TRAIT RUMINATION, DEPRESSION, AND EXECUTIVE FUNCTIONS
IN EARLY ADOLESCENCE

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ABSTRACT

Trait Rumination, Depression, and Executive Functions in Early Adolescence

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This study examined the association between executive functions (EF), trait rumination, and symptoms and diagnosis of unipolar depression in early adolescence. In addition, the current study examined associations between executive functions and risk for depression due to maternal history of depression. Participants were 486 early adolescents (230 males, 256 females; M age = 12.88 years; SD = .62) and their mothers, who were recruited through local schools as part of the Temple University (TU) Adolescent Cognition and Emotion (ACE) Project. Measures included (a) a semi-structured diagnostic interview of mother and adolescent, (b) youth self-report forms assessing depressive symptoms and trait rumination, (c) mother-report forms assessing demographic information and offspring pubertal status, and (d) behavioral tests of EF (sustained, selective and divided attention, attentional set shifting, and working memory). Deficits in executive function were not associated with higher levels of trait rumination. Better sustained attention was marginally significantly positively associated with higher levels of trait rumination after controlling for concurrent depressive symptoms.

Conversely, poorer sustained attention was significantly associated with depressive symptoms after controlling for rumination. Gender moderated the set shifting-rumination association, such that better set shifting accuracy was significantly associated with higher levels of trait rumination in boys only. Diagnosis did not moderate EF-rumination associations. No association was found between EF deficits and adolescent lifetime history of unipolar depression. Likewise, no association was found between EF deficits and maternal lifetime history of unipolar depression. In conclusion, findings do not provide evidence that EF deficits are associated with trait rumination in early adolescence, either alone or in interaction with diagnosis or gender. Rather, findings suggest dissociable patterns of association between EF and depression versus rumination and are more consistent with theories postulating that trait rumination is associated with enhanced performance on some tests of EF. In addition, findings do not provide evidence for an association between EF deficits and lifetime history of depression in early adolescence and do not suggest that deficits are present in adolescents at high risk (due to maternal history of disorder) of depression prior to first onset of disorder. Implications and directions for future research are discussed.

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CHAPTER 1 INTRODUCTION

Major Depressive Disorder is a commonly occurring disorder, with 12-month prevalence rates estimated at 6.7% in adults (Kessler, Chiu, Demler, & Waters, 2005) and 8.2% in adolescents (Kessler et al., 2012). The disorder imposes a high economic burden (Berto, D'Ilario, Ruffo, DiVirgilio, & Rizzo, 2000). Approximately 20% of adolescents are likely to experience a depressive episode by age 18 (Hankin et al., 1998). This is of concern, as depression in youth has been associated with concurrent impairment in functioning across a broad range of domains, as well as subsequent negative outcomes, including a high rate of relapse (Kovacs, Obrosky, Gatsonis, & Richards, 1997; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Lewinsohn, Rohde, Klein, & Seeley, 1999), suicide attempts, substance abuse, and impaired psychosocial functioning in adulthood (Kovacs, Obrosky, Gatsonis, & Richards, 1997; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2003; Weissman et al., 1999). Given the high prevalence rates and negative impact of unipolar depression, a clearer understanding is needed of the expression, causes, and consequences of the disorder.

Cognitive theories of depression have postulated that cognitive variables confer vulnerability for onset and more severe course of the disorder. Such theories have posited a number of different cognitive vulnerability factors and have garnered support in adult as well as child and adolescent samples (for review, see Abela & Hankin, 2008). Cognitive theories central to the understanding of unipolar depression are primarily

concerned with “the relationship between human mental activity and the experience of depressive symptoms and episodes” (Abela & Hankin, 2008; Ingram, Miranda, & Segal, 1998). Given that an understanding of mental processes is integral to the understanding of unipolar depression, an integration of a cognitive theoretical approach to depression with the study of its neuropsychological correlates appears warranted. Despite the fact that impaired concentration is part of the phenotypic expression of the disorder (Goodyer & Cooper, 1993), and despite neuropsychological studies documenting an association between deficits in executive functions and unipolar depression (Rogers et al., 2004), relatively few studies have integrated cognitive vulnerability-stress models of the disorder with the use of emotionally neutral measures of executive functions. Research examining associations between deficits in executive function and unipolar depression is reviewed below.

Executive Functions and Unipolar Depression

Executive functions are conceptualized as separable but interrelated skills (Asato, Sweeny, & Luna, 2006; Miyake et al., 2000) necessary to purposeful, goal-directed activity and behavior (Anderson, 1998; Welsh, Pennington, & Groisser, 1991). Although theories of executive control vary (e.g., Anderson, 2002; Duncan, 2001; Miller & Cohen, 2001; Shallice, 1988), and executive functions have not been operationalized consistently, they are typically thought to involve higher level cognitive processes modulating or exerting control over lower level ones according to task demands so that behavior is not always driven solely by immediate environmental triggers (Alvarez &

Emory, 2006; Gilbert & Burgess, 2008). They thus permit flexible behavior under novel environmental conditions or when multiple responses are possible. Subsumed under the umbrella term of executive functions are higher level cognitive processes, such as set shifting and maintenance, interference control, inhibition, integration across space and time, planning, and working memory (Pennington & Ozonoff, 1996). Three separate relatively circumscribed skills within this framework, thought to be implicated in performance on more complex classic tests of executive function include (1) shifting between tasks or mental sets (the ability to change attentional focus in a controlled fashion), (2), updating and monitoring of working memory representations (the ability to retain and manipulate information online and to delete and replace information that is no longer relevant with new information), and (3) inhibition (the ability to suppress prepotent responses (Miyake et al., 2000). Factor analytic approaches to neuropsychological task performance in adults support the conceptualization of executive functions as comprised of separable but related processes (Miyake et al., 2000).

Several theoretical approaches and programs of research highlight the potential importance of an understanding the extent to which executive function deficits characterize unipolar depression. Neurobiological models of depression have emphasized altered bottom-up processing of emotion accompanied by impaired effortful regulatory control associated with hypoactivation of regions implicated in cognitive control (including regions of the prefrontal cortex and anterior cingulate cortex; Disner, Beevers, Haigh, & Beck, 2011; Liotti et. al., 2000; Marchetti, Koster, Sonuga-Barke, & De Raedt,

2012; Mayberg, 1997, 2003; Phillips, Drevets, Rauch, & Lane, 2003a, 2003b). From a cognitive perspective, the resource allocation model (Ellis & Ashbrook, 1988) has postulated that depression occupies limited capacity cognitive resources, impairing ability to engage in effortful cognitive processes. Others have proposed that depressed individuals can perform at the level of non-depressed individuals in structured situations but have difficulty doing so of their own initiative in situations requiring cognitive control (Gotlib & Joormann, 2010), in particular in situations where attention is not constrained by task demands (Hartlage, Alloy, Vazquez, & Dykman, 1993; Hertel & Rude, 1991). Thus, it has been suggested that deficits in cognitive functioning documented in individuals with unipolar depression reflect deficits in attentional control and the ability to inhibit task-irrelevant thoughts (Gotlib & Joormann, 2010).

Executive Functions and Unipolar Depression in Adulthood

Neuropsychological Profiles in Acute Phase of Disorder

A vast array of studies has examined aspects of cognitive functioning including executive functions in adults with unipolar depression. These are characterized by a number of methodological inconsistencies rendering interpretation difficult, including wide age ranges, use of psychiatric inpatient samples, failure to differentiate between presence and absence of psychosis and mania, lack of clarity regarding diagnostic criteria employed, and failure to control for Type I error. Several reviews of the adult literature have concluded that in adulthood, unipolar depression is characterized by specific deficits in attention and executive control (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari,

& Lonnqvist, 2008; Rogers et al., 2004). Although much of the adult literature has employed samples with wide age ranges (into the 70s), studies focusing exclusively on young adult samples with unipolar depression have obtained evidence for deficits on tests of cognitive set-shifting on the Trail Making Test (TMT) and Wisconsin Card Sorting Test (WCST; Grant, Thase, & Sweeney, 2001; Mahurin et al., 2006; Merriam, Thase, Haas, Keshavan, & Sweeney, 1999; Smith, Muir, & Blackwood, 2006; Stordal et al., 2004), selective attention on the Stroop (Egeland et al., 2003), verbal fluency (Fossati, Amar, Raoux, Ergis, & Allilaire, 1999; Stordal et al., 2004), and impaired performance on tests of working memory (Fossati et al., 1999; Stordal et al., 2004).

Attentional deficits also have been found on tests of visuomotor processing speed and attention (Hill, Keshavan, Thase, & Sweeney, 2004; Smith et al., 2006), and sustained attention (Egeland et al., 2003). More recently, a meta-analysis (Wagner, Doering, Helmreich, & Lieb, 2011) of neuropsychological deficits in adult unipolar depression, examining verbal fluency, inhibition (on the Stroop) and cognitive flexibility (on the TMTB and WCST) obtained evidence for significantly worse performance in patients with MDD compared with healthy controls on tests of verbal fluency, WCST performance, and TMT part B, leading the authors to conclude that adults with unipolar depression are characterized by deficits in inhibition, verbal fluency, set shifting and cognitive flexibility. A recent, methodologically strong meta-analysis obtained evidence for deficits on a range of executive functions, including inhibition, set-shifting, verbal

fluency, working memory, and planning in those with major depressive disorder relative to healthy controls (effect sizes ranging from .45-.58; Snyder, 2013).

This neuropsychological research has been complemented by electrophysiological and neuroimaging studies, which often yield evidence for abnormal activation in neural networks subserving cognitive control and involving lateral PFC and ACC despite comparable levels of task performance. Studies yielding such findings have employed tests of working memory updating on the n-back task (Harvey et al., 2005; Matsuo et al., 2007; Rose, Simonotto, & Ebmeier, 2006; Walter, Wolf, Spitzer, & Vasic, 2007), inhibition on the Stroop (Holmes & Pizzagalli, 2008; Krompinger & Simons, 2011; Wagner et al., 2006), and attentional set shifting (Heinzel, Northoff, Boeker, Boesiger, & Grimm, 2010) and have led to the suggestion that depressed individuals must recruit additional neural resources to achieve levels of performance comparable to that of controls (Krompinger & Simons, 2011; Wagner et al., 2006).

Differentiation: State-Dependent Deficits versus Trait Markers

There is ongoing debate as to whether deficits in executive control precede first onset of unipolar depression and are trait markers that are etiologically linked to the disorder, and as to whether they persist following remission, or dissipate when individuals are no longer in the acute phase of a depressive episode (Hasselbalch, Knorr, & Kessing, 2011). In adults with a lifetime history of unipolar depression currently in remission, deficits have been found in executive functions, attention, memory, and psychomotor speed (for review, see Austin, Mitchell, & Goodwin, 2001; Douglas &

Porter, 2009; Hammar & Ardal, 2009; Kessing, 1998), and there has been some suggestion that deficits in attention and executive function are trait markers of the disorder that persist following remission (Boeker et al., 2012; Douglas & Porter, 2009; Kessing, 1998; Yamamoto & Shimada, 2012). Based on a qualitative review of 11 methodologically strong studies comparing remitted individuals with MDD and controls, Hasselbalch and colleagues (2011) noted deficits in sustained and selective attention, memory, and executive functions but concluded there is insufficient evidence regarding patterns of deficits to date without further studies employing hypothesis-driven selection of neuropsychological tests and controlling for potential confounds, including age, education, and subclinical symptoms of psychopathology.

A recent meta-analysis (Bora, Harrison, Yucel, & Pantelis, 2012) of studies employing remitted samples (those in remission for 2 months and who exhibited concurrent depressive symptoms below clinical cutoffs) obtained evidence for impairments among those with depression relative to healthy controls, with particularly robust findings for Stroop interference control ($d = .74$). Those with depression showed impairments on measures of Stroop interference control, set shifting (TMA A and B), semantic fluency, working memory (digit span backwards), and verbal learning and recall ($ds = .39-.74$), but did not differ from healthy controls on measures of verbal fluency, WCST, digit span forwards, or verbal recognition. Different patterns of findings were obtained for early versus late onset depression, such that only modest impairments were found for early onset depression with the exception of Stroop inhibitory control ($d = .82$),

whereas those with late onset depression exhibited a wider range of cognitive deficits of greater magnitude, particularly with regard to processing speed and verbal memory.

Studies of individuals at high risk for depression who have not yet developed the disorder can likewise help to clarify whether observed deficits in executive control in the acute phase of disorder are trait markers that are etiologically linked to the disorder or are state dependent. Studies examining aspects of executive control in high risk adults who have not yet developed depression have obtained evidence for poorer performance among high risk twins (e.g., unaffected adult monozygotic twins with a co-twin with unipolar depression) on tests of psychomotor speed, attention and set shifting (TMT part B and A), selective attention and inhibitory control (Stroop), and working and declarative memory and language processing (Christensen, Kyvik, & Kessing, 2006).

Executive Functions and Depression in Childhood or Adolescence

Compared with the plethora of studies examining neuropsychological profiles of adults with unipolar depression, far fewer studies have examined those of children or adolescents. Those that have done so have yielded evidence that depression in adolescence is characterized by deficits in behavioral performance on tests of executive control (Baune, Czira, Smith, Mitchell, & Sinnamon, 2012; Cataldo, Nobile, Loruso, Battaglia, & Moltem, 2005; Gunther, Konrad, De Brito, Herpertz-Dahlmann, & Vloet, 2011; Han et al., 2012; Klimkeit, Tonge, Bradshaw, Melvin, & Gould, 2011; Maalouf et al., 2011; Matthews, Coghill, & Rhodes, 2008; Micco et al., 2009; Wilkinson & Goodyer, 2006). At the same time, some studies have obtained null findings (Gunther, Holtkamp,

Jolles, Herpertz-Dahlmann, & Konrad, 2004) or only marginally significant or qualitative patterns of behavioral differences (Brooks, Iverson, Sherman, & Roberge, 2010; Favre et al., 2009; Frost, Moffitt, & McGee, 1989; Halari et al., 2009). The preponderance of these studies has employed cross-sectional designs and small treatment-seeking samples of children and adolescents. These studies have yielded evidence of deficits on tests of sustained attention (Cataldo et al., 2005; Han et al., 2012; Maalouf et al., 2011; Wilkinson & Goodyer, 2006), and attentional set shifting (Gunther et al., 2011; Wilkinson & Goodyer, 2006). In addition, depressed youth have demonstrated worse performance on tests of working memory (Baune et al., 2012; Klimkeit et al., 2011; Matthews et al., 2008), prepotent response inhibition (Gunther et al., 2011), verbal fluency (Cataldo et al., 2005; Klimkeit et al., 2011), and planning (Maalouf et al., 2011), more perseverative errors on the WCST (Micco et al. 2009), and more total (but not perseverative) errors and fewer categories completed on a similar card sorting task (Baune et al., 2012). Memory-related deficits that may reflect problems with attention and executive control, including difficulties with metamemory (Lauer et al., 1994), rehearsal of items in short-term memory (Osborn & Meador, 1990), and verbal recall (Gunther et al., 2004) also have been documented, as have marginally significant deficits on tests of selective attention (Cataldo et al., 2005; Wilkinson & Goodyer, 2006).

At the same time, null findings have been obtained, often within the same empirical studies that obtained positive findings. Studies have failed to identify diagnostic group differences on tests of alertness and sustained and divided attention

(Gunther et al., 2004, 2011; Micco et al., 2009), auditory attention and impulsivity (Cataldo et al., 2005), selective attention and inhibitory control on the Stroop (Favre et al., 2009; Micco et al., 2009), attentional set shifting (Frost et al., 1989; Klimkeit et al., 2011; Kyte, Goodyer, & Sahakian, 2005; Matthews et al., 2008), motor response inhibition (Gunther et al., 2004; Klimkeit et al., 2011), WCST performance (Favre et al., 2009; Frost et al., 1989), inhibition of irrelevant information/interference control (Klimkeit et al., 2011; van der Meere, Borger, Pirila, & Sallee, 2011), verbal fluency (Favre et al., 2009; Frost et al., 1989), and planning (Baune et al., 2012; Matthews et al., 2008). In some cases, marginally significant findings and qualitative patterns of deficits that have failed to reach statistical significance have been observed on tests of complex attention, including set shifting, motor response inhibition, selective attention/inhibition, and interference control (Brooks et al., 2010; Favre et al., 2009; Halari et al., 2009; Han et al., 2012). The only study to date employing fMRI in a sample of depressed children or adolescents obtained evidence for attenuated activation of fronto-cingulate and fronto-striatal pathways during engagement on tests of attentional set shifting, response inhibition, and selective attention and interference control (Halari et al., 2009).

Inconsistent findings may be explained in part by methodological limitations that have characterized many of these studies. Specifically, studies have been characterized by small sample sizes comprised of treatment seeking patients rather than community samples. Studies in unselected community samples are nonexistent, with one exception, which was limited in that it contained a very small sample size (only 10 early adolescents

with depression; Frost et al., 1989). Studies also have used very wide age ranges with samples comprising children from early childhood through late adolescence, have failed to take into account clinical correlates such as severity of depression, and have not employed sufficiently rigorous methods of diagnostic assessment. Additional studies examining profiles of executive function in youth with unipolar depression are needed that correct for these methodological issues.

In addition, the above studies examine samples of children in the acute phase of disorder, and thus may conflate state-dependent effects of depressed mood (for which they often fail to control) with trait profiles of EF that may persist into remission or be etiologically linked to the disorder. Only two studies have examined profiles of deficits in remitted samples of youth with a lifetime history of depression. One such study obtained no evidence for deficits on tests of planning or sustained attention (Maalouf et al., 2011), whereas the other obtained a pattern of behavioral responses and ERP data possibly indicative of cognitive inflexibility or difficulty reallocating attentional resources in response to a rare tone during an auditory oddball task (Houston, Bauer, & Hesselbrock, 2004). Both of these studies lacked clearly specified time frames for remission. Additional research is needed examining profiles of executive function in those with childhood onset depression, taking into account concurrent depressive symptoms and status of remission.

Several studies have examined neuropsychological profiles of children and adolescents who have not yet experienced a depressive episode but who are at-risk for

depression, due to parental history of unipolar depression. The rationale for studies of EF profiles in those at high risk of depression has been to clarify whether documented deficits are state-dependent or trait-dependent deficits and whether they arise as a result of disorder, or precede and possibly contribute to disorder onset (Micco et al., 2009). Micco and colleagues (2009) compared a sample of offspring of parents with a history of MDD and/or panic disorder to those with parents without a history of mood or anxiety disorders or psychiatric treatment. Surprisingly, controlling for child diagnostic status, parental diagnosis of depression was associated with *fewer* offspring perseverative errors on the Wisconsin Card Sorting Test, but did not predict performance on measures of verbal learning or selective or sustained attention.

In a sample of adolescent offspring of mothers with MDD, bipolar disorder, and parents without psychopathology, Klimes-Dougan, Ronsaville, Wiggs, and Martinez (2006) found that children of mothers with MDD did not differ from children of parents with no history of psychopathology on measures of set shifting, sustained attention, verbal learning, visuospatial planning, or IQ, although high risk children performed marginally significantly worse ($p = .06$) than controls on the measure of failure to maintain set on the WCST. Finally, in a longitudinal study, Hughes, Roman, Hart, and Ensor (2013) examined maternal self-reported depressive symptoms, assessed at four timepoints over four years, and child executive functions (a latent factor based on performance on measures of working memory, inhibitory control, and planning) at age 6 and reported that lower average levels of maternal depressive symptoms at age 2

significantly predicted better EF in offspring at age 6 and that greater rapidity in reduction in maternal depressive symptoms over time was marginally associated with higher offspring EF scores at age 6. Other research in samples of high risk youth has obtained no evidence of behavioral performance differences on a verbal n-back task, but fMRI imaging results suggesting greater allocation of processing resources to obtain comparable levels of performance (Mannie, Harmer, Cowen, & Norbury, 2010), or has obtained evidence of attentional difficulties in high risk children relative to controls only under conditions of affective challenge but not under emotionally neutral task conditions (Perez-Edgar, Fox, Cohn, & Kovacs, 2006).

Thus preliminary high risk studies and studies of youth in remission suggest that many of the cognitive deficits observed in the acute phase of depression are not present prior to episode onset and may normalize following remission. However, findings to date do not rule out the possibility that some specific subprocesses may be trait markers for the disorder. Positive findings to date in high risk samples of youth include abnormal neural activation during engagement on a verbal n-back task in high risk adolescents (Mannie et al., 2010), marginally significant impairment on a behavioral task requiring set shifting in high risk children and adolescents (Klimes-Dougan et al., 2006), and abnormal allocation of attentional resources in remitted children and adolescents based on behavioral and ERP data during an auditory oddball task (Houston et al., 2004). However, studies of high risk youth that employ a large sample size and a range of

measures of executive functions and that control statistically for concurrent symptoms are needed to further elucidate the specific types of deficits that may be present.

Rumination and Executive Functions

Response Styles Theory: Defining Rumination

As noted previously, there have been relatively few attempts to integrate cognitive vulnerability-stress models of depression with empirical findings suggesting unipolar depression may be characterized by impaired performance on emotionally neutral tasks requiring executive control. One promising avenue of research that does so is informed by the response styles theory of depression. According to the response styles theory (Nolen-Hoeksema, 1991), the way in which individuals respond to their depressed mood can influence the severity and duration of depression. According to this theory, rumination is “a mode of responding to distress that involves repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms” (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Rumination is defined as *a process of thinking perseveratively* about one’s feelings and problems rather than in terms of specific content of thoughts (Nolen-Hoeksema et al., 2008). Similarly, it has been defined as “a process whereby individuals *repetitively focus attention* on their depressive symptoms and the implications of their symptoms” (Hankin, 2008).

Rumination is trait-like, relatively stable across changes in level of depressive symptoms (Nolen-Hoeksema et al., 2008), and, although the content of rumination is

typically negatively valenced, it is the perseverative *style* (not the content) of thought that is conceptualized as central to rumination (Hankin, Lakdawalla, Carter, Abela, & Adams, 2007; Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008). Rumination has been conceptualized as an intrusive or uncontrollable “automatic response” (Silk, Steinberg, & Morris, 2003). Distraction is conceptualized as an adaptive alternative to rumination that may relieve depressive symptoms. Distracting responses are “thoughts and behaviors that help divert one’s attention away from one’s depressed mood and its consequences and turn it to pleasant or benign thoughts and activities that are absorbing, engaging, and capable of providing positive reinforcement” (Nolen-Hoeksema et al., 2008).

