven, Van Doesum, Witteman, & Hosman, 2014) and disciplinary style (Gallimore & Kurdek, 1992). In addition to environmental factors, the intergenerational transmission of mental health problems is also a function of genetic

inheritance (e.g., Harold et al., 2011) making parent depression and substance use prominent risk factors for adolescent adjustment.

### *Negative Life Events*

Specific life events may occur during adolescence that heighten risk for psychopathology. Negative life events are changes in one's social or physical environment that require an adjustment in life routines (Turner & Wheaton, 1997) and may include events such as parent separation or divorce, death in the family, and getting in trouble at school. Typically, negative life events are assessed with checklists or interviews where participants are asked to indicate which events they or their family have recently experienced (Allen, Rapee, & Sandberg, 2012). During adolescence, there is an increase in the occurrence of negative life events (Ge, Lorenz, Conger, Elder, & Simons, 1994) which may heighten vulnerability to psychopathology. Indeed, in adolescence, more negative life events are associated with both internalizing and externalizing psychopathology (Eisenbarth et al., 2019), depression (Maciejewski, Brieant, Lee, King-Casas, & Kim-Spoon, 2020), and greater substance use (Cheney, Vesely, Aspy, Oman, & Tolma, 2018).

## Adversity and Cognitive Control

Adversity may confer risk for adolescent psychopathology through various different pathways. Indeed, the consequences of adversity cut across multiple domains of functioning, and manifest on both behavioral and neurobiological levels. The ways in which different types of adversity "get under the skin" to impact brain structure and function are still unclear, but seem to be influenced by a number of factors including developmental timing. Theoretical models of stress exposure have posited that the neurobiological effects of adverse experiences may manifest differently depending on developmental stage. For example, Lupien et al. (2009) proposed that stress exposure during early childhood has strongest impact on the hippocampus given the ongoing development of the region during this period. In contrast, during adolescence

the effects of adversity are hypothesized to be strongest in the prefrontal cortex because of the ongoing and protracted development of the region as well as its high density of glucocorticoid receptors which are susceptible to stress (Lupien, McEwen, Gunnar, & Heim, 2009). Thus, cognitive control, which is primarily supported by the prefrontal cortex, may be a particularly salient pathway for the effects of adversity during adolescence.

Cognitive control is best understood as a higher-order cognitive ability involving the flexible regulation of behavior to override an inappropriate response (Casey et al., 1997) and engages both working memory and attention shifting (Carter & Krus, 2012). Given that cognitive control plays an important role in the regulation of behavior, there is considerable conceptual overlap between cognitive control and other self-regulation constructs, especially executive functioning (Nigg, 2017). Across adolescence, the maturation of the prefrontal cortex underlies improvements in self-regulatory abilities. Structurally, gray matter volume begins to decrease and white matter volume increases linearly (Mills et al., 2016). Functionally, there is a linear increase in cognitive control abilities, as evidenced by lower activation in the prefrontal cortex during cognitive control tasks (Luna, Padmanabhan, & O'Hearn, 2010; Ordaz, Foran, Velanova, & Luna, 2013).

There is clear evidence illustrating the effects of adverse experiences on behavioral indicators of cognitive control. In childhood, EF performance is compromised by a host of adverse experiences including caregiver substance use (Roos, Kim, Schnabler, & Fisher, 2016) and socioeconomic disadvantage (see Hackman & Farah, 2009 for a review). Less attention has been given to the effects of adversity on cognitive functioning in adolescence, despite the important cognitive changes that are occurring during this developmental period. Nonetheless, available research indicates that adversity similarly affects EF in adolescence, and is compromised by experiences including maltreatment (Mothes et al., 2015), household chaos

(Brieant et al., 2017), and socioeconomic disparities (Hackman et al., 2014). Initial research testing the effects of different dimensions of adversity has found that experiences characterized by deprivation, rather than threat, are most strongly associated with cognitive control performance (Lambert et al., 2017; Machlin, Miller, Snyder, McLaughlin, & Sheridan, 2019).

With recent advances in developmental cognitive neuroscience, researchers have extended the evaluation of adversity effects on cognitive control to underlying prefrontal structure and function. This has primarily been approached by examining the role of individual adverse experiences, independent of other adversities. For example, abuse and exposure to violence (Carrion, Garrett, Menon, Weems, & Reiss, 2008; Jankowski et al., 2017), socioeconomic disadvantage (Spielberg et al., 2015), chaotic family environment (Kim-Spoon, Maciejewski, Lee, Deater-Deckard, & King-Casas, 2017), and early deprivation (Mueller et al., 2010) have all been linked to aberrant functional activation during cognitive control tasks. Initial research incorporating multiple types of adversity has revealed that ACE scores are associated with lower anterior cingulate cortex (ACC) activation during a go/no-go task in adolescence (Fava et al., 2018). There is also preliminary evidence for differential effects of adverse experiences on adolescent cognitive control. Specifically, lower parent education, but not abuse, was associated with less efficient recruitment of the prefrontal cortex during a working memory task, demonstrating that only certain types of risk factors may be associated with disruptions in cognitive control (Sheridan, Peverill, Finn, & McLaughlin, 2017).

What are the underlying mechanisms linking adversity and cognitive control? Perhaps the most common hypothesis is that adverse experiences generate stress which alters function of the hypothalamus-pituitary-adrenal (HPA) axis and has downstream consequences for physiological, neurobiological, and behavioral development (McEwen, 1998). While there is considerable empirical support for this perspective, the broadness of the construct of stress severely limits

precision in intervention efforts. By contrast, the DMAP theoretical approach offers increased specificity by identifying unique pathways to maladaptation that are dependent on the nature of the adverse experience (i.e., deprivation versus threat). For example, the model posits that adverse experiences characterized by deprivation (e.g., neglect) especially limit expected social and cognitive stimulation. This limited sensory input may contribute to overpruning of synaptic connections in the association cortex, ultimately compromising the cognitive abilities that rely on these connections (Sheridan & McLaughlin, 2014). Thus, adolescents who experience patterns of adversity primarily characterized by deprivation will be particularly vulnerable to impaired prefrontal functioning.

**Cognitive Control and Psychopathology**

Deficits in higher-order cognitive abilities are a transdiagnostic risk factor for psychopathology across development (Martel et al., 2017; Snyder, Miyake, & Hankin, 2015). This is of particular interest during the period of adolescence when individuals are at heightened risk for the emergence of psychopathology due to ongoing neurobiological and behavioral changes (Lee et al., 2014; Paus et al., 2008). Indeed, task-based, behavioral measures of prefrontal functioning have demonstrated that lower cognitive control/executive functioning is associated with heightened internalizing and externalizing symptomatology among adolescents (Brieant, King-Casas, & Kim-Spoon, 2019; Friedman, du Pont, Corley, & Hewitt, 2018). Although few studies have extended this work to involve neurobiological measures of cognitive control, available evidence suggests that individual differences in functional activation during cognitive control is predictive of externalizing symptomatology. For example, lower activation in the ACC during inhibitory control was associated with higher externalizing symptomatology in early adolescence (Fava et al., 2018). It remains unclear how functional activation in the PFC during cognitive control is related to adolescent internalizing symptomatology.

Cognitive control deficits frequently underlie internalizing and externalizing symptomatology because cognitive control is necessary for successful management of negative emotions and arousal (see Banich et al., 2009 for a review). For example, cognitive control protects against core features of depression by overriding negative attention biases and preventing rumination (Davis & Nolen-Hoeksema, 2000; Gotlib & Joormann, 2010). Poor cognitive control may also manifest as impulsive thoughts and actions, thereby contributing to common phenotypes of externalizing disorders such as substance use (Casey & Jones, 2010). These regulatory processes are especially important during adolescence, when the ability to exert cognitive control in socio-emotionally salient contexts is limited (Steinberg, 2008). Thus, individual differences in cognitive control in adolescence may have important implications for long-term mental health and well-being.

**Present Study**

Taken together, the literature demonstrates the complex ways in which adverse experiences intersect to influence adolescent psychosocial adjustment. Consistent with the developmental cascades framework (Masten & Cicchetti, 2010), the effects of adversity in one domain (e.g., cognitive control) are likely to have downstream consequences for psychopathology outcomes. However, there are a number of limitations in current conceptualizations of adversity which inhibit our understanding of the role of heterogeneous adverse experiences in brain and psychopathology development. This in turn prevents practitioners from implementing targeted and precise intervention strategies to effectively disrupt the cascading effects of adversity. Thus, the proposed study had three central aims:

1. Identify different profiles of adverse experiences in adolescence based on type and severity of risk factors.

2. Determine whether adversity profiles differentially predict neural and behavioral indicators of cognitive control.
3. Test the cascading effects of earlier adversity profiles on later psychopathology outcomes, directly and indirectly through cognitive control.

These aims were addressed using latent profile analysis (LPA), a variation of LCA that uses continuous rather than categorical indicators. LPA is a data-driven, exploratory approach which precludes specific hypotheses regarding the classes that may emerge. However, extant theoretical and empirical work indicates that there is meaningful heterogeneity in adverse experiences. Thus, we expected that this person-centered approach would yield at least two distinct subgroups of individuals based on seven different types of adverse experiences: socioeconomic disadvantage, abuse, neglect, parent substance use, parent depression, household chaos, and negative life events. While there are certainly adverse experiences beyond those described here (e.g., early institutionalization, exposure to violence), these particular experiences capture many of the common stressors that adolescents may encounter, and generally align with the domains of adversity described in theoretical models (e.g., Sheridan & McLaughlin, 2014). Based on these theoretical models, we expect that profiles characterized by deprivation (e.g., neglect) will be more strongly associated with prefrontal functioning relative to other profiles. In addition to the direct association between adversity and psychopathology, we hypothesize that individual differences in cognitive control will cascade into psychopathology outcomes, such that adolescents with worse cognitive control will have higher levels of both internalizing and externalizing symptomatology.

