

Concurrent and Longitudinal Relations among Neurocognitive Functioning, Coping, and
Depressive Symptoms in Youth

By

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Dissertation

Submitted to the Faculty of the
Graduate School of Vanderbilt University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

in

Psychology

August, 2014

Nashville, Tennessee

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ACKNOWLEDGEMENTS

This work would not have been possible without the financial support of the Vanderbilt Graduate School Fellowship and the Vanderbilt Kennedy Center for Developmental Disabilities.

I am grateful to the members of my Dissertation Committee who have provided me with extensive personal and professional guidance and taught me a great deal about clinical research. I would especially like to thank Dr. Judy Garber, the chair of my committee and my mentor, who has taught what it means to be a good writer and scientist. I would also like to thank my family. Most importantly, I wish to thank my supportive husband, Russell, who provides unending encouragement and inspiration.

Finally, I am grateful to children and parents participants who generously donated their time and efforts to this project.

TABLE OF CONTENTS

	Page
ACKNOWLEDGMENTS	ii
LIST OF TABLES	iv
LIST OF FIGURES	v
Introduction.....	1
Method	7
Participants.....	7
Measures	8
Procedure	11
Data Analytic Plan.....	12
Results.....	14
Descriptive and Preliminary Analyses.....	14
Relations between Executive Functions and Coping.....	15
Relations between Executive Functions and Depressive Symptoms.....	15
Relations between Coping and Depressive Symptoms.....	15
Direct and Indirect Effects among Executive Functions, Coping, and Depressive Symptoms	16
Direct and Indirect Effects of Executive Functions and Coping through Depressive Symptoms.....	19
Discussion.....	20
Relation between Executive Functions and Depressive Symptoms	23
Does Coping Mediate the Relation of Executive Functions to Depressive Symptoms?.....	24
Direction of the Relations among EF, Coping, and Depressive Symptoms	25
Strengths, Limitations, and Future Directions	25
Appendix.....	40
REFERENCES	45

LIST OF TABLES

Table		Page
1.	Mean and Standard Deviations of Scores for Primary Variables at T1	29
2.	Means, Standard Deviations and Bivariate Correlations among Study Variables....	30
3.	Direct Paths for Working Memory, Primary Control Coping, and Depressive Symptoms Model	32
4.	Direct Paths for Working Memory, Secondary Control Coping, and Depressive Symptoms Model	32
5.	Direct Paths for Cognitive Flexibility, Primary Control Coping, and Depressive Symptoms Model	33
6.	Direct Paths for Cognitive Flexibility, Secondary Control Coping, and Depressive Symptoms Model	33
7.	Indirect Effects of Working Memory on Time 2 Depressive Symptoms through Time 2 Coping	34
8.	Indirect Effects of Cognitive Flexibility on Time 2 Depressive Symptoms through Time 2 Coping	35
9.	Indirect Effects of Working Memory on Time 2 Coping through T2 Depressive Symptoms.....	36
10.	Indirect Effects of Cognitive Flexibility on Time 2 Coping through T2 Depressive Symptoms.....	37

LIST OF FIGURES

Figure	Page
1. Indirect Effects of Working Memory on Depressive Symptoms through Primary Control Coping.....	38
2. Indirect Effects of Working Memory on Depressive Symptoms through Secondary Control Coping.....	38
3. Indirect Effects of Cognitive Flexibility on Depressive Symptoms through Secondary Control Coping.....	39
4. Indirect Effects of Cognitive Flexibility on Depressive Symptoms through Primary Control Coping.....	39

Introduction

Major depressive disorder (MDD) is a serious public health concern that affects interpersonal relationships, academic and work performance, and physical health. Lifetime prevalence estimates of MDD are 16.6% in adults (Kessler et al. 2005), 15% in adolescents (Kessler & Walters, 1998), and 2.5% in children (Costello, Foley & Angold, 2006). Early onset depression is associated with greater symptom severity and episode recurrence, suicidality, greater educational impairment, and high comorbidity (Berndt et al., 2000; Hollon et al., 2006; Zisook et al., 2004).

Multiple risk factors have been found to be associated with the increased likelihood of the onset and recurrence of depression including age and sex (e.g., Angold, Costello, Erkanli, & Worthman, 1999; Hankin, Abramson, Moffitt, Silva, & McGee, 1998), temperament (e.g., Caspi, Moffitt, Newman, & Silva, 1996; Goodwin, Fergusson, & Horwood, 2004; Nigg, 2006), hopelessness (Abramson, Metalsky, & Alloy, 1989), negative inferential and attributional styles (e.g., Abela & Hankin, 2008), cognitive ability (Weeks et al., 2013), executive functioning (e.g., Snyder, 2013), and neurobiological variables such as abnormal stress responses (Thase, 2008) and neural activation patterns (Ernst, Pine, & Hardin, 2006). In particular, the executive functions of working memory and cognitive flexibility have been hypothesized to underlie the ability to effectively select and utilize adaptive coping strategies in the presence of stress (e.g., Compas, 2006; Gotlib & Joorman, 2010). Executive function (EF) has been variously defined as a group of cortical functions that work to coordinate goal-oriented activity (Lezak, Howieson & Loring, 2004), and as a set of complex processes an individual uses to execute novel problem-solving tasks from inception to completion (Best, Miller, & Jones, 2009; Miyake, 2012).

In normative samples of children, EF has been found to be related to academic achievement (Blair & Razza, 2007), socioemotional adjustment (Rueda, Checa, & Rothbart,

2010), and better adaptation in the face of stress (Obradovic, 2010); deficits in EF have been associated with higher internalizing and externalizing problems (e.g., Nigg, Hinshaw, Carte, & Treuting, 1998; Riggs, Blair, & Greenberg, 2003; Riggs & Greenberg, 2004). In depressed samples, deficits in working memory and cognitive flexibility have been found in adults (e.g. Baudic, Tzortzis, Barba, & Traykov, 2004; Butters et al., 2004; Harvey et al., 2004; Snyder, 2013) and in children and adolescents (e.g. Kyte, Goodyer, Sahakian, 2005; Matthews, Coghill, & Rhodes, 2008; Micco et al., 2009). Thus, neurocognitive impairments have been related concurrently to internalizing symptoms in both normative and clinical samples. Less is known, however, about the relation of these deficits to changes in depression over time, particularly in children, (McClintock, Husain, Greer, & Cullum, 2010; Snyder, 2013).

Depressed youth have been found to have deficits in executive functions, particularly in working memory (Franklin et al., 2010; Matthews et al., 2008; Micco et al., 2009). One possible mechanism through which executive function deficits may contribute to depression is by disrupting the cognitive processes needed for coping with stress. The primary aim of the current study was to examine the links among executive functions, coping, and depressive symptoms in children. Executive processes are of particular interest because they are potentially modifiable (Diamond & Lee, 2011) and thus could be targets for intervention.

The EF domains of working memory and cognitive flexibility have been hypothesized to be associated with both coping and depression (Austin, Mitchell, & Goodwin, 2001; Castaneda et al., 2008; Compas, 2006). Working memory is a core cognitive process that involves the short-term storage of information while executing cognitive tasks that use this information. Working memory has been described as the brain's "scratch-pad," and involves the holding of pieces of information "on line" until they can be dealt with or manipulated physically or mentally (Baddeley & Hitch, 1974), such as remembering a phone number while dialing it.

Cognitive flexibility involves the ability to alter short- and long-term goals and strategies in response to changes in situations and contexts (Miyake et al., 2000). This ability to “shift” or “switch” between tasks involves top down, effortful control. Cognitive flexibility is generally considered one of the most “complex” executive functions (Davidson, Amso, Anderson, & Diamond, 2006) because it requires not only holding information in mind about new rules (i.e. working memory), but also inhibiting previously learned knowledge to carry out a new rule.

Both working memory and cognitive flexibility are central to learning and may provide the foundation for engaging in complex, adaptive behaviors in the presence of stress (Compas, 2006; Siegle & Hasselmo, 2002). Deficits in these particular executive functions may affect the cognitive processing necessary for effective selection and utilization of adaptive coping strategies (Campbell, Scaduto, Van Slyke, Niarhos, Whitlock, & Compas, 2009; Eisenberg, Fabes, Sherpard, et al., 1997; Lengua, Sandler, West, Wolchik, & Curran, 1999). Specifically, deficits in working memory have been linked to cognitive processes implicated in depression, such as intrusive thoughts (Joorman & Gotlib, 2008), difficulty problem-solving and executing multi-step plans (Gathercole et al., 2008), interpersonal problems (Fahie & Symons, 2003), and negative cognitive style (Verdejo-Garcia, Lopez-Torrecillas, Aguilar de Arcos, & Perez-Garcia, 2005). Cognitive inflexibility has been associated with impairments in generating alternative points of view and problem-solving (Lundqvist, 1995), increases in repetitive thoughts (Davis & Nolen-Hoeksema, 2000, Whitmer & Banich, 2007), and difficulty taking another’s perspective (Hughes & Ensor, 2007).

