Growth Trajectories of Neurocognitive Self-Regulation and Adolescent Adjustment

Alexis Brieant, B.A.

Thesis submitted to the faculty of Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of

Master of Science

in

Psychology

Jungmeen Kim-Spoon, Chair

Brooks King-Casas, Committee Member

Thomas Ollendick, Committee Member

12/13/2017

Blacksburg, VA

Keywords: Adolescence, executive functioning, internalizing symptomatology, externalizing symptomatology

Growth Trajectories of Neurocognitive Self-Regulation and Adolescent Adjustment

Alexis Brieant

ABSTRACT (Academic)

Adolescence is a period of social, physical, and neurobiological transitions that may leave individuals more vulnerable to the development of adjustment problems such as internalizing and externalizing symptomatology. Extant research demonstrates how self-regulation can predict adjustment outcomes in adolescence; however, it has yet to be examined how longitudinal *growth* in self-regulation may predict individual differences in symptomatology. That is, adolescents who develop self-regulatory capacities such as executive functioning (EF; including shifting, working memory, and inhibitory control) more slowly than their peers may be at increased risk for maladjustment. Data were collected from 167 adolescents and their primary caregiver over approximately three years. At each time point, adolescents completed three behavioral tasks that capture the underlying dimensions of EF, and both adolescents and their primary caregiver completed measures of adolescent symptomatology. Parallel process growth curve modeling was used to test the associations between initial levels and trajectories of both EF and adjustment. Results did not reveal any significant associations between initial levels of EF and adjustment or between growth in EF and growth in adjustment. Furthermore, there were no differential associations between the different EF dimensions. However, post-hoc analyses revealed that longitudinal increases in growth of EF predicted lower externalizing (but not internalizing) symptomatology at Time 3 (controlling for Time 1). Findings suggest that those with more rapid EF development may be better able to regulate behavioral and affective states and thus be less likely to develop externalizing symptoms, and that both early levels and *growth* in EF may be important predictors of adolescent outcomes.

Growth Trajectories of Neurocognitive Self-Regulation and Adolescent Adjustment

Alexis Brieant

ABSTRACT (Public)

Adolescence is a period of social, physical, and neurobiological transitions that may leave individuals more vulnerable to the development of symptoms such as anxiety, depression, aggression, and delinquency. Self-regulation affects these outcomes in adolescence; however, individuals demonstrate growth in self-regulation abilities at different rates. Thus, the current study sought to examine how differences in self-regulation (specifically, executive functioning (EF)) development over time may contribute to different behavioral and emotional symptoms in adolescence. Data were collected from 167 adolescents and their primary caregiver over approximately three years. At each time point, adolescents completed three behavioral tasks that capture EF, and both adolescents and their primary caregiver completed measures of adolescent symptoms. Results showed that there were no significant associations between initial levels of EF and symptoms, or between growth in EF and growth in symptoms. Furthermore, different aspects of EF (such as memory, attention, and inhibitory control) did not differentially predict symptomatology. However, additional analyses revealed that increases in growth of EF over time predicted lower symptoms of aggression and delinquency at Time 3. Findings suggest that those with more rapid EF development may be better able to regulate behavioral and emotional states and thus be less likely to develop these types of symptoms, and that both early levels and *growth* in EF may be important predictors of adolescent outcomes.

Acknowledgments

This work was supported by grants awarded to Jungmeen Kim-Spoon and Brooks King-Casas from the National Institute on Drug Abuse (DA036017). Funding source had no involvement in study design, data collection and analysis, or writing of the report. We are grateful to the adolescents and parents who participated in this study.

Table of Contents

List of Tables & Figures

1. Introduction

The developmental period of adolescence marks important social, physical, and neurobiological transitions which leave individuals vulnerable to a host of psychosocial and adjustment problems (Steinberg, 2005). These transitions are related in part to rapid and dramatic changes in structure and function of the brain. Neurodevelopmental models of adolescent brain development emphasize the imbalance in developmental trajectories between subcortical, limbic regions and prefrontal regions (Casey, Jones, & Hare, 2008). Specifically, limbic regions related to emotion and reward seeking develop earlier and more rapidly than the prefrontal regions that are necessary to effectively regulate these subcortical systems. This imbalance often manifests in the form of challenges in self-regulation, which has profound implications for adolescent adjustment and well-being. Despite accumulating evidence elucidating the development of self-regulation during adolescence, there is limited research examining how self-regulation and adjustment problems develop transactionally, as well as the potential that variation in *growth* of self-regulatory capacities (beyond initial levels) may contribute to emerging changes in adolescent mental health.

Adolescent Executive Functioning

Broadly, self-regulation is the capacity to flexibly regulate behavior, cognition, and emotion in order to successfully pursue long term goals (Baumeister, Vohs, & Tice, 2007). Self-regulation may encompass a number of processes, including both top-down and bottom-up control, emotion regulation, and cognitive control (Nigg, 2017). Self-regulation requires specific cognitive capacities in order to alter one's internal state, thoughts, goals, or emotions. Top-down processing of information is necessary to achieve these states or goals, and involves effortful, deliberate exertion of control. Top-down aspects of self-regulation are represented by executive

functioning (EF). EF refers to neurocognitive abilities that enable self-regulation, and these higher order cognitive abilities guide goal-directed behaviors. According to the theoretical model by Miyake et al. (2000), these abilities include updating and monitoring of information (working memory), shifting between tasks or mental sets (shifting), and the ability to inhibit prepotent responses (inhibitory control). These three indicators are distinct but correlated, and are thought to together reflect the latent construct of EF (Friedman & Miyake, 2017). EF is not exclusively employed in the service of self-regulation, and supports other skills such as solving math problems or planning future tasks. For the purposes of the current study, we examine EF within the framework of self-regulation, understanding that these top-down cognitive abilities *enable* self-regulation (Barkley, 2012). However, given the overlap in definitions of related constructs in research on self-regulation (see Nigg, 2017), we acknowledge and draw on related self-regulation literature (e.g., effortful control, cognitive control) in order to inform our hypotheses regarding the linked development of EF and adjustment.

Individual differences in EF emerge over time as a function of both genetic and environmental influences (Deater-Deckard, 2014). Throughout early and middle childhood, EF shows linear growth, but seems to be unidimensional (Lee, Bull, & Ho, 2013). As individuals transition into adolescence, the protracted development of the prefrontal cortex (PFC) brings EF toward maturity (Klingberg, Forssberg, & Westerberg, 2002; Kwon, Reiss & Menon, 2002) and EF begins to differentiate into the three factor structure involving shifting, inhibitory control, and working memory (Lee et al., 2013). Despite the commonalities between these indicators of EF, many studies yield low intercorrelations among them (Willoughby, Holochwost, Blanton, & Blair, 2014). A study from Huizinga, Dolan, and van der Molen (2006) further demonstrated that each of these abilities develops at different rates and follows a distinct developmental trajectory.

Specifically, shifting developed into adolescence, but working memory continued to develop into young adulthood. Performance on some aspects of inhibitory control (Flankers and Stop-signal task) reached adult levels by age 11, but other aspects (Stroop task) continued to develop throughout adolescence. Generally, children were able to perform working memory, shifting, and inhibition tasks at adult levels between 11 and 15 years of age. Taken together, it is important to consider both the unity and diversity of EF in the development of self-regulation.

Though EF is improving during adolescence, evidence suggests that cognitive control abilities related to prefrontal functioning do not reach full maturity until young adulthood (Casey et al., 2008; Steinberg et al., 2008). Evidence from human neuroimaging work illustrates linear increases in EF during adolescence, indexed by lower activation in prefrontal areas during cognitive control tasks (Luna, Padmanabhan, & O'Hearn, 2010; Ordaz, Foran, Velanova, & Luna, 2013). However, development in prefrontal areas is considerably slower as compared to the rapid development of subcortical areas (Galvan et al., 2006; Hare et al., 2008; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). According to the imbalance theory of adolescent brain development, this discrepancy may account for challenges in self-regulation during adolescence (Casey et al., 2008; Somerville et al., 2010).

Despite considerable support for the general patterns of the trajectories of EF development, it should be noted that there is substantial variation in the rate of development. That is, some children may develop these cognitive capacities more rapidly than their peers, while others may demonstrate slower rates of growth or stability. In addition to levels of EF, individual *variation* in change rates may have important implications for psychosocial outcomes. For example, Hughes and Ensor (2010) investigated latent growth in EF in children transitioning into school. Results indicated that individual differences in improvements in EF were predictive

of teacher-rated internalizing and externalizing behaviors, such that greater gains in EF between 4 and 6 years of age predicted lower emotional and conduct problems at age 6. Furthermore, growth rates were predictive of both emotional and conduct problems at age 6, over and above the effects of initial levels of EF (accounted for by covarying the intercept factor with the outcome variables). These findings emphasize the importance of considering developmental *change* in self-regulation in explaining individual differences in adjustment. However, there is limited research regarding within-person change in EF in adolescence, as well as how this change may be linked to adjustment outcomes.

Deficiencies in EF may contribute to a developmental cascade of risk, with interactions across multiple levels exerting progressive and cumulative effects on emerging psychopathology (Masten & Cicchetti, 2010). Indeed, EF has important implications for development across a number of domains, including academic (Becker, Miao, Duncan, & McClelland, 2014), social (Holmes, Kim-Spoon, & Deater-Deckard, 2016), and psychological (Letkiewicz et al., 2014) domains. In adolescence, individuals are particularly vulnerable to problems in psychological domains, such as internalizing and externalizing symptomatology, as reflected by the increase in prevalence of psychological disorders after age 12 (Costello, Mustillo, Keeler, & Angold, 2003). Since self-regulation is integral to the development of psychopathology (Nigg, 2017), elucidating individual differences in the development of cognitive abilities that facilitate successful self-regulation is imperative in order to identify adolescents who may be vulnerable to internalizing and externalizing symptomatology.

Joint Development of Executive Functioning and Internalizing and Externalizing Symptomatology

Adolescence has been popularly referred to as a period of "storm and stress" (Hall, 1904). In addition to dramatic neurobiological changes, adolescents are exposed to increasing autonomy, novel social influences, and environmental stressors (Steinberg, 2005). These changes can elicit powerful emotional responses, and adolescents experience increases in negative emotion and decreases in positive emotion during this period (Maciejewski, van Lier, Branje, Meeus, & Koot, 2016). Together, these influences expose adolescents to considerable risk for the development of psychopathology, including internalizing and externalizing symptomatology. Internalizing symptomatology includes anxiety, depression, withdrawal, and somatic complaints. Anxiety and depressive disorders increase in prevalence from childhood, across adolescence, and into adulthood (Costello, Copeland, & Angold, 2011). Externalizing symptomatology is characterized by aggression and rule-breaking behaviors. At clinical levels, these symptoms manifest as ADHD, oppositional defiant disorder (ODD), and conduct disorder (CD). In the transition from childhood to adolescence, the prevalence of externalizing disorders decreases (Costello et al., 2011).