## Chapter 2: Method

The current study used five waves of data from the Adolescent Brain Study, an ongoing longitudinal study including 167 adolescents (53% males, 47% females) and their primary caregiver (82% biological mothers, 13% biological fathers, 2% grandmothers, 1% foster, 2% other). Adolescents were 13 or 14 years of age at Time 1 ($M$ = 14.07, $SD$ = 0.54), 14 or 15 years of age at Time 2 ($M$ = 15.05, $SD$ = 0.54), 15 or 16 years of age at Time 3 ($M$ = 16.07, $SD$ = 0.56), and 16 or 17 years of age at Time 4 ($M$ = 17.01, $SD$ = 0.55), with approximately one year in between each time point. Approximately two years later, participants returned for a follow-up study at Time 5 when they were 18 or 19 years of age ($M$ = 18.87, $SD$ = 0.61). Adolescents primarily identified as Caucasian (82%), African-American (12%), and other (6%). Median family income was $35,000 - $49,999 per year at all time points, consistent with the median for the region (United States Census Bureau, 2010). Income ranged from less than $1,000 to greater than $200,000 per year.

At Time 1, 157 families participated. At Time 2, 10 adolescents aged 14-15 years were added to offset annual attrition, for a final sample of 167 parent-adolescent dyads. Across Times 1 through 4, 26 dyads did not participate at all possible time points for reasons including: declined participation ($n$ = 13), lost contact ($n$ = 10), ineligibility for fMRI tasks ($n$ = 2), and extenuating circumstances ($n$ = 1). Thirty-five dyads did not return for the Time 5 follow-up study for reasons including: lost contact ($n$ = 23), declined participation ($n$ = 6), extenuating circumstances ($n$ = 4), and moved away ($n$ = 2). We performed attrition analyses using binomial logistic regression to determine whether there were systematic predictors of missing data. Results indicated that participation was not significantly predicted by baseline demographic (i.e., age, income, sex, and race) or study variables at any of the five time points ($p$s > .05).

### Procedures

Participants were recruited from the community in the Southeastern United States via flyers, recruitment letters, and e-mail. Individuals were ineligible for the study if they had a history of head injury resulting in loss of consciousness for more than 10 minutes, claustrophobia, orthodontia impairing image acquisition, or other contraindications to functional magnetic resonance imaging (fMRI). At Times 1-4, data collection occurred at university offices where adolescents agreed to participation via written assent, parents provided written consent, and the protocol was administered by trained research assistants. Adolescents and their parents received monetary compensation for their time. At Time 5, adolescents followed the same procedures but were all at least 18 years of age and able to provide consent. Parents participated remotely and completed questionnaires via mail or an online survey.

## Measures

### *Socioeconomic Status*

Family SES was measured at Time 1 with a demographic interview administered to parent participants. Total household income before taxes for the previous year was used to calculate an income-to-needs (ITN) ratio for each family. Specifically, income was divided by the poverty threshold for the given family size (according to guidelines from the U.S. Census Bureau). Greater ITN ratios reflect higher socioeconomic status, with ITN $\leq 1$ being defined as poor and ITN $\leq 2$ as near-poor. According to this ratio, 48% of the sample qualified as poor or near-poor. Parents also provided information regarding years of education for themselves and their spouse (if applicable). In addition to these objective indicators, we collected information regarding parents' subjective SES. There were three subjective SES items including, "How often do you worry about your financial situation?" and "How well off would you say your family is?". Subjective SES was significantly correlated with the objective indicators ($r = .34$ with

education; $r$ = .54 with ITN ratio). Objective and subjective indicators were standardized and averaged together to create an overall composite score of SES.

***Household Chaos***

Adolescents reported levels of household chaos at Time 1 on the Confusion, Hubbub, and Order Scale (CHAOS; Matheny, Wachs, Ludwig, & Phillips, 1995). The short version of the scale consists of 6 statements about the individual's home environment *("1 = definitely untrue"* to *"5 = definitely true"*) with higher scores indicating higher levels of chaos. Sample items include "We are usually able to stay on top of things" and "You can't hear yourself think in our home". The scale demonstrates relatively low reliability within the current sample at $\alpha$ = .59, though this is consistent with previous research (Coldwell, Pike, & Dunn, 2006; Pike, Iervolino, Eley, Price, & Plomin, 2006).

***Parent Substance Use***

Parent substance use at Time 1 was assessed based on self-report of three items asking about frequency of use of alcohol, cigarettes, and marijuana. Items were phrased as, "Which is most true for you about smoking cigarettes?" with responses ranging from "*1 = never used*" to "*6 = usually use every day"*. Responses were averaged across the three items and standardized, with higher scores representing more frequent substance use ($\alpha$ = .44).

***Parent Depression***

Parents provided self-report on their symptoms of depression at Time 1 using the Beck Depression Inventory (BDI-II; Beck, Steer, Ball, & Ranieri, 1996). The BDI-II consists of 21 questions, with higher scores representing stronger symptoms of depression. For example, an item assessing sadness includes the following response options: "*0 = I do not feel sad", "1 = I feel sad much of the time", "2 = I am sad all the time",* or *"3 = I am so sad or unhappy that I*

*can't stand it".* The total summed score was used and demonstrated strong reliability in our sample ($\alpha$ = .92).

***Negative Life Events***

At Time 1, parents reported on life events that their child had experienced using the Child and Adolescent Survey of Experiences (CASE; Allen et al., 2012). The CASE is a checklist with 38 items describing positive and negative life events that may disrupt life routines. Parents were asked to report whether or not a given event had occurred in the past 12 months (e.g., "Someone in our family was really sick or injured", "My partner and I split up", "My child had a big argument with someone in our family"). If the event had occurred, parents then rated how good or how bad that event was for the child, from "*1 = really good"* to "*6 = really bad"*. An event was categorized as a negative event if it was rated by the parent as a little bad, quite bad, or really bad. We used the sum of the total number of negative life events that were endorsed. The measure has previously demonstrated predictive validity for anxiety and depression (Allen & Rapee, 2009) and good agreement between parents and children (Allen et al., 2012).

***Maltreatment***

Adolescents' retrospective reports of maltreatment were collected at Time 5 using the Maltreatment and Abuse Chronology of Exposure scale (MACE; Teicher & Parigger, 2015). This scale includes 52 items which evaluate exposure to different types of maltreatment from ages 1 to 18. For each type of maltreatment, participants are asked to indicate at what age(s) they experienced a given event. We used participants' reports of maltreatment experienced at age 13, corresponding with their participation at Time 1. Two composites were calculated: abuse (average of sexual abuse, parental verbal abuse, non-verbal emotional abuse, and physical maltreatment) and neglect (average of emotional neglect and physical neglect). This measure has demonstrated strong test-retest reliability in previous work (Teicher & Parigger, 2015).

***Psychopathology***

At Time 5, adolescents and their parent reported on the adolescent's internalizing and externalizing symptomatology using the Adult Self Report (ASR; Achenbach & Rescorla, 2003) and the Adult Behavior Checklist (ABCL; Achenbach & Rescorla, 2003). Each measure includes 126 items that assess behavior problems on a 3-point scale, ranging from *"0 = not true"* to *"2 = very true or often true"*. The internalizing scale includes withdrawn, anxious/depressed, and somatic complaints syndrome scales. The externalizing scale is comprised of aggressive, rule-breaking, and intrusive behavior scales. In our sample, both the ASR (externalizing scale $\alpha$ = .87; internalizing scale $\alpha$ = .91) and the ABCL (externalizing and internalizing scales $\alpha$ = .92) demonstrated strong reliability. Bivariate correlations between *T*-scores for parent and adolescent reports of psychopathology were moderate and statistically significant ($r$ = .39, $p < .001$ for externalizing symptomatology; $r$ = .44, $p < .001$ for internalizing symptomatology). Thus, *T*-scores were averaged to create a multi-informant report variable for internalizing symptomatology and externalizing symptomatology. If both reports were not available (i.e., only parent report or only adolescent report was acquired), we used single-informant reports instead of an average (n = 19)[1].

***Cognitive Control***

Cognitive control was measured at each time point with the Multi-Source Interference Task (MSIT; Bush & Shin, 2006). Participants were presented with sequences of three digits, two of which were identical (Figure 1A). Participants were instructed to indicate the identity (but not the position) of the unique, target digit. In the neutral condition, target digits were congruent with position (e.g., "2" is in the second position in the sequence "020"). In the interference

[1] Models were also tested using only adolescent report of psychopathology and only parent report of psychopathology. Results were highly consistent. To capitalize on the multi-informant data available, we report the results with the combined parent-adolescent report here.

condition, target digits were incongruent with position (e.g. “3” was in the second position in the sequence “131”). Four blocks of 24 interference trials and 4 blocks of 24 neutral trials were interleaved with an interstimulus interval of 1.75 seconds. To assess task performance, we calculated intraindividual variability in reaction time, indexed as intraindividual standard deviations (ISDs; MacDonald, Karlsson, Rieckmann, Nyberg, & Backman, 2012) for correct responses in the interference condition. Studies examining the role of intraindividual variability (IIV) of reaction time demonstrate the importance of IIV as an indicator of cognitive functioning (e.g., Hultsch, MacDonald, & Dixon, 2002). In our sample, accuracy was negatively correlated with ISD, suggesting that greater variability in reaction time during the MSIT was associated with worse performance on the task and thus worse inhibitory control. ISDs were reverse-scored such that higher scores represented better cognitive control. Blood-oxygenation-level-dependent (BOLD) response was monitored during the task using functional magnetic resonance imaging (fMRI).

**Imaging Acquisition and Analysis.** Functional neuroimaging data for the MSIT were acquired on a 3T Siemens Tim Trio MRI scanner with a standard 12-channel head matrix coil. Structural images were acquired using a high-resolution magnetization prepared rapid acquisition gradient echo sequence with the following parameters: repetition time (TR) = 1200 ms, echo time (TE) = 2.66 ms, field of view (FoV) = 245x245 mm, and 192 slices with the spatial resolution of 1x1x1 mm. Echo-planar images were collected using the following parameters: slice thickness = 4mm, 34 axial slices, FoV = 220x220mm, TR = 2 s, TE = 30 ms, flip angle = 90 degrees, voxel size = 3.4x3.4x4 mm, 64x64 grid, and slices were hyperangulated at 30 degrees from anterior-posterior commissure. Imaging data were preprocessed and analyzed using SPM8 (Wellcome Trust Neuroimaging Center). For each scan, data were corrected for head motion using a six-parameter rigid body transformation and realigned. The mean functional

image was co-registered to the anatomical image, then the anatomical image was segmented and registered to the MNI template and functional volumes were normalized using parameters from the segmented anatomical image, and were smoothed using a 6mm full-width-half-maximum Gaussian filter.