The ability to hold different thoughts simultaneously in order to evaluate or modify them (i.e., working memory) underlies both primary and secondary control coping strategies, which are dimensions of voluntary, engagement coping responses to stress (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000). For instance, changing a stressor through problem-

solving (primary control coping) requires that individuals consider multiple solutions while simultaneously evaluating each possible outcome. Consistent with the notion that working memory may be integral to engaging in effective coping, neuroimaging studies (e.g., Ochsner, Bunge, Gross, & Gabrieli, 2002) have revealed that the prefrontal regions of the brain associated with working memory are activated during utilization of cognitive coping strategies such as reframing or reappraisal (i.e., secondary control coping).

Another important executive function is cognitive flexibility, which enables switching of focus or mental engagement in response to changing situational demands. Such flexibility is crucial to functioning fluidly in the environment (Korkman, et al., 2007). Successful navigation of stressful situations and engagement of coping skills may require the ability to flexibly adapt both cognitively and behaviorally to the changing demands of a situation (Compas, 2006). Working memory and cognitive flexibility have been linked to primary control coping skills such as problem-solving and deductive reasoning (Fletcher, Marks, & Hine, 2011; Handley, Capon, Beveridge, Dennis, & Evans, 2004; Lundqvist, 1995) and to secondary control coping strategies such as cognitive restructuring (Andreotti et al., 2011; Campbell et al., 2009). Additionally, greater cognitive flexibility has been associated with increased understanding of alternate points of view (Fahie & Symons, 2003; Hughes & Ensor, 2007), which also may facilitate secondary control coping strategies such as acceptance and positive thinking.

Although some evidence exists of a relation between executive functioning and various cognitive skills utilized during coping, few empirical studies have directly examined the link between the particular executive functions of working memory and cognitive flexibility and specific coping strategies. A recent study of young adults (Andreotti et al., 2011) found a significant relation between working memory ability and the use of secondary control coping strategies (e.g., cognitive restructuring). In a study of children receiving treatment for acute

lymphocytic leukemia, Campbell and colleagues (2009) reported that greater cognitive flexibility predicted more frequent use of secondary control coping strategies (e.g., acceptance and positive thinking), which in turn, were associated with fewer behavior problems. Moreover, the relation between cognitive flexibility and behavior problems was, in part, accounted for by coping. Finally, in a study of children with functional abdominal pain, Hocking and colleagues (2011) showed that attention regulation, which is related to cognitive flexibility, predicted secondary control coping, although a measure of general EF abilities did not predict coping. Overall, there is some evidence that working memory and cognitive flexibility are related to the ability to adaptively respond to stress through secondary control coping strategies such as cognitive restructuring, acceptance, and distraction. The relation of cognitive flexibility to primary control coping (e.g., problem-solving, emotional expression, emotion modulation) is less clear, however.

Thus, an association between cognitive inflexibility and poor coping would be expected. “Executive dysfunction” has been defined as deficits in the ability to inhibit well-learned patterns of behavior and to engage in new methods of problem-solving that require a shift to using new strategies (Elliott, 2003). Coping has been defined as the “conscious, volitional effort to regulate emotion, cognitive, behavior, physiology, and the environment in response to stressful events or circumstances” (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001, p. 89), and includes attempts to change the situation or one’s emotional reactions to the situation by identifying and implementing new problem-solving strategies. Thus, individuals who lack flexibility are apt to become trapped in repetitive cycles of well-learned behaviors (i.e., perseveration) and unable to summon new strategies for coping effectively with novel situations (Henry & Bettenay, 2010). Individuals with other clinical diagnoses [e.g., substance abuse, attention deficit hyperactivity disorder (ADHD)], who have deficits in cognitive flexibility also show difficulties with perspective taking and modifying thought patterns during problem-solving

(Lundqvist, 1995). For example, among children with ADHD, those with greater impairment on a measure of cognitive flexibility -- the Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993) -- were not able to easily shift to secondary control coping and instead relied on disengagement strategies when situations went from controllable to uncontrollable (Babb, Levine, & Arseneault, 2010).

In summary, cognitive abilities such as working memory and flexibility may provide a foundation for engaging in complex thinking and for effectively selecting and utilizing adaptive coping strategies (Compas, 2006). When these executive functions are delayed or aberrant, and when the development of coping skills is slow or fails to reach full capacity, a child may become locked into repetitive patterns of behavior or thinking, or be unable to engage in complex cognitive and regulatory processes. Although executive function skills are implicated in the successful engagement of coping skills, only limited empirical evidence exists of a direct relation between EF and coping in children. The purpose of the present study was to address this gap in the literature.

The aim of this longitudinal study was to examine the concurrent and prospective relations of the executive functions of working memory and cognitive flexibility with coping (primary, secondary) and depressive symptoms in children. We tested the following hypotheses: (1) Better executive functioning (i.e., working memory and cognitive flexibility) would be significantly associated with greater use of primary control coping strategies (e.g., problem-solving, emotional modulation) and secondary control coping strategies (e.g., cognitive restructuring, acceptance). (2) We examined the direct relation between EF abilities and symptoms of depression to test the hypothesis that deficits in EF abilities would be associated with higher levels of depressive symptoms. (3) Based on previous evidence of a link between coping and depressive symptoms (Compas, Connor-Smith et al., 2001; Fear et al., 2009;

Wadsworth, Raviv, Compas, & Connor-Smith, 2005), we expected that coping strategies would be significantly associated with depressive symptoms (hypothesis 3), and would mediate the hypothesized link between EF and depressive symptoms. Specifically, primary control coping would mediate the relation between working memory and depressive symptoms (hypothesis 4), and secondary control coping would mediate the relation between cognitive flexibility and depressive symptoms (hypothesis 5). We also explored whether secondary control coping mediated the relation between working memory and depressive symptoms, and whether primary control coping mediated the relation between cognitive flexibility and depressive symptoms. Finally, to address questions about the direction of the observed relations among executive functions, coping, and depression, we tested alternative models of executive function to coping through depressive symptoms.

Method

Participants

Participants were 192 children, ages 9 to 15 (mean age = 12.36 years; $SD = 1.77$) recruited (a) from local public schools. Letters and emails explaining the study and consent forms were sent to parents of children in grades 5-9; and (b) from a university-based ListServ through which parents were emailed information about the study. The sample consisted of 100 females (52.1%) and 92 males (47.9%), and was 71.4% Caucasian, 18.2% African-American, 2.6% Asian-American, 3.6% Hispanic, and 4.2% self-reported mixed race/ethnicity. Exclusion criteria, based on parents' reports, were traumatic brain injury, neurological conditions (e.g. seizures, stroke), developmental delay (e.g. autism spectrum disorder), and significant learning or reading problems that might prevent them from understanding and completing the assessment. One child was excluded from the study prior to enrollment due to serious learning disabilities.

Measures

Executive Functioning. (a) Working Memory – Children completed the Forward and Backward Digit Span tasks of the Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV; Wechsler, 2003). The WISC-IV is widely used and has well-established psychometric properties (WISC-IV; Wechsler, 2003). Commonly used working memory tasks are Digit Span Forward and Backward (i.e. Baddeley, 1992). The Digit Span subtest requires the examinee to repeat a series of digits presented orally (Digits Forward) and then to repeat a series of digits in reversed order (Digits Backward). Both tests presumably tap short-term auditory working memory. Backward Digit Span also requires manipulation, or reordering, of mental information. A recent meta-analysis of studies of adults found deficits in both Forward and Backward Digit Span in patients with Major Depressive Disorders (Snyder, 2013). In the present analyses, children’s Digit Span Total scores were used in the working memory composite. This score represents the participant’s *span*, or the longest sequence successfully repeated, based on both the Forward and Backward Digit Span tasks.

(b) Cognitive Flexibility – A computerized version of the Wisconsin Card Sorting Test (WCST; Heaton et al., 1993) was used to assess children’s ability to flexibly adapt behavior in response to changing rules. In this task, children are presented with a series of playing cards and instructed to sort the cards into piles below one of four stimulus (key) cards. They are not given instructions about how to sort the cards, but are informed by the computer whether each sort is “right” or “wrong.” The child is required to first sort according to one sorting principle (e.g. color), and after 10 consecutive correct responses, the sorting principle changes, without the child being informed as such. This procedure continues until the child has successfully completed six sorting categories, or until all 128 cards have been placed (Strauss, Sherman, & Spreen, 2006). The WCST can be administered to individuals ages 6.5 to 89 and takes

approximately 15 minutes to complete. In child and adolescent samples, reliability generalizability coefficients, which are comparable to traditional reliability coefficients (Cronbach, Gleser, Nanda, & Rajaratnam, 1972), have ranged from .37 (percent perseverative errors) to .72 (nonperseverative errors) (Heaton et al., 1993).