In samples of children and adolescents, ruminative tendencies have been associated with greater depressive symptoms (Abela, Brozina, & Haigh, 2002; Abela, Vanderbilt, & Rochon, 2004; Ziegert & Kistner, 2002) and found prospectively to predict increases in depressive symptoms over time (Abela et al., 2002; Roelofs et al., 2009). Distraction has been associated with lower levels of depressive symptoms in some studies (Abela, Vanderbilt, & Rochon, 2004; Ziegert & Kistner, 2002) but not others (Abela et al., 2002, 2004; Garber, Braafladt, & Weiss, 1995) and has not been found to prospectively predict change over time in depressive symptoms (Abela et al., 2002; Roelofs et al., 2009). Theoretical and empirical conceptualizations of rumination have indirectly implicated deficits in executive control, linking rumination with perseverative thinking and attentional inflexibility (Hankin et al., 2007; Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008) and suggesting that high levels of rumination may prove

most detrimental when paired with an inability voluntarily to reallocate attention through distraction (Abela, Aydin, & Auerbach, 2007).

Causal Association between Rumination and Executive Functions

Theoretical conceptualizations of the exact nature of the relation between rumination and aspects of executive function as well as the direction of causality have varied. There is evidence that dysphoric individuals exhibit specific deficits in task performance under conditions in which attention is not externally controlled and mind wandering is possible (Hertel, 1998; Hertel & Hardin, 1990; Hertel & Rude, 1991). In their recently formulated “impaired disengagement hypothesis,” Koster, De Lissnyder, Derakshan, & DeRaedt (2011) postulated that difficulty exercising attentional control (e.g., difficulty selectively attending to task relevant information and inhibiting task irrelevant information) in response to negative thoughts increases the likelihood of persisting in rumination. They propose that the normal process of emotion regulation may be disrupted either by (1) impaired conflict signaling whereby negative thoughts are consistent with negative self-schemas and do not elicit cognitive conflict that would signal reallocation of attentional resources or (2) impairment in attentional control. In addition, evidence from induction paradigms has led to the conceptualization of rumination as reducing executive capacity through depletion of cognitive resources (Watkins & Brown, 2002).

In contrast, Whitmer and Banich (2007) have proposed that underlying attentional inflexibility causes rumination. There is some evidence that deficits in task performance

are not solely attributable to concurrent rumination. For example, deficits in inhibition were observed only following induced rumination, but deficits in cognitive flexibility were observed more generally regardless of rumination induction (Phillippot & Brutoux, 2008). Altamirano, Miyake, and Whitmer (2010) found that trait rumination was associated with impaired performance in task switching, a test requiring flexibility in goals, but *enhanced* performance on a version of the Stroop requiring goal maintenance. Such differential patterns of findings indicate that a pattern of attentional inflexibility may contribute both to ruminative tendencies and to impairment on tests of executive control. In line with this possibility, Altamirano, Miyake, and Whitmer (2010) have posited a “trait inflexibility view” of rumination, whereby once certain thoughts are established in working memory, they are not easily removed, so that individuals have difficulty switching to more positive thoughts or other goals. Although this would frequently take the form of difficulty disengaging from negatively valenced information when ruminators are in a negative mood state, it would also (in the absence of negative mood) take the form of difficulty disengaging from emotionally neutral goals.

Both possibilities (rumination interfering with executive control or deficits in executive control contributing to ruminative tendencies) are also viable. An underlying deficit in executive control may contribute to the development of ruminative tendencies, and habitual rumination may deplete cognitive resources, make it more difficult for individuals to disengage from self-referent and depressogenic thinking, and impede effective engagement on tasks. Levens, Muhtadie, and Gotlib (2009) have suggested that

executive control impairment may interfere with effortful controlled allocation of cognitive resources in depression and likewise make individuals more likely to ruminate, but that conversely, rumination or negative schemata may add to overall cognitive load experienced at the onset of task performance, lowering the threshold for interference in task performance and reducing cognitive flexibility in depressed individuals relative to controls. They note that it is possible that a “dynamic interplay” occurs between executive processing and rumination such that an underlying impairment is exacerbated in the presence of rumination, further impairing task performance and increasing or perpetuating rumination. Despite varying conceptualizations of the causal direction of the association, all of these perspectives converge in positing that greater tendency to ruminate is likely to be associated with greater impairment in executive functions.

Rumination and Executive Functions in Adults

In addition to substantial evidence for an association between rumination and deficits in executive control over emotionally valenced information (for review, see Gotlib & Joormann, 2010), empirical research has documented an association between rumination and deficits in executive control on emotionally neutral tasks in nonclinical samples of adults (for additional recent review, see Whitmer & Gotlib, 2012a). Comparing those high and low in self-reported rumination, Davis and Nolen-Hoeksema (2000) obtained evidence for more perseverative errors and greater failure to maintain set on the WCST among ruminators, controlling for concurrent depressive symptoms. More recently, in a sample of university students, (Miranda, Valderrama, Tsypes, Gadol, &

Gallagher, 2013), brooding rumination mediated the prospective association between cognitive inflexibility on the WCST at baseline and increases in suicidal ideation over a two-year follow-up. Whitmer and Banich (2007) employed a modified task switching paradigm in a sample of undergraduates selected as high or low in self-reported rumination, and found that depressive rumination was associated with decreased ability to inhibit a previously relevant task set, controlling for set switching costs, suggesting rumination is associated with a deficit in ability to inhibit previously relevant task sets. In a subsequent experiment with an unselected sample of undergraduates, findings from the first experiment were replicated, and anger rumination and intellectual reflection were significantly associated with set switching costs but not with inhibition. Thus, attentional inflexibility (difficulties with set shifting) appears to be associated with trait rumination in adults, with specific deficits (set switching versus inhibition of previously relevant task sets) differentially associated with different types of rumination. A recent study examined whether reversal learning (the ability to learn from feedback of the need to reverse stimulus reward associations) might potentially drive perseverative tendencies in ruminators (Whitmer & Banich, 2012) by examining the association between reversal learning and adaptive and maladaptive repetitive thought (measured via averaged z-scores of scales assessing anger rumination, intellectual reflection, depressive rumination, and worry) in an undergraduate sample. Findings indicated that both maladaptive and adaptive repetitive thought were associated with significantly slower reversal learning, a finding which became marginally significant controlling for concurrent depressive symptoms and affect. Likewise, in a sample of unselected undergraduates rumination was

associated with inhibitory deficits in long-term memory (controlling for depressive symptoms and affect) as assessed via a retrieval-induced forgetting paradigm (Whitmer & Banich, 2010). Using a mixed antisaccade paradigm in a sample of undergraduates selected as high or low in self-reported depressive symptoms, De Lissnyder, Derakshan, DeRaedt, & Koster (2011) found that high ruminators exhibited deficits in inhibition (as evidenced by slower latencies on correct antisaccades) relative to low ruminators, controlling for depressive symptoms. Altamirano and colleagues (2010) reported that greater ruminative tendency was positively associated with *lower* accuracy in task switching (involving rapid switching between letter naming and cue interpretation) but *better* goal maintenance (e.g., greater accuracy on Stroop performance on incongruent trials). In a sample of university students selected as dysphoric or non-dysphoric based on BDI screening, Owens and Derakshan (2013) employed a spatial location task switching paradigm in which participants were required to switch between two tasks that required either congruent or incongruent responses and varied in terms of preparation time (e.g., cue target interval). Patterns of findings suggested impaired inhibition in high ruminators, such that high ruminators had difficulty filtering the currently irrelevant task set as evidenced by reduced accuracy on task performance in switch trials and tended to maintain a particular mode of responding regardless of relevance as evidenced by lower accuracy on incongruent response conditions. High ruminators likewise did not exhibit reductions in the reaction time congruency effect with increases in preparation time when the dominant (left/right) task was no-longer-relevant, a finding suggesting difficulty

filtering the dominant but no-longer-relevant task set. In contrast, evidence for impaired switching performance was not associated with dysphoria.

Factor analytic approaches have identified reflection and ruminative brooding as two separate components of rumination in adults. Reflection is a more adaptive voluntary or intentional turning inward to engage in problem solving, whereas ruminative brooding is a passive maladaptive practice of comparing one's situation with an unachieved standard (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Hertel, Benbow, and Geraerts (2012) employed a sample of university students selected as high or low on self-reported ruminative brooding. Participants were exposed to an orienting task consisting of a series of trials in which they were required to (1) view neutral target words and (2) then concentrate on phrases designed to focus on phrases about the self. In the unfocused condition of this task, participants were required to report the neutral target word they had just seen before concentrating on the subsequent phrase, whereas in the focused condition the target was reported after concentration on the subsequent phrase. Following this orienting task, participants were surprised by a free and forced recall test of neutral target words. Brooders exhibited more errors in subsequent recall than nonbrooders only in the unfocused condition, suggesting more difficulty remembering neutral information when it is presented in the context of self-focus but ONLY when attention is not constrained by task demands. Correlational analyses revealed a significant negative association between brooding and free recall performance in the unfocused task condition

only, after controlling for depressive symptoms, whereas depressive symptoms were not correlated with free recall performance in either task condition.

Associations also have been found between rumination and impaired executive control in clinical samples of adult patients with unipolar depression. In a sample of patients with MDD and OCD compared with demographically matched control groups, Meiran, Diamond, Toder, and Nemets (2011) employed a test of selective attention and inhibition (Stroop) and a working memory updating task, as well as a task switching paradigm. Controlling for diagnostic grouping variables, rumination was significantly associated with less preparation-related reductions in switching costs on the task switching paradigm and was marginally significantly associated with less preparation-related reductions in mixing costs, suggesting rumination is associated with lesser preparation towards a new task and lesser ability to translate advance task information into performance (i.e. to reduce switching and mixing cost by utilizing preparation). Rumination was also associated with poorer performance on the working memory updating task, but was not associated with Stroop performance.

In a study primarily examining performance on an affectively valenced interference control task during fMRI, Berman and colleagues (2011) administered an automated operation span task requiring participants to remember words while solving math problems. They noted a marginally significant association between operation span scores and ruminative tendencies, as well as significantly poorer operation span scores in individuals with unipolar depression relative to controls. Comparing young adults with

MDD to healthy controls, Levens and colleagues (2009) examined the association between rumination and performance on a dual-task paradigm (incorporating both a primary recency probes task and tracking task) designed to resemble a typical multitasking scenario and to capture ability to resolve competing task demands and reroute cognitive resources from one task to another. Rumination (in particular, brooding) was associated with greater cross-task rerouting interference among depressed participants only.

As noted previously, rumination-induction paradigms have suggested that inducing rumination in depressed individuals may deplete executive resources leading to perseverative errors on a random number generation task (Watkins & Brown, 2002) and impaired inhibition on the Stroop (Philippot & Brutoux, 2008) and exacerbating existing cognitive inflexibility (Philippot & Brutoux, 2008). However, induced rumination may be qualitatively different than preexisting trait-like ruminative tendencies and may thus have different correlates. Indeed, noting this possibility, Whitmer and Gotlib (2012b) employed a backward inhibition task to examine both noninhibitory task switching and backward inhibition in a sample of adults with MDD and healthy controls who both completed measures of trait rumination and underwent a state rumination and distraction induction paradigm. Induced state rumination was associated with impaired ability to switch between attentional sets among MDD participants only, but was not associated with inhibitory deficits. In contrast, trait rumination was associated with poorer inhibition (a finding that remained marginally significant after controlling for depressive symptoms

and diagnostic status) but was not associated with switching. Such findings led the authors to suggest that these two types of rumination (state and trait) are distinct and are associated with different cognitive deficits.

It should be noted that some preliminary research has examined the association between rumination and deficits in processing of neutral and emotionally-valenced information in a single experimental design. Some such studies have found that rumination is not associated with deficits in the processing of neutral information, but is associated only with deficits in executive control over emotional information (Bernblum & Mor, 2010; DeLissnyder, Koster, & De Raedt, 2012; DeLissnyder, Koster, Derakshan, & De Raedt, 2010; Joormann, Levens, & Gotlib, 2011; Joormann, Nee, Berman, Jonides, & Gotlib, 2010). Additionally, deficits in set shifting for emotionally valenced (but not neutral) information have been found to predict prospective increases in rumination (DeLissnyder, Koster, Goubert, Onraedt, Vanderhasselt, & De Raedt, 2012). Other research has found evidence for impairment in attentional control for self-referent information regardless of emotional valence (Daches, Mor, Winkvist, & Gilboa-Schechtman, 2010) and inhibitory deficits only for negatively valenced emotional information but general set shifting deficits regardless of task emotional valence (DeLissnyder, Koster, Derakshan, & De Raedt, 2010).

A recent study employing adults with clinical diagnoses of depression obtained evidence for an association with general set shifting deficits rather than valence-specific deficits (DeLissnyder, Koster, Everaert, Schacht, Van den Abeele, & De Raedt, 2012).

Thus, in adults, rumination may be associated with some types of general deficits (e.g., task switching; De Lissnyder et al., 2010, 2012a), whereas other deficits may be specific to emotional information (e.g., inhibition; De Lissnyder et al., 2010), and findings may differ in samples with clinical depression versus community samples (e.g., DeLissnyder et al., 2012a).

Interestingly, dissociations have been reported in associations between executive control and rumination versus depressive symptoms. Hertel, Benbow, and Geraerts (2012) reported that after controlling for depressive symptoms, brooding was significantly associated with poorer recall of words when task demands didn't constrain attention, whereas depressive symptoms were not associated with word recall. Whitmer and Banich (2007) found that depressive symptoms were not associated with inhibition or set switching after controlling for rumination. Likewise, examining inhibitory deficits in long-term memory, Whitmer and Banich (2010) reported that depression appeared to act as a suppressor variable, in that it was unrelated to inhibition, unless rumination was taken into account, after which it was marginally positively related to inhibition. Moreover, associations between aspects of rumination and inhibitory deficits became stronger when depression was taken into account, suggesting the link between rumination and inhibition is not associated with depression. In their examination of the association between rumination, depressive symptoms, and goal maintenance and set switching Altamirano and colleagues (2010) reported similar dissociations. Specifically, greater symptoms of depression were not associated with task switching, whereas rumination

was. In addition, depressive symptoms were significantly associated with poorer goal maintenance (accuracy on Stroop task) after controlling for rumination, whereas rumination was associated with *better* goal maintenance. Meiran and colleagues (2011) found that controlling for rumination, diagnosis was associated with a different pattern of findings than was rumination, including abnormal Stroop, performance suggestive of impaired conflict monitoring, task switching abnormalities, an association between longer preparation time and greater reductions in mixing costs, and continued readiness to switch set when this was no longer necessary. Finally, Whitmer and Banich (2012) reported that maladaptive repetitive thought was significantly associated with slowed reversal learning, but that depressive symptoms were not.

Unique patterns of association with specific types of deficits in executive function (Altamirano et al., 2010; De Lissnyder et al., 2011; Whitmer and Banich, 2007) have led researchers to suggest rumination may be more proximally related to deficits in cognitive control than is depression per se (De Lissnyder et al., 2011), that rumination may be associated with deficits in some tasks requiring flexibility but associated with enhanced performance on tasks requiring goal maintenance (Altamirano et al., 2010), and that in some cases depression acts as a suppressor variable, obscuring the relation between rumination and deficits in executive function (specifically, inhibition; Whitmer & Banich, 2010). Interestingly, evidence for dissociations in the relation between deficits in executive control and rumination and depressive symptoms may help to explain inconsistencies in findings with regard to the profile of executive deficits in individuals

with depression. If rumination is more proximally associated with some types of deficits in executive control (e.g., task switching), whereas depression is more proximally associated with other types of deficits in executive control, patterns of deficits within depressed populations may appear inconsistent, varying with individual differences in tendencies to ruminate.

Rumination and Executive Functions in Children and Adolescents

Few studies have examined the association between rumination and executive function in youth. Wilkinson and Goodyer (2006) assessed sustained attention, attentional control/set shifting, and selective attention via the Test of Everyday Attention for Children (TEA-Ch; Manly, Anderson, Nimmo-Smith, Turner, & Robertson, 2001), self-reported rumination, and self-reported and rater-assigned depressive symptoms in a sample of demographically matched healthy controls and adolescents with current unipolar depression (age 11-17). Whereas associations were obtained between depression diagnosis and attentional deficits (including sustained attention, attentional set shifting, and selective attention), self-reported rumination was not associated with attentional set shifting accuracy or latency in either depressed or control participants, and both rumination and attentional set shifting latency uniquely predicted diagnostic group membership. However, this study does not report exploring whether diagnostic status interacted with rumination to predict attentional difficulties or controlling for concurrent depressive symptoms in examining the association between rumination and attention. Moreover, the measure of set shifting latency, the construct that has received the most

attention in the adult literature, could not be fully examined as a large number of participants made too many set shifting accuracy errors to allow for a valid timing score. Finally, the study did not report associations between rumination and aspects of attention other than attentional switching (e.g., sustained or selective attention).

In addition to this single study, several studies have examined rumination in relation to effortful control, a later-emerging self-regulatory component of temperament that reflects the “ability to voluntarily regulate behavior and attention, as seen in the inhibition of a dominant response and activation of a subdominant response” (Rothbart, Ahadi, Hershey, & Fisher, 2011) and encompasses “inhibitory control, the ability to focus and shift attention, and sensitivity to and pleasure in low-intensity stimuli” (Rothbart, 2007). This dimension (often measured via self or parent-report questionnaire) has been conceptualized as the outcome of the development of executive attention and has been positively associated with performance on tests of executive function in children (Rothbart, Ellis, Rueda, & Posner, 2003). Studies have suggested that effortful control moderates associations between (1) various other aspects of vulnerability and the development of rumination, and (2) tendency to ruminate and the subsequent development of depression (but for an exception in a large scale sample of young adults, see Arger, Sanchez, Simonson, & Mezulis, 2012). For example, Hilt, Armstrong, and Essex (2012) obtained evidence that effortful control (as assessed via a coded behavioral task requiring attention to a specific stimulus) moderated associations between over-controlling parenting and negative affect and increased risk for rumination, such that risk

was higher among those low in effortful control. Verstraeten, Vasey, Raes, and Bijttebier (2009) found that low effortful control (as assessed by questionnaire) moderated associations between negative affect, depressive symptoms and rumination both concurrently and prospectively, such that stronger associations were found among those low in effortful control. At the same time, a methodologically stronger prospective study in an adolescent sample (the only study to assess both rumination and depressive symptoms at multiple time points) obtained no evidence for moderating effects of self-reported effortful control on associations between negative emotionality, rumination, and depressive symptoms (Mezulis & Rudolph, 2012).

In summary, few studies have examined associations between trait rumination, depression, and executive control in children or adolescents. Only two such studies have used behavioral measures (Hilt et al., 2012; Wilkinson & Goodyer, 2006), and each of these have only examined rumination in relation to a single outcome measure of attention (e.g., coded sustained attention to a visual stimulus and attentional set shifting, respectively). No studies to date have examined associations between rumination and other types of executive function, including working memory or other aspects of attention in children or adolescents.

Moderating Effects of Gender

Women are approximately twice as likely as men to have experienced a depressive episode, whereas rates are similar or higher for boys than girls prior to age 13 (Allgood-Merten, Lewinsohn, & Hops, 1990; Twenge & Nolen-Hoeksema, 2002). This

difference, which reflects higher risk for first onsets, not recurrence, of depression among females (Hankin et al., 1998, Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993), first appears between the ages of 13 and 15, with the greatest increase in the gender difference observed between the ages of 15 and 18 (Hankin et al., 1998). Beginning in early adolescence, between the ages of 13 and 16, girls report increasingly higher levels of depressive symptoms than boys (Allgood-Merten et al., 1990; Holsen, Kraft, & Vitterso, 2000), and by mid-to-late adolescence, they are at higher risk for depressive episodes (Hankin et al., 1998).

Given these known sex differences in depression, Hankin, Wetter, and Cheely (2008) have recommended that researchers evaluate and report potential sex differences routinely, even when they are not the primary focus of the study. They note that many published articles in the depression literature have statistically controlled for sex and removed any potential influence of sex on effects, a practice that implies that sex is a nuisance variable to be controlled, when in fact sex differences are an opportunity for studying causal processes that can illuminate the complex etiological influences contributing to depression. In the study of rumination this is of particular relevance because there is some evidence that girls exhibit higher levels of rumination than boys beginning in early adolescence (Broderick, 1998; Jose & Brown, 2008).

Indeed, the literature on depression, rumination, and executive control has frequently controlled for gender differences but has not reported whether gender moderates associations. In their study of an unselected community sample of high and

low ruminators, Davis and Nolen Hoeksema (2000) found that gender moderated the association between rumination and perseverative errors and inflexible sorts on the advanced section of the WCST, which consisted of 3 levels added to increase the difficulty of the task. Post-hoc tests indicated male ruminators made more inflexible sorts than nonruminators. In contrast, female ruminators and nonruminators did not differ from one another. The authors suggested that, given evidence that men are less likely to ruminate than women, when they do become ruminators, this coping style may be part of a more pervasive problem with cognitive inflexibility and allocation of cognitive resources, whereas sources of rumination for women may be more rooted in the social environment.

Interestingly, some studies with adults that have found evidence for an association between rumination and executive control, have not reported controlling for gender, matching groups on gender, or examining whether gender moderates hypothesized associations (e.g., Altamirano et al., 2010; Whitmer & Banich, 2007, 2010), even in cases where the majority of the sample was male (e.g., 65% of the sample reported by Altamirano et al., 2010, and 67% of non-depressed and 75% of depressed sample reported by Levens et al., 2009) or female (e.g., 73% of the sample reported by Bernblum & Mor, 2010; 65% of the sample reported by Berman et al., 2011; 61% of the sample reported by Daches et al., 2010; 84% of the sample reported by De Lissnyder et al., 2012b). The few studies examining rumination and emotionally neutral tests of executive control that have employed a sample consisting of both males and females and controlled

for gender (DeLissnyder et al., 2010, 2011, 2012a) have reported that dysphoric and nondysphoric groups or groups of high and low ruminators did not differ with respect to gender but have not examined whether gender moderates effects or controlled for gender in analyses examining associations between continuous scores on measures of rumination and performance on tasks measuring executive control.

In the only neuropsychological study examining rumination in children and adolescents, Wilkinson and Goodyer (2006) matched depressed and nondepressed groups for gender but did not report controlling for gender in examining associations between rumination and executive functions and did not examine whether gender moderated outcomes. Within the temperament literature two studies have reported testing moderating effects of gender. Hilt and colleagues (2012) found that gender did not moderate associations between effortful control and rumination. Verstraeten and colleagues (2009) found some evidence that gender moderated associations between negative affect, effortful control, and depressive symptoms, such that associations were stronger in boys than girls, but did not report that gender moderated associations between effortful control and rumination.