Preprocessed scans were entered into a GLM for each participant using SPM8. Interference and neutral trials were modeled using a boxcar function convolved with a canonical hemodynamic response function. A low-pass filter with applied cutoff of 168 seconds was used to remove low-frequency noise. To control for the effect of motion, six motion realignment parameters were included as nuisance covariates. Framewise displacement (FD) was calculated using the head realignment parameters (Power, Barnes, Snyder, Schlaggar, & Petersen, 2012; Siegel et al., 2014). Volumes with FD > 0.9mm were scrubbed by adding a TR-specific regressor to the GLM for each censored volume. For each participant and for each time point, beta maps for the neutral condition were subtracted from the beta maps of the interfere condition to create interference minus neutral contrasts. For each time point, interfere minus neutral contrast images were entered into a second level one-sample *t*-test using the root mean square of frame displacement as a regressor of no interest to account for age-related changes to within-scanner motion (Satterthwaite et al., 2012). A significant MSIT interference effect on BOLD response was observed at each time point (Figure 1B-D), consistent with previously reported effects of the MSIT.

Individual-level ROI values were extracted for each participant (within-wave at Times 2, 3, and 4) using a gray matter mask and an a priori 6mm radius spherical mask centered at the MNI coordinate of peak activation in three clusters identified in the meta-analysis reported by Deng, Wang, Wang, and Zhou (2018), including dACC/MPFC/SMA (dorsal anterior cingulate cortex/medial prefrontal cortex/supplementary motor area; $x = 4$, $y = 14$, $z = 48$), R insula/R

IFG/R PUT (right insula, right inferior frontal gyrus, right putamen; $x = 42$, $y = 20$, $z = -2$), and L preCG/L IFG (left precentral gyrus and left inferior frontal gyrus; $x = -52$, $y = 2$, $z = 22$). The first eigenvariate values of the contrast images were extracted after adjusting for an $F$-contrast of the effects of interest. These three clusters were averaged together to create a composite score of neural activation during the MSIT at each wave. At each wave, composite neural activation scores were weakly or non-significantly correlated with task performance indexed by ISD (Time 2 $r = .03$, $p = .78$; Time 3 $r = .19$, $p = .05$; Time 4 $r = .11$, $p = .23$). However, the positive valence of these correlations and evidence from prior MSIT studies using whole-brain analyses (e.g., Kim-Spoon, Maciejewski, et al., 2017) suggest that higher ISD/worse cognitive control performance was associated with higher BOLD response in the regions of interest. The neural variable was reverse-scored such that higher values represented better cognitive control.

## Chapter 3: Plan of Analysis

Skewness and kurtosis were examined for all variable distributions with acceptable levels less than 3 and 10, respectively (Kline, 2011) and all variables were normally distributed. Outliers were identified as values ≥ 3.29 SD from the mean. In these cases, values were winsorized to retain statistical power and attenuate bias resulting from elimination. GLM indicated that demographic covariates including age, race, and gender were not significantly associated with cognitive control or psychopathology outcomes (*p*s > .05), with the exception of the association between race and externalizing symptomatology. Specifically, non-White participants reported significantly higher externalizing symptomatology compared to White participants. Thus, we controlled for this effect in all models. Little's MCAR test (Little, 1988) indicated that patterns of missing data on model variables were completely random ($\chi^2 = 59.92$, $df = 54$, $p = .27$), so full information maximum likelihood (FIML) estimation was used to handle missing data. FIML uses maximum likelihood estimation based on all available data, and is superior to alternative missing data methods such as listwise deletion or imputation (Enders & Bandalos, 2001). Variables with variances greater than 10 were rescaled (i.e., divided by a constant of 10) in order to facilitate model convergence. This included the measures of parent depression and internalizing and externalizing symptomatology.

### Latent Profile Analysis

Latent profiles of adversity at Time 1[2] were estimated using LPA in M*plus* version 8.3 (Muthén & Muthén, 1998-2019). LPA serves to identify the smallest number of latent profiles that describe the associations between the continuous indicators. We progressively fit models with an increasing number of profiles. The optimum number of profiles was determined by

[2] Ten participants that did not have data at Time 1 because they were recruited at the second time point; thus, we used their data from their first visit (Time 2). T-tests indicated that there were no significant differences on the adversity indicators between those with baseline data at Time 1 versus at Time 2.

model fit based on criteria as recommended by Tofighi and Enders (2007) and Dziak, Lanza, and Tan (2014). Sample-size adjusted Bayesian information criterion (SABIC) maximizes likelihood and parsimony, and lower SABIC values indicate better model fit. The Lo-Mendell-Rubin (LMR) test and bootstrap likelihood ratio test (BLRT) were used to compare the *k*-class model to a *k-1* class solution. Finally, entropy values were used to demonstrate the probability that a model accurately classified participants. Theoretical meaningfulness of the profiles was also considered, and profiles that included less than 5% of the sample were not considered meaningful. Class membership was exported as a manifest variable for inclusion in the full path model.

**Developmental Cascade Model**

Path analysis was conducted in M*plus* to test the effects of the categorical adversity variable at Time 1 on cognitive control (averaged across Times 2, 3, and 4), and the effect of cognitive control on psychopathology outcomes at Time 5. We also tested the direct effect of the adversity profiles on psychopathology outcomes. Two separate models were tested for 1) behavioral indices and 2) neural indices of cognitive control. RMSEA values of less than .05 were considered a close fit while values less than .08 were considered a reasonable fit (Browne & Cudeck, 1993). CFI values of greater than .90 were considered an acceptable fit while values greater than .95 were considered an excellent fit (Bentler, 1990).

**Supplementary Analyses**

After identifying the final model, we tested the same model with the cumulative risk approach in order to compare the efficacy of each approach in predicting cognitive control and psychopathology. We tested a common latent factor approach, using confirmatory factor analysis to verify the structure of the latent factor based on the seven adversity indicators. If the model demonstrated good fit and factor loadings were significant, we planned to test the latent factor as

a predictor of cognitive control and psychopathology. If the latent factor did not demonstrate acceptable fit, we planned to use an average score of the standardized indicators based on their face validity.

**Power Analysis**

To estimate power, we started with hypothetical effect sizes representing bivariate correlations of .10, .30 and .50 (or values for $f^2 = r^2/[1-r^2]$ as .02, .15 and .35, as described by Cohen, 1988), using sample sizes of 167 in G-power. Statistical power for each effect size—even for the smallest estimated effects—was sufficient and approached asymptote (> .995). There is no minimum sample size requirement for latent class analyses, and sample size requirement for LCA has been shown to rely upon several factors including relative sample size and measurement strength (i.e., discrepancy in item endorsement across classes) (Dziak et al., 2014). Monte Carlo simulation studies have been conducted in an effort to specify power requirements for identifying stable estimates for latent subgroups. Based on these simulations, when measurement strength is high and when the class sizes are similar, 87% power for the BLRT is achieved for a medium effect size (Cohen's $w = .35$) with a sample size of 131 and 93% power is achieved for a large effect size (Cohen's $w = .51$) with a sample size of 62 (Dziak et al., 2014). Thus, our sample size of 167 was sufficient to detect latent subclasses of adversity.

# Chapter 4: Results

Descriptive statistics and correlations are presented in Table 1. Cognitive control was significantly correlated across Times 2, 3, and 4 ($r$ = .51 - .60 for behavioral; $r$ = .20 - .26 for neural). Scores were averaged across the three time points to create composite variables for behavioral and neural cognitive control, separately.

## Latent Profile Analysis

Model fit statistics for each class solution from the LPA are presented in Table 2. SABIC decreased with each additional class. While the ALMR suggested that model fit did not significantly improve with increased classes, the BLRT was significant. It is not uncommon for different fit indices to point to different solutions in mixture modeling, and so we also considered substantive usefulness and theoretically-based expectations in our interpretations of model fit (Collins & Lanza, 2010; Grimm, Mazza, & Davoudzadeh, 2017). The three-class solution had better fit than the two-class solution, had acceptable entropy (.81), and had clearer interpretability than the four-class solution. Classification accuracy was also high, with class membership probabilities ranging from .81 to .94. For these reasons, the three-class solution was selected as the final model.

Class differences on the seven indicators were tested using one-way multivariate analysis of variance (MANVOA). $F$-tests were significant for all indicators of adversity ($p$s < .01). Post-hoc comparisons were tested with Tukey's Honestly Significant Difference (HSD) test; mean differences, standard errors, and $p$ values for all contrasts are presented in Table 3, and inter-class differences are presented visually in Figure 2. The majority of participants belonged to Class 1 (71%) and demonstrated the lowest level of risk on all variables relative to the other two classes. Thus, Class 1 was labeled as the "Low Risk" group. Class 2 included 21% of the sample and had moderate levels of risk, but was particularly characterized by the lowest levels of SES

and highest parent substance use (SU). This class was labeled “Low SES/High Parent SU”. Class 3 was the smallest (8%) and was labeled as “High Risk” given that it demonstrated the highest level of risk on all other variables.

Pearson chi-square tests were used to test group differences on gender and race (White versus non-White). Group membership was related to gender ($\chi^2 = 8.83$, $df = 2$, $p = .01$), and females were overrepresented in the high-risk group (n = 11 females, n = 2 males). Follow-up *t*-tests indicated that across the entire sample, females reported significantly higher levels of abuse ($t(102.39) = -2.53$, $p = .01$) and parent depression ($t(165) = -2.17$, $p = .03$) relative to males. There were no significant differences in race between the groups ($\chi^2 = 5.61$, $df = 2$, $p = .06$).

**Associations between Adversity, Cognitive Control, and Psychopathology**

***Behavioral Cognitive Control***

The path model with behavioral cognitive control as a mediator demonstrated acceptable model fit ($\chi^2 = 6.05$, $df = 3$, $p = .11$, RMSEA = .078, CFI = .96). The categorical adversity profile variable significantly predicted both internalizing symptomatology ($b = 0.50$, $SE = 0.11$, $p < .001$) and externalizing symptomatology ($b = 0.48$, $SE = 0.13$, $p < .001$). However, adversity profile did not predict performance on the MSIT ($b = -0.01$, $SE = 0.01$, $p = .08$) and MSIT performance did not predict internalizing symptomatology ($b = -0.26$, $SE = 2.10$, $p = .90$) or externalizing symptomatology ($b = -2.23$, $SE = 1.92$, $p = .25$). See Figure 3 for standardized estimates.