The WCST test yields four primary score indices, including perservative and nonperseverative errors, number of categories completed, failure to maintain set, and the learning to learn index. The most common measures used to assess executive control on the WCST are the number of categories achieved and perseverative errors (Strauss et al., 2006). The number of perseverative errors is considered to be the best metric of executive function if a single score from the WCST is to be used (Rhodes, 2004). Perseverative errors represent the inability to relinquish an old category for a new one or an inability to see a new possibility (Heaton et al., 1993). For the present analyses, children's cognitive flexibility was represented by total number of perserverative errors, with higher values indicating lower flexibility.

(c) The Behavior Rating Inventory of Executive Function – Self-Report (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000) was used to assess impairment in several domains of executive functioning. Children (ages 5 to 18 years old) rate their own behavior frequency using a three-point Likert scale (0 to 2) on 75 items covering nine non-overlapping clinical scales. The clinical scales comprise two broader indices of Behavioral Regulation (Inhibit, Shift, Emotional Control) and Metacognition (Initiate, Working Memory, Plan/Organize, Organization of Materials, Self-Monitor, Task Monitor). The BRIEF has satisfactory internal consistency reliability and has been normed on appropriate census populations in the United States (Roth, Isquith, & Gioia, 2005). In the present analyses, the BRIEF Working Memory index was included in the Working Memory composite and the BRIEF Shift Index was included in the

Cognitive Flexibility composite. In this sample, the alpha for the BRIEF Working Memory Index was $\alpha \geq .83$ and for the BRIEF Shift Index was $\alpha \geq .84$.

We created separate composite indices for working memory and cognitive flexibility by reverse scoring the WCST and BRIEF such that higher scores indicated better EF abilities on all measures, converting raw scores to standardized scores (z-scores) and combining the behavioral and self-report measures for each domain. The WISC-IV Digit Span and BRIEF Working Memory Index correlated significantly ($r = .16, p = .03$), and the WCST Perseverative Errors and BRIEF Shift Total Score also correlated significantly ($r = .15, p = .03$). The internal consistency for the working memory composite was $\alpha \geq .82$ and for the cognitive flexibility composite was $\alpha \geq .81$.

Coping. The revised peer stress version of the Responses to Stress Questionnaire (RSQ; Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000) was used to assess children's responses to stress related to peer interactions. The peer stress version of the RSQ includes 12 questions about common social stressors for children and adolescents (e.g. fighting with other kids; not having as many friends as you want). The measure then includes 57 items describing ways in which a child might respond to stressful peer interactions; children are asked to rate each item using a Likert scale (1 = not at all; 2 = a little; 3 = some; 4 = a lot) regarding how much they respond to peer stress in the manner described. The two RSQ subscales studied here were: primary control engagement coping (e.g., problem-solving, emotional expression, emotional modulation) and secondary control engagement coping (e.g., cognitive restructuring, acceptance, distraction, positive thinking). The RSQ uses proportional scoring, which takes into account the total number of items endorsed when reporting the factor statistics (e.g., Connor-Smith et al., 2000). Internal consistency reliabilities at both time points were $\alpha \geq .81$ for primary control coping and $\alpha \geq .80$ for secondary control coping.

Depressive Symptoms. The Children's Depression Inventory (CDI; Kovacs, 1992) was used to measure children's report of symptoms of depression (excluding the item about suicidal ideation). The 26 items list three statements in order of severity. Internal consistency, test-retest reliability, and convergent validity have been found to be adequate for the CDI (Kovacs, 1992). Internal consistency for the current sample at both times points was $\alpha \geq .86$.

Intelligence Quotient (IQ). Given that executive functioning has been found to be associated with intelligence in children (Friedman et al., 2006), we obtained an estimated IQ score to use as a control variable. The Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) is a widely-used, individual brief intelligence test for children. The short form of the WASI contains one subtest from the Verbal Comprehension Index (i.e., Vocabulary subtest) and one subtest from the Perceptual Reasoning Index (i.e., Matrix Reasoning subtest). WASI scores have been shown to correlate about .93 with the Full Scale IQ (Wechsler, 1999). The Vocabulary subtest measures word knowledge and verbal comprehension; the Matrix Reasoning subtest taps nonverbal reasoning and visual problem-solving ability. These two subtests are combined to provide an estimate of children's overall IQ. In the present study, a minimum estimated Full Scale IQ (FSIQ) of 70 was required to ensure adequate comprehension of instructions for questionnaires and behavioral tasks. One participant was excluded from all data analyses due to her having an IQ score below 70.

Procedure

Children participated in two sessions, one in person baseline (T1) session lasting 3 hours, and a briefer (30 minutes) online or phone follow-up session approximately 4 months post-baseline (T2). The average duration between the baseline and follow-up was 4.35 months ($SD = .69$). At the first session, the CDI, BRIEF, and RSQ, computerized and examiner-administered executive function tasks (WCST and Digit Span, respectively), and the WASI were

administered. Self-report questionnaires, computerized tasks, and the IQ testing were completed in a random order. To limit fatigue, children were encouraged to take breaks as needed. A total of 66 group testing sessions took place with 1 to 5 children at a time ($M = 2.9$) and were conducted either on Vanderbilt's campus, or at a local school. All participants were given \$20 for the first session. At the follow-up, children completed the CDI and RSQ, again and were given \$10. The follow-up assessment allowed us to examine the extent to which the baseline executive functions predicted changes in coping and depressive symptoms over this time.

Data Analytic Plan

First, we examined the means and standard deviations among all study variables. We next conducted separate regression analyses with each composite index of executive function (i.e., working memory and cognitive flexibility) as the independent variable and the two coping subscales as the dependent variables, controlling for age and estimated IQ. Results of these simple linear regressions are presented in Supplemental Tables 1-3 (see Appendix). Finally, to examine direct and indirect effects of EF, coping, and symptoms of depression we used SPSS macros designed by Preacher and Hayes (2008), which incorporate several regression equations to obtain path coefficients of each relation.

Direct Effects. Following the procedures of Preacher and Hayes (2008), four separate models were tested to examine the direct effects among each T1 executive function composite index, each T2 coping subscale, and T2 depressive symptoms. Models controlled for T1 levels of coping and depressive symptoms as well as concurrent correlations among variables to reduce potential bias and to provide a more stringent test of longitudinal total, direct, and indirect effects (Cole & Maxwell, 2003). To determine the direct effects, we examined path coefficients in each model (a , b , c are path coefficients). All coefficients reported here are unstandardized, unless otherwise noted, and $\alpha = .05$ two-tailed was the criterion for statistical significance.

Indirect Effects. We then tested the extent to which coping accounted for the relation between each EF variable (i.e., working memory and cognitive flexibility) and depressive symptoms. In the current study, coping and depressive symptoms were assessed at two time points. Although an ideal design would involve collecting all measures at three time points, the current “half-longitudinal design” was superior to a purely cross-sectional approach (Cole & Maxwell, 2003). According to Cole and Maxwell, the strategy for testing a mediation model using a “half-longitudinal” design with only two time points involves the assumption of stationarity; that is, “an unchanging causal structure” (Kenny, 1979, p. 232). Stationarity assumes that the influence of one variable on change in another variable is stable over time. That is, the effect of T1 coping on T2 depressive symptoms would be equivalent to the effect of T2 coping on T3 depressive symptoms under the stationarity assumption. The mediation analyses conducted here assumed such stationarity.

A bias-corrected bootstrap procedure was used to generate confidence intervals for the total indirect effect of T1 executive functioning on T2 depressive symptoms through each T2 coping subscale separately. In the bootstrap procedure, the original data set is used to create a large number of randomly drawn additional data sets of the same size. Over multiple bootstrap re-samples, an empirical approximation of the sample distribution can be generated and used for hypothesis testing. A bootstrap test is preferable to the traditional Sobel test, because the Sobel assumes that the estimate of the indirect effect follows a normal distribution, which often is not the case, and can lead to low power and high type I error rates (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). In the present analyses, each bootstrapping model used 5000 iterations and 95% confidence intervals (CI) to determine the specific indirect effects of each coping variable. Mediation was significant if the 95% bias corrected confidence intervals for the indirect effect did not include zero (Preacher & Hayes, 2004).

Results

Descriptive and Preliminary Analyses

Table 1 presents descriptive statistics including means, standard deviations, and minimum and maximum scores for the measures of executive functions, coping, and depressive symptoms. Mean scores on the EF tasks (WISC-IV Digit Span and WCST Perserverative Errors) were within the average range and similar to those observed in normative samples (Kirkwood, Hargrave, & Kirk, 2011; Klimes-Dougan et al., 2006). Scores for the RSQ Primary and Secondary Control Coping Scales represent the proportion of the total score for each coping domain. In the present study, the proportion of secondary control coping ($M = .26, SD = .05$) was significantly higher than primary control coping ($M = .19, SD = .04$) [$t(189) = -15.48, p < .001$].

Age was negatively associated with estimated FSIQ ($r = -.36, p < .001$) and positively associated with primary control coping ($r = .14, p < .05$). Therefore, age was included as a covariate in all analyses. At baseline, females reported a significantly lower proportion of secondary control coping strategies ($M = .25, SD = .05$) than males [$M = .27, SD = .05; t(188) = 2.42, p < .05$]. Inclusion of sex, however, did not alter the results of the prospective analyses, and therefore sex was not included as a covariate.