It is well-established that top-down self-regulatory abilities are related to child and adolescent adjustment outcomes. In a sample of preschool children, Espy et al. (2011) investigated executive control (working memory, attention, and inhibitory control), which is conceptually similar to EF, and its relation to different dimensions of externalizing symptomatology. Results suggested that worse executive control was associated with greater problems in hyperactivity, attention, and disinhibition behaviors. Similar patterns of results have been demonstrated in older samples, where EF (indexed by decision making tasks and an emotional Go-Nogo task) has been shown to significantly predict externalizing symptomatology in pre-adolescents (Woltering, Lishak, Hodgson, Granic, & Zelazo, 2016). Similarly, deficits in

self-reported EF (inhibition, shifting, and working memory) have been linked to heightened internalizing symptomatology in emerging adults (Letkiewicz et al., 2014). In all prior studies, executive control or EF was assumed to be a unitary dimension (i.e., composite averaged across individual dimensions), and it was not tested whether individual dimensions (such as working memory, shifting, and inhibitory control) may differentially contribute to externalizing and internalizing outcomes.

The link between EF and internalizing and externalizing symptomatology may be explained by evidence suggesting that the cognitive abilities encompassed by EF are necessary for successful management of negative emotions and arousal. For example, cognitive control may protect individuals against depression by allowing them to override attention biases and prevent rumination (Gotlib & Joormann, 2010). Furthermore, strategies such as reframing, behavioral planning, and conflict-monitoring are key components of cognitive behavioral therapies (which are used to address both internalizing and externalizing disorders) and require capacities for working memory, shifting, and inhibitory control (Mischel, 2004). Thus, individual differences in EF development in adolescence may have important implications for long-term mental health and well-being.

While there are broad EF impairments associated with psychopathology, variation in individual EF components may predict internalizing and externalizing symptomatology in distinct ways. The components of EF have been shown to recruit partially distinct neural areas (Collette et al., 2005) and demonstrate unique developmental trajectories across adolescence (Boelema et al., 2014), which may suggest that they contribute to maladaptation differentially. First, working memory allows individuals to hold information long enough to be used in the service of goal-directed behaviors. However, for individuals with internalizing symptomatology,

emotional stimuli can interfere with information in working memory, perpetuating symptomatology such as rumination (Joormann & Gotlib, 2008). A recent meta-analysis demonstrated that across 177 samples, working memory impairment was reliably associated with anxiety symptoms (Moran, 2016). In terms of externalizing symptomatology, a meta-analysis found that working memory had one of the strongest and most consistent effects on ADHD in relation to other executive functions (Wilcutt, Goyle, Nigg, Faraone, & Pennington, 2005).

Second, the shifting component of EF allows one to flexibly modify their attention. In the context of psychopathology, this may translate to an ability to adjust thoughts and actions away from negative or arousing thoughts or stimuli. Thus, individuals with poor shifting abilities may also have internalizing symptomatology due to their inability to flexibly manage internal states. For example, pre-adolescents with higher levels of internalizing symptomatology performed less efficiently on an emotional set-shifting task than those with lower internalizing symptomatology (Mocan, Stanciu, & Visu-Petra, 2014). In terms of externalizing symptomatology, Hatoum, Rhee, Corley, Hewitt, and Friedman (in press) found that shifting-specific components of EF were positively associated with change in teacher-reported externalizing symptomatology from age 7 to 15, supporting the value of considering longitudinal growth in adjustment.

Finally, inhibitory control works to regulate impulsive, prepotent responses which are particularly characteristic of externalizing symptomatology. Children with externalizing problems demonstrate significantly worse inhibitory control than children with internalizing problems (Eisenberg et al., 2001). Furthermore, Young et al. (2009) found that inhibitory control is more strongly related to behavioral disinhibition than working memory and shifting in adolescents. These results indicate that there are important commonalities between cognitive inhibition and behavioral inhibition, and that inhibitory control may be an underlying factor in

the development of externalizing disorders. Taken together, the distinct results regarding individual components of EF call for specificity in our conceptualization of the way that these top-down cognitive abilities may be contributing to the emergence of adolescent internalizing and externalizing symptomatology.

The extant literature on EF and psychopathology primarily considers EF at a single time point predicting later maladaptation. However, since adolescence is a period of dramatic change in cognitive capacities (Powers & Casey, 2015) and heightened emotionality (Maciejewski, van Lier, Branje, Meeus, & Koot, 2015), the way that *growth* in EF may affect the development of psychopathology is a particularly important consideration. Most prior studies do not address variance in EF development, which is potentially informative for adaptive psychological development. That is, individual differences in the developmental trajectories of self-regulation may in part account for variances in internalizing and externalizing symptomatology during adolescence.

To our knowledge, only two studies have attempted to elucidate the simultaneous development of self-regulation and adjustment. Piehler et al. (2014) implemented an intervention program with formerly homeless children (ages 6 – 12) which was shown to promote increases in EF growth across four time points (indexed by parent-reported composite based on 10 items from the Behavior Assessment System for Children scale). Using parallel process growth curve modeling, it was further found that longitudinal improvements in EF predicted reduced growth in conduct problems. These results offer initial evidence for the importance of individual differences in EF trajectories contributing to child adjustment outcomes.

Using similar growth modeling techniques, King, Lengua, and Monahan (2013) examined variability in the development of effortful control (indexed by mother- and adolescent-

reported composite of the attention regulation subscale of the Early Adolescent Temperament Questionnaire and the inhibitory control subscale of the Child Behavior Questionnaire) among pre-adolescents (mean age = 9 years at Time 1). Effortful control is an aspect of self-regulation closely tied to EF, but is conceptualized as a temperamental construct which is typically examined distinctly from EF in the existing literature (Zhou, Chen, & Main, 2011). King et al. (2013) found that better initial levels of effortful control significantly predicted lower levels of internalizing and externalizing symptomatology two years later. Furthermore, individual differences in the rate of effortful control development explained variation in both internalizing and externalizing symptomatology, over and above the effects of initial levels. The findings indicate that variations in developmental trajectories of self-regulation may account for the emergence of adjustment problems in pre-adolescence. However, these results were subject to some limitations. First, self-regulation measurement relied solely on questionnaires. Additionally, the study primarily focused on individuals ages 9 – 12, which warrants further investigation of the extensions of these developmental trajectories throughout adolescence.

Present Study

The purpose of the proposed study was to determine variation in the development of neurocognitive self-regulation and symptomatology within and between individuals during the period of adolescence. Furthermore, we seek to elucidate how changes in neurocognitive self-regulation may impact changes in symptomatology. The study utilized data from three time points across adolescence (ages 13 through 16 years) with a one-year interval in between each time point. The specific aims of this study were to:

1. Determine whether variation in the development of the latent construct of EF (based on inhibitory control, shifting, and working memory) predicts developmental changes in internalizing and externalizing symptomatology.

2. Determine growth in individual dimensions of EF and if they differentially predict trajectories of internalizing and externalizing symptomatology.

Hypotheses

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

1. Lower initial levels of EF will predict increases in both internalizing and externalizing symptomatology. Similarly, although it is expected to see positive growth in EF as the whole group, those with decreases or slower increases in EF will exhibit increases in internalizing and externalizing symptomatology.

2. The individual dimensions of EF—working memory, shifting, and inhibitory control—will differentially predict the development of internalizing and externalizing symptomatology. Though no study has examined the relative contributions of each EF dimension to adjustment, available evidence suggests that working memory may be the strongest predictor for internalizing symptomatology, and inhibitory control may be the strongest predictor for externalizing symptomatology.

2. Method

Participants

The proposed study used three waves of data that have been collected as part of an ongoing longitudinal study. The sample includes 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 = 14.13, SD = 0.54), 14

or 15 years of age at Time 2 (M = 15.05, SD = 0.54), and 15 or 16 years of age at Time 3 (M = 16.07, SD = .56), with approximately one year in between each time point. Adolescents primarily identified as Caucasian (82%), 12% African-American, and 6% other. Caregivers also primarily identified as Caucasian (88%), 10% African American, and 2% other. Caregiver ages ranged from 31 to 61 years (M = 42.02, SD = 6.63). For the city and counties sampled, 2010 US Census data showed median annual household income to be in the $36,000-59,000 range, and in the current sample, median household income was in the $35,000-$50,000 range (United States Census Bureau, 2010).Overall, the annual income in the sample ranged from less than $1,000 to greater than $200,000 per year. At Time 1, 157 families participated. At Time 2, 10 families were added for a final sample of 167 parent-adolescent dyads. However, 24 families did not participate at all possible time points for reasons including: ineligibility for tasks (n = 2), declined participation (n = 17), and lost contact (n = 5) during the follow-up assessments. We performed attrition analyses using general linear model (GLM) univariate procedure to determine whether there were systematic predictors of missing data. Results indicated that rate of participation (indexed by proportion of years participated to years invited to participate) was not significantly predicted by demographic and intelligence covariates at Time 1 (p = .88 for age, p = .79 for income, p = .81 for sex, p = .73 for race, contrasted as White vs non-White, and p = .85 for intelligence) or study variables at Time 1 (p = .91 for internalizing symptomatology, p = .55 for externalizing symptomatology, p = .40 for EF factor score).

Measures

Demographic interview. In a verbal interview, participants reported sex, age, race, and ethnicity at each time point. Parents provided a report of total household income.

Externalizing and internalizing symptomatology. Adolescents' self-reported levels of externalizing and internalizing symptomatology were assessed with the Youth Self-Report (YSR; Achenbach & Rescorla, 2001), a 102-item questionnaire that assesses behavior problems in children ages 11 to 17. Behaviors were rated on a 3-point scale ranging from "0 = Not true" to "2 = Very true". Raw summed scores from the externalizing (aggressive behavior and rule-breaking behavior) and internalizing (anxious-depressed, withdrawal-depressed, and somatic complaints) scales were used. The YSR has shown strong psychometric properties for internalizing and externalizing (α = .90; Achenbach & Rescorla, 2001) and demonstrates similar reliability in the current sample for both internalizing symptomatology (α = .90 at each time point) and externalizing symptomatology (α = .86 at Time 1, α = .84 at Time 2, α = .89 at Time 3).