***Neural Cognitive Control***

The path model with neural cognitive control as a mediator demonstrated acceptable model fit ($\chi^2 = 4.89$, $df = 3$, $p = .18$, RMSEA = .06, CFI = .98). The categorical adversity profile variable significantly predicted both internalizing symptomatology ($b = 0.52$, $SE = 0.11$, $p < .001$) and externalizing symptomatology ($b = 0.49$, $SE = 0.13$, $p < .001$). However, adversity

profile did not predict neural activation during the MSIT ($b$ = 0.04, $SE$ = 0.03, $p$ = .15) and neural activation during the MSIT did not predict internalizing symptomatology ($b$ = -0.22, $SE$ = 0.34, $p$ = .52) or externalizing symptomatology ($b$ = 0.09, $SE$ = 0.28, $p$ = .75). See Figure 4 for standardized estimates.

***Post-hoc Contrasts***

Means of the outcome variables for each group are presented in Figure 5. To examine the specific effects of the different adversity profiles on psychopathology, dummy-coded grouping variables were entered as predictors in the full path model. Estimates for class differences in internalizing and externalizing symptomatology were highly similar between the model with the behavioral mediator versus the model with the neural mediator; estimates are presented in parentheses for behavioral / neural. First, we tested contrasts with the low risk group as the reference group. Results indicated that internalizing symptomatology was significantly higher for the low SES/high parent SU group ($b$ = 0.53/0.56, $SE$ = 0.22/0.22, $p$ = .02/.01) and the high risk group ($b$ = 0.98/1.00, $SE$ = 0.29/0.29, $p$ = .001/.001) relative to the low risk group. Similarly, externalizing symptomatology was significantly higher for the low SES/high parent SU group ($b$ = 0.39/0.41, $SE$ = 0.19/0.20, $p$ = .04/.04) and the high risk group ($b$ = 1.03/1.04, $SE$ = 0.26/0.26, $p$ < .001/< .001) relative to the low risk group. Next, we tested the same contrasts with the high risk group as the reference group. The low SES/high parent SU group had significantly lower externalizing symptomatology compared to the high risk group ($b$ = -0.64/-0.64, $SE$ = 0.29/0.30, $p$ = .03/.03). The low SES/high parent SU group and the high risk group did not significantly differ on internalizing symptomatology ($b$ = -0.45/-0.44, $SE$ = 0.34/0.34, $p$ = .18/.19). We note that power may have been low for contrasts with the high risk group given its small size; thus, we interpret non-significant differences with caution.

Post-hoc contrasts also indicated group differences in behavioral cognitive control. With the low risk group as the reference group, results indicated that the low SES/high parent SU group had significantly worse behavioral cognitive control relative to the low risk group ($b$ = -0.02, $SE$ = 0.01, $p$ = .04). The difference in behavioral cognitive control between the low risk group and the high risk group was not significant ($b$ = -0.01, $SE$ = 0.01, $p$ = .27). Finally, with the high risk group as the reference group, the low SES/high parent SU group was not significantly different from the high risk group ($b$ = -0.003, $SE$ = 0.01, $p$ = .80).

None of the groups were significantly different in regards to neural cognitive control. With the low risk group as the reference group, results indicated that neither the low SES/high parent SU group ($b$ = 0.07, $SE$ = 0.05, $p$ = .16) nor the high risk group ($b$ = 0.06, $SE$ = 0.08, $p$ = .41) were significantly different from the low risk group. Finally, with the high risk group as the reference group, the low SES/high parent SU group was not significantly different from the high risk group ($b$ = 0.01, $SE$ = 0.08, $p$ = .95).

**Comparison to Cumulative Risk Approach**

We tested a CFA model with the seven adversity indicators loading onto a single latent factor. All parameters were freely estimated and the measurement model demonstrated poor fit ($\chi^2$ = 34.41, $df$ = 14, $p$ = .002, RMSEA = .09, CFI = .78). Thus, we instead created a composite score based on the seven adversity variables to represent cumulative risk. Each adversity variable was standardized and variables were reverse-scored as needed so that a higher composite score represented greater cumulative risk.

We tested the full path models as described above using the cumulative risk composite rather than the categorical profile variable. For the behavioral model, model fit was acceptable ($\chi^2$ = 5.26, $df$ = 3, $p$ = .15, RMSEA = .07, CFI = .98). Results indicated that higher cumulative risk was associated with higher internalizing symptomatology ($b$ = 0.64, $SE$ = 0.12, $p$ < .001) and

externalizing symptomatology ($b$ = 0.63, $SE$ = 0.13, $p < .001$). Furthermore, higher cumulative risk was associated with worse behavioral cognitive control ($b$ = -0.02, $SE$ = 0.01, $p$ = .002). The neural model also demonstrated acceptable fit ($\chi^2$ = 4.37, $df$ = 3, $p$ = .22, RMSEA = .05, CFI = .98) but cumulative risk was not significantly associated with neural activation during cognitive control ($b$ = 0.03, $SE$ = 0.04, $p$ = .40). Standardized estimates are presented in Figure 6.

To test empirical similarity between the cumulative risk composite and categorical latent profiles, we explored differences in cumulative risk between the three groups identified in the latent profile analysis. MANOVA indicated that the groups were significantly different from each other on levels of cumulative risk [$F(2)$= 126.96, $p < .001$]. The low risk group had significantly lower cumulative risk relative to the low SES/high parent SU group and the high risk group ($p < .001$). The high risk group had significantly higher cumulative risk relative to the low SES/high parent SU group ($p < .001$).

# Chapter 5: Discussion

By the age of 18, most adolescents have been exposed to at least one type of adversity (Merrick et al., 2018) and these experiences are predictive of long-term physical and mental health. However, it remains unclear how and why adversity exerts its influence on these domains of functioning, especially during adolescence when individuals are experiencing dramatic social and neurobiological changes. Traditional variable-centered approaches to the conceptualization of adversity may not be well-suited to delineate the mechanisms whereby adverse experiences confer risk for adolescent outcomes because they do not fully account for heterogeneity in experiences. Thus, the current study sought to leverage a person-centered approach in order to identify unique constellations of adversity exposure in adolescence and examine prospective associations with behavioral and neural cognitive control and psychopathology. Specifically, through a developmental psychopathology framework, we examined how the effects of adversity unfold from early adolescence to late adolescence, cascading into different domains of functioning over time and across multiple levels of analysis (Masten & Cicchetti, 2010). Results indicated three unique profiles of adversity: a low risk group, a low SES/high parent substance use group, and a high risk group. These groups significantly differed on levels of behavioral cognitive control as well as levels of psychopathology five years later. These findings offer a level of nuance that was not captured with a cumulative risk approach.

## Patterns of Adolescent Adversity

Using LPA, we identified qualitatively distinct profiles of adversity in early adolescence based on seven forms of risk: socioeconomic disadvantage, abuse, neglect, parent substance use, parent depression, household chaos, and negative life events. The low risk group was characterized by the lowest levels of risk across all indicators. The low SES/high parent SU group was characterized by the highest socioeconomic disadvantage and the highest parent

substance use. Finally, the high risk group was characterized by the most severe exposure to adverse experiences (except socioeconomic disadvantage and parent substance use). The low risk group had the greatest prevalence (71%) which was not surprising given the community sample, and the high risk group was the smallest group (8%). Each of the seven indicators significantly differed between the classes, suggesting that the constructs that we selected meaningfully contributed to the characterization of adolescent adverse experiences.

Prior studies using person-centered approaches to differentiate adverse experiences have similarly identified unique subgroups. Despite considerable variations in sample size, adversity exposure, and participant demographics, most studies have identified three to four qualitatively distinct profiles. Much of this work has been conducted with children (e.g., Hagan et al., 2015) or adults (e.g., Cavanaugh, Petras, & Martins, 2015; McLafferty et al., 2015), and does not capture the critical developmental period of adolescence. However, there are a couple of studies that have used person-oriented approaches (LCA) to characterize adversity in early adolescence. In a community sample of 9-13 year olds, King et al. (2019) found three classes of early life stress which were distinguished by family instability (e.g., parental divorce, family fights) and victimization (e.g., abuse and neglect). Similar to our findings, they found the greatest prevalence (60%) in a typical/low group which had relatively low probability of family instability or victimization. In early adolescence (ages 11 to 14), Dunn et al. (2011) identified four classes based on experiences including family relationships, family physical and mental health, economic circumstances, and abuse. Again, the low exposure class had the greatest prevalence relative to moderate, severe, and atypical parenting classes. In these studies, distinctive patterns of adverse experiences significantly predicted early adolescent outcomes, including variations in regional brain volume and psychopathology. The current study is the first to extend prior research by investigating whether distinctive patterns of adverse experiences in

early adolescence may be associated with cognitive control during middle-to-late adolescence and subsequently with psychopathology during the transition to young adulthood.

While we hypothesized that latent profiles may be distinguished by experiences of threat versus deprivation according to dimensional theoretical models (DMAP; Sheridan & McLaughlin, 2014), the subgroups identified in our sample did not clearly align with these dimensions. For example, we identified a group that was characterized by particularly high parent substance use and low SES. However, the DMAP does not account for the experience of parent substance use. Furthermore, socioeconomic disadvantage is not always included in empirical tests of DMAP because it can manifest as either threat or deprivation. However, our inclusion of risk factors beyond threat and deprivation yielded results indicating that each of these factors are important considerations for the conceptualization of adversity exposure during adolescence.

Prior studies using LCA have not found significant differences between adversity classes on demographic factors including gender (e.g., Dunn et al., 2011; Hagan et al., 2015; King et al., 2019). However, our results indicated that females were more likely to belong to the high risk group. Across the entire sample, females reported significantly higher levels of abuse and parent depression relative to males, which may have contributed to their overrepresentation in this group. Indeed, female children and adolescents experience higher rates of sexual abuse (Stoltenborgh, Van Ijzendoorn, Euser, & Bakermans-Kranenburg, 2011) which may have contributed to these differences. Yet, at the same time, male adolescents experience higher rates of physical abuse than females (Titus et al., 2003). We find no particular reason why parents' self-reported levels of depression would differ by their adolescent's gender. Given the small size of the high risk group, future replication will be necessary in order to determine whether female adolescents are more likely to experience certain patterns of adversity.