The sample included 16 children who reported having been diagnosed with Attention Deficit Hyperactivity Disorder (ADHD). Of these children, 12 reported that they were currently taking medication to treat ADHD. Children with a diagnosis of ADHD obtained significantly lower working memory composite scores, $t(190) = 3.04, p < .05$, and higher baseline depressive symptoms, $t(190) = -1.94, p = .054$. Results did not differ, however, when these 16 children were included versus excluded, and therefore they were retained in the analytic sample.

Relations between Executive Functions and Coping.

Correlations among study variables are presented in Table 2. As hypothesized, executive functioning correlated significantly and positively with coping at both baseline and follow-up. The working memory composite correlated significantly with primary control coping at T1 ($r = .25; p < .01$) and T2 ($r = .38; p < .01$), and with secondary control coping at T1 ($r = .23; p < .01$) and T2 ($r = .26; p < .01$). Similarly, the cognitive flexibility composite correlated significantly with primary control coping at T1 ($r = .16; p < .05$) and T2 ($r = .24; p < .01$), and with secondary control coping at T1 ($r = .21; p < .01$) and T2 ($r = .30; p < .01$). Thus, a positive association was found between executive functioning and coping.

Relation between Executive Functions and Depressive Symptoms

Consistent with hypothesis 2, executive functions were associated negatively with depressive symptoms both at Time 1 and at follow-up (see Table 2). Specifically, significant correlations were found between working memory composite scores and depressive symptoms at T1 ($r = -.42; p < .01$) and T2 ($r = -.37; p < .01$). Significant correlations also were found between cognitive flexibility composite scores and depressive symptoms at T1 ($r = -.40; p < .01$) and T2 ($r = -.33; p < .01$). Thus, better executive functioning was linked with lower levels of depressive symptoms.

Relations between Coping and Depressive Symptoms

As predicted (hypothesis 3), both primary and secondary control coping correlated negatively with depressive symptoms (see Table 2). Significant correlations were found between T1 primary control coping and depressive symptoms at T1 ($r = -.22; p < .05$) although not at T2 ($r = -.12; p = .12$). Time 2 primary control coping significantly correlated with depressive symptoms at T1 ($r = -.42; p < .01$) and T2 ($r = -.47; p < .01$). Similarly, Time 1 secondary control coping significantly correlated with depressive symptoms at T1 ($r = -.47; p < .01$) and T2 ($r = -.35; p < .01$), and T2 secondary control coping significantly correlated with depressive

symptoms at T1 ($r = -.34; p < .01$) and T2 ($r = -.44; p < .01$). Thus, less use of primary or secondary control coping was associated with higher levels of depressive symptoms at baseline and follow-up.

Direct and Indirect Effects among Executive Functions, Coping, and Depressive Symptoms

Mediation models were tested following the procedures of Preacher and Hayes (2008) to examine the direct effects between each study variable and to test the extent to which coping (primary; secondary) accounted for the relation between executive function (i.e., working memory; cognitive flexibility) and depressive symptoms at follow-up (see Tables 3-6).

Working Memory. We examined the relation of T1 working memory to T2 depressive symptoms through coping. We tested the model (hypothesis 4) that included T1 working memory, T2 primary control coping, and T2 depressive symptoms (see Figure 1). Examining the individual path coefficients in the model including working memory and primary control coping, the total effect of working memory on T2 depressive symptoms was significant, $c = -.553, t(159) = -1.977, p = .049$, such that each 1-point increase in working memory predicted approximately a 0.5-point decrease in T2 depressive symptoms. The analysis of working memory predicting the hypothesized mediator, T2 primary control coping, yielded a nonsignificant trend; $a = .004, t(159) = 1.918, p = .056$. Next, the b path from T2 primary control coping to T2 depressive symptoms was significant, $b = -41.232, t(158) = -4.011, p < .001$. Finally, when T2 primary control coping was included in the model, the estimated direct effect of working memory on T2 depressive symptoms was no longer significant, $c' = -.39, t(158) = -1.422, p = .151$. The overall model predicting T2 depressive symptoms was significant, with adjusted $R^2 = .552$ and $F(6, 158) = 34.665, p < .001$.

Bootstrap analyses based on 5000 resamples were used to test the indirect effect of working memory on T2 depressive symptoms through T2 primary control coping. Confidence

intervals of the indirect effects of working memory on depressive symptoms (95% CI: -.39 to -.024) did not include zero. A significant indirect effect between working memory at T1 and depressive symptoms at T2 was found indicating that the relation between working memory and depressive symptoms was partially mediated by primary control coping. The point estimate of this indirect effect, ab , was -.163 (see Table 7).

We next conducted an exploratory analysis of a model that included T1 working memory, T2 secondary control coping, and T2 depressive symptoms (see Figure 2). Examining the individual path coefficients in the model including working memory and secondary control coping, the total effect of working memory on T2 depressive symptoms showed a nonsignificant trend, $c = -.541$, $t(159) = -1.948$, $p = .053$ such that each 1-point increase in working memory predicted approximately a 0.5-point decrease in T2 depressive symptoms. The path between working memory and the hypothesized mediator, T2 secondary control coping was not significant; $a = .004$, $t(159) = 1.526$, $p = .129$. The b path from T2 secondary control coping to T2 depressive symptoms was significant, $b = -30.474$, $t(158) = -3.773$, $p < .001$. When T2 secondary control coping was included in the model, the estimated direct effect of working memory on T2 depressive symptoms decreased and was not significant, $c' = -.419$, $t(158) = -1.559$, $p = .121$. The overall model significantly predicted T2 depressive symptoms, with an adjusted $R^2 = .548$ and $F(6, 158) = 34.127$, $p < .001$.

Bootstrap analyses based on 5000 resamples were used to test the indirect effect of working memory on T2 depressive symptoms through T2 secondary control coping. Confidence intervals of the indirect effects of working memory on depressive symptoms (95% CI: -.346 to .015) included zero, thus indicating that there was not a significant indirect effects of working memory on depressive symptoms through secondary control coping (see Table 7).

Cognitive Flexibility. Next, we tested a model in which cognitive flexibility assessed at Time 1 predicted depressive symptoms at T2, with T2 secondary control coping as a mediator (hypothesis 5). Examining the individual path coefficients in this model, the total effect of cognitive flexibility on T2 depressive symptoms was not significant, $c = -.434, t(158) = -1.649, p = .10$. Cognitive flexibility significantly predicted the hypothesized mediator of T2 secondary control coping; $a = .006, t(158) = 2.30, p = .02$. Additionally, T2 secondary control coping significantly predicted T2 depressive symptoms, $b = -30.266, t(157) = -3.67, p < .001$. When T2 secondary control coping was included in the model, the estimated direct effect of cognitive flexibility on T2 depressive symptoms was not significant; $c' = -.264, t(157) = -1.026, p = .31$. The overall model significantly predicted T2 depressive symptoms, with an adjusted $R^2 = .542$ and $F(6, 157) = 33.096, p < .001$.

The bootstrap bias-corrected and accelerated confidence interval (95% CI: $-.418$ to $-.030$) for the indirect effect, ab , of T1 cognitive flexibility on T2 depressive symptoms through T2 secondary control coping did not include zero. A significant indirect effect between cognitive flexibility at T1 and depressive symptoms at T2 was found indicating greater cognitive flexibility predicted higher levels of secondary control coping, which in turn predicted lower levels of depressive symptoms (see Figure 3). The point estimate of this indirect effect, ab , was $-.17$. Thus, these results were consistent with hypothesis 5.

Finally, we conducted an exploratory analysis to test the model that included cognitive flexibility as a predictor of T2 depressive symptoms with T2 primary control coping as a mediator (Figure 4). Examining the individual path coefficients in this model, the total effect of cognitive flexibility on T2 depressive symptoms was not significant, $c = -.439, t(158) = -1.67, p = .097$. Cognitive flexibility did not significantly predict the hypothesized mediating variable, T2 primary control coping; $a = .001, t(158) = .366, p = .71$. However, T2 primary control coping

significantly predicted T2 depressive symptoms, $b = -43.529$, $t(157) = -4.28$, $p < .001$. When T2 primary control coping was included in the model, the estimated direct effect of cognitive flexibility on T2 depressive symptoms was not significant; $c' = -.408$, $t(157) = -1.631$, $p = .11$. The overall model significantly predicted T2 depressive symptoms, with an adjusted $R^2 = .553$ and $F(6, 157) = 34.668$, $p < .001$.

The bootstrap bias-corrected confidence interval (95% CI: $-.225$ to $.138$) for the indirect effect, ab , of T1 cognitive flexibility on T2 depressive symptoms through T2 primary control coping included zero. Thus, there was not a significant indirect effect of cognitive flexibility on depressive symptoms through primary control coping (see Table 8).