Likewise, the majority of studies examining profiles of executive control in children and adolescents with unipolar depression have either matched groups on gender or included gender as a covariate. Several studies (e.g., Houston et al., 2004; Micco et al., 2009; Osborn & Meador, 1990; Van der Meere et al., 2011) do not report controlling for gender at all. In addition, only two studies employing small samples of children or

adolescents have examined whether gender moderates associations between depression and executive control. Frost and colleagues (1989) did so and found no moderating effects of gender and found that gender did not interact with diagnostic group to predict anything other than visuospatial abilities. Likewise, Halari and colleagues (2009) found no gender by diagnosis interactions. Given the consistent evidence for sex differences in depression and rumination, the scarcity of research examining whether gender moderates associations between these constructs and executive control is quite surprising. Additional research is needed that fully explores whether gender moderates associations between executive control and rumination or unipolar depression.

Developmental Framework

The preponderance of empirical research examining unipolar depression, rumination, and executive functions reviewed above has used samples of adults. From a developmental perspective, there are many reasons why the study of samples of children and adolescents is needed. First, there is evidence that pre-pubertal pediatric depression may differ in important ways from post-pubertal depression that emerges in adolescence or adulthood (for discussion, see Avenevoli, Knight, Kessler, and Merikangas, 2008). Therefore, it cannot be assumed that the patterns of deficits observed in adults can be extended downward to unipolar depression in youth. From a clinical perspective, research examining profiles in remitted youth may also provide unique information not available from remitted adult samples, given that remitted youth are less likely to have

experienced a prolonged course of disorder, whereas deficits observed in remitted adults may represent “scars” of prior episodes.

In addition, studies of youth at high risk for depression are of interest as they provide unique information not available from studies of high risk adults who have not developed the disorder. Because rates of depression increase dramatically between early adolescence and young adulthood (Hankin et al., 1998), a greater proportion of high risk youth than high risk adults are likely to go on to develop full blown unipolar depression. High risk studies of adults have employed much older samples where the mean age is beyond the window of highest risk for disorder onset (e.g., age ranges in the 30s or 40s; Christensen et al., 2006). Studies using high risk samples of early adolescents are therefore uniquely situated to examine profiles of executive function in individuals who have not yet developed unipolar depression but are developmentally poised to do so.

Moreover, the transition from childhood to adulthood is particularly important in understanding vulnerability to depression. Whereas lifetime prevalence of depressive disorders in prepubertal school-aged children is estimated to be quite low (less than 5%; Hankin et al., 1998; Kashani & Sherman, 1988), lifetime prevalence increases in adolescence (Hankin et al., 1998; Lewinsohn et al., 1993), particularly between the ages of 15 and 18 (Hankin et al., 1998), so that by young adulthood, lifetime prevalence has been estimated to be approximately 10-20% in females and 5-12% in males (APA, 2001). Rates begin to increase in early adolescence with the highest window of risk between age 15 and young adulthood (Hankin et al., 1998). For those without a past history of depression, the greatest risk for developing the disorder occurs between the ages of 15-19

and 25-29 (Burke, Burke, Regier, & Rae, 1990), and peak age of onset has been estimated to be between 15 and 20 years of age (Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). In addition, the oft-noted gender difference in depression begins to emerge in early adolescence and peaks in mid-adolescence, with girls twice as likely as boys to develop the disorder (Lewinsohn, Clarke, Seeley, & Rohde, 1994). As with onset of unipolar depression, the period of entry into adolescence may be particularly important for understanding ruminative tendencies. For example, a large cross-sectional study of rumination in a nonclinical sample of adolescents suggested that rumination increases significantly between the ages of 11 and 18 for both males and females, but does so more modestly and starting at a later age for males (after age 14) than females (for whom rumination increases in a linear fashion after age 12; Jose & Brown, 2008).

In addition, development of executive abilities necessary for self-regulation is protracted and continues through young adulthood (Casey, Giedd, & Thomas, 2000; Casey, Galvan, & Hare, 2005; Diamond 2002). Performance on behavioral tests of executive control has indicated distinct trajectories of development for different subprocesses, which often emerge in infancy or early childhood, show different rates of development across childhood and adolescence (for reviews see Anderson, 2002; Diamond, 2002; Spear, 2010; Steinberg, 2005, 2008; Welsh, 2002), and undergo refinement throughout adolescence (Luna & Sweeney, 2004). Executive skills that continue to show development on behavioral tasks at least into early adolescence or

beyond include working memory (for review, see Bunge & Wright, 2007; Conklin, Luciana, Hooper, & Yarger, 2007; De Luca et al., 2003; Hooper, Luciana, Conklin, & Yarger, 2004; Huizinga & Van der Molen, 2007; Kwon, Reiss, & Menon, 2002; Luciana, Conklin, Hooper, & Yarger, 2005; Luna, Garver, Urban, Lazar, & Sweeney, 2004; Luna, Padmanabhan, & O’Hearn, 2010; Siegel, 1994), sustained attention (Greenberg & Waldman, 1993), selective attention (Leon-Carrion, Garcia-Orza, & Perez-Santamaria, 2004) and inhibition of irrelevant information or prepotent responses (Casey, Thomas, Davidson, Kunz, & Franzen, 2002; Casey et al., 2005), set shifting and maintenance (Cepeda, Kramer, & Gonzalez de Sather, 2001; Huizinga, Dolan, & Van der Molen, 2006; Kray, Eber, & Lindenberger, 2004), verbal fluency (Klenberg, Korkman, & Lahti-nuuttila, 2001; Levin et al., 1991; Welsh et al., 1991), multitasking (Blakemore & Choudhury, 2006), and planning and complex problem solving (Albert & Steinberg, 2011; Asato, Sweeney, & Luna, 2006; De Luca et al., 2003; Demetriou et al., 2002; Huizinga, Dolan, & van der Molen, 2006; Levin et al., 1991; Luciana & Nelson, 2002; Welsh et al., 1991).

Development of these cognitive competencies co-occurs with ongoing structural brain maturation throughout adolescence (particularly of the PFC and cortical association areas, as well as subcortical regions that receive projections from higher order association areas; Blakemore & Choudhury, 2006, Casey et al., 2000, 2005; Diamond 2002; Durston & Casey, 2006; Giedd et al., 1999; Giedd, 2004, 2008; Gogtay et al., 2004; Lenroot & Giedd, 2006; Poletti, 2009, Somerville & Casey, 2010; Sowell et al., 2004; Spear, 2010).

Ongoing synaptic pruning and myelination reflected in increased ratio of white to gray matter across adolescent development (Casey et al., 2008), as well as increasing integrity of white matter tracts connecting frontal, parietal and subcortical regions (Asato, Terwilliger, Woo, & Luna, 2010; Casey et al., 2005; Liston et al., 2006) is observed with increasing age. Likewise, as previously noted, functional imaging during engagement in tests of executive function suggests a developmental shift occurs from childhood to adulthood, characterized by more focal or fine-tuned recruitment of regions implicated in cognitive control in conjunction with attenuated recruitment of task irrelevant regions (Brown et al., 2005; Durston et al., 2006; Luna, Padmanabhan, & O'Hearn, 2010), as well as increases in coordinated activity between neural networks (Rubia, Smith, Taylor, & Brammer, 2007), and increases in functional connectivity between distant regions (Fair et al., 2007, 2009). Overall, findings suggest increased reliance on widely distributed brain regions functioning in collaborative networks of activity with the PFC (Luna et al., 2010; Luna & Sweeney, 2004; Spear, 2010).

In contrast, subcortical limbic regions mature earlier, such that adolescence is characterized by functionally mature subcortical limbic regions in conjunction with immature regions subserving top-down cognitive control, as well as immature functional connections between PFC and subcortical regions (for review see Casey et al., 2008). As a result of this imbalance between prefrontal and limbic development, neurobiological models conceptualize adolescence as unique relative to childhood or adulthood, in that it is a period characterized by particular sensitivity to reward and to emotionally salient

situations (Casey, Getz, & Galvan, 2008; Somerville & Casey, 2010) due to insufficient top down cognitive control over regions subserving reward and affective processing.

Thus, during the transition from childhood to adulthood individuals experiencing greater sensitivity to reward and to emotionally salient situations must navigate an increasingly challenging and stressful environmental context (Ge, Lorenz, Conger, Elder, & Simons, 1994; Ge, Conger, & Elder, 2001; Rudolph & Hammen, 1999; Schwartz & Koenig, 1996), while they do not yet possess mature executive functions necessary for self-regulation. The shift from childhood to early adolescence may be a time when the impact of preexisting individual differences in ruminative tendencies and/or executive control becomes amplified – that is, these differences may have greater power to confer vulnerability to or resilience against depression, as adolescents begin to have more independence to navigate and shape an increasingly stressful and complex environment. Preexisting individual differences may also be exacerbated by the challenges of the adolescent transition; some vulnerabilities may stabilize or come online and individual differences may emerge with development.

Given the status of adolescence as a period of unique vulnerability to depression and as a period when executive functions have come online but are still immature, it is surprising how little research to date has examined associations between depression, rumination, and executive function in this age group. Research that has done so has employed wide age ranges encompassing heterogeneous samples of children and adolescents and has not taken pubertal development into account. A goal of the current

study was therefore to examine associations between rumination, depression, and executive function in a large sample of early adolescents with a circumscribed age range, controlling for pubertal development. The aim was to provide a snapshot of how individual differences in executive function are related to individual differences in rumination and depressive symptoms in a sample of youth, the majority of whom have not yet developed full blown unipolar depression, even though many of them may be poised to do so over the course of adolescence. This age range was thought to be likely to display substantial variability in trait rumination and also in performance on measures of executive function, permitting elucidation of associations between these constructs prior to onset of unipolar depression. In addition, the moderating role of gender and depression diagnosis was explored.

Specific Aims and Hypotheses

The current study examined associations between executive functions, trait rumination, depressive symptoms, and depression diagnosis and risk in a large scale community sample of early adolescents. A primary question was the nature of the association between executive function, trait rumination, and depressive symptoms in this age group, and the role of gender and diagnosis as moderators of these associations. Additional questions included the nature of the association between executive functions and diagnosis of and risk for unipolar depression in this age group and the role of gender as a moderator of these associations.

Primary Goal

The primary goal (Goal 1) of the current study was to examine the association between trait rumination, depressive symptoms, and executive control in a sample of early adolescents. Our sample consisted of individuals, the majority of whom have not yet developed a depressive episode due to their young age, but who are likely to be experiencing developmental increases in rumination as they enter early adolescence and thus should exhibit a wide range of rumination scores. Strongest support for an association between rumination and deficits on neutral tests of executive control has come from research with nonclinical samples of undergraduates and has suggested a pattern of mental inflexibility (Altamirano et al., 2010). This was the first study to examine whether a similar pattern is observable in non-treatment-seeking samples of children or adolescents. Examination of associations in this nonclinical community sample of adolescents was thought to be useful given the possibility that examination of profiles in individuals already diagnosed with unipolar depression might reflect a heterogeneous group of high and low trait ruminators, which would obscure potentially dissociable relations between rumination versus depression and executive control deficits. In addition, the current study explored associations with a variety of aspects of executive function for emotionally-neutral stimuli, including sustained attention, selective attention, attentional set shifting, and working memory. Examination of separate associations between rumination and depression and these related but separable constructs is of particular interest given evidence from nonclinical samples of young adults suggesting

rumination may be associated with better performance on some aspects of executive function but weaker performance on others (Altamirano et al., 2010) and that rumination and depressive symptoms may exhibit dissociable patterns of associations with different aspects of executive control (Altamirano et al., 2010; De Lissnyder et al., 2011; Whitmer & Banich, 2007, 2010). Very few studies have systematically examined this in adults, and none have done so in samples of children and adolescents.

It was hypothesized that trait rumination would be associated with deficits in executive control, controlling for concurrent depressive symptoms in the overall sample (hypothesis 1). Because so few studies have systematically examined whether associations between rumination, depression, and aspects of executive function are dissociable, our hypotheses were exploratory. However, based on studies that have done so (Altamirano et al., 2010; Meiran et al., 2011; Wilkinson & Goodyer, 2006), it was expected that somewhat different patterns of findings might emerge, with rumination more closely associated with deficits in set shifting tasks requiring inhibition of irrelevant or no-longer-relevant information. In contrast, it was expected that depressive symptoms might be more closely associated with deficits on tasks requiring goal maintenance over time and in the face of distraction information (e.g., selective and sustained attention).

Secondary Goals

A secondary goal of the current study (Goal 2) was to examine whether worse performance on measures of executive function would predict greater likelihood of a lifetime history of diagnosis of unipolar depression in a well-defined socioeconomically

and racially diverse community sample of male and female early adolescents. This study aimed to advance previous studies, which have yielded inconsistent findings, in that it employed a large ($N = 486$) unselected, non-treatment-seeking sample of early adolescents, and well-validated diagnostic interview techniques, and investigated aspects of executive function that have been particularly implicated in depression and depressive rumination, including selective attention, sustained attention, divided attention, attentional set-shifting, and working memory.

It was hypothesized that poorer performance on measures of executive function would be associated with a greater likelihood of lifetime history of unipolar depression (hypothesis 2). Given inconsistent findings to date, our hypotheses with regard to specific subprocesses that would be impaired were exploratory. Controlling for depressive symptoms, diagnostic status was expected to be associated with poorer performance on tests of selective attention, set shifting and working memory, reflective of inhibitory deficits. An additional goal (Goal 3) was to examine whether aspects of executive function are associated with higher risk of future onset of depression (as indicated by maternal history of disorder), controlling for child diagnosis and depressive symptoms. Although studies examining this question to date have yielded null findings for the most part (Micco et al., 2009; Perez-Edgar et al., 2006), abnormal neural activation has suggested the possibility of compensatory activity to achieve comparable levels of task performance on the n-back in a small ($N = 17$) sample of youth at-risk for depression (Mannie et al., 2010), and a marginally significant difficulty with set maintenance was

observed in performance of high risk youth on the WCST (Klimes-Dougan et al., 2006). Additional reason to expect deficits in high risk individuals in the absence of current depression comes from studies of adults at genetic high risk who have exhibited impairment on tests of set shifting, conflict resolution, working memory, and selective attention (Christensen et al., 2006; Holmes, Bogdan, & Pizzagalli, 2010). Moreover, this study improved methodologically on previous studies of youth with parental history of depression in that it combined a large unselected community sample of mothers with versus without a lifetime history of depression, used a range of measures of executive function, and took into account child psychopathology and concurrent depressive symptoms.

Given previous findings in samples of offspring of parents with unipolar depression suggesting compensatory neural activity to achieve comparable levels of n-back working memory task performance (Mannie et al., 2010) and marginally significant difficulties with set maintenance on the WCST (Klimes-Dougan et al., 2006), it was tentatively hypothesized that high risk youth might exhibit specific deficits on tests of working memory and attentional set shifting (hypothesis 3). However, studies to date have yielded far less convincing evidence for executive deficits in individuals not currently experiencing a depressive episode. Our hypotheses with regard to Goal 3 were therefore exploratory, and it was expected that deficits, if identified, would likely be qualitatively less pervasive or severe than those characterizing youth who have previously experienced or were currently experiencing a depressive episode.

Exploratory Analyses: Moderating Variables

The current study explored whether rumination interacted with diagnostic status or depressive symptoms to predict deficits in executive function. Studies using samples with depression diagnoses have not consistently examined this question, and our hypotheses are therefore exploratory. Those that have done so have found evidence for a significant interaction whereby rumination was associated with deficits only among individuals with depression diagnoses in a high interference multitasking scenario (Levens et al., 2009) and that induced rumination was associated with deficits in selective attention and perseveratory counting errors only among those with depression diagnoses (Philippot & Brutoux, 2008; Watkins & Brown, 2002).

At the same time, other studies have reported associations between rumination and working memory collapsing across diagnostic status (Berman et al., 2011) or between rumination and task switching and working memory deficits controlling for diagnostic status (Meiran et al., 2011). It was expected that some executive deficits might be associated with rumination only among those with diagnoses or high current symptoms of depression, whereas others might be associated with rumination across the entire sample or among those who have no lifetime history of disorder or current depressive symptoms. In particular, there is evidence in adults that attentional switching is associated with trait rumination in nonclinical samples of young adults, and it was therefore expected that set shifting would be associated with rumination in the overall sample, and in the subset of adolescents with no lifetime history or current symptoms of

depression. In contrast, rumination was expected to be associated with deficits in dual-task performance, which is somewhat analogous to the multitasking scenario (Levens et al., 2009), only among those with unipolar depression diagnoses or current depressive symptoms.

Given recommendations by Hankin and colleagues (2008), the current study explored the possibility that gender moderates associations between rumination and executive function. It was suggested that associations might be found between rumination and executive control only among males or females. Studies have typically controlled for gender and rarely examined whether gender moderated associations. Davis and Nolen-Hoeksema (2000) did so in adults and found evidence for some associations between rumination and cognitive flexibility among males only. As socialization may contribute more to rumination in females (Davis & Nolen-Hoeksema, 2000), it was thought to be possible that executive control deficits might account for a greater proportion of the variance in males. Conversely, it was thought to be possible that among females, who are believed to be at higher risk for rumination, greater executive control might serve to ameliorate risk.

CHAPTER 2

METHOD

Participants

Participants were recruited as part of the Temple University (TU) Adolescent Cognition and Emotion (ACE) Project, a NIMH-funded prospective longitudinal study of the development of depressive disorders in adolescence headed by Dr. Lauren Alloy. Measures were collected during the Time 1 phase of the study. Participants consisted of a community sample of early adolescents. Caucasian and African American male and female adolescents and their mothers or primary female caretakers were recruited from the School District of Philadelphia and other Philadelphia area public and private middle schools. Inclusion criteria included age (age 12-13), fluency in English, and the absence of developmental, cognitive, and emotional disability that would preclude comprehension of study measures. Adolescents who were within 5 months of the age inclusion criterion were also permitted to participate, resulting in a final sample comprised primarily of 12 to 13-year-olds (93% of the sample), but ranging in age from 11 to 14 (M age = 12.88 years; SD = .62). Mother-child dyads who met study inclusion and exclusion criteria were invited to Temple University to complete the Time 1 assessment. Overall sample characteristics are presented in Table 1.

Depression diagnoses for adolescents are presented in Table 2. Of those who met criteria for a current or lifetime history of unipolar depression ($n = 68$), 11 (16.18%) met criteria for a current depressive episode. Among those with current depression, diagnoses

included the following: DSM and RDC Major Depressive Disorder, Definite ($n = 5$), DSM Major Depressive Disorder, Definite and RDC Major Probable Depression ($n = 2$), DSM Subthreshold and RDC Minor Probable Depression ($n = 1$), Current RDC Minor Probable ($n = 1$), DSM Dysthymia and RDC Intermittent Depressive Disorder ($n = 1$), and current DSM Dysthymia and RDC Intermittent Depressive Disorder superimposed on a single past episode meeting criteria for DSM subthreshold and RDC Major Probable ($n = 1$). An additional 57 participants met criteria for a past (but not current) episode of depression currently in remission. Among these participants, diagnoses were as follows: DSM and RDC Major Definite MDE ($n = 26$), DSM Definite and RDC Major Probable ($n = 3$), DSM Definite MDE ($n = 1$), DSM Subthreshold and RDC Major Probable ($n = 12$), RDC Minor Definite ($n = 2$), RDC Minor Probable ($n = 8$), DSM Dysthymia and RDC IDD ($n = 2$), DSM DNOS ($n = 2$), and DSM DNOS and Past DSM subthreshold ($n = 1$).

Maternal diagnostic data were available for 463 participants, 24 of whom were excluded from Goal 3 analyses due to a history of psychosis or bipolar spectrum disorder. Table 3 summarizes depression diagnoses in mothers with a lifetime history of unipolar depression ($n = 188$), who met criteria for a lifetime history of DSM or RDC Definite MDE ($n = 131$), DSM Subthreshold and RDC Major Probable ($n = 39$), and DSM Dysthymia or RDC IDD ($n = 18$). Table 4 summarizes comorbidity (with regard to anxiety disorders, oppositional defiant or conduct disorders, ADHD, eating disorders, substance use disorders, and bipolar spectrum disorders) in the overall adolescent sample

and frequency of disorder among adolescents with and without a lifetime history of unipolar depression. Chi square analyses indicated that lifetime history of depression in adolescents was associated with greater likelihood of an additional comorbid disorder, $\chi^2(1, N = 486) = 5.45, p = .02$.

Measures

Demographic Information was assessed via parent self-report questionnaire.

Parents were asked to report parents' years of education, family income, child eligibility for school lunch, and their own age, race and ethnicity and that of their child. Given that all individuals in the sample identified as (1) white or European, (2) African American, or (3) Biracial, or Multiracial, two dummy variables were created for race (Dummy variable Race 1: African American = 1, Not African American = 0; Dummy variable Race 2: Biracial or multiracial = 1, Not Biracial or Multiracial = 0) and used in all analyses controlling for race. A single dichotomous SES variable was computed based on parent reported income and child eligibility for school lunch. Those in the top three brackets (with a household income of \$60,000 and higher) were coded as high in SES; those in the bottom three brackets (with a household income under \$50,000) were coded as low in SES, and those in the middle income bracket (with a household income of \$45,000-59,000) were coded as of high SES unless they reported eligibility for school lunch (45.3% of those in this income bracket reported eligibility for school lunch), in which case they were coded as low in SES.

Table 1. *Sample Characteristics*

	N	%
Gender		
Male	230	47.3
Female	256	52.7
Race		
White	237	48.8
African American	229	47.1
Biracial or Multiracial	20	4.2
SES		
High	217	53.1
Low	258	44.7
Unavailable	11	2.3
Income		
0-14,999	71	14.6
15-29,000	73	15
30-44,999	80	16.5
45-59,000	78	16
60-74,000	53	10.9
75-89,999	34	7
90,000 and over	86	17.7
Unavailable	11	2.3

Table 2. *Diagnoses of Adolescents With Lifetime History of Depression*

	Current	Past
DSM/RDC Definite MDE	7	30
DSM Subthreshold and RDC Major/Minor Probable	1	12
RDC Minor Definite	0	2
RDC Minor Probable	1	8
DSM Dysthymia and RDC IDD	2*	2
DSM D-NOS	0	3**
Any Unipolar Depression Diagnosis	11	57

Note. DSM = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. D-NOS = Depressive Disorder Not

Otherwise Specified. IDD = Intermittent Depressive Disorder. RDC = Research Diagnostic Criteria. * In one case this diagnosis was superimposed on a single past episode meeting DSM subthreshold and RDC Major Probable Criteria. ** One person reported a past episode meeting criteria for DSM D-NOS and an additional episode meeting DSM Subthreshold criteria.