**Associations with Cognitive Control**

Given the protracted development of the prefrontal cortex during adolescence (Casey, 2015), stress or trauma may have particularly pronounced effects on the cognitive control system. Our results revealed that there were significant differences on behavioral indices of cognitive control between the profiles. Specifically, the low SES/high parent SU group demonstrated the worst cognitive control. Both of these risk factors have independently been linked to deficits in self-regulation. In children, lower SES has consistently been linked to lower executive functioning (Lawson, Hook, & Farah, 2017) and in adolescence, poverty has been shown to predict lower cognitive control performance (Lambert et al., 2017). Adolescents from poor families are less likely to receive environmental stimulation when their brain is still malleable and plastic (Bradley & Corwyn, 2002; Rosen, Sheridan, Sambrook, Meltzoff, & McLaughlin, 2018), and they may allocate substantial cognitive resources to navigating challenges associated with low SES (Mani, Mullainathan, Shafir, & Zhao, 2013). These effects may be compounded by the co-occurrence of parent substance use in the home. While parent substance use may not inflict direct harm on the adolescent, it can affect more proximal factors such as caregiving behaviors and attachment (Solis, Shadur, Burns, & Hussong, 2012) which have been linked to adolescent self-regulation (Brody & Ge, 2001). In particular, a recent study demonstrated a significant longitudinal association between a family multiple-risk index (measured by socioeconomic adversity, household chaos, and family risk-taking behaviors including parent substance use) and adolescent self-regulation, measured by delay discounting performance (Kim-Spoon et al., 2019). There may also be genetic factors at play; parents' use of substances may be due in part to their own self-regulatory deficits, and these deficits may be genetically transmitted to their offspring (Deater-Deckard, 2014). Our findings underscore that

the co-occurrence of low SES and high parent SU may exacerbate the effects on adolescent self-regulation, thereby resulting in the lowest behavioral cognitive control between the groups.

While there were meaningful group differences for behavioral performance during cognitive control, these differences did not emerge on a neurobiological level. That is, participants demonstrated similar BOLD responses during the MSIT, regardless of their adversity profile. This seems to contradict previous work that has demonstrated the effects of adversity on adolescent brain structure (e.g., King et al., 2019; Walsh et al., 2014) and function (e.g., Fava et al., 2018; Sheridan et al., 2017). At the same time, there are key methodological differences that may account for these discrepancies. Most importantly, this is the only study to our knowledge to isolate the effects of adversity experienced *during adolescence* on the adolescent brain. Other studies have collected reports of adversity based on childhood exposure (Fava et al., 2018), cumulative lifetime exposure (King et al., 2019), or a combination (Sheridan et al., 2017). These temporal differences in measurement of adversity likely have important implications for effects on brain structure and function, and early/cumulative stress may have stronger effects than stress modeled at just one time point. Indeed, in a sample of young adults, Birn, Roeber, and Pollak (2017) found that functional activation during reward processing was predicted by early childhood stress exposure, but not concurrent stress exposure. Although we expected the effects of adolescent adversity to predict brain function given the ongoing development of the prefrontal cortex during adolescence (Lupien et al., 2009), other levels of complexity such as adversity chronicity or latency of effects on the brain may also be at play. Additional longitudinal research will be necessary to clarify these nuances, spanning childhood and adolescence in order to identify sensitive periods where adversity is most likely to exert its influence on different regions of the developing brain.

From a measurement viewpoint, the discrepancy between behavioral and neural cognitive control was not surprising given that behavioral performance during the MSIT was weakly or not significantly correlated with the magnitude of the BOLD response in the selected regions of interest. In Bush et al.'s (2003) development of the MSIT, they found significant associations between imaging and performance data, such that higher reaction times were associated with greater BOLD response. However, the ROIs in the current study were selected based on reliable functional activation across studies (Deng et al., 2018), independent of behavioral performance. Thus, the ROIs selected based on this meta-analysis may be reliable and replicable indicators of functional activation, but not necessarily clear correlates of behavioral performance in adolescence.

**Associations with Psychopathology**

The three adversity profiles were differentially associated with reports of psychopathology five years later. Specifically, the low SES/high parent SU group and the high risk group reported significantly higher levels of both internalizing and externalizing symptomatology relative to the low risk group. This is consistent with extensive literature which demonstrates that stressful experiences broadly confer risk for later psychopathology in youth (Evans et al., 2013). Relative to the low risk group, the other two groups demonstrated elevated risk on each of the adversity indicators. Exposure to multiple different forms of risk has been associated with greater adolescent internalizing and externalizing symptomatology across both person-centered (Dunn et al., 2011; St Clair et al., 2015) and variable-centered (Fava et al., 2018; Luby, Barch, Whalen, Tillman, & Belden, 2017) approaches. Importantly, this literature has largely examined early childhood adversity and its association with later adolescent or adult adjustment; few studies have focused on adversity occurring during the period of adolescence. However, developmental timing of stress exposure is a critical factor in the prediction of

psychosocial outcomes (Dunn, Nishimi, Powers, & Bradley, 2017) and adolescence has been given far less attention than early childhood adversity. Our findings offer evidence that adverse experiences in early adolescence have important long-term implications for psychological adjustment.

Comparisons between the low SES/high parent SU group and the high risk group revealed additional nuance in psychopathology outcomes following adversity exposure. While the two groups were comparable on levels on internalizing symptomatology, the high risk group reported significantly higher externalizing symptomatology. The specific features of adversity experienced by the high risk group may explain their heightened susceptibility to externalizing problems. Compared to the low SES/high parent SU group, this group demonstrated significantly higher levels of abuse, neglect, parent depression, and household chaos. This finding is consistent with prior research indicating that maltreatment was linked to behaviors such as aggression and rule-breaking (Kim, Cicchetti, Rogosch, & Manly, 2009). However, few studies have examined the effects of abuse and neglect relative to other types of adverse experiences. As one exception, Negriff (2020) found that maltreatment was related to adolescent internalizing and externalizing symptomatology above and beyond other adversity factors, including caregiver substance use. Furthermore, household chaos has been linked to adolescent externalizing behaviors (e.g., substance use), even after adjusting for depression (Chatterjee et al., 2015). Finally, while parent depression is a clearly-established risk factor for offspring internalizing disorders (e.g., Garber & Cole, 2010), less research has tested whether it also increases risk for externalizing disorders. However, there is some evidence that maternal depression is associated with both internalizing and externalizing symptoms in youth (O'Connor, Langer, & Tompson, 2017). Together, the co-occurrence of these risk factors may particularly increase risk for adolescent externalizing symptomatology. However, additional research will be necessary to

more clearly delineate how and why maltreatment, parent psychopathology, and maladaptive household environments operate in concert to place youth at particular risk for developing externalizing symptomatology.

**Cognitive Control and Psychopathology**

Contrary to our hypotheses, neither behavioral nor neural indicators of cognitive control were significantly associated with internalizing or externalizing symptomatology. This appears to contradict previous findings demonstrating the link between cognitive control and adolescent adjustment (Fava et al., 2018; Friedman et al., 2018). At the same time, contemporary models of adolescent brain development emphasize the *joint development* of cognitive control with socioemotional systems (Casey, Getz, & Galvan, 2008; Steinberg, 2008). Our cognitive control task was affectively neutral and was not designed to explicitly recruit limbic regions associated with emotion processing. However, youth who have experienced trauma are more likely to have heightened emotion reactivity (McLaughlin et al., 2010; Weissman et al., 2019). This heightened reactivity requires modulation by the prefrontal cortex, which is still developing during adolescence. Thus, while we did not observe a direct effect between cognitive control and psychopathology, it may serve as an important moderator. This idea is consistent with theoretical perspectives and empirical evidence illustrating the interface between the valuation system (e.g., emotional and motivational reactivity) and the regulatory system (e.g., cognitive control) (Kim-Spoon, Kahn, et al., 2017). For example, the interaction between cognitive control and self-reported affect was predictive of adolescent psychopathology, such that higher positive affect was associated with lower externalizing symptomatology, but only for adolescents with poor neural cognitive control (Brieant et al., 2018). Thus, future work may benefit from the consideration of cognitive control over emotionally salient events/information in pathways to adolescent psychopathology. This perspective is also consistent with a transdiagnostic model of

trauma and psychopathology positing emotion processing (i.e., heightened emotion reactivity and poor emotion regulation) as a primary mechanism whereby adverse experiences impact internalizing and externalizing symptomatology (McLaughlin, Colich, Rodman, & Weissman, 2020). Importantly, additional putative pathways are proposed in this transdiagnostic model, including accelerated biological aging (e.g., pubertal timing) and social information processing (e.g., enhanced threat detection), which were not tested in our study and may serve as alternative explanations.

**Comparison to Cumulative Risk Approach**

In order to evaluate the utility of a person-centered approach to characterizing adversity, we tested the final models with the alternative cumulative risk approach. Given our access to continuous indicators of adversity, we were able to calculate a composite score that accounted for the severity of exposure on each indicator. Results were largely similar to the models that used the latent profile approach. Specifically, higher cumulative risk was associated with worse behavioral cognitive control and higher levels of internalizing and externalizing psychopathology. This finding corroborates extensive prior work demonstrating the dose-response effect of youth adverse experiences (Evans et al., 2013).

While the two approaches yielded similar broad conclusions, our analyses based on a person-oriented approach revealed distinct subgroups within the sample which were obscured by the cumulative risk approach. Importantly, these subgroups demonstrated unique associations with adolescent outcomes, indicating that there was meaningful, unobserved heterogeneity within the sample. The cumulative risk approach has been essential in establishing a foundation of knowledge regarding how adversity broadly impacts cognition and mental health in children and adolescents. However, the challenge facing developmental and clinical science going forward will be introducing additional precision in these predictions in order to identify distinct

pathways to maladjustment and optimize intervention and prevention efforts. Person-centered approaches can aid in these efforts by identifying which types of psychosocial outcomes are most strongly associated with what specific patterns of adversity.

**Strengths and Limitations**

The current study benefitted from a number of strengths that help advance our understanding of adolescent adversity, cognitive control, and psychopathology. First, we used a prospective, longitudinal study design. In this way, we strengthened inferences regarding directionality of effects and limited potential bias introduced by retrospective reports of adverse experiences. We also adopted multiple levels of analysis, using self- and other-report, behavioral performance, and neurobiological data in order to better understand these associations. Finally, prior studies using person-centered approaches to the study of adversity have used categorical indicators to assess exposure to adversity. This is the first study to use continuous indicators of risk in an LPA approach, thereby accounting for both type and severity of adverse experiences.