Direct and Indirect Effects of Executive Functions on Coping through Depressive Symptoms

To explore alternative models in which the direction of the relation between coping and depression was reversed, we tested whether T2 depressive symptoms mediated the relation between executive functions and changes in coping. We conducted separate analyses for the two executive function components and the two coping domains. Exploratory bootstrap analyses revealed neither significant indirect effects of T1 working memory on T2 secondary control coping through T2 depressive symptoms, nor any indirect effects of cognitive flexibility on either T2 coping strategies through T2 depressive symptoms (see Tables 9-10). Thus, no evidence emerged of an indirect effect of executive functions on T2 coping through T2 depressive symptoms, except for the model that included working memory and primary control coping. Inspection of individual path coefficients in this model indicated that, similar to the model of working memory to depression through primary coping, the total effect of working memory on T2 coping yielded a nonsignificant trend, $c = .004$, $t(159) = 1.918$, $p = .056$; working memory significantly predicted the hypothesized mediator, T2 depressive symptoms; $a = -.553$, $t(159) = -1.977$, $p = .049$, and T2 depressive symptoms significantly predicted T2 primary

control coping, $b = -.002$, $t(158) = -4.011$, $p < .001$. When T2 depressive symptoms were entered into the model, the estimated direct effect of working memory on T2 primary control coping was not significant; $c' = .003$, $t(158) = 1.36$, $p = .18$. The overall model of working memory predicting T2 primary control coping through depressive symptoms was significant, with an adjusted $R^2 = .354$ and $F(6, 158) = 16.006$, $p < .001$. The bootstrap bias-corrected and accelerated confidence interval for the indirect effect, ab , of working memory on T2 primary control coping through T2 depressive symptoms was .0001 to .0031 (i.e., it did not contain zero). The point estimate of the indirect effect, ab , was .0012.

Discussion

The present study investigated the concurrent and prospective relations among executive functioning, coping, and depressive symptoms in children. We examined the associations between each executive function domain (i.e., working memory, cognitive flexibility) and each coping strategy (i.e., primary, secondary), between the EF domains and depressive symptoms, and between primary and secondary control coping and depressive symptoms, both at the initial assessment and at the four-month follow-up. Analyses predicting Time 2 coping or Time 2 depressive symptoms controlled for the Time 1 level of the respective variable. We then tested mediation models of the extent to which coping accounted for the relations between EF and depressive symptoms. Finally, to explore the direction of the observed relations, we tested another set of models in which the mediator was depressive symptoms and the outcome was coping.

Relation of Executive Function to Coping

The correlational evidence supported Hypotheses 1 that better executive functioning (i.e., working memory and cognitive flexibility) would be significantly associated with greater use of primary control coping strategies (e.g., problem-solving, emotional modulation) and secondary

control coping strategies (e.g., cognitive restructuring, acceptance). Specifically, both executive function composite scores were significantly positively correlated with primary and secondary control coping at baseline and follow-up. These cross-sectional associations are consistent with findings of a previous study of EF and coping in youth with cancer (Campbell et al., 2009), although different from a study of children with functional abdominal pain (FAP) (Hocking et al., 2011). The small sample size in the study by Hocking et al. may partially explain the different results. It also is possible that EF abilities are less central to successful coping with the particular chronic health condition of FAP.

Analysis of the direct effects in the working memory models, revealed that the association between working memory and primary control coping was marginal. Examination of the full model, however, indicated a significant indirect relation between working memory and depressive symptoms through the mediator of primary control coping. In mediation models, an indirect effect may be detectably different from zero even though one of its constituent paths is not (Hayes, 2009). That is, although the direct pathway between working memory and primary control coping yielded a nonsignificant trend, the significant indirect effect in the overall model indicated that working memory was relevant to the use of primary control coping strategies. Working memory abilities may be important for implementing primary control coping strategies such as developing a plan, implementing multi-step solutions, and carrying-out steps to actively modify events or conditions. Experimental studies that systematically manipulate working memory are needed to more precisely specify the strength and nature of its relation to primary control coping strategies in children and adolescents.

Working memory and secondary control coping also showed significant bivariate correlations, which is similar to the significant but small to medium correlations found in previous studies (Campbell et al., 2009; Hocking et al., 2011). Results of the analyses of the

direct effects in the working memory models revealed that the relation between working memory and secondary control coping was not significant. This finding differs from previous investigations (Andreotti et al., 2013; Campbell et al., 2009), which may be partially due to differences between child and adult samples, or to the inclusion of FSIQ as a covariate in our analyses. Campbell and colleagues, however, also found no significant association between working memory and secondary control coping in their healthy control subsample. Investigations with larger samples that include both at-risk clinical and normal controls may provide greater variability and more power to detect these associations.

Consistent with previous studies (e.g., Campbell et al., 2009), we found a significant direct effect between cognitive flexibility and secondary control coping. Controlling for baseline coping, age, and estimated FSIQ, better cognitive flexibility significantly predicted secondary control coping at follow-up. This result provides support for the suggestion of Compas (2006) that cognitive flexibility facilitates the use of secondary control coping strategies involving shifting thoughts and behaviors (i.e. cognitive restructuring, distraction, acceptance) and fluidly adapting to stressful life events. Children who lack flexibility may become trapped in repetitive cycles of well-learned behaviors (perseveration) and be less able to recruit new strategies for coping effectively with novel situations. In turn, such children who have difficulty engaging in complex cognitive and regulatory processes may be particularly vulnerable to the deleterious effects of stress.

Finally, there was a small significant bivariate correlation between cognitive flexibility and primary control coping, but the direct effect was not significant when controlling for baseline coping, age, and estimated FSIQ. Few previous investigations have examined the association between cognitive flexibility and primary control coping. This result suggests that cognitive flexibility may not be central to the ability to enact primary coping strategies.

Thus, the present longitudinal study revealed several important concurrent and prospective links between executive functioning and coping. These findings expand upon previous evidence that executive functions are associated with multiple skills that characterize primary control coping such as problem-solving and deductive reasoning (Fletcher, Marks, & Hine, 2011; Handley, Capon, Beveridge, Dennis, & Evans, 2004; Kail, 2007; Lundqvist, 1995) as well as secondary control coping skills such as cognitive restructuring (Andreotti et al., 2011). An important direction for future research is to determine whether improving children's executive functioning increases the likelihood of their using more adaptive strategies for coping with stress.

Relation between Executive Function and Depressive Symptoms

We next found evidence in both the bivariate correlations and regression analyses in line with hypothesis 2 that deficits in executive functioning abilities would be associated with higher levels of depressive symptoms at baseline and follow-up. In particular, even when controlling for T1 depressive symptoms, age, and estimated full scale IQ, difficulties in working memory significantly predicted increases in depressive symptoms at Time 2. With regard to cognitive flexibility, the bivariate correlations indicated significant associations with depressive symptoms at both baseline and follow-up, but this relation was not significant in the analyses controlling for T1 depressive symptoms, age, and estimated full scale IQ predicting depression at Time 2. Whereas some studies have shown a link between EF and depressive symptoms in youth (e.g. Kyte, Goodyer, Sahakian, 2005; Micco et al., 2009) others have not (e.g., Favre et al., 2009; Korhonen et al., 2002; Maalouf et al., 2011). In particular, some studies have reported deficits in working memory, but intact cognitive flexibility, among children and adolescents with depression (Brooks, Iverson, Sherman, & Roberge, 2010; Matthews et al., 2008; Micco et al.,

2009). Thus, improving working memory may be a particularly important target for interventions aimed at preventing or treating depression in youth.

Does Coping Mediate the Relation of Executive Functions to Depressive Symptoms?

We constructed mediation models to examine the extent to which these executive function abilities were *indirectly* related to subsequent depressive symptoms through coping. We examined each path in the model and conducted bootstrap analyses to obtain a confidence interval for the indirect effect. As predicted, analyses of the *indirect* effects revealed that primary control coping strategies mediated the relation between working memory and depressive symptoms (hypothesis 4), and secondary control coping mediated the relation between cognitive flexibility and depressive symptoms (hypothesis 5). These results provide further evidence to support the theoretical model of Compas (2006) that coping is closely tied to higher order executive functions.

In the present study, working memory predicted engaging in active strategies to change a stressor or one's emotional responses to a stressor, which in turn predicted depressive symptoms. Additionally, the ability to think flexibly and shift cognitive set was related to children's reported use of secondary coping strategies such as cognitive restructuring and acceptance, which in turn was related to children's levels of depressive symptoms. Thus, these results further demonstrated that coping may be one salient pathway through which deficits in executive functions contribute to children's symptoms of depression.

Exploratory analyses of the mediation models revealed some unique relations. That is, the association between working memory and depressive symptoms was mediated through primary control but not secondary control coping. The relation between cognitive flexibility and depressive symptoms was mediated through secondary but not primary control coping. These results may indicate some specificity in the links among EF, coping, and depressive symptoms. It

also is possible, however, that a larger sample would have yielded significant findings for variables that had a weaker, yet still significant relation (e.g., working memory and secondary control coping).