Table 3. Diagnoses of Mothers with Lifetime History of Depression

	Current	Past Only	Lifetime
DSM/RDC Definite MDE	6	125	131
DSM Subthreshold/RDC Major Probable	3	36	39
DSM Dysthymia/RDC IDD	7	11	18
Depression Diagnosis	16	172	188

Note. DSM = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. IDD = Intermittent Depressive Disorder. RDC = Research Diagnostic Criteria.

Table 4. Frequency of Axis I Disorders in Adolescents With and Without a Lifetime History of Unipolar Depression

	Depression History	No Depression History	Overall Sample
Anxiety Disorder	21(30.9%)	71(17%)	92(18.9%)
Oppositional Defiant or Conduct Disorders	10 (14.7%)	31 (7.4%)	41(8.4%)
ADHD	21(30.9%)	61(14.6%)	82(16.9%)
Eating Disorder	1(1.5%)	2(0.5%)	3(0.6%)
Alcohol or Drug Use	0(0%)	0(0%)	0(0%)
Bipolar Spectrum Disorder	0(0%)	10(2.4%)	10(2.1%)
Any Axis I Disorder	35(51.5%)	153(36.6%)	188(38.7%)

Pubertal Status was assessed via the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). This scale was completed by mothers at Time 1, and requires that they rate indices of pubertal development (e.g., growth spurt in height, body hair, skin change, voice change and facial hair growth for boys, and breast change and menstruation for girls) on a 4-point Likert scale. Scores are summed to create a scale ranging from 0 to 20, with higher scores indicating more mature pubertal development. The PDS has demonstrated good convergent validity and test-retest reliability in diverse samples (Siegel, Yancey, Anashensel, & Schuler, 1999). Parent report was used rather than youth report, because of more complete data for parent report, due to omissions by youth on this scale. There is evidence that parent report of pubertal development is moderately correlated with the results of a physical exam ($r_s = .57-.87$; Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987). In addition, previous research has indicated that parents and youth tend to converge on ratings of pubertal development, with significant correlations between both individual items and overall scores on parent and youth report (Carskadon & Acebo, 1993). In the current study, parent and youth report was significantly positively correlated ($r = .65, p = .000$), such that parent report of more mature pubertal development was associated with child report of more mature pubertal development. As separate items were completed for males and females, separate internal consistencies were calculated for this measure within the male and female samples. Internal consistency of the PDS parent report in the current sample was acceptable ($\alpha = .73$ for females, $\alpha = .81$ for males).

Measures of Executive Function

Attentional Control

Test of Everyday Attention for Children (TEA-Ch; Manly et al., 2001). The TEA-Ch assesses the attentional capacities of children age 6 to 16. It is a game-like test that was designed to provide an ecologically valid measure of attention in line with Posner and Petersen's (1990) theory of attention networks. The TEA-Ch has demonstrated a 3-factor structure across multiple studies and cultural contexts. Specifically, 3 latent factors corresponding to the theoretical constructs of sustained and selective attention and attentional control/switching have been identified through confirmatory factor analysis (Chan et al., 2008; Manly et al., 2001). The test has demonstrated satisfactory test-retest reliability in Western samples over a 5-20 day interval (r_s .64-.92; Manly et al., 2001) and Chinese samples over a 4-week interval, for the majority of subtests (r_s .045-.79; Chan et al., 2008). It also has demonstrated satisfactory convergent (with measures of selective attention; Manly et al., 2001) and discriminant validity (WISC and WRAT subtests; Manly et al., 2001). Consistent with previous research (Wilkinson & Goodyer, 2006), TEACH raw scores were transformed to age and gender-scaled scores. Scores were derived from a normative sample, with a mean of 10 and a standard deviation of 3. This conversion was made to render means more easily interpretable, to facilitate comparison with previous research findings and across adolescents of different ages and gender, and to convert the data to a normal distribution appropriate for multiple

regression. Thus, for each subtest described below, higher scores are indicative of better performance.

Creature Counting is a subtest assessing ability to shift attention. Children are instructed to count the number of “creatures in their burrow,” and count visually presented pictures of creatures interspersed along a winding path. In order to do so, they must alternate/switch between counting forwards and backwards (from 1 up to 12 and vice versa) in response to visual cues (pictures of upwards and downwards arrows interspersed among the “creatures”). This subtest thus requires inhibition of no-longer-relevant task sets, shifting to new and previously inhibited task sets, and inhibition of prepotent mental sequencing. This subtest yields an accuracy score (number of correctly counted trials out of 7), with higher scores indicating greater accuracy, as well as a set shifting time score (completion time scores for correct trials), with higher scores indicating shorter completion time (e.g., better performance).

Sky Search is a timed visual search task requiring selective attention and inhibition of irrelevant visual stimuli/interference control. Children are required to view an array of 128 pairs of space ships and are instructed to find and circle targets (pairs of matching space ships) while ignoring very similar distracters (pairs of dissimilar space ships) and instructed to work as quickly as possible. A second, motor control condition requires children to circle targets as quickly as possible in the absence of distracters, providing a control for individual differences in psychomotor speed. This subtest yields measures of selective attention accuracy (number of the 20 possible targets correctly

identified), time per target, and an Attention score which subtracts the ratio of targets found to time elapsed in the motor control condition (containing no distractors) from the selective attention condition (containing distractors). The Attention score was used as a measure of selective attention for the current study, as it controls for individual variability in motor speed. Higher scores indicate better selective attention performance.

Score is a test of auditory sustained attention, which consists of a 5.5 minute audiorecording of identical tones (ranging from 9 to 15) presented at irregular intervals in 10 trials of variable length. Children are asked to count silently the scoring sounds and to tell the recorder how many sounds they heard when a siren sound signals the end of a trial. Higher scores indicate better performance.

Sky Search Dual Task is a cross-modal dual task test requiring sustained and divided attention. Children, who had previously completed *Score* and *Sky Search* Subtests, were instructed to simultaneously perform both tasks. Specifically children are instructed to circle targets (matching ship pairs) while ignoring distractors (dissimilar ship pairs), and to count the number of scoring sounds and to provide a count when a siren sound signals the end of each trial. This task yields a dual task decrement score, computed by subtracting the time per target score for *Sky Search DT* from the time per target score for *Sky Search*. As scores are standardized, higher scores indicate better performance.

Working Memory

Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV; Wechsler, 2003) Digit Span Subtest. The WISC-IV has been standardized and normed for children age 6 to 18. The digit span subtest assesses auditory-verbal working memory. In the “forward” condition, children are instructed to listen to and then repeat a series of numbers presented by the experimenter at approximately 1 second intervals. In the “backwards” condition, children are asked to listen to a series of numbers presented by the experimenter, but are told to repeat the numbers in reverse order. Scores from the two conditions were summed to create a total score. As with TEACH scores, WISC Digit Span scores were transformed to age scaled scores derived from a normative sample with a mean of 10 and a standard deviation of 3. Thus, higher digit span scores indicate better working memory performance relative to same age peers.

Measures of Depressive Symptoms and Mother and Child Diagnosis

The Kiddie – Schedule for Affective Disorders and Schizophrenia – Epidemiological Version (K-SADS-E; Orvaschel, 1995a) is a semistructured diagnostic interview that assesses current and lifetime Axis I disorders in youth based on Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition criteria (DSM-IV-TR; American Psychiatric Association, 2000). Interviewers met with mothers first followed by youth and then assigned summary symptom and diagnostic ratings based on integrated parent and child report. Given evidence of disagreement between parent and child report of symptoms and lack of current consensus on how to integrate discrepant parent and

child reports (e.g., Braaten et al., 2001; Cantwell, Lewinsohn, Rohde, & Seeley, 1997; Jensen et al., 1999), the interviewer's summary ratings were based on his/her 'best-estimate' clinical judgment from interviewing both mother and child. The K-SADS-E is a well-validated diagnostic measure that has demonstrated good inter-rater and retest reliability (Orvaschel, 1995), with κ 's of .73 and .72 for Major Depression and Dysthymia, respectively, .63-.75 for anxiety disorders, and .51-.77 for other disorders (Orvaschel, 1995). The K-SADS-E interviews were administered by Clinical Psychology postdoctoral fellows, Clinical Ph.D. students, individuals with Clinical or Counseling Masters degrees, and trained research staff with BAs. Interviewers participated in a 2-day intensive training session at Temple University led by Dr. Orvaschel, a creator of the K-SADS-E, which was videotaped and used to train interviewers recruited later in the project. In addition to this training, interviewers participated in approximately 200 hours of instruction on diagnostic assessment methods, including reading and didactic instruction, exposure to case vignettes and videotaped interviews, role-playing, and observation of live interviews by experienced interviewers. They then conducted live interviews with supervision and feedback from the diagnostic trainers prior to commencing independent interviewing. Inter-rater reliability (based on 120 pairs of ratings consisting of 5 raters for each of 24 diagnoses from 10 K-SADS interviews) in the current project was $\kappa = .85$.

Expanded Schedule for Affective Disorders and Schizophrenia – Lifetime Version (Endicott & Spitzer, 1978) The exp-SADS-L (Endicott & Spitzer, 1978) semi-structured

diagnostic interview was administered to mothers during the Time 1 second session. The exp-SADS-L assesses the occurrence, duration, and severity of symptoms related to mood, anxiety, eating, psychotic, and substance use disorders over the lifetime. This version of the original SADS-L was developed in consultation with diagnostic experts (Drs. Endicott, Akiskal, Angst, Clayton, and Gruenberg), and included a number of modifications. These included (1) probes to permit the assignment of DSM-IV diagnoses as well as RDC diagnoses; (2) improved probes in the depression, mania, (hypo)mania, and cyclothymia sections based on Depue's (1985) Behavioral Variability Interview; (3) additional items and probes in the depression, mania, (hypo)mania, and cyclothymia sections regarding frequency and duration of symptoms and episodes, and cyclothymic periods' switch rapidity (4) additional items exploring the extent to which participants' symptoms/behavior changes were noticeable to other observers; (5) an expanded 5-point symptom severity rating scale and (6) additional or appended sections on eating disorders, ADHD, acute stress disorder, medical history, family history, and organic rule-out conditions. The exp-SADS-L interview has demonstrated good inter-rater reliability for diagnoses of major depressive episodes ($k's > .95$) and all unipolar depression diagnoses ($k's > .90$) based on 80 jointly rated interviews (Alloy et al., 2000). Interviewers completed over 200 hours of diagnostic training, and interviews were audiotaped in order to obtain consensus diagnoses and to monitor inter-rater reliability. Participants were assigned a diagnosis if they met DSM and/or RDC criteria for an Axis I disorder.

The Children's Depression Inventory (CDI; Kovacs, 1992) is a self-report measure of depressive symptoms in children and adolescents (age 7-17). Participants rated 27 items, assessing affective, behavioral, and cognitive symptoms of depression on a 3-point scale (0 = "never," 1 = "sometimes," 2 = "always"). Ratings were summed, yielding total scores ranging from 0 to 54, with higher scores indicating higher depressive symptoms. The CDI has demonstrated good reliability and validity as a measure of youths' depressive symptoms (Klein, Dougherty, & Olino, 2005). Internal consistency in the sample was $\alpha = .85$.

Rumination

The Children's Response Styles Questionnaire (CRSQ; Abela, Vanderbilt, & Rochon, 2004) is a 25-item self-report questionnaire that asks participants to rate the frequency with which they experience various thoughts and feelings when they are sad on 4-point scales of never, sometimes, often, or almost always. Example items include: "When I am sad" I think 'Why can't I handle things better?'" "When I am sad, I think about other times when I have felt sad," "When I am sad, I think there must be something wrong with me or I wouldn't feel this way," and "When I am sad, I think I'm disappointing my friends, family, or teachers." Responses are summed to yield total scores representing tendencies to ruminate, distract, or problem solve, with higher scores indicating stronger tendencies. The rumination subscale used in the current study consists of 13 items indicating self-focused responses to depressed mood. Past research with the CRSQ indicated good validity and moderate internal consistency (Abela et al., 2004).

The rumination subscale demonstrated good internal consistency in the current sample ($\alpha = .87$).

Procedures

Mother-child dyads who met study inclusion and exclusion criteria were invited to Temple University to complete the Time 1 assessment. During the Time 1 assessment, the following information relevant to my dissertation was collected (along with other data not relevant here). Current and lifetime history of DSM-IV-TR Axis I psychopathology for mothers and children was assessed via semi-structured diagnostic interview.

Adolescents were assessed based on integration of parent and child report on The Kiddie-Schedule for Affective Disorders and Schizophrenia-Epidemiological Version (K-SADS-E; Orvaschel, 1995). Mothers were assessed via an expanded version of the SADS-L (Endicott & Spitzer, 1978). Child self-report was obtained for depressive symptoms using the Children's Depression Inventory (CDI; Kovacs, 1992). Child report on the Children's Response Styles Questionnaire (CRSQ; Abela et al., 2004) was used to assess rumination. Executive functions were assessed as follows. Working memory was assessed via the Digit Span subtest of the Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV; Wechsler, 2003). Components of attention, including auditory sustained attention, selective attention, attentional set shifting, and divided attention were assessed via subtests from the Test of Everyday Attention for Children (TEA-Ch; Manly et al., 2001). Demographic information and adolescents' pubertal development was obtained via parent questionnaire. Participants completed measures in two consecutive 3-

hour sessions. Initially, mothers and children completed informed consent and assent, respectively, and were subsequently reimbursed as a thank you for their time. The current study was comprised of participants and their mothers who completed both Time 1 sessions ($N = 486$). Analyses for Goal 1 were conducted in this overall sample.

Some questionnaire data and behavioral task data were missing due to omissions by participants or administration errors. Analyses used listwise deletion to take missing data into account, which assumes that data are missing completely at random (Tabachnik & Fidell, 2007). Demographic variables including race, sex, and age, and child diagnostic information were complete for all participants. Examination of missingness for each individual variable indicated that the majority of variables were missing less than 5% of the data. Less than 5% of the data were missing for income ($n = 11$), child depressive symptoms ($n = 1$), symptoms of anxiety ($n = 3$), rumination ($n = 4$), child pubertal status ($n = 1$), set shifting accuracy ($n = 2$), sustained attention ($n = 1$), selective attention ($n = 4$), and divided attention ($n = 10$). Five percent of the data were missing for working memory ($n = 25$), and 7% ($n = 36$) of the data were missing for set shifting time. The high missingness for the latter variable can be explained by the fact that the TEACH manual specifies that set shifting time cannot be calculated for those with raw set shifting accuracy scores lower than 3. Thus, 2 participants actually had missing data for this measure, and the other 34 could not be calculated due to raw accuracy scores lower than 3.

As would be expected, independent samples t-tests indicated those missing set shifting time had significantly lower set-shifting accuracy scores ($M = 3.97$) than those with complete data ($M = 10.11$), $t(482) = 12.96$, $p = .000$. In addition, those with missing set shifting time data had marginally significantly lower working memory scores ($M = 9.06$) than those with complete scores ($M = 9.94$), $t(459) = 1.66$, $p = .09$. Those missing set shifting time data did not differ from those with complete data on any other continuous variable (all $ps > .19$). Chi-square analyses indicated that those missing set shifting time data were disproportionately likely to be of lower SES, $\chi^2 = 6.07$, $p = .01$, a finding that can be explained by the fact that lower set-shifting accuracy performance, which was associated with lower SES (findings presented below) resulted in greater likelihood that switch cost scores could not be calculated and were thus missing. Those missing set shifting time data did not differ from those with complete data on any other categorical variable, including, race, sex, or child or mother lifetime history of depression (all $ps > .27$). Due to the high level of missingness, including systematic missingness, and consistent with previous research (Wilkinson & Goodyer, 2006), rather than excluding participants with low set shifting accuracy scores from analyses, analyses for hypothesis 1 included set shifting accuracy but not set shifting time as a predictor in the model.

With regard to working memory, independent samples t-tests were used to examine whether those missing working memory scores differed from those with complete working memory scores on any continuous variable. No significant differences were obtained. Chi-square analyses indicated that those missing data did not differ from

those with complete data on any categorical variable, including race, sex, SES, child or mother lifetime history of depression (all $ps > .29$). Diagnostic interviews were not completed for twenty-three (5% of) mothers. These mothers did not differ significantly from the overall study sample on any variable of interest. Chi-square analyses indicated missingness was independent of status on any categorical variable, including race, sex, SES, or child lifetime history of depression (all $ps > .34$).

CHAPTER 3

RESULTS

Descriptive Statistics

Variable means and standard deviations are presented in Table 5. Bivariate correlations are presented in Table 6. Greater age was positively correlated with more advanced pubertal status ($r(483) = .23, p = .000$). Independent samples t-tests indicated girls ($M = 14.17, SD = 3.57$) were of more advanced pubertal status than boys ($M = 10.03, SD = 3.87$), $t(483) = 12.17, p = .000, d = 1.11$). An independent samples t-test yielded no significant difference between those high and low in SES in level of pubertal development ($p = .39$).

Independent samples t-tests indicated that those high and low in SES did not differ in levels of pubertal development ($p = .39$). Chi square analyses indicated race and SES were not independent of one another, $\chi^2(2) = 89.70, p = .000$, Cramer's $V = .435$, such that African American participants were disproportionately likely to be of lower SES, and Caucasian participants were disproportionately likely to be of higher SES.

With regard to associations between demographic variables and predictor and outcome variables of interest, higher age was associated with poorer sustained attention ($r(483) = -.20, p = .000$) and working memory ($r(459) = -.13, p = .006$) scaled scores, indicating that in this sample, an association existed between older age and poorer

Table 5. *Variable Means and Standard Deviations*

	Mean	SD
Age	12.88	0.62
Pubertal Status	12.22	4.25
Rumination	24.60	7.80
Depressive Symptoms	7.19	6.04
Selective Attention	10.04	2.70
Set Shifting Accuracy	9.68	3.09
Set Shifting Time	8.45	2.60
Sustained Attention	9.50	3.07
Divided Attention	7.69	2.54

Note. Age = age. Depressive Symptoms = CDI Total Scores. Divided Attention = TEACH Dual Task Decrement Score. Pubertal Status = Maternal Report on Pubertal Development Scale. Rumination = CRSQ Rumination Scale Score. Selective Attention = TEACH Selective Attention Score. Set Shifting Accuracy = TEACH Creature Count Accuracy Score. Set Shifting Time = TEACH Creature Count Time Score. Sustained Attention = TEACH Auditory Sustained Attention Score.

Measure	1	2	3	4	5	6	7	8	9
1 Age	-								
2 Pubertal Status	0.23***	-							
3 Selective Attention	0.00	0.08†	-						
4 Sustained Attention	-0.20***	-0.04	0.10*	-					
5 Set Shifting Accuracy	0.08†	0.02	-0.06	0.14**	-				
6 Set Shifting Time	-0.05	0.00	0.20***	0.19***	0.13**	-			
7 Divided Attention	-0.06	0.01	-0.19***	0.16***	0.15***	0.10*	-		
8 Working Memory	-0.13**	0.04	0.11*	0.23**	0.16***	0.24***	0.10*	-	
9 Depressive Symptoms	0.00	0.07	0.02	-0.08†	0.03	-0.01	0.04	-0.07	-
10 CRSQ – Rumination	0.00	0.03	-0.03	0.03	0.09†	-0.02	0.09*	-0.03	0.49***

Note. Pubertal Status = Maternal Report on Pubertal Development Scale. Selective Attention = TEACH Selective Attention Score. Sustained Attention = TEACH Auditory Sustained Attention Score. Set Shifting Accuracy = TEACH Creature Count Accuracy Score. Set Shifting Time = TEACH Creature Count Set Shifting Time Score. Divided Attention = TEACH Dual Task Decrement Score. Working Memory = WISC Digit Span Total Score. Depressive Symptoms = CDI Total Score. CRSQ-Rumination = The Children's Response Styles Questionnaire Rumination Subscale. *** $p < .001$, ** $p < .01$, * $p < .05$, † $p < .10$

performance on tests of sustained attention and working memory relative to same-age (and in the case of sustained attention, same-gender) peers. In addition, age was marginally positively correlated with set shifting accuracy ($r(484) = .08, p = .07$), indicating that in this sample, older age was associated with better set shifting performance relative to same age peers. More advanced pubertal status was marginally significantly associated with better selective attention ($r(479) = .08, p = .08$).

Some gender differences were observed in performance relative to same-age same-gender peers. Girls ($M = 25.22, SD = 8.25$) had marginally higher rumination scores than boys ($M = 23.91, SD = 7.22$), $t(480) = -1.31, p = .06, d = 0.17$ and significantly higher levels of depressive symptoms ($M = 7.74, SD = 6.85$) than boys ($M = 6.58, SD = 4.94$), $t(483) = -2.14, p = .03, d = 0.20$. Girls ($M = 7.95, SD = 2.38$) also performed significantly better than boys ($M = 7.4, SD = 2.68$) on divided attention, $t(474) = -2.34, p = .02, d = 0.22$. In addition, girls ($M = 10.32, SD = 2.88$) outperformed boys ($M = 9.37, SD = 3.13$) on working memory, $t(459) = -3.41, p = .001, d = 0.32$. Girls ($M = 9.74, SD = 3.02$) likewise performed marginally significantly better than boys ($M = 9.23, SD = 3.11$) on sustained attention, $t(483) = -1.84, p = .07, d = 0.17$.

Independent samples t-tests indicated that participants of high SES did not differ from those of low SES on measures of rumination or depressive symptoms (all $ps > .36$). Adolescents of high SES ($M = 8.89, SD = 2.47$) performed significantly better than those of low SES ($M = 8.07, SD = 2.64$) on set shifting time, $t(438) = -3.36, p = .001, d = 0.32$. Likewise, those of high SES ($M = 10.07, SD = 2.88$) performed significantly better than

those of low SES ($M = 9.29$, $SD = 3.22$) on measures of set shifting accuracy, $t(471) = -2.76$, $p = .006$, $d = 0.26$. Those of high SES ($M = 9.75$, $SD = 3.05$) performed marginally significantly better than those of low SES ($M = 9.26$, $SD = 3.09$) on the test of sustained attention, $t(472) = 1.72$, $p = .09$, $d = 0.16$.

Means and standard deviations as a function of child diagnostic status are presented in Table 7, and categorical variable frequencies are presented in Table 8. Preliminary independent samples t-tests indicated that children with a current or lifetime history of unipolar depression reported significantly more depressive symptoms ($t(483) = -4.12$, $p = .000$, $d = .53$) and rumination ($t(480) = -3.07$, $p = .003$, $d = .40$) and poorer working memory ($t(459) = 2.39$, $p = .02$, $d = .32$), but groups did not differ on other continuous variables of interest (all $ps > .27$). Chi-square analyses indicated child diagnostic status was independent of race, $\chi^2(2) = 3.68$, $p = .16$, Cramer's $V = .09$, sex, $\chi^2(2) = .94$, $p = .19$, Cramer's $V = .06$ and SES, $\chi^2(2) = .06$, $p = .90$, $\phi = .03$.