Parent-report of adolescent internalizing and externalizing symptomatology was assessed with the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), a 118-item questionnaire that assesses behavior problems for children 4 – 16 years of age. As in the YSR, behaviors are rated on a 3-point scale and we used raw sums for the externalizing and internalizing scales. The CBCL has shown strong psychometric properties for internalizing (α = .90) and externalizing (α = .94) and demonstrates similar reliability in the current sample for both internalizing symptomatology (α =.84 at Time 1, α = .88 at Time 2 and Time 3) and externalizing symptomatology (α = .91 at Time 1 and Time 2, α = .90 at Time 3).

In addition to parent-only and adolescent-only report of symptomatology, we created a composite score based on combined parent and adolescent report for both internalizing and externalizing symptomatology.

Executive functioning. EF factor scores for each time point were based on confirmatory factor analysis (CFA) of three behavioral tasks that capture the underlying constructs of EF,

according to the theoretical model proposed by Miyake and colleagues (2000): working memory, shifting, and inhibitory control. Working memory was measured with the Stanford-Binet memory for digits (Roid, 2003) in which participants were asked to repeat back a series of numbers read by the experimenter (first forward, then backwards). An age standardized composite score for combined forward and backward digit-span was calculated and used in the EF factor score.

The shifting component of EF was captured with the Wisconsin Card Sorting Test (WCST; Heaton, & Staff, 2003) which requires participants to sort a series of cards based on color, number, and shapes of the patterns on the card under changing schedules of reinforcement. The number of perseverative errors was used in the EF factor score.

Finally, inhibitory control was measured with the Multi-Source Interference Task (MSIT; Bush, Shin, Holmes, Rosen, & Vogt, 2003), a cognitive interference task shown to activate the anterior cingulate cortex and the parietal, premotor, and dorsolateral prefrontal cortices. In the MSIT, participants are presented with sequences of three digits, two of which are identical. Participants are instructed to indicate the identity (but not the position) of the unique, target digit. In the neutral condition, target digits are congruent with position (e.g., "2" is in the second position in the sequence "121"). In the interference condition, target digits are incongruent with position (e.g. "2" is in the first position in the sequence "211"). To assess task performance, we used accuracy and intraindividual variability in reaction time, indexed as intraindividual standard deviations (ISDs; Macdonald, Karlsson, Rieckmann, Nyberg, & Bäckman, 2012) for correct responses in the interference condition.

Intelligence. Adolescents' intelligence was assessed at Time 1 with the Kaufman Brief Intelligence Test (2nd Edition, KBIT; Kaufman & Kaufman, 2004). The KBIT is a short

intelligence test appropriate for both adults and children. We specifically tested for verbal intelligence using the Verbal Knowledge and Riddles subscales. Based on the standardized scores of these two scales, we calculated a composite variable of verbal intelligence for adolescents, with higher scores indicating higher intelligence.

Data Analytic Plan

For all study variables, descriptive statistics were examined to determine normality of distributions and outliers. 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 SD from the mean. In these cases, values were winsorized to retain statistical power and attenuate bias resulting from elimination (Ghosh & Vogt, 2012). Demographic covariates were tested within each model, including age, income, gender, and race. Intelligence was also included as a covariate for EF variables given that previous research demonstrates the significant association between intelligence test performance and EF performance (e.g., Jester et al., 2009). We were unable to test possible moderating effects of gender due to limited degrees-of-freedom resulting in model nonconvergence. The hypothesized models were tested via Structural Equation Modeling (SEM) using M*plus* statistical software version 7.4 (Muthén & Muthén, 2012). Overall model fit indices were determined by χ^2 value, degrees of freedom, corresponding *p*-value, Root Mean Square Error of Approximation (RMSEA), and Confirmatory Fit Index (CFI). RMSEA values of less than .05 were considered a close fit while values less than .08 were considered an acceptable 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). Full information maximum likelihood (FIML) estimation procedure (Arbuckle, 1996) was used for missing data

since FIML estimates are superior to those obtained with listwise deletion or other ad hoc methods (Schafer & Graham, 2002).

The hypothesized model is depicted in Figure 1. Parallel process growth curve modeling was used to simultaneously test the slopes and intercepts of EF and adjustment outcomes. Models were tested separately for (1) EF factor and (2) individual EF components (i.e., separately for working memory, shifting, and inhibitory control). These models separately examined internalizing and externalizing symptomatology for a total of eight models.

First, for each construct, univariate GCM was performed to fit the baseline model (a two-factor growth model) across the three time points. The first latent factor was the intercept, with all factor loadings fixed to one. The second latent factor was the slope, indicating growth of the function and change over time. Nested model comparisons were used to compare the latent growth model to the linear growth and no growth models in order to determine the shape of the trajectories. In the no growth model, non-significant change in the slope were assumed. In the linear growth model, a linear pattern of change was assumed and factor loadings for the latent slope factor were fixed to 0, 1, and 2. Finally, the latent growth model allowed the data to estimate the shape of growth by fixing the first and last time points (to 0 and 1, respectively) and freely estimating the second. The χ^2 difference test was used to compare these nested models and the most parsimonious model with acceptable fits was chosen as the best-fitting model.

Next, bivariate GCM was tested involving both EF and adjustment constructs (e.g., EF and internalizing symptomatology or EF and externalizing symptomatology). As shown in Figure 1, correlations were estimated between the intercept and the slope factors within the EF and the adjustment factor and between the EF and the adjustment intercept factors. A regression path from the EF intercept to the adjustment slope and a regression path from the adjustment

slope to the EF slope were estimated to test bidirectional prospective prediction of changes. Finally, in line with the hypothesis of EF development predicting developmental changes in adjustment outcomes, a regression path from the EF slope to the adjustment slope was estimated.

3. Results

Correlations and descriptive statistics for study variables are in Table 1. We explored the correlations between adolescent and parent report of internalizing and externalizing symptomatology. However, the correlations between adolescents' report and parents' reports were small to moderate (r = 0.27 – 0.35) and main analyses testing proposed hypotheses tended to be more strongly supported by using adolescents' report than using combined parent-adolescent report. Thus, we report results from the analyses using adolescents' self-report of symptomatology (results using combined parent-adolescent report are available upon request).

Baseline Growth Curve Models

Executive functioning. CFA was used to calculate factor scores for EF at each time point. Model fits were good and all factor loadings were significant. Next, three alternative models were fit in order to determine the shape of the trajectories of EF as well as each of the individual EF dimensions (see Table 2). For the EF factor score, the χ^2 difference test indicated that a linear growth model provided the best fit to the data. Significant variance of intercept (σ^2 = 0.09, SE = .01, p < .001) and slope (σ^2 = .01, SE = .004, p = 0.01) indicated individual differences in initial levels and change in EF. EF was a factor score thus the mean of both the slope and intercept was zero[1].

[1] In growth curve modeling, it is not ideal to use factor scores or standardized scores since these scores have a mean of 0 (composite scores based on averaging the three standardized dimensions also had a mean of 0). We tried a number of alternative approaches to avoid this. (1) We estimated each EF factor simultaneously in a longitudinal CFA model in order to impose measurement invariance over time and yield comparable factor scores. However, linear

For inhibitory control, a latent growth model provided the best fit to the data. There was significant variance for intercept ($\sigma^2 = 0.001$, $SE = 0.00$, $p < .001$) but not for slope ($\sigma^2 = 0.00$, $SE = 0.00$, $p = 0.13$), indicating individual differences in initial levels of inhibitory control. The means of the intercept ($M = -0.24$, $SE = 0.003$, $p < .001$) and slope ($M = 0.05$, $SE = 0.004$, $p < .001$) were significantly different from zero, showing that inhibitory control increased over time.

For shifting, a latent growth model provided the best fit to the data and significant variance of intercept ($\sigma^2 = 0.10$, $SE = 0.01$, $p < .001$) and slope ($\sigma^2 = 0.09$, $SE = 0.01$, $p < .001$) indicated individual differences in initial levels and change in shifting. The means of the intercept ($M = -0.71$, $SE = 0.03$, $p < .001$) and slope ($M = 0.23$, $SE = 0.03$, $p < .001$) were significantly different from zero, showing that shifting increased over time.

For working memory, a linear growth model provided the best fit to the data. The variance of intercept ($\sigma^2 = 1.13$, $SE = 0.18$, $p < .001$) was significant but not the variance of slope ($\sigma^2 = 0.07$, $SE = 0.05$, $p = 0.16$), indicating significant individual differences in initial levels of working memory. The means of the intercept ($M = 9.69$, $SE = 0.10$, $p < .001$) and slope ($M = 0.11$, $SE = 0.05$, $p = 0.02$) were significantly different from zero, showing that working memory increased over time.

Adjustment. As with the EF variables, three alternative models were fit in order to determine the shape of the trajectories of internalizing symptomatology and externalizing symptomatology (see Table 3). For externalizing symptomatology, a linear growth model

---

dependency between the factors across time made the model uninterpretable. (2) We attempted to produce factor scores based on the CFA using Time 2 and Time 3 scores that were "standardized" to the means and standard deviations at Time 1, but were unable to yield factor scores with a non-zero mean. (3) CFA models that were based on EF composite scores (i.e., average scores across the three EF dimensions, instead of factor scores) based on the scores that were "standardized" relative to the means and standard deviations at Time 1; but negative variance of the slope made the model uninterpretable. (4) A second-order factor model may also address this issue, but we did not have sufficient power to test for this.

provided the best fit to the data. Significant variances of intercept (σ^2 = 28.53, *SE* = 4.23, *p* < .001) and slope (σ^2 = 3.11, *SE* = 1.11, *p* = 0.01) indicated that there were significant individual differences in initial levels as well as change in externalizing symptomatology. The mean of the intercept was significantly different from zero (*M* = 9.41, *SE* = 0.48, *p* < .001), but the mean of the slope was not significant (*M* = 0.25, *SE* = 0.24, *p* = 0.29) indicating no significant changes across waves. For internalizing symptomatology, none of the baseline models demonstrated good fit to the data. To improve the fit of the latent growth model, we introduced covariances between internalizing symptomatology Time 1 and Time 2, and covariances between internalizing symptomatology Time 2 and Time 3. The resulting model was saturated. There was no significant variance of intercept (σ^2 = 32.14, *SE* = 27.31, *p* = .24) or slope (σ^2 = 7.88, *SE* = 52.05, *p* = .88), indicating no significant individual differences in the initial level or the slope of change. The mean of the intercept (*M* = 11.24, *SE* = 0.10, *p* < .001) was significant, but not the mean of the slope (*M* = 0.31, *SE* = 0.59, *p* = .60), indicating no significant changes across waves.