Direction of the Relations among EF, Coping, and Depressive Symptoms

Finally, to address the direction of the observed relations, we tested exploratory alternative models examining depressive symptoms as a possible mediator of the relation between EF and coping. One previous longitudinal study found that coping predicted symptoms over time, but symptoms did not predict coping (Wadsworth & Berger, 2006). The current study found one significant model from working memory to primary coping through depressive symptoms. Other data analytic methods such as dynamic latent change score (LCS) modeling across multiple time points should be used in future studies to evaluate possible bidirectional relations among these variables over time (McArdle & Hamagami, 2001).

The other tests of the indirect effect of executive functions on T2 coping through T2 depressive symptoms were not significant. Of course, these null results do not rule out the possibility that depressive symptoms mediate the prospective path from EF to coping. The “half-longitudinal” design (Cole & Maxwell, 2003) used in the present study included two concurrently collected measures (T2 coping and T2 depressive symptoms) in the mediation models, and required the assumption of stationarity. Future studies that include three or more time points are needed to eliminate potential bias due to violations of stationarity and to allow for direct examination of longitudinal mediation effects.

Strengths, Limitations, and Future Directions

The current investigation addressed several methodological problems of previous studies by utilizing a longitudinal design, a moderate size sample, well-validated measures of executive functions and coping, and controlling for intellectual abilities, demographic variables, and prior

levels of the dependent variables. Each EF domain was assessed using multiple methods -- a behavioral task and a self-report measure -- which comprised the EF composite scores. In addition, whereas some prior studies had not controlled for IQ (Campbell et al., 2009; Hocking et al., 2011), we controlled for WISC-IV FSIQ in all analyses. That is, because intellectual abilities are related but separable from executive functions (Friedman et al., 2006), we examined the unique contribution of EF, over and above IQ, to coping and depressive symptoms. Finally, the present study also advances beyond previous cross-sectional findings by employing a “half-longitudinal design” (Cole & Maxwell, 2003) that controlled for T1 levels of coping and depressive symptoms as well as concurrent correlations among variables to reduce potential bias and to provide a more stringent test of longitudinal total, direct, and indirect effects among EF, coping, and depression.

Limitations of this study also should be noted as they provide directions for future research. First, we assessed executive function skills using both behavioral measures and child report. Although composite scores are likely better than using only one method of measurement, additional behavioral tasks might allow for even more precise identification of executive functioning (Chase-Carmichael, Ris, Weber, & Schefft, 1999). Moreover, we used only children’s self-report of their coping and psychopathology. Children may not be accurate reporters of behaviors, particularly those that are in the executive function domain. Using multiple informants, particularly parents’ reports of children’s executive functions and psychopathology, would decrease the common method variance and the possibly inflated correlations that may occur when only one informant is used.

The current study examined links between two domains of executive function – working memory and cognitive flexibility – and coping. We have suggested that these two cognitive abilities provide a foundation for utilizing effective coping strategies. Other executive functions

(e.g. inhibition, and attention) and cognitive abilities (e.g., scientific reasoning, metacognition, and processing speed), as well as social and emotional abilities (e.g., emotion understanding and expression) also may be related to coping and should be the focus of future investigations.

Finally, another limitation of the current study was the relatively short duration between the two assessments. The four-month follow-up period may not have been long enough for significant changes in coping or depressive symptoms to occur. Future studies should include more than two waves of data collection over a longer time period to increase the chances of observing change in the variables of interest, and to allow for the use of state-of-the-art mediation analyses (Cole & Maxwell, 2003) to test the relations among EF, coping, and depressive symptoms in children.

In conclusion, the present study demonstrated prospective associations among cognitive flexibility, coping, and depressive symptoms in children. Mediation models revealed that deficits in working memory predicted subsequent symptoms of depression, in part, through primary control coping, and cognitive flexibility predicted depressive symptoms through secondary control coping, over and above the contribution of prior symptom levels. Thus, working memory and cognitive flexibility may be promising targets for interventions aimed at improving children's ability to utilize regulatory strategies that predict greater well-being in the context of social stress. Specifically, working memory may be central to the use of primary strategies, and cognitive flexibility may be particularly linked to the ability to enact secondary coping strategies.

Recently, evidence has been accumulating indicating that executive functions are malleable and can be modified through intervention (Diamond & Lee, 2011; Zelazo & Carlson, 2012). Although current studies of children with ADHD indicate that the effects of these interventions may not consistently generalize to other contexts (Dunning, Holmes, & Gathercole, 2013; Melby-Lervag & Hulme, 2013), few studies have examined the efficacy of such

interventions in depressed children (e.g. Riggs, Greenberg, Kusche, & Pentz, 2006). An important next step would be to investigate whether interventions that manipulate executive functions result in improvements in coping strategies and reductions in symptoms of depression.

The current study sets the stage for exploration of links between the developmental trajectories of coping and executive functions, particularly in the context of specific types of stressors. The developing brain undergoes periods of great plasticity, which increases both vulnerability to the effects of stress (Andersen & Teicher, 2008; Davidson, Jackson, & Kalin, 2000) and potential for response to intervention. Children with delayed or aberrant EF may have coping skills that fail to reach full capacity. Identifying when developmental shifts in coping occur and how they differ for children with and without executive function deficits could help researchers and clinicians target youth who are at greatest risk for negative outcomes.

Table 1

Mean and Standard Deviations of Scores for Primary Variables at Time 1

	Mean (<i>SD</i>)	Minimum	Maximum
WASI Full Scale IQ	111 (13.41)	75	145
Children's Depression Inventory Total Score	6.61 (5.73)	0	32
Working Memory Composite	0.00 (1.52)	-3.42	3.34
WISC-IV Digit Span Total Score	16.9 (3.8)	8	27
BRIEF Working Memory Index Score	19.01 (4.47)	12	30
Cognitive Flexibility Composite	0.07 (1.36)	-7.01	2.38
WCST Perseverative Errors Total Score	13.27 (9.48)	4	62
BRIEF Shift Index Score	15.54 (3.81)	10	26
RSQ Primary Control Coping	0.19 (0.04)	0.1	0.28
RSQ Secondary Control Coping	0.26 (0.05)	0.08	0.43

Note. WASI = Wechsler Abbreviated Scale of Intelligence; WISC-IV = Wechsler Intelligence Scale for Children - 4th edition; BRIEF = Behavior Rating Inventory of Executive Function; WCST = Wisconsin Card Sorting Test; RSQ = Responses to Stress Questionnaire; RSQ uses proportion scores reflecting the percentage of the total score on that measure that fell in each type of coping. Raw scores are reported for the BRIEF and WISC-IV Digit Span. Higher scores on the BRIEF indicate poorer performance; higher scores on EF composite indices indicate better performance. EF composites were computed by reverse scoring the WCST and BRIEF, converting all raw scores to z-scores, and summing the z-scores for the corresponding EF behavioral task and self-report.

Table 2

Means, Standard Deviations and Bivariate Correlations among Study Variables

	<i>M</i>	<i>SD</i>	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Age	12.36	1.77	--								
2. Full Scale IQ	111	13.41	-.36**	--							
3. T1 Working Memory Composite	.001	1.52	.18*	.29**	--						
4. T1 BRIEF WM Index	19.01	4.47	.02	.15*	.76**	--					
5. T1 WISC-IV Digit Span Total	16.90	3.80	.25**	.29**	.76**	.16*	--				
6. T1 Cognitive Flexibility Composite	.07	1.36	.10	.25**	.46**	.56**	.14	--			
7. T1 WCST Perseverative Errors	13.77	10.59	-.16*	-.21**	-.16*	-.10	-.15*	-.76**	--		
8. T1 BRIEF Total Shift Index	15.54	3.81	-.02	.16*	.53**	.74**	.07	.76**	-.15*	--	
9. T1 Children's Depression Inventory	6.61	5.73	.11	-.30**	-.42**	-.49**	-.15*	-.40**	.12	-.49**	--
10. T1 Primary Control Coping	.19	.04	.14*	.19*	.25**	.25**	.13	.16*	-.06	.18*	-.22**
11. T1 Secondary Control Coping	.26	.05	.00	.11	.23**	.23**	.12	.21**	-.04	.28**	-.47**
12. T2 Children's Depression Inventory	6.70	6.61	.09	-.20**	-.37**	-.43**	-.13	-.33**	.14	-.39**	.69**
13. T2 Primary Control Coping	.19	.04	.10	.24**	.38**	.33**	.25**	.24**	-.11	.28**	-.42**
14. T2 Secondary Control Coping	.27	.05	.09	.07	.26**	.32**	.09	.30**	-.13	.34**	-.36**

Table 2 (continued)

	10.	11.	12.	13.	14.
1. Age					
2. Full Scale IQ					
3. T1 Working Memory Composite					
4. T1 BRIEF WM Index					
5. T1 WISC-IV Digit Span Total					
6. T1 Flexibility Composite					
7. T1 WCST Perseverative Errors					
8. T1 BRIEF Total Shift Index					
9. T1 Children's Depression Inventory					
10. T1 Primary Control Coping	--				
11. T1 Secondary Control Coping	-.02	--			
12. T2 Children's Depression Inventory	-.12	-.35**	--		
13. T2 Primary Control Coping	.42**	.25**	-.47**	--	
14. T2 Secondary Control Coping	.20*	.53**	-.44**	.28**	--

* $p < .05$; ** $p < .01$

Note. T1 = Time 1; T2 = Time 2; SD = Standard Deviation; CF = Cognitive Flexibility; WM = Working Memory; WCST = Wisconsin Card Sorting Test. BRIEF = Behavior Rating Inventory of Executive Function; mean proportion scores and SD are reported for the RSQ; raw scores are reported for the BRIEF and WISC-IV Digit Span. Higher scores on the BRIEF indicate poorer performance; higher scores on the EF composite indices indicate better performance.