Means and standard deviations as a function of mother diagnostic status are presented in Table 9 and categorical variable frequencies are presented in Table 10. Chi-square analyses yielded significant findings for child race, indicating a significantly higher proportion of Caucasian children had mothers with a lifetime history of depression relative to African American children ($\chi^2(2) = 17.04$, $p = .000$, Cramer's $V = 0.19$) and a significantly higher proportion of Caucasian children of mothers with a lifetime history of MDD relative to African American children ($\chi^2(2) = 22.40$, $p = .000$, Cramer's $V = 0.22$).

Table 7. *Variable Means and Standard Deviations by Diagnostic Status*

Variable	Depression History		No Depression History	
	Mean	SD	Mean	SD
Age	12.82	0.58	12.89	0.62
Puberty	12.02	4.70	12.25	4.18
CDI	10.43	7.37	6.66	5.63
Rumination	27.66	9.35	24.10	7.40
Selective Attention	10.24	2.39	10.00	2.74
Sustained Attention	8.91	3.34	9.59	3.01
Divided Attention	7.82	2.13	7.67	2.60
Set Shifting	9.29	2.80	9.74	3.13
Set Shifting Time	8.32	2.63	8.47	2.59
Working Memory	9.08	3.02	10.01	3.02

Note. Age = age. Puberty = PDS scores. CDI = Children's Depression Inventory Scores. Divided Attention = TEACH Dual Task Decrement Score. Rumination – CRSQ Rumination Subscale Score. Selective Attention = TEACH Selective Attention Score. Set Shifting Accuracy = TEACH Creature Count Accuracy Score. Set Shifting Time = TEACH Creature Count Time Score. Sustained Attention = TEACH Auditory Sustained Attention Score. Working Memory = WISC Digit Span Total Score.

Table 8. *Categorical Variable Frequencies by Diagnostic Status*

Variable	Depression History		No Depression History	
	N	%	N	%
Race				
White	40	58.8	197	47.1
African American	26	38.2	203	48.6
Biracial, Multiracial, or Other	2	02.9	17	04.1
Gender				
Male	38	55.9	192	45.9
Female	30	44.1	226	54.1
SES				
0-14,999	7	10.3	64	15.3
15-29,000	9	13.2	64	15.3
30-44,999	15	22.1	65	15.6
45-59,000	11	16.2	67	16.0
60-74,000	6	08.8	47	11.2
75-89,999	6	08.8	28	06.7
90,000 and over	13	19.1	73	17.5
No income information provided	1	01.5	10	02.4

Table 9. Means and Standard Deviations by Maternal Diagnostic Status

Variable	Depression History		No Depression History	
	Mean	SD	Mean	SD
Age	12.91	0.61	12.85	0.61
Puberty	12.33	4.18	12.18	4.33
CDI	7.55	6.68	7.05	5.67
Rumination	24.36	7.78	24.79	7.82
Selective Attention	10.09	2.53	10.00	2.85
Sustained Attention	9.64	2.95	9.49	3.13
Divided Attention	7.70	2.39	7.70	2.65
Set Shifting	9.82	3.11	9.51	3.11
Set Shifting Time	8.39	2.81	8.49	2.47
Working Memory	10.08	3.21	9.65	2.88

Note. Age = age. Puberty = PDS scores. CDI = Children's Depression Inventory Scores. Divided Attention = TEACH Dual Task Decrement Score. Selective Attention = TEACH Selective Attention Score. Set Shifting Accuracy = TEACH Creature Count Accuracy Score.. Set Shifting Time = TEACH Creature Count Time Score. Sustained Attention = TEACH Auditory Sustained Attention Score. Working Memory = WISC Digit Span Total Score.

Table 10. *Categorical Variable Frequencies by Maternal Diagnostic Status*

Variable	Depression History		No Depression History	
	N	%	N	%
Child Depression				
No Lifetime History	150	79.8	228	90.8
Lifetime History	38	20.2	23	9.2
Race				
White	112	59.6	99	39.4
African American	70	37.2	141	56.2
Biracial, Multiracial, or Other	6	3.2	11	4.4
Gender				
Male	87	46.3	121	48.2
Female	101	53.7	130	51.8
SES				
Low	94	52.0	138	54.8
High	93	47.5	105	42.1
Unavailable	1	0.50	8	3.2
Income				
0-14,999	20	10.6	40	15.9
15-29,000	28	14.9	32	12.7
30-44,999	30	16.0	49	19.5
45-59,000	40	21.3	34	13.5
60-74,000	24	12.8	24	9.6
75-89,999	5	2.7	23	9.2
90,000 and over	40	21.3	41	16.3

A higher than expected proportion of children with mothers with a lifetime history of depression also met criteria for a lifetime history of depression themselves ($\chi^2(1) = 13.45, p = .000, \phi = .17$), as did a higher than expected proportion of children with mothers with a lifetime history of MDD ($\chi^2(1) = 10.45, p = .002, \phi = .15$). A higher than expected proportion of children with mothers with a lifetime history of MDD also met criteria for the presence of an additional Axis I disorder ($\chi^2(1) = 4.03, p = .05, \phi = .09$), whereas those with a lifetime history of any episode of depression did not ($\chi^2(1) = 2.31, p = .15, \phi = .07$). All other categorical variables appeared to be independent of maternal diagnostic status (all $ps > .11$).

Independent samples t-tests indicated no significant differences between groups with and without a maternal lifetime history of depression on any continuous variable (puberty, CDI, rumination, or executive functions measures; all $ps > .14$).

Bivariate correlations between measures of EF, CDI scores, and rumination are presented in Table 6. Greater rumination was positively associated with higher levels of depressive symptoms ($r(485) = .49, p = .00$). CDI scores were marginally negatively associated with sustained attention, such that higher levels of depressive symptoms were marginally associated with poorer sustained attention ($r(484) = -.08, p = .07$). CDI scores were not significantly correlated with other measures of EF (all $ps > .37$).

Rumination was significantly positively associated with divided attention ($r(472) = .09, p = .04$), such that greater rumination was associated with better divided attention performance. Rumination was marginally significantly positively correlated with set

shifting accuracy, such that higher levels of rumination were correlated with better set shifting accuracy performance ($r(480) = .09, p = .06$). Rumination was not significantly correlated with other measures of EF (all $ps > .48$).

Primary Analyses

A hierarchical linear regression was used to examine whether measures of executive function (selective attention, sustained attention, attentional set shifting accuracy, divided attention, and working memory) predicted rumination (hypothesis 1).

Control variables, including gender, age, race (dummy coded), SES (dummy coded), pubertal development, and current depressive symptoms were entered in Step 1 and measures of executive function were entered as simultaneous predictors in Step 2. Nonsignificant control variables were removed from the model. Only depressive symptoms were significant predictors in Step 1 and were thus retained in the model. Results are presented in Tables 11 and 12.

Prior to analyses, data were examined to ensure that assumptions of multiple regression were met. The ratio of independent variables ($k = 6$) to cases ($N = 445$) was adequate to detect even a small effect ($f^2 = .02$). Residuals were normally distributed, and an examination of standardized residuals indicated an absence of outliers in the solution, with all standardized residuals less than ± 3.29 .

Examination of the scatterplot of predicted values of rumination against residuals indicated possible heteroscedasticity, a problem which may weaken the model, although

it does not invalidate it (Tabachnik & Fidell, 2007). Collinearity diagnostics were satisfactory. Pearson correlation coefficients did not suggest that problems with multicollinearity were likely (for predictors, all r s < .24), all Variance Inflation Factor (VIF) statistics were close to 1, and all tolerance values were greater than .2. Five multivariate outliers were identified as potentially exerting undue influence on the model, based on the criterion for Mahalanobis distance at $p < .001$ ($\chi^2_{\text{critical value}} = 22.458$), as well as problematically high leverage values (leverage values > $3(k+1)$; Stevens, 1992). Analyses were rerun after removing multivariate outliers, and model parameters did not change substantially. Results from the full dataset are presented in Table 11. Results are presented in Table 12 with multivariate outliers removed. The results of the final model, with CDI scores entered in Step 1 and measures of executive function entered in Step 2, indicate that all predictors explained 23.3% of the variance (Adjusted $R^2 = .233$, $F(6,438) = 23.46$, $p = .000$, $f^2 = 0.321$) in rumination.

The same model was re-run, excluding the five multivariate outliers identified in data screening. Results are presented in Table 12 and are similar to results without outliers excluded (although findings are somewhat weakened). The results of the final model indicate that all predictors explained 23.2% of the variance (Adjusted $R^2 = .232$, $F(6,438) = 23.46$, $p = .000$, $f^2 = 0.321$). After controlling for depressive symptoms, which again, significantly positively predicted rumination ($\beta = .48$, $p = .000$), better sustained attention marginally positively predicted greater rumination ($\beta = .08$, $p = .07$). However, this no longer resulted in a marginally significant increase in the predictive power of the

Table 11. *Executive Functions Predicting to Rumination*

		ΔR^2	B	SE	B	f^2
Predictor						
Step 1		.227***				
	CDI		.63	.06	.48***	0.294
Step 2		.016†				
	CDI		.63	.06	.48***	0.021
	Selective Attn		-.07	.13	-.03	
	Sustained Attn		.20	.11	.08†	
	Divided Attn		.18	.14	.06	
	Set Shifting		.14	.11	.06	
	Working Memory		-.04	.11	-.02	
Total R ²		.243***				0.321
Adjusted R ²		.233***				
N		445				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score.

Sustained Attn = TEACH Auditory Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement

Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC Digit Span Total Score.

† $p < .10$, * $p < .05$, ** $p < .02$, *** $p < .001$.

Table 12. *Executive Functions Predicting to Rumination with Outliers Excluded*

		ΔR^2	B	SE	B	f^2
Predictor						
Step 1		.228***				
	CDI		.67	.06	.48***	0.295
Step 2		.014				
	CDI		.67	.06	.48***	0.020
	Selective Attn		-.06	.13	-.02	
	Sustained Attn		.20	.11	.08†	
	Divided Attn		.14	.14	.04	
	Set Shifting		.14	.11	-.06	
	Working Memory		-.01	.11	.00	
Total R ²		.243***				0.321
Adjusted R ²		.232***				
N		440				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score

Sustained Attn = TEACH Auditory Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement

Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC Digit Span Total.

model, R^2 change = .014, $F(5,433) = 1.63$, $p = .15$, $f^2 = 0.020$. No other predictors were significant (all $ps > .19$).

When the analysis was rerun with all nonsignificant EF variables removed from the model (Table 13), better sustained attention continued to marginally positively predict greater rumination ($\beta = .07$, $p = .09$), resulting in a marginally significant increase in predictive power of the model, R^2 change = .005, $F(1,478) = 2.89$, $p = .09$, $f^2 = 0.007$. Findings remained unchanged after removing three multivariate outliers (See Table 13). Because set shifting time missingness was systematically related to other measures of executive functions, set shifting time was excluded from primary analyses. However, to explore the relation between set shifting time and rumination for those with complete data, a separate regression was run with control variables entered as predictors in Step 1 and set shifting time in Step 2. Removal of 2 multivariate outliers did not change findings. No significant association was found between set shifting time and rumination ($\beta = -.02$, $p = .63$). The results of the final model indicate that all predictors explained 23.8% of the variance (Adjusted $R^2 = .238$, $F(2,433) = 69.23$, $p = .000$, $f^2 = 0.312$). After controlling for depressive symptoms, which again, significantly predicted rumination ($\beta = .48$, $p = .000$), set shifting time was not significantly associated with rumination ($\beta = -.02$, $p = .63$), and its addition in Step 2 did not increase the predictive power of the model, R^2 change = .000, $F(1,443) = .23$, $p = .63$, $f^2 = .000$.

Table 13. *Sustained Attention-Rumination Associations with (left) and without (right) Multivariate Outliers*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Predictor	.238***				0.312	Predictor	.237***				0.311
Step 1						Step 1					
CDI		.63	.05	.49***		CDI		.68	.06	.49***	
Step 2	.005†				0.007	Step 2	.005†				0.007
CDI		.64	.05	.49***		CDI		.68	.06	.49***	
Sustained Attn		0.17	0.1	.07†		Sustained Attn		.18	.10	.07†	
Total R ²	.243***				0.321	Total R ²	.242***				0.319
Adjusted R ²	.240***					Adjusted R ²	.239***				
N	481					N	478				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Auditory Sustained Attention Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

In summary, no support was obtained for hypothesis 1. Executive function deficits were not significantly associated with rumination. Moreover, marginally significant associations between the sustained attention measure of executive function and rumination were in the opposite direction to that hypothesized, with better task performance associated with greater rumination.

Secondary Analyses

Hypothesis 2

To test hypothesis 2, a hierarchical logistic regression was used to predict adolescent diagnostic status (presence versus absence of a lifetime history of unipolar depression). Control variables (gender, age, race, SES, pubertal development, and current depressive symptoms) were entered in Step 1, and measures of executive function (selective attention, sustained attention, attentional set shifting accuracy, divided attention, and working memory) were entered in Step 2. Nonsignificant control variables were removed from the model.

Diagnostics indicated all assumptions were met for logistic regression. Dichotomous predictor variables were distributed across levels of the outcome variable; with regard to expected cell frequencies, all were greater than one and fewer than 20% were less than five (Tabachnik & Fidell, 2007). The assumption of independence of errors was met, and the relationship between continuous predictors and the logit transformation of the DV was linear (Tabachnik & Fidell, 2007). Collinearity diagnostics

yielded no evidence of multicollinearity. All VIF statistics were close to 1.0, all tolerance statistics were greater than .95, and condition indices were less than 25; in no case did two eigenvalues load onto a single dimension with variance proportions of .50 or greater (Field, 2005). The Hosmer Lemeshow's (H-L) goodness of fit test statistic was nonsignificant, indicating the model was an adequate fit to the data. Examination of residuals indicated no evidence of outliers in the solution (all Cooks values and Dfbetas less than 1; Field, 2005).

Results are presented in Table 14. In Step 1, higher CDI scores (Wald = 21.16 $p = .000$, OR = 1.10, 95% CI = 1.06-1.15) were significantly associated with greater likelihood of a lifetime history of unipolar depression diagnosis, and female child gender (Wald = 3.99, $p = .05$, OR = .56, 95% CI = .31-.99) was significantly associated with a lower likelihood of a lifetime history of unipolar depression diagnosis. Identifying as African American was marginally significantly associated with lower likelihood of a lifetime history of unipolar depression diagnosis (Wald = 3.21, $p = .07$, OR = .59, 95% CI = .34-1.05). Entry of these predictors in Block 1 resulted in a significant model, χ^2 (4, N = 448) = 26.15, $p < .001$. The addition of executive functions in Step 2 was nonsignificant, χ^2 (5, N = 448) = 6.18, $p = .29$. Selective attention (Wald = .17, $p = .68$, OR = 1.02, 95% CI = .92-1.14), sustained attention (Wald = .92, $p = .34$, OR = .95, 95% CI = .87-1.05), set shifting accuracy (Wald = 1.23, $p = .27$, OR = .95, 95% CI = .86-1.04),

Table 14. *Executive Functions Predicting Lifetime History of Depression in Adolescents Controlling for CDI*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	26.15***	4				
CDI		1	.10(.02)	21.16	1.10***	1.06-1.15
Gender		1	-.57(.29)	3.99	0.56*	0.32-0.99
Race (1)		1	-.52(.29)	3.21	0.59†	0.34-1.05
Race(2)		1	-.74(.79)	0.87	0.48	0.10-2.25
Step 2	6.18	5				
CDI		1	.10 (.02)	19.26	1.10***	1.05-1.15
Gender		1	-.51(.30)	2.88	0.60†	0.34-1.08
Race (1)		1	-.66(.30)	4.72	0.52*	0.29-0.94
Race(2)		1	-.90(.81)	1.21	0.41	0.08-2.01
Selective Attn		1	.02(.05)	0.17	1.02	0.92-1.14
Sustained Attn		1	-.05(.05)	0.92	0.95	0.87-1.05
Divided Attn		1	.06(.06)	0.91	1.06	0.94-1.19
Set Shifting		1	-.05(.05)	1.23	0.95	0.86-1.04
Working Memory		1	-.08(.05)	2.20	0.93	0.84-1.02
Overall χ^2	32.33***	9				
<i>N</i>		448				

Note. CDI = Children's Depression Inventory Scores. Race (1) = African American (yes/no). Race (2) = Biracial/Multiracial (yes/no). Selective Attn = TEACH Selective Attention Score. Sustained Attn: TEACH Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement Score.

Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

divided attention (Wald = .91, $p = .34$, OR = 1.06, 95% CI = .94-1.19), and working memory (Wald = 2.20, $p = .14$, OR = .93, CI = .84-1.02) were not significantly associated with adolescent lifetime history of depression. A separate hierarchical logistic regression was run to examine whether set shifting time predicts diagnostic status (presence versus absence of a lifetime history of unipolar depression). Control variables (gender, age, race, SES, pubertal development, and current depressive symptoms) were entered in Step 1, set shifting time was entered in Step 2. Nonsignificant control variables were removed from the model. Results are presented in Table 15. Set shifting time did not significantly predict adolescent lifetime history of depression (Wald = .15, $p = .70$, OR = .98, 95% CI = .88-1.09)¹.

Given that previous studies have not controlled for concurrent CDI scores when examining associations between EF and depression diagnosis, and that this is a somewhat stringent approach, designed to isolate trait-like characteristics of the disorder present in complete remission rather than state-dependent correlates, we examined whether EF predicted diagnostic status without controlling for CDI scores. Again, a hierarchical logistic regression was used to predict diagnostic status (presence versus absence of a lifetime history of unipolar depression). Control variables (gender, age, race, SES, and pubertal development) were entered in Step 1, and measures of executive function

¹ Given comorbidity in the sample, analyses were rerun controlling for Axis I diagnosis other than unipolar depression. Control variables, including presence of Axis I disorder were entered in Step 1, and measures of EF were entered simultaneously in Step 2. Results were unchanged and are presented in Tables A8-A9.

Table 15. *Set Shifting Time Predicting Lifetime History of Depression in Adolescents Controlling for CDI*

	χ^2	df	B(SE)	Wald	OR	95% CI
Predictor						
Step 1	27.81***	4				
CDI		1	.10(.02)	20.69	1.10***	1.06-1.15
Gender		1	-.72(.29)	6.29	0.49*	.28-.85
Race (1)		1	-.58(.29)	3.87	0.56*	.32-1.00
Race(2)		1	-.76(.79)	0.92	0.47	.10-2.20
Step 2	0.15	5				
CDI		1	.10 (.02)	20.43	1.10***	1.06-1.15
Gender		1	-.72(.29)	6.28	0.49*	.28-.86
Race (1)		1	-.59(.30)	3.99	0.56*	.31-.99
Race(2)		1	-.75(.79)	0.90	0.47	.10-2.23
Set Shifting Time		1	-.02(.06)	0.15	0.98	.88-1.09
Overall χ^2	27.96***	9				
N		448				

Note. CDI = Children's Depression Inventory Scores. Race (1) = African American (yes/no). Race(2) = Biracial or Multiracial (yes/no). Set Shifting Time = TEACH Creature CountTime Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

(selective attention, sustained attention, attentional set shifting accuracy, divided attention, and working memory) were entered in Step 2. A secondary logistic regression was used to examine set shifting time as a predictor of diagnostic status (presence versus absence of a lifetime history of unipolar depression). Control variables (gender, age, race, SES, and pubertal development) were entered in Step 1, and set shifting time was entered in Step 2. Nonsignificant control variables were again removed from the model. Results are presented in Tables 16 and 17. In both cases, results remained unchanged. Measures of EF remained nonsignificant predictors of diagnostic status. In summary, no evidence was obtained for the hypothesized association between impaired executive functions and lifetime history of unipolar depression in adolescents (hypothesis 2).

Hypothesis 3

To test hypothesis 3, a hierarchical logistic regression was used to predict maternal diagnostic status (presence versus absence of a lifetime history of unipolar depression). Control variables, including child gender, child diagnosis, and child current depressive symptoms, child pubertal status, race, and SES were entered in Step 1, and measures of executive function were entered in Step 2. Prior to analyses, data were examined to ensure that assumptions of logistic regression were met. All assumptions were met for logistic regression. Dichotomous predictor variables were distributed across groups, expected cell frequencies were all greater than one and no more than 20% were less than five (Tabachnik & Fidell, 2007), the assumption of independence of errors was met, and

Table 16. *Executive Functions Predicting Lifetime History of Depression in Adolescents*

	χ^2	Df	B(SE)	Wald	OR	95% CI
Predictor						
Step 1	4.96	3				
Gender		1	-.38(.27)	1.91	0.69	0.40-1.17
Race (1)		1	-.48(.28)	2.90	0.62†	0.36-1.08
Race (2)		1	-.60(.77)	0.60	0.55	0.12-2.49
Step 2	7.78	5				
Gender		1	-.30(.28)	1.11	0.74	0.43-1.29
Race (1)		1	-.60(.29)	4.25	0.55*	0.31-0.97
Race (2)		1	-.70(.78)	0.81	0.50	0.11-2.29
Selective Attn		1	.03(.05)	0.25	1.03	0.93-1.14
Sustained Attn		1	-.07(.05)	2.28	0.93	0.85-1.02
Divided Attn		1	.06(.06)	1.03	1.06	0.95-1.18
Set Shifting		1	-.04(.05)	0.59	0.97	0.88-1.06
Working Memory		1	-.08(.05)	2.56	0.92	0.84-1.02
Overall χ^2	12.74	8				
N	448					

Note. CDI = Children's Depression Inventory Scores. Race (1) = African American (yes/no); Race (2) = Biracial/Multiracial (yes/no). Selective Attn = TEACH Selective Attention Score. Sustained Attention = TEACH Auditory Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 17. *Set Shifting Time Predicting Lifetime History of Depression in Adolescents*

		χ^2	df	B(SE)	Wald	OR	95% CI
	Predictor						
Step 1		7.35†	3				
	Gender			-.54(.27)	3.93	0.58*	0.34-0.99
	Race (1)			-.50(.28)	3.17	0.60†	0.35-1.05
	Race (2)			-.62(.77)	0.65	0.54	0.12-2.43
Step 2		0.31	1				
	Gender			-.55(.27)	4.00	0.58*	0.34-0.99
	Race (1)			-.52(.29)	3.36	0.59†	0.34-1.04
	Race (2)			-.61(.77)	0.62	0.55	0.12-2.47
	Set Shifting Time			-.03(.05)	0.31	0.91	0.88-1.08
	Overall χ^2	7.66	4				
<i>N</i>		448					

Note. Race (1) = African American (yes/no); Race (2) = Biracial/Multiracial (yes/no); Set Shifting Time = TEACH Creature Count Time score

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

the relationship between continuous predictors and the logit transform of the DV was linear (linearity of the logit; Tabachnik & Fidell, 2007). Collinearity diagnostics yielded no evidence of multicollinearity. All VIF statistics were close to 1.0, all tolerance statistics were greater than .95, and condition indices were less than 25, and in no case did two eigenvalues load onto a single dimension with variance proportions of .50 or greater (Field, 2005). The Hosmer Lemeshow's (H-L) goodness of fit test statistic was nonsignificant, indicating the model was an adequate fit to the data. Examination of residuals indicated no evidence of outliers in the solution (no residuals greater than 1.96, all Cooks values and Dfbetas less than 1; Field, 2005). Results are presented in Table 18.