Bivariate Growth Curve Models

Bivariate growth curve analyses were used to examine the dynamic relations between growth functions of EF (and its individual dimensions) and externalizing symptomatology. Adolescent report of internalizing symptomatology did not demonstrate good fit in the no-growth, linear, or latent models, thus we focused on externalizing symptomatology. Four models were fit to test initial levels and growth in externalizing symptomatology with initial levels and growth of EF, inhibitory control, shifting, and working memory separately. Intelligence was included as a covariate for EF variable intercepts and slopes. Age and gender were included as covariates for externalizing symptomatology intercept and slope. Other demographic covariates

(race and income) were not included in the final growth models because there were no significant associations with initial levels or growth in the EF or adjustment variables.

Executive functioning. The bivariate model examining joint development of EF and externalizing symptomatology demonstrated good fit ($\chi^2 = 16.57$, $df = 24$, $p = 0.87$, RMSEA = 0.00, CFI = 1.00). However, none of the regression paths were statistically significant (see Table 4 for standardized estimates).

Inhibitory control. The bivariate model examining joint development of inhibitory control and externalizing symptomatology demonstrated good fit ($\chi^2 = 15.16$, $df = 23$, $p = 0.89$, RMSEA = 0.00, CFI = 1.00). However, none of the regression paths were statistically significant (see Table 4 for standardized estimates).

Shifting. The bivariate model examining joint development of shifting and externalizing symptomatology demonstrated good fit ($\chi^2 = 24.02$, $df = 22$, $p = 0.35$, RMSEA = 0.02, CFI = 0.99). However, a problem with the latent variable covariance matrix rendered the parameter estimates uninterpretable.

Working memory. The bivariate model examining joint development of working memory and externalizing symptomatology demonstrated good fit ($\chi^2 = 23.79$, $df = 24$, $p = 0.47$, RMSEA = 0.00, CFI = 1.00). However, none of the regression paths were statistically significant (see Table 4 for standardized estimates).

Post-Hoc Analyses

Since we did not detect any significant associations between initial levels and growth of EF and adjustment, we simplified the model (see Figure 2). Specifically, we modeled the effect of the intercept and slope of the EF factor score on adjustment outcomes at Time 3 (controlling for Time 1). In keeping with the original hypotheses, we also tested the reciprocal effect of

adjustment at Time 1 on the slope of EF. Age and gender were included as covariates for adjustment outcomes. Separate models were tested for externalizing and internalizing symptomatology.

The model for externalizing symptomatology demonstrated excellent fit ($\chi^2 = 6.74$, $df = 9$, $p = 0.66$, RMSEA = 0.00, CFI = 1.00). There was a significant association between the slope of EF and externalizing symptomatology at Time 3, such that growth in EF predicted lower externalizing symptomatology at Time 3 ($b = -15.85$, $SE = 7.88$, $p = 0.04$), controlling for baseline levels of externalizing symptomatology at Time 1 ($b = 0.76$, $SE = 0.07$, $p < 0.001$), over and above the effects of age and gender. The effect of the EF intercept on externalizing symptomatology was not significant ($b = -1.61$, $SE = 1.88$, $p = 0.39$). Furthermore, the reciprocal effect of externalizing symptomatology on the slope of EF was not significant ($b = 0.001$, $SE = 0.002$, $p = 0.71$). Standardized estimates are presented in Figure 2.

The model for internalizing symptomatology demonstrated good fit ($\chi^2 = 13.06$, $df = 9$, $p = 0.16$, RMSEA = 0.05, CFI = .98). However, there was no significant association between the slope of EF and internalizing symptomatology at Time 3 ($b = -2.30$, $SE = 8.13$, $p = 0.78$), controlling for baseline levels of internalizing symptomatology at Time 1 ($b = 0.55$, $SE = 0.07$, $p < 0.001$). Furthermore, the effect of the EF intercept on internalizing symptomatology at Time 3 ($b = 2.10$, $SE = 2.07$, $p = 0.31$) and the effect of internalizing symptomatology at Time 1 on the EF slope ($b = -0.002$, $SE = 0.002$, $p = 0.32$) were both non-significant. Standardized estimates are presented in Figure 3.

Additionally, we tested these post-hoc models separately for each dimension of EF, with externalizing and then internalizing symptomatology. However, none of the regression paths were statistically significant.

4. Discussion

Extant literature has identified the importance of neurocognitive self-regulation in psychopathology outcomes for children and adolescents. Specifically, deficits in self-regulation underlie a host of adjustment problems, including heightened internalizing and externalizing symptoms. Individual differences in self-regulatory processes such as EF emerge across development as a function of genetic and environmental conditions (Deater-Deckard, 2014). However, there is meaningful variability not just in initial levels of EF, but also in how individuals develop EF over time. That is, some individuals may demonstrate growth that is substantially faster or slower as compared to their same-age peers. This is a particularly relevant concern during the developmental period of adolescence as individuals undergo dramatic changes in prefrontal cortex development (Casey, Jones, & Hare, 2008). Thus, the current study sought to elucidate the joint developmental processes of EF and adjustment in adolescence. Specifically, we tested a parallel process growth curve model to examine whether growth in EF predicted growth in internalizing and externalizing symptomatology across three years of adolescence. Using a parallel process growth curve model further allowed us to test reciprocal effects, whereby symptomatology may perpetuate EF problems. Finally, we also tested the possibility that specific dimensions of EF (i.e., inhibitory control, working memory, and shifting) would differentially predict adjustment outcomes.

Our first hypothesis was that lower initial levels of EF would predict increases in both internalizing and externalizing symptomatology. We also expected that individuals with decreases or slower increases in EF would exhibit increases in internalizing and externalizing symptomatology. Ultimately, we were not able to test the bivariate model with internalizing symptomatology due to poor fit for the baseline models describing growth patterns of

internalizing symptomatology. For the bivariate growth curve models including externalizing symptomatology, the regression paths from EF intercept to externalizing symptomatology slope, externalizing symptomatology intercept to EF slope, and EF slope to externalizing symptomatology slope were all non-significant. Our second hypothesis was that different dimensions of EF would differentially predict growth in internalizing and externalizing symptomatology. However, as with the EF factor score, we did not detect any significant associations between inhibitory control, working memory, shifting, and externalizing symptomatology.

Though we found no evidence for significant paths between growth factors of EF and the growth factors of symptomatology in the bivariate growth curve models, significant variation around the intercept and slope of the EF factor as well as externalizing symptomatology warranted further exploration. Thus, as post-hoc analyses we tested an alternative model in which intercept and slope factors of EF predicted internalizing or externalizing symptomatology outcomes at Time 3 after controlling for the baseline levels at Time 1. The model fit was acceptable with internalizing symptomatology as the outcome, though none of the associations between internalizing symptomatology and EF were significant. However, we found a significant association between the slope of EF and externalizing symptomatology. Specifically, improvements in EF across Time 1 through Time 3 significantly predicted low levels of externalizing symptomatology at Time 3 after controlling for baseline externalizing symptomatology at Time 1. These effects of the EF slope were evident over and above initial levels of EF.

We note that, in all growth curve models, the EF scores were based on factor scores from CFA, thus the means were 0 at each time point. One clear limitation of this approach is that we

cannot evaluate the change of the group mean levels over time (i.e., developmental trend in EF levels as the whole sample from age 13-14 to age 15-16). However, we used CFA in an effort to appropriately capture the three different dimensions of EF according to Miyake et al.'s (2000) theoretical model. Alternative solutions such as multiple indicator growth models (in which the intercept and slope growth factors are estimated based on EF latent factors that are loaded on the three EF component scores at Time 1, Time 2, and Time 3) and longitudinal CFA (in which growth factors of Time 1, Time 2, and Time 3 are simultaneously estimated) were not available to us due to insufficient statistical power and linear dependency between the latent EF factors across time points, respectively. Future studies with larger samples or additional time points may increase power to allow for more sophisticated approaches by including the latent EF factors and the manifest indicators for each time point simultaneously in the same model (i.e., multiple indicator growth model) or re-specifying factor scores on a comparable metric across time points (i.e., longitudinal CFA model). The latter is a potential future avenue for this sample once the fourth wave of data becomes available. Despite the inherent limitations of using factor scores, there was nonetheless significant variation around the intercept and the slope of EF. Moreover, the EF slope was a significant predictor which accounted for individual differences in externalizing symptomatology outcomes.

Unlike the EF factor scores, for the three individual dimensions of EF we were able to use raw scores and estimated group-level developmental changes in the slope as well as individual differences in these changes. Each dimension demonstrated group level increases across the three time points, consistent with prior studies that have demonstrated improvements in performance on tasks for individual EF dimensions across adolescence (Huizinga et al., 2006). However, shifting was the only dimension that had significant *variance* in growth, suggesting

greater individual differences in development of shifting relative to inhibitory control or working memory. Notably, two of the EF dimensions, shifting and inhibitory control, fit latent patterns of growth better than a linear pattern. Specifically, for both shifting and inhibitory control, approximately 70% of growth was accounted for between Time 1 and Time 2, suggesting more rapid growth earlier in adolescence. However, counter to our predictions, specific dimensions of EF did not predict symptomatology. While some studies have found differential predictions for individual EF components (e.g. White et al., 2017), contemporary models of EF emphasize the problems with using performance on an individual task as an index of EF. Specifically, individual tasks may recruit additional cognitive processes distinct from EF which can reduce the interpretability of results (Friedman & Miyake, 2017). Taken together, both our findings and theoretical models suggest the value of considering multiple dimensions of EF simultaneously, as we did not detect any effects when considering individual tasks separately.

Our finding that growth in EF was predictive of externalizing problems in adolescence is consistent with previous work. In a younger sample (ages 4 through 6), Hughes and Ensor (2011) demonstrated that longitudinal gains in EF predicted lower teacher-rated hyperactivity and conduct problems. Similar patterns have been found in pre-adolescence, such that individuals who increased more in effortful control had fewer externalizing problems three years later (King et al., 2013). A more recent study with a sample similar to ours (ages 13 through 17) found that adolescents who had greater growth in self-regulation (based on self- and parent- report of adolescent's ability to regulate emotions, cognitions, and behaviors) were less likely to engage in risky sexual behaviors at age 18 (Crandall, Magnusson, & Novilla, 2017). Our findings corroborate these patterns of results showing longitudinal effects of self-regulation on later psychopathology.