Table 3

Direct Paths for Working Memory, Primary Control Coping, and Depressive Symptoms Model

Direct Effects				
(Between Stress and Depressive Symptoms)				
	<i>b</i>	SE	<i>t</i>	p-value
T1 Working Memory → T2 Primary Control Coping	.004	.002	1.92	.056
T1 Working Memory → T2 Depressive Symptoms	-.55	.28	-1.98	.049
T2 Primary Control Coping → T2 Depressive Symptoms	-41.23	10.28	-4.01	.0001

Table 4.

Direct Paths for Working Memory, Secondary Control Coping, and Depressive Symptoms Model

Direct Effects				
(Between Stress and Depressive Symptoms)				
	<i>b</i>	SE	<i>t</i>	p-value
T1 Working Memory → T2 Secondary Control Coping	.004	.003	1.52	.129
T1 Working Memory → T2 Depressive Symptoms	-.54	.28	-1.95	.053
T2 Secondary Control Coping → T2 Depressive Symptoms	-30.47	8.08	-3.77	.0002

Table 5

Direct Paths for Cognitive Flexibility, Primary Control Coping, and Depressive Symptoms Model

Direct Effects				
(Between Stress and Depressive Symptoms)				
	<i>b</i>	SE	<i>t</i>	p-value
T1 Cognitive Flexibility → T2 Primary Control Coping	.001	.002	.37	.715
T1 Cognitive Flexibility → T2 Depressive Symptoms	-.44	.26	-1.67	.098
T2 Primary Control Coping → T2 Depressive Symptoms	-43.53	10.17	-4.28	<.0001

Table 6.

Direct Paths for Cognitive Flexibility, Secondary Control Coping, and Depressive Symptoms Model

Direct Effects				
(Between Stress and Depressive Symptoms)				
	<i>b</i>	SE	<i>t</i>	p-value
T1 Cognitive Flexibility → T2 Secondary Control Coping	.006	.002	2.30	.023
T1 Cognitive Flexibility → T2 Depressive Symptoms	-.43	.26	-1.65	.101
T2 Secondary Control Coping → T2 Depressive Symptoms	-30.27	8.25	-3.67	.0003

Table 7

Indirect Effects of Working Memory on Time 2 Depressive Symptoms through Time 2 Coping

	Point Estimate	Bootstrapping ^a	
		Bias Corrected and Accelerated 95% CI	
		Lower	Upper
Indirect effects of WM through Primary Control Coping	-.1631	-.3899	-.0235
Indirect effects of WM through Secondary Control Coping	-.1219	-.3290	.0187

Note. WM = Working Memory; CI = Confidence Interval; Covariates included in each model: Age, IQ, Time 1 Coping, Time 1 Depressive Symptoms

^a 5000 Bootstrap Samples.

Table 8

Indirect Effects of Cognitive Flexibility on Time 2 Depressive Symptoms through Time 2 Coping

	Point Estimate	Bootstrapping ^a	
		Bias Corrected and Accelerated 95% CI	
		Lower	Upper
Indirect effects of CF through Primary Control Coping	-.0312	-.2171	.1393
Indirect effects of CF through Secondary Control Coping	-.17	-.4183	-.0304

Note. CF = Cognitive Flexibility; CI = Confidence Interval; Covariates included in each model: Age, IQ, Time 1 Coping, Time 1 Depressive Symptoms

^a 5000 Bootstrap Samples.

Table 9

Indirect Effects of Working Memory on Time 2 Coping through Time 2 Depression

	Point Estimate	Bootstrapping ^a	
		Bias Corrected and Accelerated 95% CI	
		Lower	Upper
Indirect effects of WM on Primary Control Coping through Depression	.0012	.000	.0031
Indirect effects of WM on Secondary Control Coping through Depression	.0015	-.0001	.0039

Note. WM = Working Memory; CI = Confidence Interval; Covariates included in each model: Age, IQ, Time 1 Coping, Time 1 Depressive Symptoms

^a 5000 Bootstrap Samples.

Table 10

Indirect Effects of Cognitive Flexibility on Time 2 Coping through Time 2 Depression

	Point Estimate	Bootstrapping ^a	
		Bias Corrected and Accelerated 95% CI	
		Lower	Upper
Indirect effects of CF on Primary Control Coping through Depression	.001	-.002	.0028
Indirect effects of CF on Secondary Control Coping through Depression	.0011	-.0002	.0034

Note. CF = Cognitive Flexibility; CI = Confidence Interval; Covariates included in each model: Age, IQ, Time 1 Coping, Time 1 Depressive Symptoms

^a 5000 Bootstrap Samples

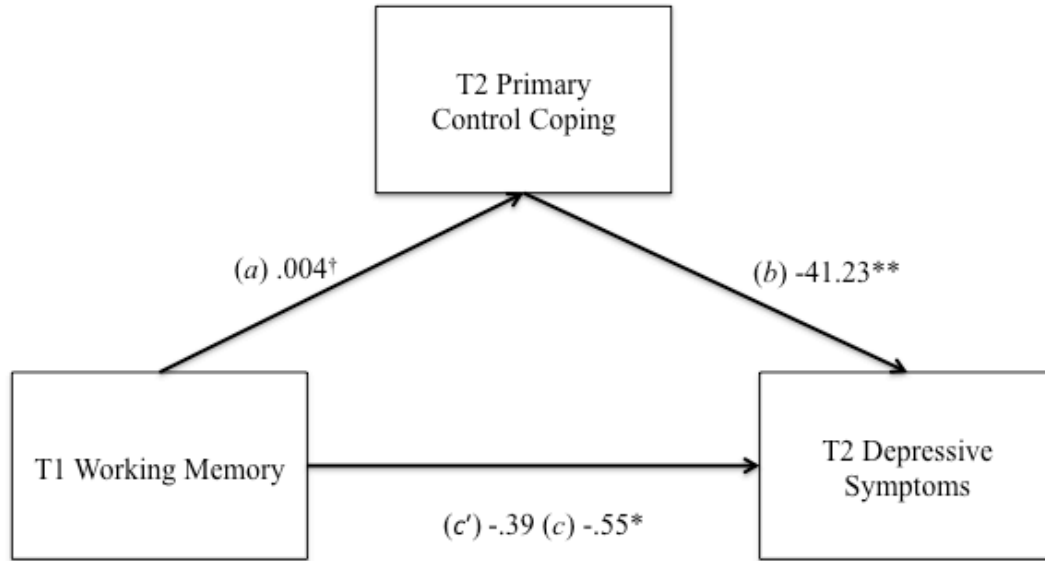


Figure 1. Indirect Effects of Working Memory on Depressive Symptoms through Primary Control Coping

Notes: † $p < .10$, * $p < .05$, ** $p < .01$

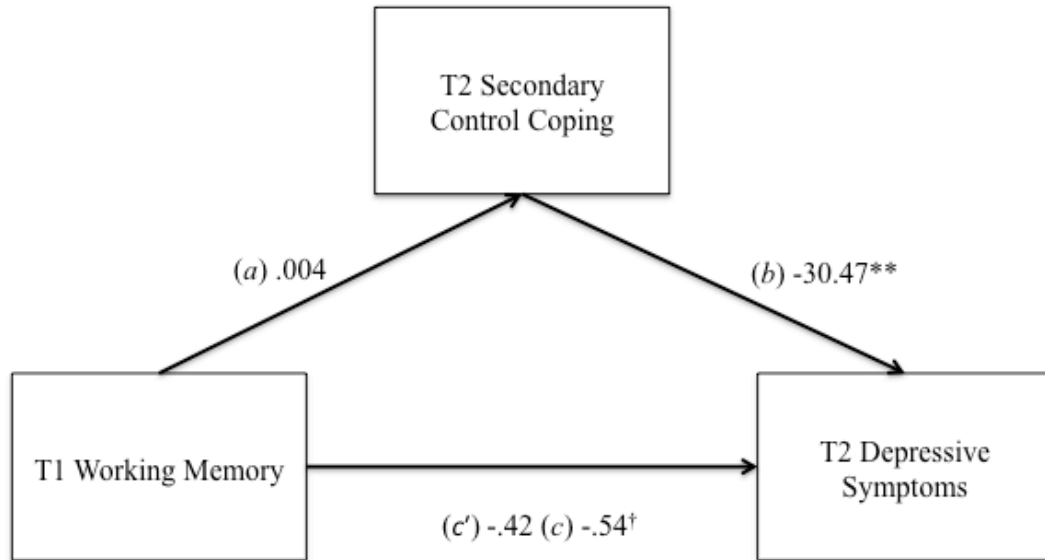


Figure 2. Indirect Effects of Working Memory on Depressive Symptoms through Secondary Control Coping