In Step 1, presence of a child diagnosis was significantly associated with greater likelihood of maternal lifetime history of depression (Wald = 9.78, $p = .002$, OR = 2.55, 95% CI = 1.42-4.58). Identifying as African American (Wald = 14.14, $p = .000$, OR = 0.46, 95% CI = 0.30-0.69) was significantly associated with lower likelihood of maternal lifetime history of depression, $\chi^2(3, N = 414) = 26.06$, $p = .000$. The addition of executive functions in Step 2 did not significantly increase the predictive power of the model, $\chi^2(5, N = 414) = 1.73$, $p = .885$. Selective attention (Wald = .08, $p = .78$, OR = .99, 95% CI = .92-1.07), sustained attention (Wald = .00, $p = .97$, OR = 1.00, 95% CI = .93-1.07), set shifting accuracy (Wald = .78, $p = .38$, OR = 1.03, 95% CI = .96-1.10), divided attention (Wald = .07, $p = .79$, OR = .99, 95% CI = .91-1.08), and working memory (Wald = .64, $p = .43$, OR = 1.03, CI = .96-1.11) were not significantly associated with maternal lifetime history of depression. A follow-up hierarchical logistic regression (Table 19) was run to examine whether set shifting predicted maternal diagnostic status

Table 18. *Offspring Executive Functions Predicting to Lifetime History of Depression in Mothers*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	26.06***	3				
Child Diagnosis		1	.94(.30)	9.78	2.55**	1.42-4.58
Race (1)		1	-.79(.21)	14.14	0.46***	0.30-0.69
Race (2)		1	-.56(.54)	1.07	0.57	0.20-1.65
Step 2	1.73	5				
Child Diagnosis		1	.97(.30)	10.26	2.63**	1.46-4.76
Race (1)		1	-.75(.21)	12.20	0.47***	0.31-0.72
Race (2)		1	-.54(.54)	0.98	0.58	0.20-1.69
Selective Attn		1	-.01(.04)	0.08	0.99	0.92-1.07
Sustained Attn		1	.00(.04)	0.00	1.00	0.93-1.07
Divided Attn		1	-.01(.04)	0.07	0.99	0.91-1.08
Set Shifting		1	.03(.04)	0.78	1.03	0.96-1.10
Working Memory		1	.03(.04)	0.64	1.03	0.96-1.11
Overall χ^2	27.79**	8				
N	414					

Note. CDI = Children's Depression Inventory Scores. Race (1) = African American (yes/no). Race (2) = Biracial/Multiracial (yes/no). Selective Attn = TEACH Selective Attention. Divided Attn = TEACH Dual Task Decrement. Sustained Attention = TEACH Sustained Attention. Set Shifting = TEACH Creature Count Accuracy. Working Memory = WISC. Digit Span Total Score. . † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 19. *Offspring Set Shifting Time Predicting to Lifetime History of Depression in Mothers*

	χ^2	Df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	24.45***	3				
Child Diagnosis		1	.92(.30)	9.39	2.52**	1.39-4.54
Race (1)		1	-.78(.21)	13.33	0.46***	0.30-0.70
Race (2)		1	-.53(.54)	0.96	0.59	0.21-1.70
Step 2	0.12	1				
Child Diagnosis		1	.92(.30)	9.33	2.51**	1.39-4.52
Race (1)		1	-.79(.21)	13.43	0.46***	0.30-0.69
Race (2)		1	-.36(.49)	0.54	0.70	0.21-1.71
Set Shifting Time		1	-.01(.04)	0.12	0.99	0.91-1.07
Overall χ^2	24.57***	4				
<i>N</i>		403				

Note. CDI = Children's Depression Inventory Scores. Race(1) = African American (yes/no). Race (2) = Biracial/Multiracial (yes/no).

Set Shifting Time = TEACH Creature Count Time Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

(presence versus absence of a lifetime history of unipolar depression). Control variables (child gender, child age, race, SES, pubertal development, child diagnosis, and child current depressive symptoms) were entered in Step 1, and set shifting time was entered in Step 2. Nonsignificant control variables were removed from the model. Results are presented in Table 19. Set shifting time did not significantly predict mothers' lifetime history of depression (Wald = .12, $p = .72$, OR = .99, 95% CI = .91-1.07).^{2,3}

Exploratory Analyses

Moderation by Gender

Separate hierarchical linear regressions were conducted to examine whether gender interacted with measures of EF to predict rumination. Control variables and depressive symptoms were entered in Step 1, a single measure of executive function (mean centered) and gender was entered in Step 2, and the interaction term (computed using gender and the mean centered measure of executive function) was entered in Step 3. A Bonferroni correction was used to control for familywise error (criterion alpha = .008). Results are presented in Tables 20-22.

² Analyses were rerun with only biological mothers included. Analyses were likewise rerun predicting to presence/absence of lifetime history of MDD (DSM or RDC major depressive disorder only) in the entire sample and among biological mothers and their offspring only. Results are presented in further detail in the appendix (Tables A10-A15). Findings remained unchanged.

³ Given that presence of an Axis I disorder in offspring was significantly associated with presence of lifetime history of MDD in mothers, all analyses for hypothesis 3 predicting to maternal lifetime of MDD were likewise run controlling for presence of an Axis I disorder other than unipolar depression (Tables A16-A17). Control variables including child depression diagnosis and presence of an Axis I disorder were entered in Step 1 and measures of EF were entered in Step 2. Results were also unchanged predicting to history of unipolar depression in general and using only biological mothers and their offspring.

Examining gender as a moderator of associations between executive functions and rumination yielded no significant findings for selective attention ($\beta = .04, p = .46$), sustained attention ($\beta = .00, p = .97$), set shifting time ($\beta = .01, p = .63$), divided attention ($\beta = .02, p = .77$), or working memory ($\beta = -.01, p = .80$). In these cases, depressive symptoms significantly predicted rumination, resulting in a statistically significant model, but measures of executive function did not result in a significant increase in the predictive power of the model. Significant findings (presented in Table 21) were obtained with regard to set shifting accuracy. Specifically, after entering depressive symptoms in Step 1, set shifting accuracy (mean centered) and gender in Step 2, and the interaction term in Step 3, a marginally significant main effect for set shifting accuracy was obtained in Step 2 ($\beta = .07, p = .08$), but this did not result in a significant increase in the predictive power of the model, $F(2, 476) = 1.92, p = .15, f^2 = .008$. The addition of the interaction term in Step 3 resulted in a statistically significant increase in the predictive power of the model, $R^2 \text{ change} = .016, F(1, 475) = 10.47, p = .001, f^2 = .023$. In this step, the significant main effect of set shifting accuracy ($\beta = .20, p = .000$) was qualified by a higher order interaction, whereby gender significantly moderated the association between set shifting accuracy and rumination ($\beta = -.18, p = .001$). The results of the final model indicate that all predictors explained 25.6% of the variance ($R^2 = .256, F(4, 475) = 40.83, p = .000, f^2 = .344$) in rumination.

Table 20. *Gender Moderating EF-Rumination Associations: Selective and Sustained Attention*

Table 20: Gender Moderating Effects on Rumination Associations: Selective and Sustained Attention											
	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Predictor						Predictor					
Step 1	.234**				.306	Step 1	.238**				.312
CDI		.63	.05	.48**		CDI		.63	.05	.49**	
Step 2	.004				.005	Step 2	.005				.008
CDI		.63	.05	.48**		CDI		.64	.05	.49**	
Gender		.67	.63	.04		Gender		0.46	0.63	0.03	
Selective Attn (MC)		-0.13	0.12	-0.04		Sustained Attn (MC)		.17	.10	.07	
Step 3	.001				.001	Step 3	.000				.000
CDI		.63	.05	.48**		CDI		.64	.05	.49**	
Gender		.67	.63	.04		Gender		.46	.63	.03	
Selective Attn (MC)		-.21	.16	-.07		Sustained Attn (MC)		.17	.15	.07	
Gender X Selective Attn		.17	.23	.04		Gender X Sustained Attn		-.01	.20	.00	
Total R ²	.239**				.314	Total R ²	.244**				.323
Adjusted R ²	.232**					Adjusted R ²	.237**				
N	479					N	481				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Auditory Sustained Attention Score.

†† $p < .05$, † $p < .01$, * $p < .008$, ** $p < .001$.

Table 21. *Gender Moderating EF-Rumination Associations: Set Shifting Time and Accuracy*

		ΔR^2	B	SE	β	f^2			ΔR^2	B	SE	β	f^2
<u>Predictor</u>							<u>Predictor</u>						
Step 1		.233**				0.304	Step 1		.238**				0.312
	CDI		0.66	.052	0.48***			CDI		.64	.05	.49**	
Step 2		.006				0.008	Step 2		.001				0.000
	CDI		0.62	0.05	0.48***			CDI		.64	.05	.49**	
	Gender		0.46	0.63	0.03			Gender		.29	.65	.02	
	Set Shifting (MC)		0.18	0.10	0.07†			Set Shifting Time (MC)		-.06	.13	-.02	
Step 3		.016**				0.023	Step 3		.000				0.001
	CDI		0.62	0.05	0.48**			CDI		.64	.06	.49**	
	Gender		0.43	0.62	0.03			Gender		.29	.65	.02	
	Set Shifting (MC)		0.49	0.14	0.20**			Set Shifting Time (MC)		-.09	.18	-.03	
	Gender X Set Shifting		-0.64	0.20	- 0.18*			Gender X Set Shifting Time		.06	.25	.01	
Total R ²		.256**				.344	Total R ²		.239**				0.314
Adjusted R ²		.250**					Adjusted R ²		.232**				
N		480					N		446				

Note . CDI = Children's Depression Inventory Scores. MC = Mean Centered. Set Shifting = TEACH Set Shifting Accuracy Score. Set Shifting Time = TEACH Set Shifting Timing Score.

†† $p < .05$, † $p < .01$, * $p < .008$, ** $p < .001$.

Table 22. Gender Moderating EF-Rumination Associations: Working Memory and Divided Attention

Predictor		ΔR^2	B	SE	β	f^2	Predictor		ΔR^2	B	SE	β	f^2
Step 1		.235**				0.307	Step 1		.241**				0.318
	CDI		0.64	0.05	0.49**			CDI		.640	.050	0.49**	
Step 2		.001				0.003	Step 2		.006				0.008
	CDI		0.64	0.06	0.48**			CDI		0.63	0.05	0.49**	
	Gender		0.58	0.66	0.04			Gender		0.51	0.63	0.03	
	Working Memory (MC)		-0.02	0.11	-0.01			Divided Attn(MC)		0.22	0.12	0.07	
Step 3		0.000				0.000	Step 3		0.000				0.001
	CDI		0.64	0.06	0.48**			CDI		0.63	0.05	0.49**	
	Gender		0.58	0.66	0.04			Gender		0.51	0.64	0.03	
	Working Memory (MC)		.010	0.15	0.00			Divided Attn(MC)		0.18	0.17	0.06	
	Gender X Working Memory		-0.05	0.22	-0.01			Gender X Divided Attn		0.07	0.25	0.02	
Total R ²		.237**				0.311	Total R ²		.248**				0.330
Adjusted R ²		.230**					Adjusted R ²		.241**				
N		457					N		472				

Note . CDI = Children's Depression Inventory Scores. Divided Attn = TEACH Dual Task Decrement Score. Working Memory = WISC Digit Span Total Score.

†† $p < .05$, † $p < .01$, * $p < .008$, ** $p < .001$.

To elucidate the nature of the interaction, separate multiple regressions were run for males only and females only, predicting to rumination, with depressive symptoms entered in Step 1 and set shifting accuracy in Step 2.

Results are presented in Table 23. Among females, set shifting accuracy was not significantly associated with rumination ($\beta = -.06, p = .29$). Although predictors explained 31.1% of the variance ($R^2 = .311, F(2,250) = 56.44, p = .000, f^2 = .451$) in rumination among females, this was attributable to depressive symptoms predicting rumination ($\beta = .56, p = .000$), while the addition of set shifting accuracy did not significantly increase the predictive power of the model, R^2 change = .003, $F(1, 250) = 1.10, p = .29, f^2 = .004$.⁴

A different pattern of findings was obtained among males. CDI was entered in Step 1 and significantly predicted rumination ($\beta = .35, p = .000$). The addition of set shifting accuracy in Step 2 was significant ($\beta = .22, p = .000$), such that better set shifting accuracy was associated with greater rumination, resulting in a significant increase in the predictive power of the model, R^2 change = .05, $F(1, 224) = 13.48, p = .000, f^2 = .059$. Predictors explained 17.3% of the variance ($R^2 = .173, F(2,224) = 23.48, p = .000, f^2 = .209$) in rumination among males.⁵

⁴ Removing CDI scores from Step 1 did not result in any increase in significance of prediction for set shifting accuracy for females ($\beta = -.05, p = .40$), indicating it is not simply controlling for CDI scores that resulted in nonsignificant findings.

⁵ Follow-up analyses explored whether gender moderated EF-diagnosis associations. A Bonferroni correction was used to correct for familywise error (alpha criterion = .008). No interaction terms were significant (all $ps > .06$).

Table 23. *Set Shifting Predicting Rumination in Males (Left) versus Females (Right)*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
	<u>Predictor</u>						<u>Predictor</u>				
Step 1	.124**				0.142	Step 1	.308**				.445
		CDI	0.53	0.09	0.35**			CDI	0.67	0.06	0.56**
Step 2	.050**				0.059	Step 2	.003				0.004
		CDI	0.51	0.09	0.34**			CDI	0.67	0.06	0.56**
		Set Shifting	0.50	0.14	0.22**			Set Shifting	-0.15	0.14	-0.06
Total R ²	.173**				0.209	Total R ²	.311**				0.451
Adjusted R ²	.166**					Adjusted R ²	.306**				
N	227					N	253				

Note. CDI = Children's Depression Inventory Scores. Set Shifting = TEACH Set Shifting Accuracy Score.

†† $p < .05$, † $p < .01$, * $p < .008$, ** $p < .001$.

Moderation by Diagnosis

Separate hierarchical linear regressions were conducted to examine whether child diagnostic status interacted with each measure of executive function to predict rumination. Age, SES, race, and pubertal development were entered in Step 1, a single measure of executive function (mean centered) and diagnosis in Step 2, and the executive function X diagnosis interaction term in Step 3. Nonsignificant control variables were removed from the model. A Bonferroni correction was used for familywise error (alpha correction = .008). Results are presented in Tables 24-26.

Diagnosis did not significantly moderate EF-rumination associations. Diagnostic status did not interact with sustained attention ($\beta = .03, p = .53$), selective attention ($\beta = -.02, p = .65$), divided attention ($\beta = .00, p = .93$), set shifting time ($\beta = .03, p = .56$), set shifting accuracy ($\beta = .00, p = .96$), or working memory ($\beta = -.06, p = .21$) to predict rumination.

Of note, several multivariate outliers were identified in the original regression model. Outliers were removed and analyses were re-run with outliers removed and findings continued to be nonsignificant. Results are therefore reported for the complete dataset.

Table 24. *Diagnosis Moderating EF-Rumination Associations: Selective and Sustained Attention*

		ΔR^2	B	SE	β	f^2			ΔR^2	B	SE	β	f^2
	Predictor							Predictor					
Step 1		.234				0.306	Step 1		.238**				0.312
	CDI	0.63	0.05		0.48**			CDI	0.63	0.05		0.49**	
Step 2		.004				0.005	Step 2		.008				0.011
	CDI	0.63	0.05		0.47**			CDI	0.62	0.05		0.48**	
	Diagnosis	1.13	0.91		0.05			Diagnosis	1.32	0.91		0.06	
	Selective Attn (MC)	-0.17	0.37		-0.02			Sustained Attn (MC)	0.18	0.10		0.07	
Step 3		.000				0.001	Step 3		.001				0.001
	CDI	0.62	0.05		0.47**			CDI	0.63	0.05		0.48**	
	Diagnosis	1.16	0.92		0.05			Diagnosis	1.40	0.92		0.06	
	Selective Attn (MC)	-0.11	0.12		-0.04			Sustained Attn (MC)	0.15	0.11		0.06	
	Diagnosis X Selective Attn	-0.17	0.37		-0.02			Diagnosis X Sustained Attn	0.17	0.27		0.03	
Total R ²		.239**				0.314	Total R ²		.247**				0.328
Adjusted R ²		.232**					Adjusted R ²		.241**				
N		479					N		481				

Note . CDI = Children's Depression Inventory Scores. MC = Mean Centered. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Auditory Sustained Attention Score. ††p < .05, †p < .01, *p < .008, **p < .001 .

Table 25. *Diagnosis Moderating EF-Rumination Associations: Set Shifting Accuracy and Time*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Predictor						Predictor					
Step 1	.233***				.304	Step 1	.238***				0.312
CDI		0.63	0.05	0.48**		CDI		0.64	0.05	0.49**	
Step 2	.009				.012	Step 2	.003				0.003
CDI		0.61	0.05	0.47**		CDI		0.62	0.06	0.48**	
Diagnosis		1.38	0.01	0.06		Diagnosis		1.07	0.93	0.05	
Set Shifting (MC)		0.19	0.10	0.08†		Set Shifting Time (MC)		-0.06	0.12	-0.02	
Step 3	.000				.000	Step 3	.001				0.001
CDI		0.62	0.05	0.48**		CDI		0.63	0.06	0.48**	
Diagnosis		1.38	0.92	0.06		Diagnosis		1.08	0.93	0.05	
Set Shifting (MC)		0.19	0.11	0.08		Set Shifting Time (MC)		-0.09	0.14	-0.03	
Diagnosis X Set Shifting		0.02	0.32	0.00		Diagnosis X Set Shifting Time		0.21	0.35	0.03	
Total R ²	.242**				.319	Total R ²	.241**				0.318
Adjusted R ²	.236**					Adjusted R ²	.234**				
N	480					N	446				

Note . CDI = Children's Depression Inventory Scores. MC = Mean Centered. Set Shifting = TEACH Set Shifting Accuracy Score. Switch Costs = TEACH Set Shifting Timing Score.

Score. †† $p < .05$, † $p < .01$, * $p < .008$, ** $p < .001$.

Table 26. *Diagnosis Moderating EF-Rumination Association: Working Memory and Divided Attention*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Predictor						Predictor					
Step 1	.235**				0.307	Step 1	.241**				0.318
CDI	0.64	0.05		0.49**		CDI	0.64	0.05		0.49**	
Step 2	0.004				0.005	Step 2	.008				0.011
CDI	0.64	0.06		.48**		CDI	0.62	0.06		0.47**	
Diagnosis	1.10	0.92		0.05		Diagnosis	1.36	0.94		0.01	
Working Memory (MC)	-0.02	0.11		-0.01		Divided Attn(MC)	0.22	0.12		0.07	
Step 3	0.003				0.004	Step 3	0.000				0.000
CDI	0.62	0.06		.47**		CDI	0.62	0.05		0.48**	
Diagnosis	1.36	0.94		0.06		Diagnosis	1.10	0.92		0.05	
Working Memory (MC)	0.02	0.11		0.01		Divided Attn(MC)	0.23	0.13		0.07	
Diagnosis X Working Memory	-0.39	0.31		-0.06		Diagnosis X Divided Attn	-0.04	0.42		0.00	
Total R ²	.242**				0.319	Total R ²	.249**				0.332
Adjusted R ²	.235**					Adjusted R ²	.242**				
N	457					N	472				

Note . CDI = Children's Depression Inventory Scores. Divided Attn = TEACH Dual Task Decrement Score. MC = Mean Centered. Working Memory = WISC Digit Span Total Score. ††p < .05, †p < .01, *p < .008, **p < .001 .

Moderation by Depressive Symptoms

A similar set of separate hierarchical linear regressions was conducted to examine whether current depressive symptoms interacted with executive function measures to predict rumination. In this latter set of regressions, diagnostic status was entered in Step 1 instead of depressive symptoms, a single measure of executive function and depressive symptoms (mean centered) were entered in Step 2, with their interaction term entered in Step 3. To control for family-wise error rate, a Bonferroni correction was used and the criterion alpha was .008. Findings are presented in Tables 27 through 30. Depressive symptoms did not moderate associations between executive functions and rumination. Findings were nonsignificant with regard to selective attention ($\beta = .00, p = .97$), sustained attention ($\beta = -.06, p = .17$), divided attention ($\beta = .04, p = .35$), set shifting time ($\beta = .05, p = .20$), and working memory ($\beta = .01, p = .84$). Of note, in each set of analyses, multivariate outliers were identified, and regression analyses were rerun after removing them. As interaction terms remained nonsignificant after excluding multivariate outliers from the model, findings are presented for the full dataset.⁶

⁶ To ensure findings did not reflect a subset of participants with low IQ scores,, follow-up analyses were conducted eliminating those with WISC digit span scores two or more standard deviations below the mean ($n = 8$). All reported findings remained unchanged after excluding these individuals from analyses.