Adolescents who are making more rapid progress in their cognitive development may be better equipped to appropriately manage the challenges they encounter during this developmental period. In addition to neurobiological and pubertal maturation, adolescents must also master novel social contexts, intellectual challenges, and navigate increasing autonomy (Blakemore, 2008; Steinberg, 2005). These challenges often occur in conjunction with heightened emotionality and stress reactivity (Powers & Casey, 2015). This combination of conditions has contributed to the historical view of adolescence as a period of "storm and stress" (Hall, 1904). However, adolescents are not equally susceptible to the stressful nature of this developmental period, with many individuals successfully navigating these challenges (Arnett, 1999). Thus, more recent research supports the idea that adolescence is a period of both vulnerabilities *and* opportunities (Dahl, 2004). Nonetheless, individuals with deviant patterns of self-regulation growth may be more susceptible to "vulnerabilities". Rather than capitalizing on opportunities in a way that promotes autonomy and successful adaptation, individuals with more gradual, prolonged growth in EF may struggle to adaptively respond to challenges. EF is essential for behavior planning and management of arousal (Blair & Ursache, 2011) and those who are not experiencing growth in these abilities at the same rate as their peers may be more susceptible to the development of behavioral problems including substance use, aggression, and delinquency.

While our post-hoc analyses revealed associations between growth in EF and externalizing symptomatology, we did not find any significant associations with internalizing symptomatology. These results appear to contradict a recent study by White et al. (2017) which demonstrated stronger effects of EF (general EF as well as individual dimensions of response inhibition and working memory) on internalizing symptomatology relative to externalizing symptomatology. However, there are a number of aspects of their study that may account for this

discrepancy, including (1) the large sample (n > 9,000) with greater prevalence of clinical-level symptomatology, (2) the cross-sectional design, and (3) the wide age range of the sample (8 to 21 years). Our much smaller, community sample focused on a specific window of adolescent development where patterns of EF and psychopathology may manifest differently, in part due to the changes in prefrontal functioning that are occurring during this particular age range (Casey, Jones, & Hare, 2008). Other studies examining the relation between specific dimensions of EF and internalizing symptomatology have often utilized emotionally-salient tasks (Mocan, Stanciu, & Visu-Petra, 2014), whereas the cognitive tasks implemented in our study were emotionally-neutral tasks. The nature of cognitive processes involved in managing the emotional arousal that is characteristic of internalizing disorders may not have been detected with our set of tasks. In addition, tasks such as the MSIT involve behavioral control (i.e., reaction time) which may better translate to behavioral regulation and be more relevant to externalizing problems than internalizing problems, as supported by our findings.

Our original hypotheses regarding effects among EF and internalizing and externalizing symptomatology growth factors were not supported in our sample. This finding is inconsistent with most prior studies that have found significant associations between growth in self-regulation and adjustment, which have utilized questionnaire-based assessments of both self-regulation and adjustment (e.g. King et al., 2013; Piehler et al., 2013). However, it is possible that the effects in these studies may have been inflated due to method invariance, and our efforts to incorporate both self-report and behavioral measures may have attenuated any effects in our sample.

Limitations to the current study should be addressed in future analyses. First, we utilized factor scores to index EF which limited the evaluation of mean-level developmental changes of the whole sample. Second, our limited sample size prevented us from testing effects of factors

that have been shown to moderate the associations between self-regulation and adjustment, such as gender (Hatoum et al., in press). Third, we used only adolescent report of adjustment, rather than multiple informants. However, the correlations between parent and adolescent report were relatively low in our sample and by this age, adolescents may be better reporters of internalizing and externalizing symptomatology because with increased autonomy parents may not be aware of certain emotions they are experiencing or behaviors they are exhibiting in different contexts. Finally, our sample predominantly identifies as White and thus generalizability of the findings to more racially diverse populations awaits further replication.

Despite these limitations, our study design demonstrates a number of strengths that can be retained in future analyses. First, we attempted to model change and growth in self-regulation to better reflect the developmental and dynamic nature of this construct, compared to prior studies that examine self-regulation at a single time point. Next, we used multiple behavioral tasks to index EF. Studies of EF are frequently limited by the use of self-report or single-indicator measurement of EF. By using multiple measures and CFA to model the latent variable of EF, we were able to extract the common variance and minimize problems related to task impurity (Miyake et al., 2000; Rabbitt, 1997). Finally, we identified significant variance in trajectories of EF development and its individual dimensions during a period when this aspect of development is especially important. That is, the emerging neural imbalance between subcortical regions related to emotion and motivation relative to prefrontal, regulatory areas during this period (Casey, Jones, & Hare, 2008) makes this topic of research particularly germane to adolescence. Moving forward, it is important for future work to examine antecedents to variability in EF development during adolescence. Little is known about specific aspects of adolescents' environmental contexts that contribute to variability in longitudinal growth of EF.

However, available studies on self-regulation indicate that maternal parenting (Moilanen & Rambo-Hernandez, 2017) and stressful life events (Hughes & Ensor, 2009) are related to longitudinal change in self-regulation in pre- and early adolescence. Few studies have investigated how early environment is associated with growth in self-regulation and future work should evaluate how these contexts may contribute to individual differences in EF *development*.

The current study demonstrates the value of a developmental perspective in understanding the relations between neurocognitive processes and adjustment in adolescence. That is, levels of EF *and* change over time may have important implications for how externalizing symptomatology unfolds during adolescence. The few studies that have adopted this perspective have been done in younger samples (e.g., King et al., 2013; Hughes & Ensor, 2009); our findings offer the first evidence that developmental growth in EF continues to be important for externalizing outcomes into adolescence. Specifically, our results suggest that adolescents who are experiencing slower *growth* (rather than earlier levels) in prefrontal functioning may be more vulnerable to the development of behavioral problems. If these findings are replicated, they suggest that targeting *trajectories* of neurocognitive self-regulation may be crucial in mitigating adolescent externalizing symptomatology. For example, there is evidence that an intervention program for children predicted increased growth in EF, which in turn predicted decreases in conduct problems (Piehler et al., 2013). Applied to adolescents, interventions such as this may be able to alter trajectories of EF development for those who are demonstrating slower growth than their peers. Taken together, the results of this study highlight the importance of a dynamic, developmental approach to studying neurocognitive development in order to better understand the nature of neurocognitive self-regulation and its association with adjustment in adolescence.

References

Achenbach, T.M. & Rescorla, L. (2001). *Manual for the ASEBA School-Age Forms and Profiles.* Burlington, VT: Department of Psychiatry, University of Vermont.

Arbuckle, J. L. (1996). Full information estimation in the presence of incomplete data. In G. A. Marcoulides & R. E. Schumacker (Eds.), *Advanced structural equation modeling: Issues and techniques* (pp. 243-277). Mahwah, NJ: Erlbaum.

Arnett, J. J. (1999). Adolescent storm and stress, reconsidered. *American Psychologist, 54,* 317–326.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science*, *16*, 351-355. doi: 10.1111/j.1467-8721.2007.00534.x

Becker, D. R., Miao, A., Duncan, R., & McClelland, M. M. (2014). Behavioral self-regulation and executive function both predict visuomotor skills and early academic achievement. *Early Childhood Research Quarterly*, *29*, 411–424. http://doi.org/10.1016/j.ecresq.2014.04.014

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238–246. doi:10.1037/0033-2909. 107.2.238.

Bentler, P. M., & Chou, C. P. (1987) Practical issues in structural modeling. *Sociological Methods & Research, 16*, 78-117.

Blair, C., & Ursache, A. (2011). A bidirectional model of executive functions and self-regulation. In K. D. Vohs & R. F. Baumeister (Eds.), Handbook of self- regulation: Research, theory, and applications (2nd ed., pp. 300–320). New York, NY: Guilford Press.

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

Boelema, S. R., Harakeh, Z., Ormel, J., Hartman, C. A., Vollebergh, W. A. M., & van Zandvoort, M. J. E., (2014). Executive functioning shows differential maturation from early to late adolescence : Longitudinal findings from a TRAILS study, *28*, 177–187. doi:10.1037/neu0000049

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In Bollen & S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Beverly Hills: Sage.

Bush, G., Shin, L. M., Holmes, J., Rosen, B. R., & Vogt, B. A. (2003). The Multi-Source Interference Task: Validation study with fMRI in individual subjects. *Molecular Psychiatry*, *8*, 60-70. doi:10.1038/sj.mp.4001217

Casey, B., Jones, R. M., & Hare, T. A. (2008). The adolescent brain. *Annals of the New York Academy of Sciences, 1124*, 111–126. doi:10.1196/annals.1440.010

Collette, F., Van der Linden, M., Laureys, S., Delfiore, G., Degueldre, C., Luxen, A., & Salmon, E. (2005). Exploring the unity and diversity of the neural substrates of executive function. *Human Brain Mapping*, *25*, 409–423. doi:10.1002/hbm.20118

Costello, E. J., Copeland, W., & Angold, A. (2011). Trends in psychopathology across the adolescent years: What changes when children become adolescents, and when adolescents become adults? *Journal of Child Psychology and Psychiatry, 52*, 1015–1025.

Costello, E., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60*, 837-844. doi: 10.1001/archpsyc.60.8.837

Crandall, A. A., Magnusson, B. M., & Novilla, M. L. B. (2017). Growth in adolescent self-regulation and impact on sexual risk-taking: A curve-of-factors analysis. *Journal of Youth and Adolescence*, 1–14. http://doi.org/10.1007/s10964-017-0706-4

Dahl, R. E. (2004), Adolescent brain development: A period of vulnerabilities and opportunities. Keynote Address. *Annals of the New York Academy of Sciences, 1021*, 1–22. doi:10.1196/annals.1308.001

Deater-Deckard, K. (2014). Family matters: Intergenerational and interpersonal processes of executive function and attentive behavior. *Current Directions in Psychological Science*, *23*, 230–236. doi:10.1177/0963721414531597

Espy, K. A., Sheffield, T. D., Wiebe, S. A., Clark, C. A. C., & Moehr, M. J. (2011). Executive control and dimensions of problem behaviors in preschool children. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *52*, 33–46. doi:10.1111/j.1469-7610.2010.02265.x

Friedman, N. P., & Miyake, A. (2017). Unity and diversity of executive functions: Individual differences as a window on cognitive structure. *Cortex*, 1–19. doi:10.1016/j.cortex.2016.04.023

Galvan, A., Hare, T.A., Parra, C.E., Penn, J., Voss, H., Glover, G., & Casey, B.J. (2006). Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience, 26*, 6885–6892.