Despite these strengths, the findings should be interpreted in light of several limitations. First, although we captured a variety of distinct forms of adversity relevant to adolescent development, there are additional risk factors that were not captured in our study. Other common forms of adversity (e.g., incarceration of a family member) may further distinguish latent subgroups. Second, our model did not control for baseline levels of psychopathology. While the study does include earlier data on adolescent internalizing and externalizing symptomatology, we chose not to control for these effects given our interest in the cascading effects of adversity, illustrating the developmental flow from earlier adversity to later levels of functioning. Our findings provide a foundation for future work to explore more complex developmental processes, such as bidirectional and autoregressive effects. Third, mixture modeling approaches such as LPA have inherent limitations, including the partially subjective nature of model selection and

interpretation. That is, our selection of the three-profile solution was based not only on objective model fit indices, but also perceived interpretability of the profiles. Furthermore, we assigned descriptive labels for each subgroup based on the empirical results. While this is standard practice in mixture modeling, such subjectivity may limit replication of the findings. Relatedly, it is possible that these profiles are specific to our sample; future research using similar indicators of adversity will be necessary to verify whether these groups generalize to broader populations.

Finally, the largely non-significant associations with cognitive control may reflect limitations in the MSIT and its ability to capture cognitive control processes. In particular, its limited ecological validity may have attenuated any associations with adversity and psychopathology. That is, the MSIT is a lab-based task which was administered in the scanner; the behaviors and neural responses we observed may not sufficiently reflect how adolescents exert cognitive control in their daily lives. Future research may benefit from the use of more ecologically valid measures of cognitive control, or from incorporating multiple indicators of cognitive control/self-regulation (i.e., additional behavioral tasks or related self-report measures) in order to better index this construct.

**Future Directions**

The current findings point to potential directions for future research. For example, we modeled profiles of adversity at only one time point. Adversity experiences may have fluctuated across adolescence and individuals may have transitioned into different profiles over time. However, prior research using latent class analysis has found that adversity class membership remains relatively stable from childhood into early adolescence (Dunn et al., 2011). Nonetheless, latent transition analyses could be leveraged in future work to capture any shifts across adolescence, especially in the transition from adolescence to young adulthood when individuals are often transitioning into new environments. Another important avenue for future research is

the consideration of timing and chronicity of adverse experiences. We do not know if the indicators of adversity that we measured emerged during early adolescence, or if they had already been present earlier in childhood. Consideration of chronicity of stress exposure may further distinguish subgroups. Finally, we acknowledge that not all adolescents who are exposed to adversity will experience deficits in cognitive control or heightened psychopathology. Rather, there are important protective factors that may buffer against these effects (such as a warm and consistent adult figure) and should be examined in future work.

**Conclusion**

This study aimed to test the utility of a person-centered approach to the characterization of adversity during adolescence. The results offer some of the first evidence that adolescent exposure to certain patterns of risk may differentially predict their later cognitive functioning and mental health outcomes. Adversity experiences in early adolescence have nuanced and lasting consequences for adjustment, even into young adulthood, indicating the importance of clearly delineating the specific features of stress that confer risk for different types of psychopathology. The results further highlight that adolescents with household environments characterized by low SES and high parent substance use were at risk for deficits in cognitive control. Thus, cognitive control may be a potential target for intervention efforts for adolescents whose experiences align with this particular risk profile. As the science of person-centered approaches to the understanding of stress and adversity evolves, practitioners will increasingly be able to move towards precision screening and individualized treatment planning that will optimize outcomes for vulnerable youth.

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**Tables**

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Min-Max | *M* (SD) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Socioeconomic Status | | | | | | | | | | | -1.84-2.34 | 0.00 (0.80) |
| 2. Abuse | -.19* | | | | | | | | | | 0.00-8.00 | 1.84 (2.01) |
| 3. Neglect | -.30** | .39** | | | | | | | | | 0.00-9.00 | 1.79 (2.35) |
| 4. Parent Substance Use | -.25** | .25** | .17 | | | | | | | | -1.65-2.92 | -0.01 (0.97) |
| 5. Parent Depression | -.32** | .26** | .10 | .09 | | | | | | | 0.00-31.00 | 8.27 (7.63) |
| 6. Household Chaos | -.21** | .41** | .15 | .03 | .13 | | | | | | 1.17-4.67 | 2.45 (0.69) |
| 7. Negative Life Events | -.09 | .06 | .18* | .10 | .22** | -.01 | | | | | 0.00-11.00 | 3.20 (2.50) |
| 8. Behavioral Cognitive Control Time 2-4 Average | .20* | -.05 | -.14 | -.18* | -.12 | -.11 | -.17* | | | | -0.29 - -0.11 | -0.19 (0.04) |
| 9. Neural Cognitive Control Time 2-4 Average | -.02 | .10 | -.09 | .12 | .07 | .05 | .01 | .09 | | | -0.98-0.52 | -0.30 (0.24) |
| 10. Internalizing Symptomatology Time 5 | -.14 | .38** | .10 | .30** | .16 | .16 | .23** | -.07 | -.01 | | 30.00-72.50 | 50.06 (9.66) |
| 11. Externalizing Symptomatology Time 5 | -.19* | .34** | .29** | .26** | .06 | .22* | .38** | -.17 | .08 | .58** | 29.00-73.00 | 48.69 (8.97) |

Table 1. *Correlations and Descriptive Statistics for Adversity Variables, Cognitive Control, and Psychopathology*

*Note.* $*p < .05$, $**p < .0$

| Number of latent classes | SABIC | ALMR | BLRT | Entropy | Profile Prevalence | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | 1 | 2 | 3 | 4 |
| 1 | 3500.32 | - | - | - | | | | |
| 2 | 3390.47 | 122.48 ($p$ =.13) | < .001 | .92 | .92 | .08 | | |
| **3** | **3361.64** | **43.39 (*p* = .13)** | **< .001** | **.81** | **.71** | **.21** | **.08** | |
| 4 | 3283.09 | 77.88 ($p$ = .26) | < .001 | .80 | .68 | .11 | .14 | .08 |

Table 2. *Latent Profile Analysis Model Fit Statistics*

*Note.* Final selected model is in boldface. ALMR = Adjusted Lo-Mendell-Rubin test; BLRT = Bootstrap Likelihood Ratio Test; SABIC = Sample-size Adjusted Bayesian Information Criterion

| Dependent Variable | Comparison (Class X minus Class Y) | Mean Difference | Standard Error | Significance |
|---|---|---|---|---|
| Socioeconomic Status | Class 1 - Class 2 | 1.07 | 0.13 | $p < .001$ |
| | Class 2 - Class 3 | -0.53 | 0.22 | $p = .04$ |
| | Class 1 - Class 3 | 0.54 | 0.20 | $p = .02$ |
| Abuse | Class 1 - Class 2 | -1.20 | 0.28 | $p < .001$ |
| | Class 2 - Class 3 | -4.80 | 0.45 | $p < .001$ |
| | Class 1 - Class 3 | -5.99 | 0.39 | $p < .001$ |
| Neglect | Class 1 - Class 2 | -1.28 | 0.48 | $p = .02$ |
| | Class 2 - Class 3 | -3.10 | 0.77 | $p < .001$ |
| | Class 1 - Class 3 | -4.38 | 0.67 | $p < .001$ |
| Parent Substance Use | Class 1 - Class 2 | -1.57 | 0.14 | $p < .001$ |
| | Class 2 - Class 3 | 0.91 | 0.24 | $p = .001$ |
| | Class 1 - Class 3 | -0.66 | 0.21 | $p = .01$ |
| Parent Depression[+] | Class 1 - Class 2 | -2.49 | 1.41 | $p = .18$ |
| | Class 2 - Class 3 | -5.71 | 2.38 | $p = .05$ |
| | Class 1 - Class 3 | -8.20 | 2.14 | $p = .001$ |
| Household Chaos | Class 1 - Class 2 | -0.18 | 0.12 | $p = .28$ |
| | Class 2 - Class 3 | -0.97 | 0.20 | $p < .001$ |
| | Class 1 - Class 3 | -1.16 | 0.18 | $p < .001$ |
| Negative Life Events | Class 1 - Class 2 | -0.61 | 0.47 | $p = .40$ |
| | Class 2 - Class 3 | -1.56 | 0.79 | $p = .12$ |
| | Class 1 - Class 3 | -2.17 | 0.71 | $p = .01$ |

Table 3. *Mean Differences between Latent Classes on Indicators of Adversity*

[+]Transformed (divided by 10) to facilitate model convergence.
*Note.* Class 1 = Low risk, Class 2 = Low SES/High parent SU, Class 3 = High Risk.