Table 27. *Depressive Symptoms Moderating EF-Rumination Associations: Selective and Sustained Attention*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Step 1	Predictor					Step 1	Predictor				
		.023**			.024			.026**			0.027
	Diagnosis	3.37	1.01	0.15*			Diagnosis	3.59	1.00	0.16**	
Step 2		.216**			.282	Step 2		.220**			0.292
	Diagnosis	1.13	0.91	0.05			Diagnosis	1.32	0.91	0.06	
	CDI (MC)	0.62	0.05	0.47**			CDI (MC)	0.62	0.05	0.48**	
	Selective Attn (MC)	-0.13	0.12	-0.05			Sustained Attn (MC)	0.18	0.10	0.07	
Step 3		.000			.000	Step 3		.003			0.004
	Diagnosis	1.13	0.91	0.05			Diagnosis	1.26	0.91	0.06	
	CDI	0.62	0.05	0.47**			CDI (MC)	0.61	0.05	0.47**	
	Selective Attn (MC)	-0.13	0.12	-0.04			Sustained Attn (MC)	0.19	0.10	0.07	
	CDI X Selective Attn	0.00	0.02	0.00			CDI X Sustained Attn	-0.02	0.02	-0.06	
Total R ²		.238**			.312	Total R ²		.249**			0.332
Adjusted R ²		.232**				Adjusted R ²		.243**			
N		479				N		481			

Note. CDI = Children's Depression Inventory Scores. MC = Mean Centered. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Auditory Sustained Attention Score. ††p < .05, †p < .01, *p < .008, **p < .001.

Table 28. *Depressive Symptoms Moderating Set Shifting-Rumination Associations With (left) and Without (right) Multivariate Outliers*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Predictor						Predictor					
Step 1	.027**				0.028	Step 1	.013††				0.013
Diagnosis		3.63	0.99	0.16***		Diagnosis	.2.51	1.02	0.11††		
Step 2	.215**				0.284	Step 2	.189**				0.235
Diagnosis		1.38	0.91	0.06		Diagnosis	0.95	0.93	0.04		
CDI		0.61	0.05	0.47**		CDI	0.61	0.06	0.43**		
Set Shifting (MC)		0.19	0.10	0.08		Set Shifting (MC)	0.16	0.10	0.06		
Step 3	.005				0.007	Step 3	.001				0.001
Diagnosis		1.37	0.91	0.06		Diagnosis	0.97	0.93	0.04		
CDI		0.61	0.05	0.47**		CDI	0.61	0.06	0.43***		
Set Shifting (MC)		0.20	0.10	.08††		Set Shifting (MC)	0.17	0.10	0.07		
Diagnosis X Set Shifting		0.03	0.02	.07		Diagnosis X Set Shifting	0.01	0.02	0.03		
Total R ²	.247**				0.328	Total R ²	.202**				0.253
Adjusted R ²	.241**					Adjusted R ²	.195**				
N	480					N	471				

Note . CDI = Children's Depression Inventory Scores. Set Shifting = TEACH Set Shifting Accuracy Score. ††p < .05, †p < .01, *p < .008, **p < .001 .

Table 29. *Depressive Symptoms Moderating Set Shifting Time -Rumination Association*

		ΔR^2	B	SE	β	f^2
<u>Predictor</u>						
Step 1		.023**				.024
	Diagnosis		3.31	1.03	0.15**	
Step 2		.218***				.286
	Diagnosis		1.07	0.93	0.05	
	CDI (MC)		0.62	0.06	0.48***	
	Set Shifting Time (MC)		-0.06	0.12	-0.02	
Step 3		.003				.004
	Diagnosis		1.09	0.93	0.05	
	CDI		0.64	0.06	0.49***	
	Set Shifting Time (MC)		-0.06	0.12	-0.02	
	CDI X Set Shifting Time		0.03	0.02	0.05	
Total R^2		.243***				.321
Adjusted R^2		.236***				
N		446				

Note . CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count

Set Shifting Time Score. †† $p < .05$, † $p < .01$, * $p < .008$, ** $p < .001$.

Table 30. *Depressive Symptoms Moderating EF-Rumination Associations: Divided Attention and Working Memory*

	ΔR^2	B	SE	β	f^2		ΔR^2	B	SE	β	f^2
Predictor						Predictor					
Step 1	.025*				0.026	Step 1	0.030**				0.030
Diagnosis		3.51	1.02	0.16**		Diagnosis		3.77	1.03	0.17**	
Step 2	.224**				0.298	Step 2	.210**				0.276
Diagnosis		1.10	0.92	0.05		Diagnosis		1.36	0.94	0.06	
CDI (MC)		0.62	0.05	0.48**		CDI		0.62	0.06	0.47**	
Divided Attn (MC)		0.22	0.12	0.07†		Working Memory (MC)		0.02	0.11	0.01	
Step 3	.001				0.001	Step 3	.000				0.000
Diagnosis		1.05	0.92	0.05		Diagnosis		1.38	0.95	0.06	
CDI (MC)		0.62	0.05	0.48**		CDI		0.63	0.06	0.47**	
Divided Attn(MC)		0.24	0.13	0.08		Working Memory (MC)		0.01	0.11	0.01	
CDI X Divided Attn		0.02	0.02	0.04		CDI X Working Memory		0.00	0.02	0.01	
Total R ²	.250**				0.333	Total R ²	.239**				0.314
Adjusted R ²	.244**					Adjusted R ²	.232**				
N	472					N	457				

Note . CDI = Children's Depression Inventory Scores. MC = Mean Centered. Divided Attn = TEACH Dual Task Decrement Score. Working Memory = WISC Digit Span Total Score. †p < .05, *p < .01, **p < .008, ***p < .001 .

Concurrent Associations between EF and Depressive Symptoms

Given previously reviewed evidence of dissociable patterns of association between measures of EF and concurrent depressive symptoms versus rumination (Whitmer & Gotlib, 2012a), an exploratory regression analysis was run, examining whether measures of EF would be significantly associated with CDI scores after controlling for concurrent trait rumination. Results are presented in Tables 31 and 32. Rumination was entered as a control variable in Step 1, and measures of EF were entered simultaneously in Step 2. Four multivariate outliers were identified, but the results remained unchanged after excluding multivariate outliers, and are thus presented for the full sample. Sustained attention was significantly negatively associated with CDI scores, such that poorer sustained attention was associated with higher levels of depressive symptoms when entered simultaneously with other measures of EF in Step 2 ($\beta = -.11, p = .02$) and when other nonsignificant measures of EF were removed ($\beta = -.09, p = .02$). No other measures of EF were associated with depressive symptoms (all $ps > .21$).

Table 31. *Executive Functions Predicting to Depressive Symptoms*

Predictor	ΔR^2	B	SE	β	f^2
Step 1	.227***				0.294
Rumination		0.36	0.03	0.48***	
Step 2	0.013				0.017
Rumination		0.36	0.03	0.48***	
Selective Attn		0.12	0.10	0.05	
Sustained Attn		-0.21	0.09	-0.11*	
Divided Attn		0.04	0.10	0.02	
Set Shifting		0.01	0.08	0.00	
Working Memory		-0.03	0.09	-0.01	
Total R^2	.240***				0.316
Adjusted R^2	.230***				
N	445				

Note . CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Auditory Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC Digit Span Total Score. †p < .10, *p < .05, **p < .01, ***p < .001.

Table 32. *Sustained Attention-CDI Association Controlling for Rumination*

Predictor	ΔR^2	B	SE	β	f^2
Step 1	.238***				0.312
Rumination		0.38	0.03	0.49***	
Step 2	.009*				0.012
Rumination		0.38	0.03	0.49***	
Sustained Attn		-0.18	0.08	-.09*	
Total R^2	.247***				0.328
Adjusted R^2	.244***				
N	481				

Note . CDI = Children's Depression Inventory Scores. Sustained Attn = TEACH Auditory Sustained Attention Score. †p < .10, *p < .05, **p < .01, ***p < .001.

CHAPTER 4 DISCUSSION

The current study examined associations between performance on tests of executive functions, depressive symptoms, lifetime history of unipolar depression, and trait rumination in a large community sample of early adolescents. In addition, associations between executive functions and high risk for depression due to maternal lifetime history of the disorder.

Executive Functions and Trait Rumination

First, it was hypothesized (hypothesis 1) that, controlling for depressive symptoms, impaired executive functions, and particularly set shifting, would be positively associated with greater levels of trait rumination. Results provided no support for this hypothesis. No evidence was obtained for an association between poorer performance on measures of executive function (including selective attention, divided attention, set shifting, or working memory) and trait rumination. Although sustained attention was marginally significantly associated with rumination, the effect size of this association was small; moreover, it was in the opposite direction to that hypothesized, with *better* sustained attention predicting higher levels of rumination. Likewise, a small but significant gender by set shifting interaction was found, such that *better* set shifting accuracy was associated with higher levels of trait rumination among boys only.

With regard to specific associations between different types of EF and rumination, it was hypothesized that differential patterns of association between executive functions

and depressive symptoms versus rumination might emerge, with rumination more closely associated with deficits in set shifting tasks, and depressive symptoms more closely associated with deficits on tasks requiring goal maintenance over time and in the face of distraction information (e.g., selective and sustained attention). Results are only partially consistent with this hypothesis. Differential patterns of association emerged between depression versus rumination and (1) sustained attention in the overall sample, and (2) set shifting in boys only. Controlling for depressive symptoms (CDI scores), *better* sustained attention was associated with *higher* levels of rumination, whereas controlling for rumination, *better* sustained attention was marginally significantly associated with *lower* levels of depressive symptoms. Likewise, among boys only, after controlling for depressive symptoms, *better* set shifting accuracy was associated with *higher* levels of rumination. Conversely, after controlling for rumination, set shifting accuracy was not associated with depressive symptoms in either boys or girls. While this pattern of findings is theoretically interesting, in that it is consistent with the hypothesized pattern, it should be interpreted with caution. Given the small effect sizes of observed associations, it is possible that findings are a function of the large sample size (all f^2 's = .01-.06). No other measures of EF exhibited similar differential patterns of association.

Previous research in adult samples has pointed to a potential association between cognitive inflexibility and trait rumination. Specifically, previous research obtained evidence for concurrent and prospective associations between WCST performance and rumination (Davis & Nolen-Hoeksema, 2000; Miranda et al., 2013). Rumination also has

exhibited associations with deficits in set switching and inhibition of previously relevant task sets (Altamirano et al., 2010; Owens & Derakshan, 2013; Whitmer & Banich, 2007; Whitmer & Gotlib, 2012b), reversal learning (Whitmer & Banich, 2012), and inhibitory deficits in long term memory (Whitmer & Banich, 2010) and on an antisaccade paradigm (De Lissnyder et al., 2011).

This is the first study to examine rumination-EF associations in a community-based unselected sample of children or adolescents. Current findings are not consistent with previous research in adults. They are, however, more consistent with the only previous study to examine rumination-EF associations in a sample of treatment-seeking depressed adolescents and controls (Wilkinson & Goodyer, 2006), which obtained no evidence for the expected association between rumination and impaired set shifting. It is possible that documented EF-rumination associations in adults are not generalizable to samples of youth.

Previous research in adult clinical samples has obtained evidence for an EF-rumination association, after controlling for diagnosis (Meiran et al., 2011), and has documented significant interactions whereby rumination was associated with EF deficits only in participants with clinical diagnoses of depression but not controls (Levens et al., 2009). This pattern of findings was not obtained in the current study. Controlling for presence of depression diagnosis did not change findings with regard to rumination-EF associations, and executive functions did not interact with diagnosis to predict rumination.

With regard to hypothesis 1, the current study has several limitations that suggest directions for future research. One limitation of the current study is its correlational nature and reliance solely on a self-report measure of rumination. Studies using rumination-induction paradigms have documented impairments in executive function following rumination induction among depressed adults (Philippot & Brutoux, 2008; Watkins & Brown, 2002), and recent research has suggested that state (induced) and trait (habitual) rumination may be associated with differing patterns of deficits (Whitmer & Gotlib, 2012b). The current study focused on correlates of trait rumination and cannot speak to whether rumination induction in depressed youth may result in specific impairments in EF.

The current study relied on emotionally neutral tests of executive control. Some previous studies in adult samples have found that rumination is not associated with deficits in the processing of neutral information, but is associated only with deficits in executive control over emotional information (Bernblum & Mor, 2010; DeLissnyder, Koster, & De Raedt, 2012; DeLissnyder, Koster, Derakshan, & De Raedt, 2010; Joormann, Levens, & Gotlib, 2011; Joormann et al., 2010). However, research on this topic is inconsistent, and there has been growing consensus that empirical findings regarding rumination and EF are not consistent with valence-specific accounts, given that the preponderance of evidence for such accounts has focused on clinically depressed samples (Whitmer & Gotlib, 2012a). In addition, there is evidence that trait rumination is associated with deficits in performance on some types of emotionally neutral tasks as

well as better performance on others, and it has been suggested that trait ruminators may only experience valence-specific deficits when they are clinically depressed (Whitmer & Gotlib, 2012a). Thus, it is unlikely that our failure to obtain significant findings is attributable to our use of an emotionally neutral task.

Moreover, the current study employed a relatively circumscribed array of measures of EF. There is some evidence that rumination may be associated with deficits only on complex tasks or when attention is unconstrained by task demands (Hertel & Rude, 1991; Hertel et al., 2012). In the TEACH, tasks permit the test administrator to elicit responses relatively frequently and to engage frequently with the adolescent being tested. For example, on the test of attentional set shifting, the adolescent is constrained to follow the interviewer's finger when counting, and is prompted to provide responses at frequent intervals. Thus, these tasks may not be complex and open-ended enough to capture the attentional difficulties experienced by ruminators.

Consistent with this possibility, Davis and Nolen-Hoeksema (2000) obtained evidence for an association between trait rumination in adults and perseverative errors on the WCST, but did not find an association between trait rumination and performance on another test of task switching. Another consideration is evidence that trait ruminators' difficulties on the WCST may be attributable to impaired ability to learn from negative feedback about the need to switch. A recent study found that, when explicitly instructed to switch, ruminators did not have difficulty with a set shifting task (Whitmer & Banich, 2012). The TEACH attentional set shifting task provides feedback regarding correct

responses on two initial practice rounds, but visual up and down arrows explicitly instruct the child when to switch task set.

Related to this issue, much of the previous research linking EF deficits on emotionally neutral tasks with rumination that has obtained support has employed tasks designed to reveal inhibitory deficits. In particular, evidence from studies using backward inhibition paradigms has led to the suggestion that difficulty with removing no longer relevant task sets from working memory during attentional switching tasks may be associated with greater trait rumination (Whitmer & Gotlib, 2012b). In the current study, such inhibitory deficits should have been evident in performance on the TEACH attentional set shifting measure, which requires that participants switch between task sets. However, this task is not designed to isolate differences in task switching versus inhibitory deficits.

An additional limitation was the necessity of excluding set shifting time from the primary analyses because of systematic missingness due to the large number of participants whose performance on this variable could not be scored due to their low set shifting accuracy scores. The only other study to examine TEACH performance and rumination in adolescents reported the same limitation (Wilkinson & Goodyer, 2006). Future research is needed permitting examination of set shifting latency in relation to trait rumination in adolescents. In addition, future research should employ tasks such as backward inhibition paradigms that are designed to isolate inhibitory abilities more specifically. In summary, it remains possible that findings might have been different had

the current study employed emotionally valenced measures of EF, more fine grained measures able to differentiate between specific abilities such as task switching and inhibition (e.g., Whitmer & Banich, 2007), or more demanding complex tasks that, for example, required reversal learning through feedback or imposed fewer constraints on attention.

An intriguing aspect of the current pattern of findings is that the direction of associations between rumination and EF was opposite to that hypothesized, with better sustained attention marginally associated with greater rumination and better set shifting associated with greater rumination among boys only. This is partially consistent with Altamirano and colleagues' (2010) findings in adult samples that trait rumination was associated with better goal maintenance (albeit greater difficulty switching to a different task goal on a letter switching task). Likewise, on emotionally valenced tasks, evidence has been found that individuals who are trait ruminators do not show difficulties ignoring distractors (Joormann et al., 2010; Zetsche et al., 2012; for discussion and review, see Whitmer & Gotlib, 2012a), whereas depression diagnosis has been associated with difficulties ignoring distractors (Joormann et al., 2010). Current findings with regard to better sustained attention among trait ruminators are consistent with these findings, although, as previously noted, effect sizes are small. Specifically, controlling for CDI scores, better sustained attention was marginally associated with higher levels of rumination, whereas controlling for rumination, better sustained attention was significantly associated with lower CDI scores.

From a theoretical perspective, current findings are not consistent with a resource depletion account (Watkins & Brown, 2002), which would predict that rumination would be associated with widespread difficulties on EF tasks (particularly complex ones such as the divided attention and attentional set shifting task in the current study). However, another pertinent theory is Whitmer and Gotlib's (2012a) recently formulated attentional scope model. The authors note that findings from studies in unselected samples or nondepressed participants (who are less likely to be in a state of rumination) are not consistent with a resource depletion account of rumination, but rather suggest that trait ruminators are characterized by *better* performance on a variety of tasks, maintaining task goals, ignoring external distractors, and showing less task-irrelevant thought during task engagement. They suggest that this pattern of strengths and deficits reflects a narrowed attentional scope that increases susceptibility to rumination. According to this formulation, trait ruminators have a narrowed attentional scope, and when they become depressed or enter a negative mood state, negative mood further constricts their attentional scope while simultaneously biasing attention toward negative self-relevant information. Because attentional scope aids individuals in ignoring distracting, irrelevant information, it may actually facilitate problem solving when all information is provided and only deductive reasoning is required, but will impair performance on tasks requiring insight or creativity, tasks that rely on use of inaccessible information in long term memory, or tasks that require broad attention (Whitmer & Gotlib, 2012a). Although effect sizes of observed associations in the current study are small and should be replicated before conclusions are drawn, the direction of these associations is consistent

with the attentional scope model. As previously noted, one explanation for lack of significant findings with regard to other EF tasks is that they are relatively simple. All information needed for correct performance was provided and therefore would not be likely to expose the types of deficits postulated to be associated with narrowed attentional scope.

Findings are also consistent with Alloy and Abramson's (2007) biocognitive vulnerability-stress model of depression, which embeds the cognitive vulnerability-stress model within the context of normative cognitive and brain development in adolescence. This view suggests that normative brain maturation, including maturation of prefrontal cortex and concomitant normative cognitive development of self-regulatory executive functions (sustained and selective attention and executive control over attentional switching as well as working memory) serve as a prerequisite for adolescents to engage in attempts to self-regulate negative affect and full blown rumination. As adolescents develop attentional competence, they will be better able to focus on negative inferences and thus be more likely to suffer depressogenic effects. The current study focused on individual differences in trait rumination and executive functions in a community sample of adolescents, many of whom are poised to develop first onset of depression, but the majority of whom have not done so. Thus, it was not possible to test longitudinal associations or examine the causal relationship between development of executive functions and development of trait rumination over time. However, the direction of associations between sustained attention and set shifting and trait rumination is consistent

with this formulation. Replication is needed to verify that significant findings are not simply a function of sample size, and additional longitudinal research is needed to tease apart the relations between developmental changes in EF, individual differences in EF, rumination, and depression over time.

Although the reported associations are consistent with the theories discussed above, the effect sizes for all rumination-EF associations identified in the current study were small. Depressive symptoms and sustained attention together explained only approximately 24% of the variance in trait rumination, and sustained attention explained only an additional 0.5% of the variance in rumination after controlling for depressive symptoms. Likewise, within the male sample, depressive symptoms and set shifting explained approximately 17% of the variance in trait rumination, and set shifting accounted for only an additional 5% of the variance in rumination after controlling for depressive symptoms. These findings highlight the possibility that vulnerability factors unrelated to EF may also play a role in the development of trait rumination and that evaluation of and inclusion of such factors as predictors might increase the explanatory power of future models. These may include factors rooted in the social environment (as suggested by Davis & Nolen-Hoeksema, 2000), such as peer, family, and parenting influences, as well as individual differences in negative affect or cognitive style. It is possible that EF deficits are associated with trait rumination when they are examined in interaction with other such vulnerability factors. Consistent with this possibility are findings from the temperament literature suggesting stronger associations between

negative affect, rumination, and depressive symptoms among those low in effortful control (Hilt et al., 2012; Verstraeten et al., 2009) and that high effortful control is a protective factor against developing rumination only among those who do not experience overcontrolling parenting (Hilt et al., 2012). Future research is needed to examine whether measures of EF interact with other temperamental and social risk factors to predict the development of rumination over time.

Executive Functions and Depression in Adolescence

A secondary goal of the current study (Goal 2) was to examine whether worse performance on measures of executive function was associated with greater likelihood of a lifetime history of diagnosis of unipolar depression. It was hypothesized that poorer performance on measures of executive function would be associated with a greater likelihood of lifetime history of unipolar depression (hypothesis 2). Given inconsistent patterns of findings to date, hypotheses with regard to the specific pattern of deficits in EF in depressed youth were exploratory, but it was tentatively hypothesized that, after controlling for concurrent depressive symptoms, diagnostic status would be associated with poorer performance on tests of selective attention, set shifting and working memory, reflective of inhibitory deficits. No support was obtained for this hypothesis.

Research to date examining whether depressed children and/or adolescents are characterized by deficits in EF has yielded inconsistent findings. The current study hypotheses were informed by previously obtained evidence of deficits in EF in depressed samples of children and adolescents (Baune et al., 2012; Cataldo, Nobile, Loruso,

Battaglia, & Moltem, 2005; Gunther, Konrad, De Brito, Herpertz-Dahlmann, & Vloet, 2011; Han et al., 2012; Klimkeit, Tonge, Bradshaw, Melvin, & Gould, 2011; Maalouf et al., 2011; Matthews, Coghill, & Rhodes, 2008; Micco et al., 2009; Wilkinson & Goodyer, 2006). The current findings are not consistent with those of these studies, but are instead consistent with previously noted contradictory studies reporting null findings or obtaining only marginally significant or qualitative patterns of behavioral differences in child and adolescent samples (Brooks, Iverson, Sherman, & Roberge, 2010; Favre et al., 2009; Frost, Moffitt, & McGee, 1989; Gunther, Holtkamp, Jolles, Herpertz-Dahlmann, & Konrad, 2004; Halari et al., 2009). No clear methodological differences differentiate studies obtaining evidence for EF deficits in depressed youth from studies (including the current study) that have not obtained significant evidence of EF deficits in depressed youth. However, findings from the current study are consistent with the only other study to date to use an unselected community sample of youth with unipolar depression, which obtained no differences in performance between depressed youth and healthy controls on measures of EF including cognitive flexibility, set shifting, problem solving, and planning (Frost et al., 1989).

The current study improved upon many such previous studies in that it employed a much larger unselected community-based sample and assessed and controlled for comorbid diagnosis. Findings did not change after controlling for the presence of comorbid disorders, including ADHD, anxiety disorders, and conduct or oppositional defiant disorders. In addition, given that the majority of the adolescents with a history of

depression were in remission and we controlled for concurrent depressive symptoms, we aimed to differentiate between state-dependent impairments and trait-markers of disorder. In this sample, controlling for CDI scores did not change our findings. No significant associations were found between measures of EF and lifetime history of unipolar depression in adolescents whether or not we controlled for concurrent depressive symptoms.