Ghosh, D., & A. Vogt. (2012). Outliers: An evaluation of methodologies. In *Joint Statistical Meetings* (pp. 3455–3460). American Statistical Association, San Diego, CA.

Gotlib, I.H., Joormann, J., 2010. Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology, 6*, 285 - 312.

Hall, G. S. (1904). Adolescence: In psychology and its relation to physiology, anthropology, sociology, sex, crime, religion, and education, Vol. I & II, Englewood Cliffs, NJ: Prentice-Hall.

Hare, T.A., Tottenham, N., Galvan, A., Voss, H.U., Glover, G.H., & Casey, B.J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biological Psychiatry*, *63*, 927–934.

Hatoum, A. S., Rhee, S. H., Corley, R. P., Hewitt, J. K., & Friedman, N. P. (in press). Do executive functions explain covariance between internalizing and externalizing behaviors? *Development and Psychopathology*. http://doi.org/10.1017/S0954579417001602

Heaton, R. K., & Staff, P. A. R. (2003). Wisconsin card sorting test: Computer version 4-research edition (WCST: CV4). Lutz, FL: Psychological Assessment Resources.

Holmes, C. J., Kim-Spoon, J., & Deater-Deckard, K. (2016). Linking executive function and peer problems from early childhood through middle adolescence. *Journal of Abnormal Child Psychology*, *44*, 31–42. doi:10.1007/s10802-015-0044-5

Hughes, C., & Ensor, R. (2011). Individual differences in growth in executive function across the transition to school predict externalizing and internalizing behaviors and self-perceived academic success at 6 years of age. *Journal of Experimental Child Psychology*, *108*, 663–676. doi:10.1016/j.jecp.2010.06.005

Huizinga, M., Dolan, C. V., & van der Molen, M. W. (2006). Age-related change in executive function: Developmental trends and a latent variable analysis. *Neuropsychologia*, *44*, 2017–2036. doi:10.1016/j.neuropsychologia.2006.01.010

Jester, J. M., Nigg, J. T., Puttler, L. I., Long, J. C., Fitzgerald, H. E., & Zucker, R. A. (2009). Intergenerational transmission of neuropsychological executive functioning. *Brain and Cognition, 70*, 145–153. doi:10.1016/j.bandc.2009.01.005

Joormann, J., & Gotlib, I. H. (2008). Updating the contents of working memory in depression: Interference from irrelevant negative material. *Journal of Abnormal Psychology, 117*, 182-192. doi:10.1037/0021-843X.117.1.182

King, K. M., Lengua, L. J., & Monahan, K. C. (2013). Individual differences in the development of self-regulation during pre-adolescence: Connections to context and adjustment. *Journal of Abnormal Child Psychology*, *41*, 57–69. doi:10.1007/s10802-012-9665-0

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

Klingberg, T., Forssberg, H., & Westerberg, H. (2002). Increased brain activity in frontal and parietal cortex underlies the development of visuospatial working memory capacity during childhood. *Journal of Cognitive Neuroscience, 14*, 1-10.

Kwon, H., Reiss, A. L., & Menon, V. (2002). Neural basis of protracted developmental changes in visuo-spatial working memory. *Proceedings of the National Academy of Sciences, 99*, 13336–13341.

Lee, K., Bull, R., & Ho, R. M. H. (2013). Developmental changes in executive functioning. *Child Development*, *84*, 1933–1953. doi:10.1111/cdev.12096

Letkiewicz, A. M., Miller, G. A., Crocker, L. D., Warren, S. L., Infantolino, Z. P., Mimnaugh, K. J., & Heller, W. (2014). Executive function deficits in daily life prospectively predict increases in depressive symptoms. *Cognitive Therapy and Research*, 612–620. doi:10.1007/s10608-014-9629-5

Luna, B., Padmanabhan, A., & O'Hearn, K. (2010). What has fMRI told us about the development of cognitive control through adolescence? *Brain and Cognition*, *72*, 101-113. doi:10.1016/j.bandc.2009.08.005

MacDonald, S. W. S., Karlsson, S., Rieckmann, A., Nyberg, L., & Bäckman, L. (2012). Aging-related increases in behavioral variability: Relations to losses in dopamine D1 receptors. *Journal of Neuroscience, 32*, 8186-8191. doi: 10.1523/JNEUROSCI.5474-11.2012

Maciejewski, D. F., van Lier, P. A., Branje, S. J., Meeus, W. H., & Koot, H. M. (2015). A 5-year longitudinal study on mood variability across adolescence using daily diaries. *Child Development*, *86*, 1908-1921.

Maciejewski, D. F., van Lier, P. A. C., Branje, S. J. T., Meeus, W. H. J., & Koot, H. M. (2016). A daily diary study on adolescent emotional experiences: Measurement invariance and developmental trajectories. *Psychological Assessment*. doi: 10.1037/pas0000312

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology, 22,* 491–495. doi:10.1017/ S0954579410000222

Mischel, W. (2004). Toward an integrative model for CBT: Encompassing behavior, cognition, affect, and process. *Behavior Therapy*, *35*, 185–203. doi:10.1016/S0005-7894(04)80011-X

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology*, *41*, 49–100. doi:10.1006/cogp.1999.0734

Mocan, O., Stanciu, O., & Visu-Petra, L. (2014). Relating individual differences in internalizing symptoms to emotional attention set-shifting in children. *Anxiety, Stress & Coping*, *27*, 509-526.

Moilanen, K. L., & Rambo-Hernandez, K. E. (2017). Effects of maternal parenting and mother-child relationship quality on short-term longitudinal change in self-regulation in early adolescence. *Journal of Early Adolescence*, *37*, 618–641. http://doi.org/10.1177/0272431615617293

Moran, T. P. (2016). Anxiety and working memory capacity: A meta-analysis and narrative review. *Psychological Bulletin*, *142*, 831–864. doi:10.1037/bul0000051

Nigg, J. T. (2017). Annual Research Review: On the relations among self-regulation, self-control, executive functioning, effortful control, cognitive control, impulsivity, risk-taking, and inhibition for developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *58*, 361–383. doi: 10.1111/jcpp.12675

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

Piehler, T. F., Bloomquist, M. L., August, G. J., Gewirtz, A. H., Lee, S. S., & Lee, W. S. C. (2014). Executive functioning as a mediator of conduct problems prevention in children of homeless families residing in temporary supportive housing: A parallel process latent growth modeling approach. *Journal of Abnormal Child Psychology*, *42*, 681–692. doi:10.1007/s10802-013-9816-y

Powers, A., & Casey, B. J. (2015). The adolescent brain and the emergence and peak of psychopathology. *Journal of Infant, Child, and Adolescent Psychotherapy*, *14*, 3–15. doi:10.1080/15289168.2015.1004889

Rabbitt, P. (1997). Introduction: Methodologies and mod- els in the study of executive function. In P. Rabbitt (Ed.), Methodology of frontal and executive function (pp. 1–38). Hove, UK: Psychology Press.

Roid, G. H. (2003). *Stanford-Binet Intelligence Scales, Fifth Edition*, Itasca, IL: Riverside Publishing.

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

Somerville, L. H., Jones R. M., & Casey, B. J. (2010). A time of change: Behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain and Cognition, 72,* 124–133.

Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience, 2*, 859-861.

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Science, 9,* 69–74.

Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology, 44*, 1764–1778.

United States Census Bureau (2010). *American community survey*. Retrieved from http://factfinder2.census.gov/faces/nav/jsf/pages/searchresults.xhtml?refresh=t.

Van Dulmen, M. H. M., Egeland, B., van Dulmen, M. H. M., Egeland, B., Dulmen, V., & Egeland, B. (2011). Analyzing multiple informant data on child and adolescent behavior problems: Predictive validity and comparison of aggregation procedures. *International Journal of Behavioral Development*, *35*, 84–92. doi:10.1177/0165025410392112

White, L. K., Moore, T. M., Calkins, M. E., Wolf, D. H., Satterthwaite, T. D., Leibenluft, E., … Gur, R. E. (2017). An evaluation of the specificity of executive function impairment in developmental psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry, 56,* 975-982. http://doi.org/10.1016/j.jaac.2017.08.016

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of Attention-Deficit/Hyperactivity Disorder: A meta-analytic review. *Biological Psychiatry*, *57*, 1336–1346. doi:10.1016/j.biopsych.2005.02.006

Willoughby, M., Holochwost, S. J., Blanton, Z. E., & Blair, C. B. (2014). Executive functions: Formative versus reflective measurement. *Measurement: Interdisciplinary Research and Perspectives*, *12*, 69–95. doi:10.1080/15366367.2014.929453

Woltering, S., Lishak, V., Hodgson, N., Granic, I., & Zelazo, P. D. (2015). Executive function in children with externalizing and comorbid internalizing behavior problems. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *1*, 30–38. doi:10.1111/jcpp.12428

Zhou, Q., Chen, S. H., & Main, A. (2012). Commonalities and differences in the research on children's effortful control and executive function: A call for an integrated model of self-regulation. *Child Development Perspectives*, *6*, 112–121. doi:10.1111/j.1750-8606.2011.00176.x

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	1
Time 1																			
1. Intelligence																			(
2. Inhibitory control	.31*																		.
3. Working memory	.45**	.21**																	(
4. Shifting	.24**	.25**	.21**																(
5. EF factor score	.46**	.74**	.62**	.72**															(
6. Internalizing symptomatology	-.04	.11	.004	.03	.08														(
7. Externalizing symptomatology	-.02	-.10	-.04	-.04	-.09	.46**													(
Time 2																			
8. Inhibitory control	.30**	.49**	.25**	.17*	.45**	-.03	-.15												(
9. Working memory	.39**	.12	.67**	.17*	.42**	-.16	-.10	.22**											(
10. Shifting	.22**	.28**	.29**	.50**	.52**	-.11	-.14	.23**	.23**										(
11. EF factor score	.43**	.42**	.57**	.39**	.66**	-.14	-.18	.69**	.69**	.71**									(
12. Internalizing symptomatology	.01	.19*	-.04	.05	.11	.69**	.27**	.03	-.17*	-.07	-.10								(
13. Externalizing symptomatology	-.05	-.06	-.09	-.004	-.07	.26**	.71**	-.15	-.08	-.20*	-.21*	.31**							(

Time 3																			
14. Inhibitory control	.23**	.45**	.18*	.20*	.40**	-.05	-.13	.59**	.19*	.28**	.51**	.09	-.10						
15. Working memory	.48**	.18*	.62**	.28**	.48**	-.13	-.11	.26**	.72**	.22**	.56**	-.13	-.11	.18*					
16. Shifting	.10	.15	.15	.11	.19*	.11	.07	.23**	.10	.10	.21*	.06	-.04	.23**	.16				
17. EF factor score	.35**	.42**	.39**	.28**	.51**	-.02	-.08	.56**	.40**	.30	.60**	.04	-.12	.80**	.51**	.68**			
18. Internalizing symptomatology	.08	.33**	-.06	-.10	.09	.56**	.21*	.03	-.10	-.04	-.05	.74**	.22**	.09	-.03	.02	.06		
19. Externalizing symptomatology	-.002	.01	-.12	-.06	-.08	.22**	.66**	-.09	-.08	-.13	-.14	.25**	.76**	-.07	-.06	-.13	-.13	.37**	

Table 1.
Correlations and descriptive statistics for study variables.