## Figures

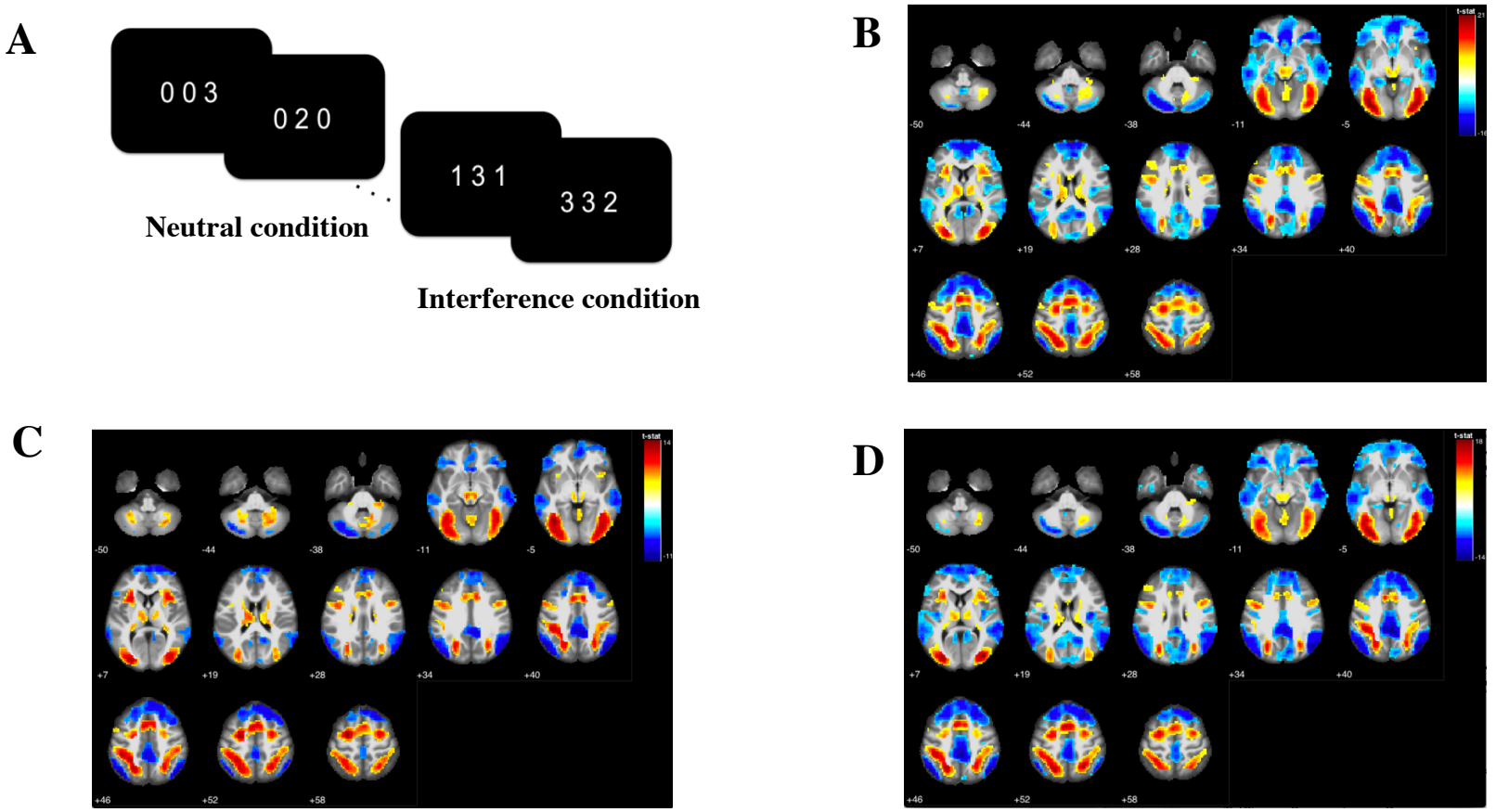


*Figure 1*. A) In the multi-source interference task (MSIT), adolescents were asked to identify the digit that differed from two other concurrently presented digits, ignoring its position in the sequence. B) Interference – neutral *t*-test at Time 2, displayed at *p* (FWE) < .001. C) Interference – neutral *t*-test at Time 3, displayed at *p* (FWE) < .001. D) Interference – neutral *t*-test at Time 4, displayed at *p* (FWE) < .001

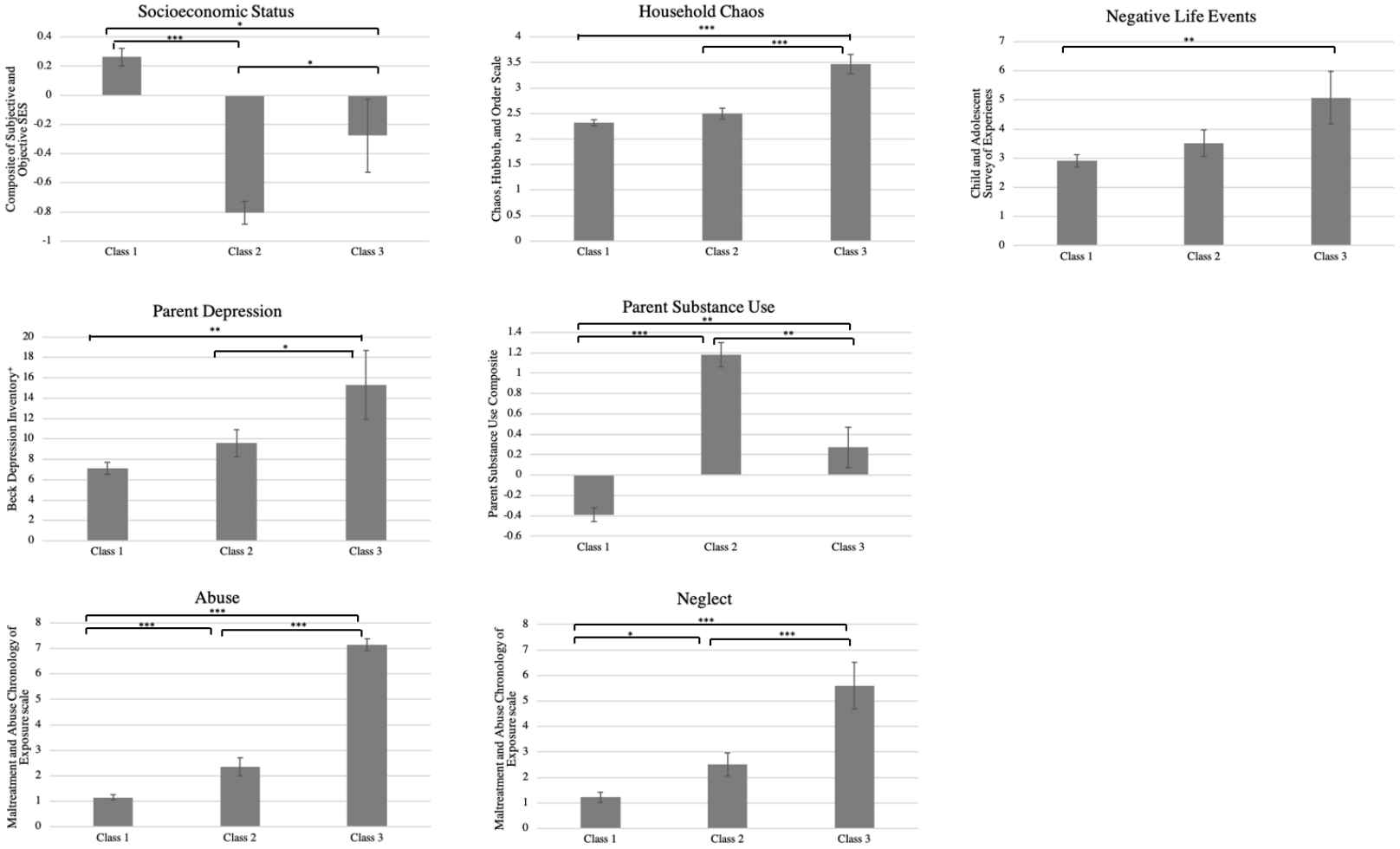


*Figure 2.* Between-class means for adversity variables.

*Note.* +Transformed (divided by 10) to facilitate model convergence. Class 1 = Low risk, Class 2 = Low SES/High parent SU, Class 3 = High Risk. *$p < .05$, **$p < .01$, *** $p < .001$

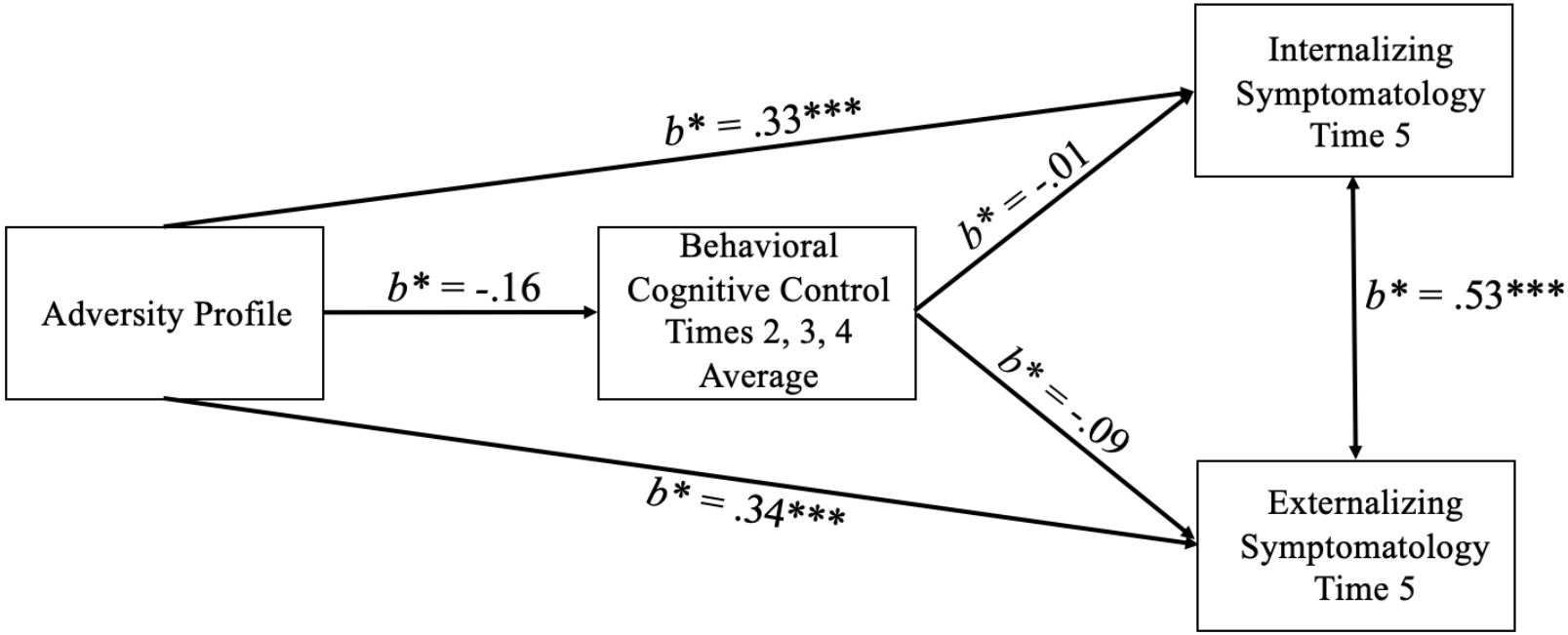


*Figure 3.* Full path model for the associations between profiles of adversity, behavioral cognitive control, and psychopathology. Model controls for the effect of race on externalizing symptomatology ($b^* = .20$, $SE = .08$, $p = .02$).