Notes: † $p < .10$, * $p < .05$, ** $p < .01$

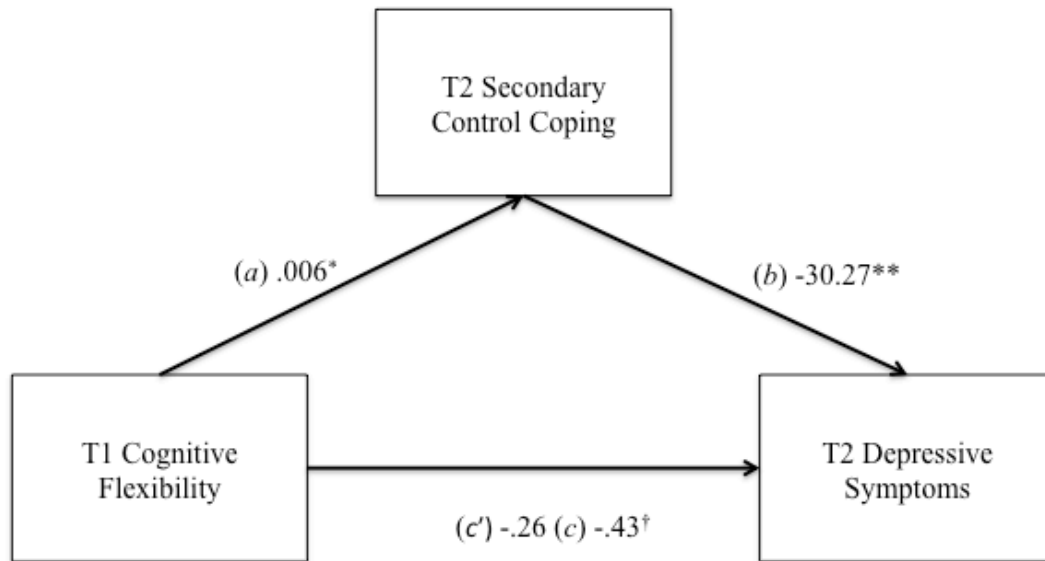


Figure 3. Indirect Effects of Cognitive Flexibility on Depressive Symptoms through Secondary Control Coping

Notes: † $p < .10$, * $p < .05$, ** $p < .01$

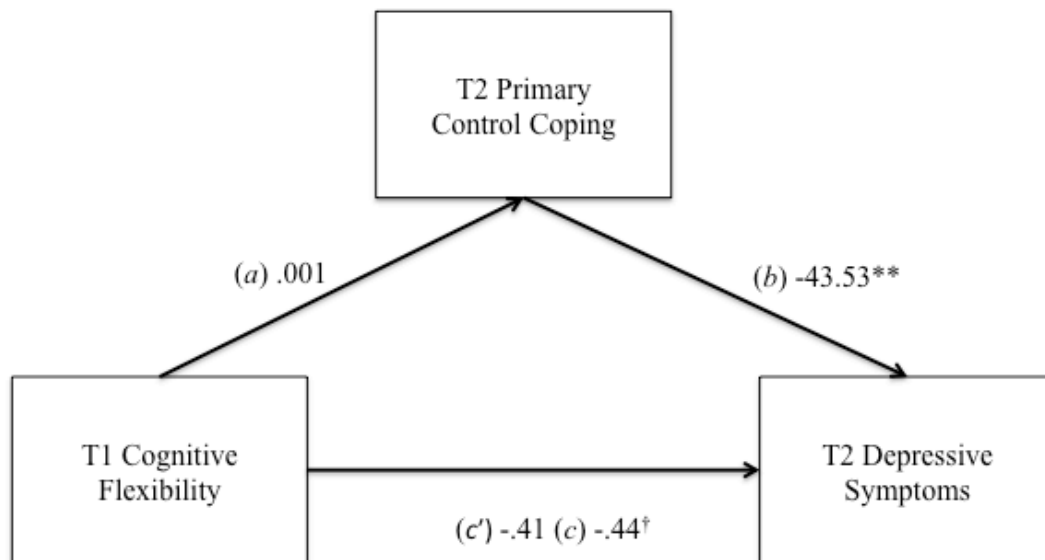


Figure 4. Indirect Effects of Cognitive Flexibility on Depressive Symptoms through Primary Control Coping

Notes: † $p < .10$, * $p < .05$, ** $p < .01$

Appendix

Supplemental Table 1

Summary of Simple Regression Analyses for Working Memory Predicting Coping Strategies at Follow-Up

Variable	T2 Primary Control Coping					T2 Secondary Control Coping					
	<i>B</i>	<i>SE B</i>	β	<i>t</i>	p^b	<i>B</i>	<i>SE B</i>	β	<i>t</i>	p^b	
Age	.001	.002	.06	.77	.441	.002	.002	.07	.90	.368	
Full Scale IQ	.001	.00	.12	1.45	.149	<.001	.00	-.001	-.01	.990	
T1 Coping	.34	.08	.32	4.43***	<.001	.51	.07	.50	7.51***	<.001	
WM Composite	.007	.002	.25	3.27**	.001	.005	.002	.15	2.08	.039	
	<i>R</i> ²					.26					.31
	<i>F</i>					14.24***					18.22***
	Cohen's <i>f</i> ²					.35					.45

* $p < .05$; ** $p < .01$; *** $p < .01$

Note. WM = Working Memory; IQ = Intelligence Quotient; T1 = Time 1; T2 = Time 2;

Supplemental Table 2

Summary of Simple Regression Analyses for Cognitive Flexibility Predicting Coping Strategies at Follow-Up

Variable	T2 Primary Control Coping					T2 Secondary Control Coping				
	<i>B</i>	<i>SE B</i>	β	<i>t</i>	<i>p</i> ^b	<i>B</i>	<i>SE B</i>	β	<i>t</i>	<i>p</i> ^b
Age	.003	.002	.11	1.42	.16	.002	.002	.07	.98	.33
Full Scale IQ	<.001	.00	.16	1.94	.054	<.001	.00	-.01	-.17	.863
T1 Coping	.37	.08	.35	4.71***	<.001	.50	.07	.50	7.46***	<.001
CF Composite	.003	.002	.12	1.67	.097	.007	.002	.20	2.81*	.006
	<i>R</i> ²									
						.23				
	<i>F</i>									
						11.66***				
	Cohen's <i>f</i> ²									
						.30				
						.33				
						19.48***				
						.49				

p* < .05; *p* < .01; ****p* < .01

Note. CF = Cognitive Flexibility, IQ = Intelligence Quotient; T1 = Time 1; T2 = Time 2; Coping was assessed with the Responses to Stress Questionnaire (RSQ)

Supplemental Table 3

Summary of Simple Regression Analysis for Working Memory Predicting Depressive Symptoms at Follow-Up

Variable	T2 Depressive Symptoms				
	<i>B</i>	<i>SE B</i>	β	<i>t</i>	p^b
Age	.29	.23	.08	1.28	.203
FSIQ	.03	.03	.06	.96	.341
T1 Depressive Symptoms (CDI)	.76	.07	.64	10.51***	<.001
Working Memory Composite	-.63	.28	-.15	-2.27	.025
	R^2				.49
	F				40.92***
	Cohen's f^2				.97

* $p < .05$; ** $p < .01$; *** $p < .01$

Note. T1 = Time 1; T2 = Time 2; Depressive symptoms were assessed with the Children's Depression Inventory (CDI); FSIQ = estimated Full Scale Intelligence Quotient

Supplemental Table 4

Summary of Simple Regression Analysis for Cognitive Flexibility Predicting Depressive Symptoms at Follow-Up

Variable	T2 Depressive Symptoms				
	<i>B</i>	<i>SE B</i>	β	<i>t</i>	p^b
Age	.20	.22	.05	.89	.38
FSIQ	.02	.03	.04	.61	.54
T1 Depressive Symptoms (CDI)	.78	.07	.66	10.63***	<.001
Cognitive Flexibility Composite	-.40	.27	-.09	-1.48	.14
	R^2				.48
	F				38.97***
	Cohen's f^2				.92

* $p < .05$; ** $p < .01$; *** $p < .01$

Note. IQ = Intelligence Quotient; T1 = Time 1; T2 = Time 2; CDI = Children's Depression Inventory (CDI); FSIQ = estimated Full Scale Intelligence Quotient

Footnote

^b Critical p -values using a Bonferroni correction were .0083 for $p < .05$; .00167 for $p < .01$; .00016 for $p < .001$

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