Note. T scores for symptomatology are reported for the correlation table for descriptive purposes, but raw sum scores were used for model testing.
*$p < .05$, **$p < .01$

Model Label	χ^2	*df*	*p*	RMSEA	CFI	$\Delta\chi^2$	Δdf	*p*(d)
EF factor score								
1. No-growth model	10.74	6	.10	.07	.97			
2. Linear growth model	**2.15**	**3**	**.54**	**.00**	**1.00**	**8.59**	**3**	**.04**
3. Latent growth model	.37	2	.83	.00	1.00	1.78	1	.18
MSIT								
1. No-growth model	165.00	6	.00	.40	.00			
2. Linear growth model	12.37	3	.01	.14	.90	152.63	3	.00
3. Latent growth model	**.34**	**2**	**.84**	**.00**	**1.00**	**12.03**	**1**	**.00**
WCST								
1. No-growth model	190.91	6	.00	.43	.00			
2. Linear growth model[+]	37.97	3	.00	.26	.09	152.94	3	.00
3. Latent growth model[++]	**.22**	**1**	**.64**	**.00**	**1.00**	**37.75**	**2**	**.00**
SB-DS								
1. No-growth model	15.34	6	.02	.10	.95			
2. Linear growth model	**3.26**	**3**	**.35**	**.02**	**1.00**	**12.08**	**3**	**.01**
3. Latent growth model	1.32	2	.52	.00	1.00	1.94	1	.16

Table 2

Fit Indices of Baseline Growth Curve Models for Executive Functioning Variables

Note. Best-fitting baseline model in boldface. CFI = comparative fit index; RMSEA = root mean square error of approximation; $\Delta\chi^2$ = difference in likelihood ratio tests; Δdf = difference in *df*; *p*(d) = probability of the difference tests. EF = Executive Functioning; MSIT = Multi-Source Interference Task; WCST = Wisconsin Card Sort Task; SB-DS = Stanford-Binet Digit Span.

[+]Correlation greater than 1 between slope and intercept factors. Freed theta to improve model fit and fixed correlation between slope and intercept to 1. This resulted in negative variance on the slope factor, which was then fixed to 0.

[++]Negative residual variance of WCST at Time 1. Released theta to improve model fit and fixed negative variance of WCST Time 1 to 0.

Model Label	χ^2	*df*	*p*	RMSEA	CFI	$\Delta\chi^2$	Δdf	*p*(d)
Externalizing symptomatology								
1. No-growth model	17.98	6	.01	.11	.95			
2. Linear growth model	**2.97**	**3**	**.40**	**.00**	**1.00**	**15.01**	**3**	**.00**
3. Latent growth model	2.83	2	24	.05	1.00	.14	1	.71
Internalizing symptomatology								
1. No-growth model	33.05	6	.00	.16	.89			
2. Linear growth model	23.24	3	.00	.20	.92	9.81	3	.02
3. Latent growth model	**0.00**	**0**	**.00**	**0.00**	**1.00**	**23.24**	**3**	**<.001**

Table 3

Fit Indices of Baseline Growth Curve Models for Adjustment Variables

Note. Best-fitting baseline model in boldface. CFI = comparative fit index; RMSEA = root mean square error of approximation; $\Delta\chi^2$ = difference in likelihood ratio tests; Δdf = difference in *df*; *p*(d) = probability of the difference tests.

	Estimate	Standard Error
EF and externalizing symptomatology		
Regression effects on intercept		
Intelligence → EF intercept	0.54**	0.07
Gender → Externalizing symptomatology intercept	-0.05	0.09
Age → Externalizing symptomatology intercept	-0.01	0.16
Regression effects on slope		
Intelligence → EF slope	-0.17	0.13
Gender → Externalizing symptomatology slope	-0.14	0.13
Age → Externalizing symptomatology slope	0.29*	0.13
Regressions between EF and externalizing symptomatology factors		
EF intercept → Externalizing symptomatology slope	-0.07	0.15
Externalizing intercept → EF slope	-0.001	0.16
EF slope → Externalizing symptomatology slope	-0.35	0.25
Factor covariances		
EF intercept ↔ Externalizing symptomatology intercept	-0.14	0.11
EF intercept ↔ EF slope	-0.35*	0.14
Externalizing symptomatology intercept ↔ Externalizing symptomatology slope	-0.01	0.19
Inhibitory control and externalizing symptomatology		
Regression effects on intercept		
Intelligence → IC intercept	0.41**	0.09
Gender → Externalizing symptomatology intercept	-0.06	0.09
Age → Externalizing symptomatology intercept	-0.01	0.09
Regression effects on slope		
Intelligence → EF slope	-0.05	0.16
Gender → Externalizing symptomatology slope	-0.12	0.14
Age → Externalizing symptomatology slope	0.28*	0.13
Regressions between IC and externalizing symptomatology factors		
IC intercept → Externalizing symptomatology slope	0.20	0.19
Externalizing symptomatology intercept → IC slope	-0.07	0.20
IC slope → Externalizing symptomatology slope	-0.36	0.31
Factor covariances		
IC intercept ↔ Externalizing symptomatology intercept	-0.14	0.14
IC intercept ↔ IC slope	-0.09	0.26
Externalizing symptomatology intercept ↔ Externalizing symptomatology slope	0.01	0.21

Working memory and externalizing symptomatology		
Regression effects on intercept		
Intelligence → WM intercept	0.50**	0.08
Gender → Externalizing symptomatology intercept	-0.05	0.09
Age → Externalizing symptomatology intercept	-0.02	0.09
Regression effects on slope		
Intelligence → WM slope	0.16	0.19
Gender → Externalizing symptomatology slope	-0.10	0.14
Age → Externalizing symptomatology slope	0.27*	0.13
Regressions between WM and externalizing symptomatology factors		
WM intercept → Externalizing symptomatology slope	-0.03	0.19
Gender → Externalizing symptomatology intercept	-0.05	0.21
WM slope → Externalizing symptomatology slope	0.04	0.37
Factor covariances		
WM intercept ↔ Externalizing symptomatology intercept	-0.05	0.12
WM intercept ↔ WM slope	0.04	0.29
Externalizing symptomatology intercept ↔ Externalizing symptomatology slope	-0.01	0.96

Table 4
Parameter Estimates and Standard Errors for Parallel Process GCM with Executive Functioning (EF) and Externalizing Symptomatology

Note. *$p < .05$, **$p < .01$. WM = working memory, IC = inhibitory control.

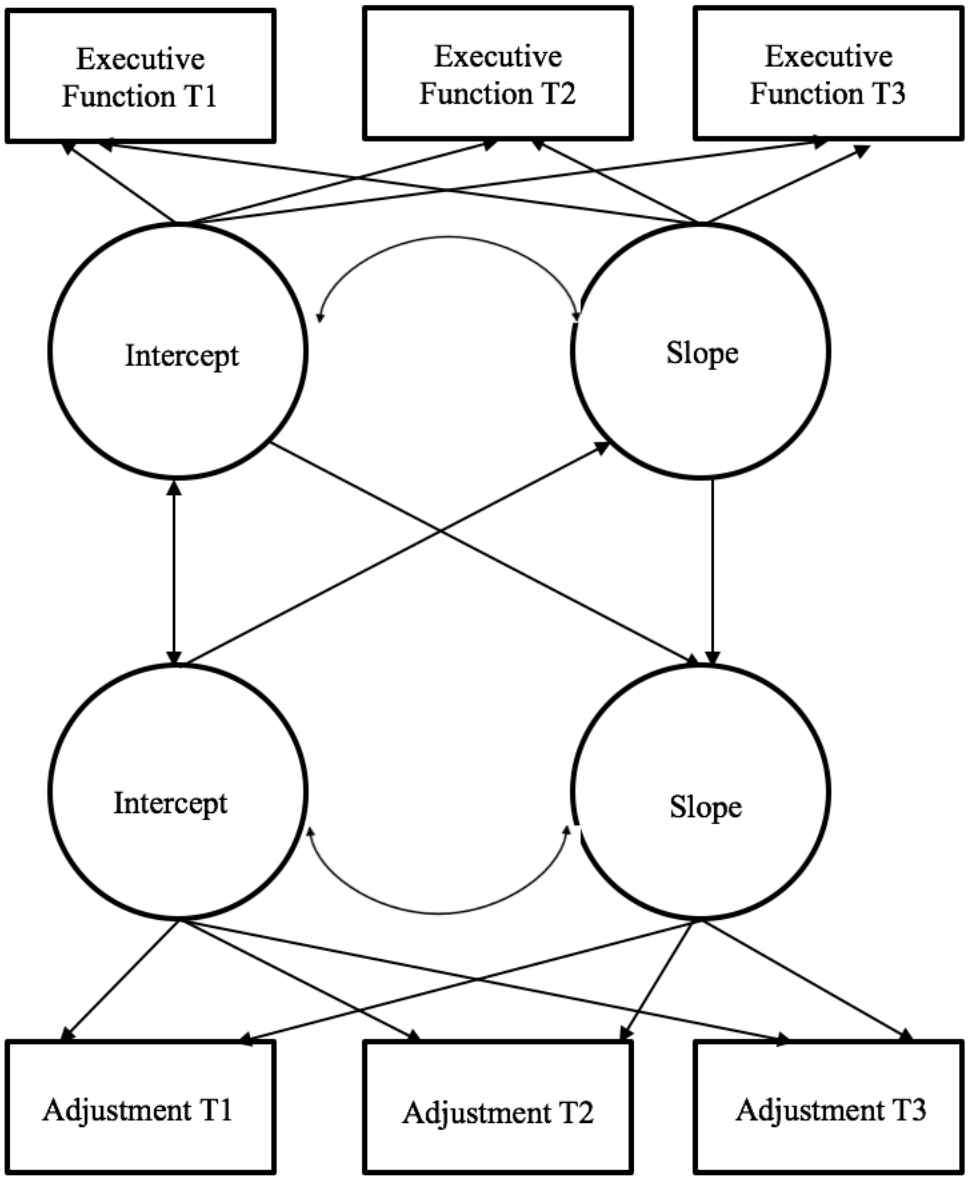

Figure 1. Proposed longitudinal model of the joint developmental trajectories of EF and adjustment (internalizing and externalizing symptomatology tested separately).