*$p < .05$, **$p < .01$, ***$p < .001$

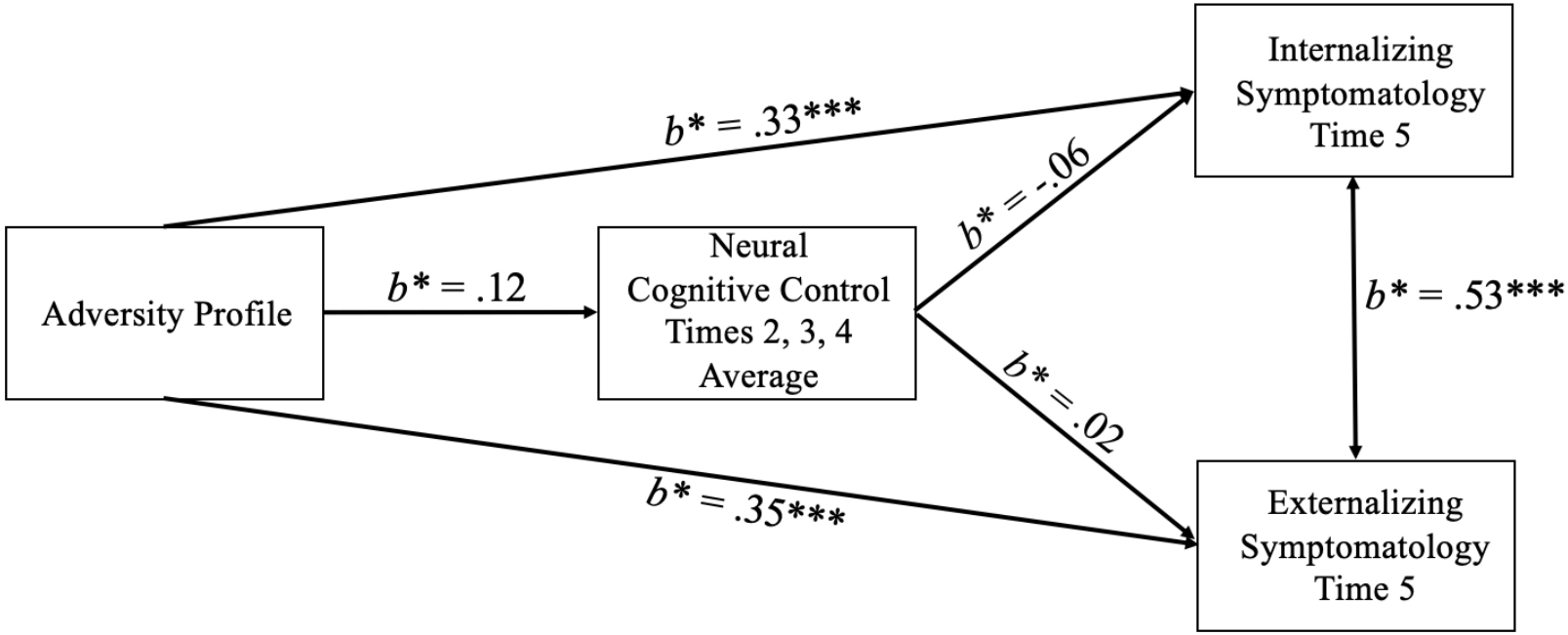


*Figure 4.* Full path model for the associations between profiles of adversity, neural cognitive control, and psychopathology. Model controls for the effect of race on externalizing symptomatology ($b^* = .21$, $SE = .08$, $p = .01$).

*$p < .05$, **$p < .01$, ***$p < .001$

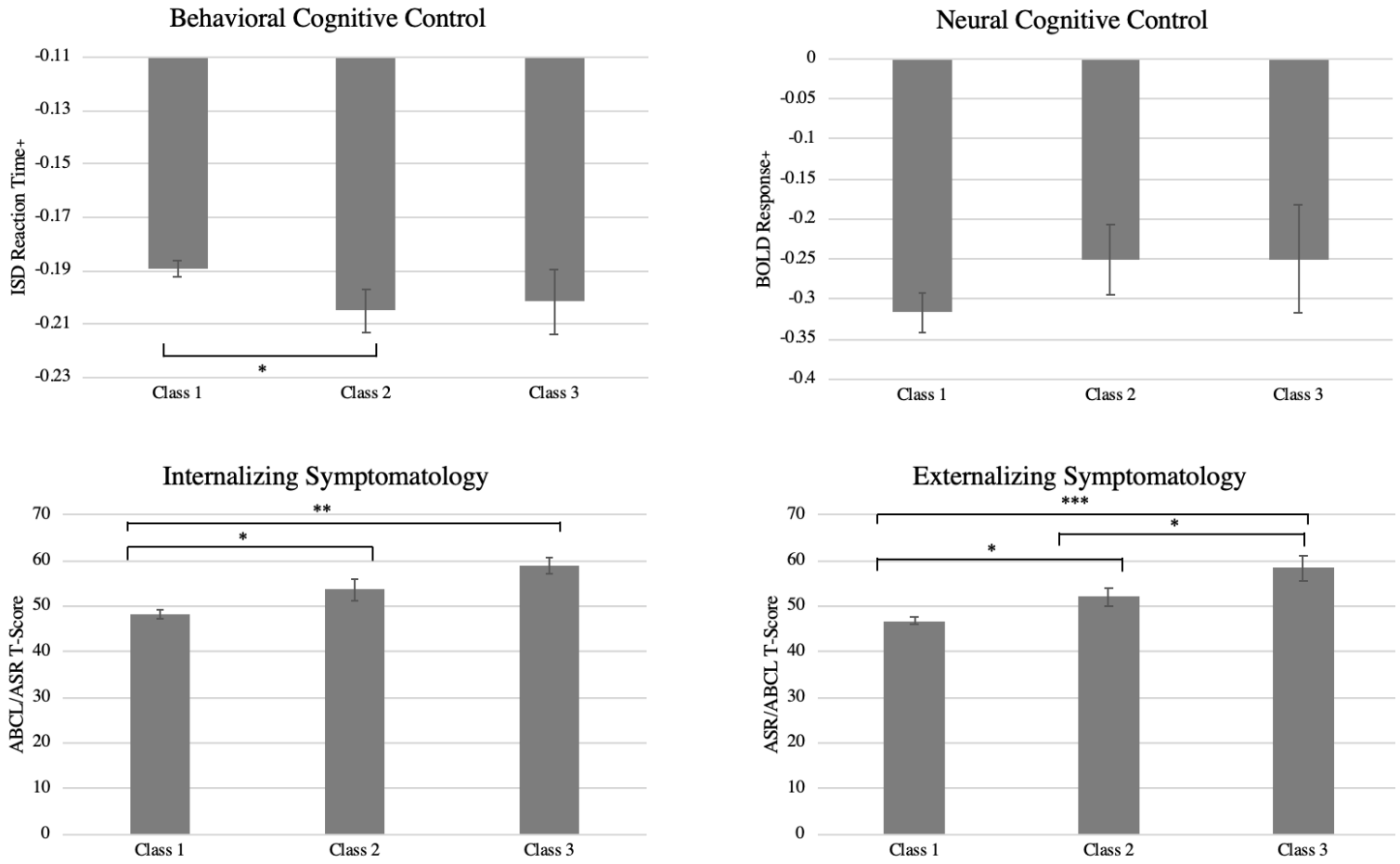


*Figure 5.* Between-class means for outcome variables.

*Note.* +Variables were reverse-scored such that lower values = lower/worse cognitive control. Class 1 = Low risk, Class 2 = Low SES/High parent SU, Class 3 = High Risk.

*$p < .05$, **$p < .01$, *** $p < .001$

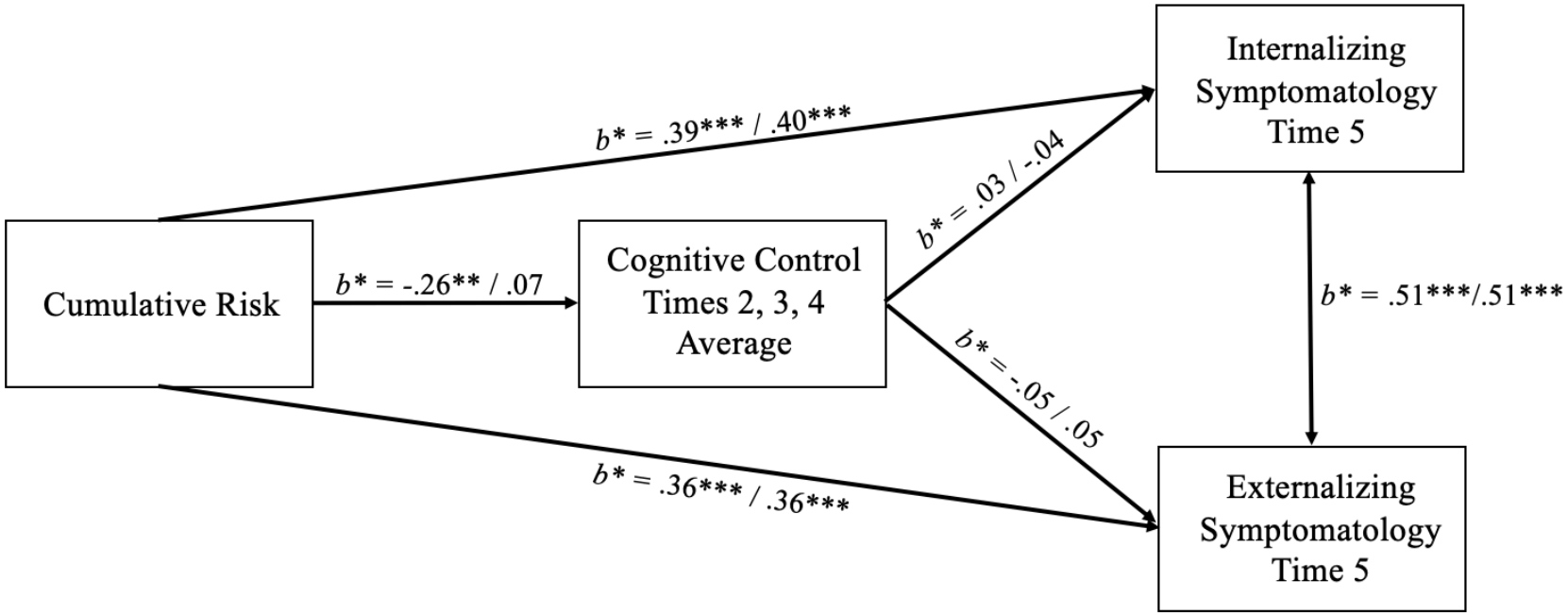


*Figure 6.* Full path model with cumulative risk approach. Estimates are for behavioral cognitive control / neural cognitive control. Model controls for the effect of race on externalizing symptomatology ($b^*$ = .19/.20, *SE* = .08/.08, $p$ = .02/.01).

*$p$ < .05, **$p$ < .01, ***$p$ < .001