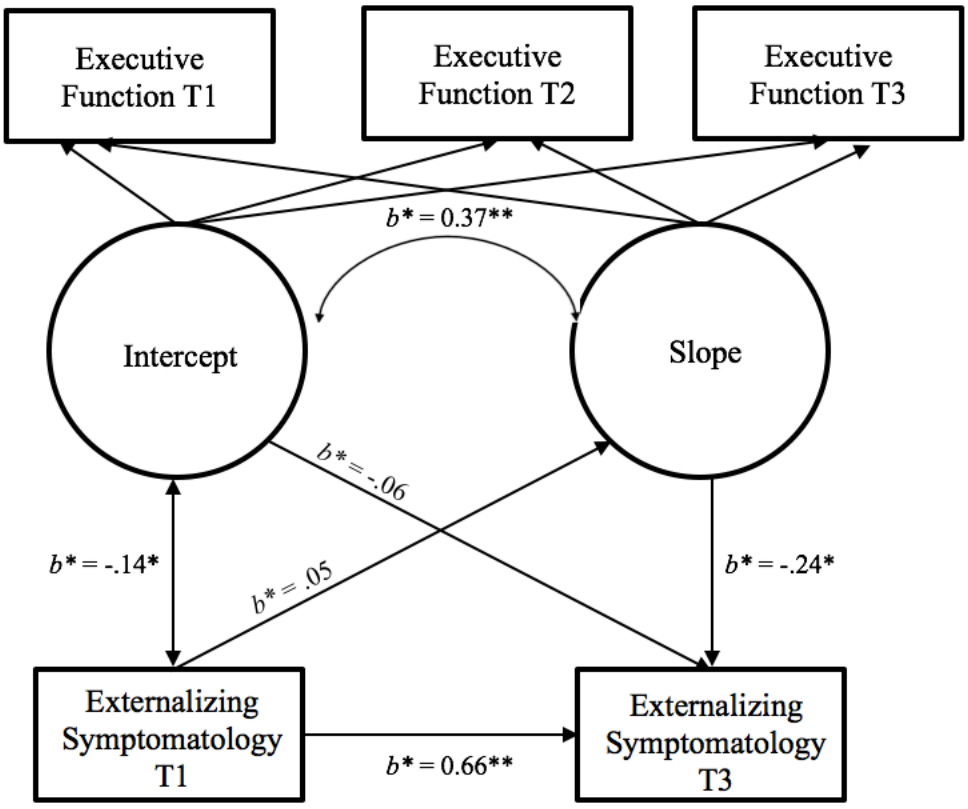

Figure 2. Post-hoc analyses of the effect of growth and initial levels of executive function on externalizing symptomatology at Time 3. Covariates age and gender were estimated but not included for clarity of presentation (age → externalizing symptomatology T3 = 0.14*, gender → externalizing symptomatology T3 = -0.09, age ↔ externalizing symptomatology T1 = -0.01, age ↔ executive function intercept = -0.01, externalizing symptomatology T1 ↔ executive function intercept = -0.14, gender ↔ executive function intercept = -0.10, gender ↔ externalizing symptomatology T1 = -0.05, age ↔ gender = -0.12).

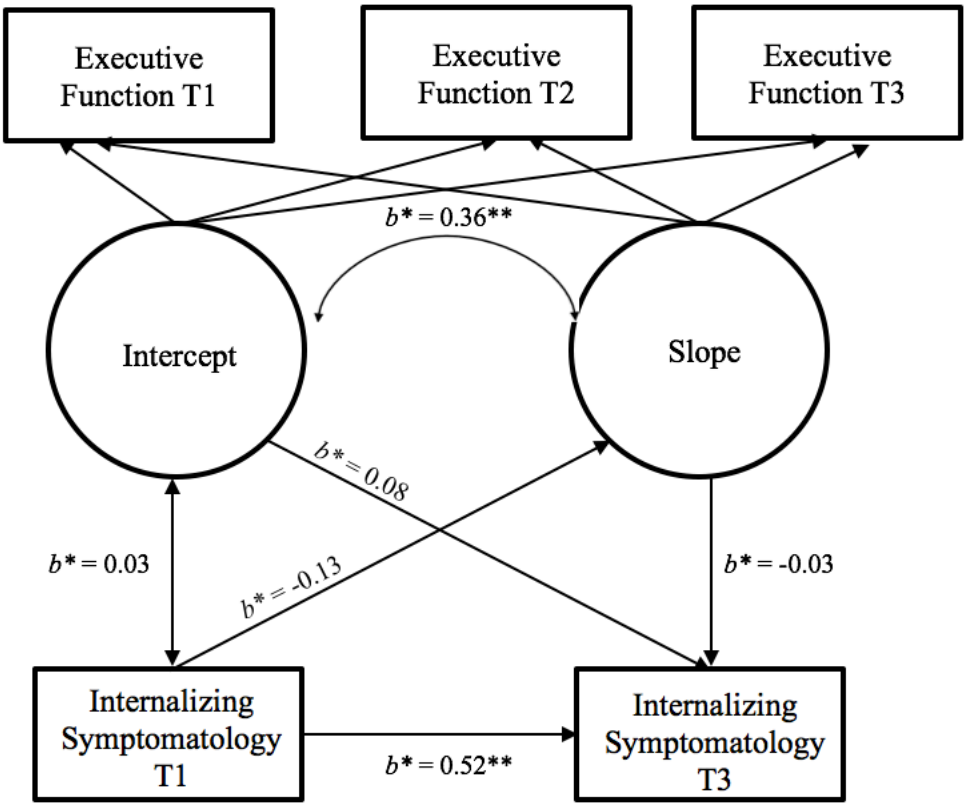

Figure 3. Post-hoc analyses of the effect of growth and initial levels of executive function on internalizing symptomatology at Time 3. Covariates age and gender were estimated but not included for clarity of presentation (age → internalizing symptomatology T3 = 0.09, gender → internalizing symptomatology T3 = 0.21*, age ↔ internalizing symptomatology T1 = -0.01, age ↔ executive function intercept = 0.00, internalizing symptomatology T1 ↔ executive function intercept = 0.03, gender ↔ executive function intercept = -0.09, gender ↔ internalizing symptomatology T1 = 0.29**, age ↔ gender = -0.12).

Appendix A- Adolescent Demographic Interview

Demographic Interview

1. How old are you? _____

2. What is your sex?

   A. Male
   B. Female

3. How would you describe your own race?

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

4. How would you describe your own ethnicity?
   1) Hispanic or Latino
   2) Not Hispanic or Latino

5. Are you in school?
   1) Middle school
   2) High School
   3) College
   4) Not in school

Other (Please specify) _____

Appendix B- Parent Demographic Interview

Demographic Interview

Introduction:

I am going to ask you some basic questions about the work and educational experiences of yourself and of the people in your household. These questions are very important and need to be answered honestly. No one outside of the project will ever have access to this information. The information that you provide us will not affect any services or assistance that you might be receiving. This information will only be used for the purposes of our research.

1. What is your relation to the child? (RESNM)

① Mother
② Father
③ Grandmother
④ Grandfather
⑤ Foster Parent
⑥ Other – Specify who other is _____

2. What is your gender? (R_GENDER)

⓪ Male
① Female

3. If respondent is not the biological parent ask:
"How long has this child been in your care?" (RCARE) _____

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

5. What are your birth month and year? (MonthBirth) __ __/__ __ __ __
Month Year

Continue on to next page.

11a. How would you describe your own race?

RRACE _____

11b. How would you describe the race of the child who is participating with you in this study?

CRACE _____

1 = American Indian/Alaska Native

2 = Asian

3 = Native Hawaiian or Other Pacific Islander

4 = Black or African American

5 = White

6 = More than one race

7 = Other _____

11c. How would you describe your own ethnicity?

RETH _____

① Hispanic or Latino

② Not Hispanic or Latino

11d. How would you describe the ethnicity of the child who is participating with you in this study?

CETH _____

① Hispanic or Latino

② Not Hispanic or Latino

For the following questions, please fill in the bubble for the number or letter that is associated with your answer.

18. Do you receive any public income assistance such as TANF (Temporary Assistance for Needy Families), AFDC (Aid to Families with Dependent Children), food stamps, fuel assistance, rent vouchers or SSI (Supplemental Security Income)? (AID)

① Yes
② No

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

Ⓐ	None	or	$0 per month
Ⓑ	Less than 1,000	or	Less than $83 per month
Ⓒ	$1,000 - $2,999	or	$83 - $249 per month
Ⓓ	$3,000 - $4,999	or	$250 - $416 per month
Ⓔ	$5,000 - $7,499	or	$417 - $624 per month
Ⓕ	$7,500 - $9,999	or	$625 - $833 per month
Ⓖ	$10,000 - $14,999	or	$834 - $1,249 per month
Ⓗ	$15,000 - $19,999	or	$1,250 - $1,666 per month
Ⓘ	$20,000 - $24,999	or	$1,667 - $2,083 per month
Ⓙ	$25,000 - $34,999	or	$2,084 - $2,916 per month
Ⓚ	$35,000 - $49,999	or	$2,917 - $4,167 per month
Ⓛ	$50,000 - $74,999	or	$4,168 - $6,249 per month
Ⓜ	$75,000 - $99,999	or	$6, 250 - $8,333 per month
Ⓝ	$100,000 - $199,999	or	$8,334 - $16,666 per month
Ⓞ	$200,000 or more	or	$16,667 or more per month

Please circle the number corresponding with your answer to the following questions about your health.

20. During the last 12 months (one year), would you say that your general health has been...(HEALTH)

① Excellent
② Good
③ Fair
④ Poor