Previous research obtaining evidence of deficits in EF in children and adolescents with depression has employed clinical treatment-seeking inpatient and outpatient samples and has not controlled for concurrent depressive symptoms. The current sample consisted of adolescents recruited from the community, the majority of whom were in remission and who reported a relatively circumscribed range of CDI scores. Many of the adolescents with a lifetime history of depression had mild forms of the disorder, meeting criteria for DSM subthreshold, or RDC major probable or minor depression rather than definite DSM or RDC criteria for definite Major Depressive Disorder. The overall lack of evidence for significant deficits in EF among those with a history of depression is consistent with previous research in a remitted sample of adolescents that failed to obtain support for attentional deficits (Maalouf et al., 2001), but inconsistent with previous evidence of cognitive inflexibility in remitted depressed youth (Houston et al., 2004).

It may be that EF deficits would have been detected in a sample of unremitted youth, in youth with more severe clinical depression, or in a larger sample. Although our sample of adolescents with a current or lifetime history of depression was large compared

to previous studies examining this question ($N = 68$), it was nevertheless relatively small, which may have limited power to detect small effects.

As previously noted, the current study is cross-sectional and can speak only to correlations between history of disorder and current EF task performance. Future research should examine whether EF deficits predict prospective onset of disorder. Moreover, it is important to note that, given the age range of our sample, we were primarily examining early onset prepubertal depression, which may differ from later onset depression. Previous research in adult samples also has identified a broader, more pervasive pattern of deficits in adults with late versus early onset depression (Bora, Harrison, Yucel, & Pantelis, 2012). It may be that early (prepubertal) onset depression has different correlates than depression characterized by first onset in adolescence or thereafter.

Executive Functions in Offspring of Depressed Mothers

A third goal (Goal 3) of the current study was to examine whether aspects of executive function are associated with higher risk of future onset of depression (as indicated by maternal history of disorder), controlling for child diagnosis and depressive symptoms. Based on previous findings in offspring of mothers with a history of depression (Klimes-Dougan et al., 2006; Mannie et al., 2010) and in adults at high genetic risk for the disorder (Christensen et al., 2006; Holmes et al., 2010), it was hypothesized that high risk youth would exhibit mild impairments in EF (albeit milder than those observed in adolescents with diagnoses of unipolar depression). It was further hypothesized that high risk youth might exhibit specific deficits on tests of working

memory and attentional set shifting (hypothesis 3). Given that studies to date have yielded far less convincing evidence for executive deficits at the behavioral level, hypotheses with regard to Goal 3 were exploratory.

The current study improved methodologically on previous studies of youth with parental history of depression in that it combined a large unselected community sample of mothers with versus without a lifetime history of depression, used a range of measures of executive function, and took into account child psychopathology and concurrent depressive symptoms. Despite this, no support was obtained for this hypothesis. After controlling for adolescent depression diagnosis, measures of executive function were not significantly associated with maternal current or lifetime diagnosis of unipolar depression generally or major depressive disorder specifically. Findings remained nonsignificant whether analyses were conducted with measures of EF entered as simultaneous predictors in the model or separately. Likewise, findings remained nonsignificant whether analyses were conducted in the overall sample, or within the sample of biological mothers and their offspring only. These null findings add to a growing body of literature that has failed to obtain compelling evidence of a profile of impaired performance on emotionally neutral tests of executive functions in youth at high risk for depression due to maternal lifetime history of the disorder (Klimes-Dougan et al., 2006; Mannie et al., 2010; Micco et al., 2009; Perez-Edgar et al., 2006). It may be that deficits do not exist prior to disorder onset, that deficits are specific to the processing of emotionally valenced information or evident only under emotionally threatening conditions (e.g., Perez-Edgar et al., 2006) or

that deficits are too subtle to detect with behavioral tasks, but rather are manifested as compensatory neural activity to achieve comparable levels of task performance to that of individuals without the disorder (Mannie et al., 2010).

In that the current study found no evidence for an association between EF deficits and lifetime history of depression or risk of future onset due to maternal history of disorder, findings are not consistent with the conceptualization of impaired executive functions as trait markers of unipolar depression. Rather, findings are consistent with patterns of findings in previous research suggesting that many of the cognitive deficits observed in the acute phase of depression are not present prior to episode onset and may normalize following remission. Consistent with this, worse sustained attention was positively correlated with higher concurrent depressive symptoms, but was not associated with lifetime history of disorder.

Conclusions and Directions for Future Research

This is the first study to examine associations between symptoms and diagnoses of unipolar depression, rumination, and performance on tests of executive function (selective attention, sustained attention, divided attention, attentional set shifting, and working memory) in a large non-treatment-seeking community sample of children or adolescents. Although one previous study examined the association between rumination and attentional set shifting in a child and adolescent sample (Wilkinson & Goodyer, 2006), this is the first to do so in a large unselected community sample of adolescents,

and is the first to examine associations between rumination and other aspects of attention and working memory in a sample of youth.

In contrast to studies linking deficits on neutral tests of executive control and rumination in nonclinical samples of young adults, this study does not provide support for the existence of such associations in early adolescents. Instead, the current study suggests that trait rumination is either unrelated to performance or is related to *better* performance on some tests of EF in this age group, and is unrelated to performance on other tests of EF. Specifically, better sustained attention was marginally significantly associated with greater trait rumination, and, among boys only, better set shifting performance was associated with greater trait rumination. Given that this is the first study to examine these associations in a sample of unselected youth and that significant effects were small, additional research is needed to replicate these findings. This study was unique in that it examined associations between EF and trait rumination in a very large, nonclinical unselected sample of adolescents. Findings are consistent with the suggestion that in the absence of depressed mood, trait ruminators may not exhibit general deficits in task performance on measures of EF, and may even exhibit strengths on some types of tasks requiring that they ignore irrelevant distractors (Whitmer & Gotlib, 2012a). Findings are also potentially consistent with the proposal that developmental increases in cognitive competencies may serve as prerequisites for the development of rumination in adolescence (Alloy & Abramson, 2007).

In addition, this study was the first to explore whether differential patterns of association between executive functions and (1) rumination and (2) depressive symptoms observed in adult samples (e.g., Altamirano et al., 2010) are observable in youth. Differential patterns of association were detected in youth, but not in the hypothesized direction. Instead, *better* sustained attentional performance was associated with greater rumination, whereas worse performance was associated with greater depressive symptoms. In boys, better set shifting performance was associated with greater rumination, but was unrelated to depressive symptoms. Given preliminary evidence suggesting that trait rumination may be associated with better task performance and possible differential patterns of association between EF and trait rumination versus depression, prospective longitudinal studies are needed examining temporal associations between changes in rumination, depressive symptoms, and executive functions over time.

An additional contribution of the current study was its examination of gender and diagnosis as potential moderators of EF-rumination associations. There was no evidence that diagnosis moderated associations. Future research with larger, more clinically severe depressed samples of adolescents is needed to replicate these findings. Gender did moderate set shifting-rumination associations, such that evidence for a positive association between better set-shifting and rumination was obtained in boys but not girls. Previous research has found associations between deficits on the WCST and trait rumination in men but not women, leading to the suggestion that different pathways to trait rumination may exist for men versus women (Davis & Nolen-Hoeksema, 2000).

However, given that no other evidence for moderation by gender was found, that the effect size was small, and that the direction of associations differed from previous findings in adults, additional research is warranted replicating these findings before conclusions are drawn.

The current study improved on previous studies examining associations between unipolar depression diagnosis and measures of EF in early adolescents by using a large unselected, non-treatment-seeking community sample and a range of measures of EF. No evidence was obtained for an association between EF deficits and lifetime history of depression in adolescence. Likewise, no evidence was obtained for a profile of EF deficits in adolescents at high risk of future first onset of depressive disorder, suggesting that high risk adolescents who have not yet experienced first onset of the disorder do not exhibit behavioral deficits in EF.

However, research using more fine grained measures of inhibitory deficits as well as pitting emotionally neutral versus emotionally valenced measures of EF against one another in adolescent samples is needed to further explore this question during the transition to adolescence. The current study examines whether individual differences in rumination are associated with individual differences in EF in a sample of early adolescents. Future research is needed that examines whether within-subject developmental changes in EF prospectively predict within-subject changes in levels of trait rumination and depression over time (or vice versa) in order to clarify temporal and causal relations between these variables.

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APPENDIX SUPPLEMENTARY RESULTS

This section contains tabulated supplementary findings referenced in footnotes in the results section.

Table A1. *Executive Functions Predicting Rumination Controlling for Depression Diagnosis*

	ΔR^2	B	SE	β	f^2
Predictor					
Step 1	.230***				0.299
CDI		0.61	0.06	0.47***	
Depression Diagnosis		1.16	0.96	0.05	
Step 2	.017†				0.021
CDI		0.62	0.06	0.47***	
Depression Diagnosis		1.31	0.96	0.06	
Selective Attn		-0.08	0.13	0.06	
Sustained Attn		0.21	0.11	0.08†	
Divided Attn		0.17	0.14	0.06	
Set Shifting		0.15	0.11	0.06	
Working Memory		-0.03	0.11	-0.01	
Total R ²	.246***				0.326
Adjusted R ²	.234***				
N	445				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC Digit Span Total Score. †p < .10, *p < .05, **p < .01, ***p < .001.

Table A2. *EF Predicting Rumination Controlling for Depression Diagnosis Without Outliers*

		ΔR^2	B	SE	β	f^2
	Predictor					
Step 1		.231***				0.300
	CDI		0.66	0.06	0.47***	
	Depression Diagnosis		1.22	0.97	0.05	
Step 2		0.015				0.020
	CDI		0.66	0.06	0.47***	
	Selective Attn		-0.04	0.13	-0.01	
	Sustained Attn		0.23	0.11	0.09*	
	Divided Attn		0.13	0.15	0.04	
	Set Shifting		0.13	0.11	0.05	
	Working Memory		0.01	0.12	0	
Total R ²		.246***				0.326
Adjusted R ²		.233***				
N		431				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC Digit Span Total Score. †p < .10, *p < .05, **p < .01, ***p < .001.

Table A3. *Set Shifting Time Predicting to Rumination Controlling for Depression Diagnosis*

		ΔR^2	B	SE	β	f^2
	Predictor					
Step 1		.240***				0.316
	CDI		0.62	0.06	0.48***	
	Depression Diagnosis		1.07	0.93	0.05	
Step 2		0.000				0.000
	CDI		0.62	0.06	0.48***	
	Depression Diagnosis		1.07	0.93	0.05	
	Set Shifting Time		-0.06	0.12	-0.02	
Total R ²		.240***				0.316
Adjusted R ²		.235***				
N		446				

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count Timing Score.

†p < .10, *p < .05, **p < .01, ***p < .001.

Table A4. *Sustained Attention-Rumination Associations Controlling for Depression Diagnosis*

		ΔR^2	B	SE	β	f^2
	<u>Predictor</u>					
Step 1		.241***				0.318
	CDI		0.62	0.05	0.48***	
	Depression Diagnosis		1.23	0.91	0.06	
Step 2		.005†				0.007
	CDI		0.62	0.05	0.48***	
	Depression Diagnosis		1.32	0.91	0.06	
	Sustained Attn		0.18	0.1	0.07†	
Total R^2		.246***				0.326
Adjusted R^2		.242***				
N		481				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Sustained Attn = TEACH Auditory Sustained Attention Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Rumination Controlling for Axis I Disorders With (left) and Without (right) Multivariate Outliers

B	SE	β	f^2		ΔR^2	B	SE	β	f^2
<u>Predictor</u>									
			.299	Step 1	.227***				0.294
0.61	0.06	0.47***				0.66	0.06	0.47***	
1.20	0.96	0.05				1.25	0.98	0.06	
-0.23	0.69	-0.01				-0.21	0.69	-0.01	
			.023	Step 2	.016†				0.021
0.62	0.06	0.47***				0.66	0.06	0.47***	
1.33	0.96	0.06				1.35	0.98	0.06	
-0.15	0.69	-0.01				-0.13	0.69	-0.01	
-0.08	0.13	-0.03				-0.07	0.13	-0.02	
0.20	0.11	0.08†				0.24	0.11	0.09*	
0.17	0.14	0.06				0.14	0.14	0.04	
0.15	0.11	0.06				0.12	0.11	0.05	
-0.03	0.11	-0.01				0.00	0.12	0.00	
			.238	Total R ²	0.243***				.321
				Adjusted R ²	.229***				
<i>N</i>					434				

ve Attn = TEACH Selective Attention Score. Sustained Attn: TEACH Sustained Attention Score. Divided Attn = TEACH Dual Task Decrement Score.

king Memory = WISC Digit Span Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A6. *Set Shifting Time Predicting Rumination Controlling for Axis I Disorders*

	ΔR^2	B	SE	β	f^2
Predictor					
Step 1	.240***				0.316
CDI		0.62	0.06	0.48***	
Depression Diagnosis		1.1	0.94	0.05	
Other Axis I Disorder		-0.21	0.68	-0.01	
Step 2	0.000				0.001
CDI		0.63	0.06	0.48***	
Depression Diagnosis		1.1	0.94	0.05	
Other Axis I Disorder		-0.24	0.68	-0.02	
Set Shifting Time		-0.06	0.13	-0.02	
Total R^2	.241***				0.318
Adjusted R^2	.234***				
N	446				

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count Timing Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A7. *Sustained Attention-Rumination Associations Controlling for Diagnosis*

	ΔR^2	B	SE	B	f^2
Predictor	.242***				0.319
Step 1					
CDI		0.62	0.05	0.48***	
Depression Diagnosis		1.23	0.91	0.06	
Other Axis I Disorder					
Step 2	.005†				0.005
CDI		0.62	0.05	0.48***	
Depression Diagnosis		1.32	0.91	0.06	
Other Axis I Disorder		-0.23	0.65	-0.01	
Sustained Attn		0.18	0.1	0.07†	
Total R^2	.246***				0.326
Adjusted R^2	.240***				
<i>N</i>	481				

Note. CDI = Children's Depression Inventory Scores. Sustained Attn = TEACH Auditory Sustained Attention Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A8. *Executive Functions Predicting Adolescent History of Depression Controlling for Comorbidity*

	χ^2	df	B(SE)	Wald	OR	95% CI
Step 1	30.14***	5				
CDI		1	0.09(0.02)	17.79	1.10***	1.05-1.14
Gender		1	-0.53(0.29)	3.37	0.59†	0.34-1.04
Race (1)		1	-0.54(0.29)	3.39	0.58†	0.33-1.04
Race(2)		1	-0.74(0.79)	0.89	0.48	0.10-2.22
Axis I Disorder		1	0.57(0.28)	4.00	1.76*	1.01-3.07
Step 2	5.91	5				
CDI		1	0.10 (0.02)	19.26	1.10***	1.05-1.15
Gender		1	-0.48(0.30)	2.57	0.62†	0.35-1.11
Race (1)		1	-0.66(0.30)	4.75	0.52*	0.29-.94
Race(2)		1	-0.89(0.81)	1.23	0.41	0.08-1.99
Axis I Disorder		1	0.55(0.29)	3.73	1.74*	0.99-3.05
Selective Attn		1	0.03(0.06)	0.34	1.03	0.93-1.15
Sustained Attn		1	-0.05(0.05)	0.84	0.96	0.87-1.05
Divided Attn		1	0.06(0.06)	0.99	1.06	0.95-1.19
Set Shifting		1	-0.05(0.05)	1.13	0.95	0.87-1.04
Working Memory		1	-0.07(0.05)	2.11	0.93	0.84-1.03
Overall χ^2	36.05***	10				
<i>N</i>		448				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Divided Attn = TEACH Dual Task Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span Total Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A9. *Set Shifting Time Predicting Adolescent History of Depression Controlling for Comorbidity*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	31.70***	5				
CDI		1	.09(.02)	17.27	1.09***	1.05-1.14
Gender		1	-.68(.29)	5.59	0.51*	0.29-0.89
Race (1)		1	-.60(.29)	4.14	0.55*	0.31-0.98
Race(2)		1	-.75(.78)	0.92	0.47	0.10-2.19
Axis I Disorder		1	.56(.28)	3.90	1.74*	1.00-3.04
Step 2	0.07					
CDI		1	.10 (.02)	20.43	1.10***	1.06-1.15
Gender		1	-.68(.29)	5.57	0.51*	0.29-0.89
Race (1)		1	-.61(.30)	4.21	0.55*	0.31-0.97
Race(2)		1	-.75(.79)	0.90	0.47	0.10-2.21
Axis I Disorder		1	.55(.28)	3.83	1.74*	1.00-3.03
Set Shifting Time		1	-.02(.06)	0.07	0.99	0.89-1.10
Overall χ^2	31.78***	6				
<i>N</i>		448				

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count

Timing Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A10. *Offspring Executive Functions Predicting to Lifetime History of Depression in Biological Mothers*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	22.53***	3				
Child Diagnosis		1	.86(.30)	8.10	2.37**	1.31-4.30
Race (1)		1	-.77(.22)	12.50	0.47***	0.30-0.71
Race (2)		1	-.56(.54)	1.06	0.57	0.20-1.66
Step 2	2.49	5				
Child Diagnosis		1	.89(.31)	8.43	2.43**	1.34-1.43
Race (1)		1	-.73(.22)	10.94	0.48**	0.31-0.74
Race (2)		1	-.56(.56)	1.06	0.57	0.20-1.66
Selective Attn		1	.00(.04)	0.00	1.00	0.93-1.08
Sustained Attn		1	-.02(.04)	0.24	0.98	0.91-1.06
Divided Attn		1	.00(.04)	0.00	1.00	0.92-1.09
Set Shifting		1	.04(.04)	1.24	1.04	0.97-1.08
Working Memory		1	.03(.04)	.82	1.03	0.96-1.11
Overall χ^2	25.02**	8				
N		385				

Note. CDI = Children's Depression Inventory Scores. Selective Attn = TEACH Selective Attention Score. Divided Attn = TEACH Dual Task Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A11. *Offspring Set Shifting Time Predicting to Lifetime History of Depression in Biological Mothers*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	20.63***	3				
Child Diagnosis		1	.85(.31)	7.71	2.33**	1.28-4.24
Race (1)		1	-.72(.22)	10.88	0.49**	0.32-0.75
Race (2)		1	-.51(.54)	0.91	0.60	0.21-1.72
Step 2	0.40	1				
Child Diagnosis		1	.84(.31)	7.67	2.33**	1.28-4.23
Race (1)		1	-.73 (.22)	10.97	0.48**	0.31-0.74
Race (2)		1	-.51(.54)	0.87	0.60	0.21-1.74
Set Shifting Time		1	-.01(.04)	0.11	0.99	0.91-1.07
Overall χ^2	21.03***	4				
<i>N</i>	377					

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count Time Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A12. *Offspring Executive Functions Predicting to Lifetime History of MDD in Biological Mothers*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	22.65***	3				
Child Diagnosis		1	.66(.30)	4.75	1.94*	1.07-3.51
Race (1)		1	-.96(.24)	16.23	0.38***	0.24-0.61
Race (2)		1	-.34(.56)	0.37	0.71	0.24-2.14
Step 2	1.61	5				
Child Diagnosis		1	.68(.31)	4.97	1.98**	1.09-3.60
Race (1)		1	-.95(.24)	15.16	0.39***	0.24-0.63
Race (2)		1	-.35(.57)	0.39	0.70	0.23-2.14
Selective Attn		1	.00(.04)	0.00	1.00	0.92-1.09
Sustained Attn		1	-.03(.04)	0.48	0.97	0.90-1.05
Divided Attn		1	-.01(.05)	0.06	1.00	0.90-1.08
Set Shifting		1	.03(.04)	0.50	1.03	0.95-1.11
Working Memory		1	.03(.04)	0.73	1.03	0.96-1.12
Overall χ^2	24.27***	8				
<i>N</i>	385					

Note. MDD = DSM OR RDC Major Depressive Disorder. Sustained Attn = TEACH Sustained Attention Score. Selective Attn = TEACH Selective Attention Score. Divided Attn = TEACH Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A13. *Offspring Set Shifting Time Predicting to Lifetime History of MDD in Biological Mothers*

	χ^2	d f	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	22.86***	3				
Child Diagnosis		1	.71(.30)	5.37	2.03*	1.12-3.70
Race (1)		1	-.97(.24)	15.88	0.38***	0.24-0.61
Race (2)		1	-.33(.56)	0.33	0.72	0.24-2.18
Step 2	0.34	1				
Child Diagnosis		1	.71(.31)	5.40	2.04*	1.12-3.72
Race (1)		1	-.95(.24)	15.20	0.39***	0.24-0.62
Race (2)		1	-.34(.57)	0.37	0.71	0.23-2.15
Set Shifting Time		1	.03(.05)	0.34	1.03	0.94-1.12
Overall χ^2	23.20***	4				
<i>N</i>	377					

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count Time Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A14. *Offspring Executive Functions Predicting to Maternal Lifetime History of MDD*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	23.52***	3				
Child Diagnosis		1	.68(.30)	5.32	1.98*	1.11-3.55
Race (1)		1	-.93(.23)	10.58	0.39***	0.25-0.62
Race (2)		1	-.32(.56)	0.33	0.73	0.24-2.18
Step 2	1.12	5				
Child Diagnosis		1	.71(.30)	5.62	2.03*	1.13-3.66
Race (1)		1	-.92(.23)	15.33	0.40***	0.25-0.63
Race (2)		1	-.31(.57)	0.30	0.74	0.24-2.23
Selective Attn		1	-.02(.04)	0.20	0.98	0.90-1.07
Sustained Attn		1	-.01(.04)	0.06	0.99	0.92-1.07
Divided Attn		1	-.03(.05)	0.39	0.97	0.89-1.06
Set Shifting		1	.02(.04)	0.21	1.02	0.95-1.10
Working Memory		1	.03(.04)	0.45	1.03	0.95-1.11
Overall χ^2	24.63***	8				
<i>N</i>	414					

Note. MDD = DSM OR RDC Major Depressive Disorder. Selective Attn = TEACH Selective Attention Score. Divided Attn = TEACH Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A15. *Offspring Set Shifting Time Predicting to Maternal Lifetime History of MDD*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	24.95***	3				
Child Diagnosis		1	.75(.30)	6.32	2.13**	1.18-3.83
Race (1)		1	-.98(.24)	17.03	0.38***	0.24-0.60
Race (2)		1	-.30(.56)	0.28	0.74	0.25-2.24
Step 2	0.245	1				
Child Diagnosis		1	.76(.30)	6.36	2.13*	1.18-3.84
Race (1)		1	-.97(.24)	16.50	0.38***	0.24-.61
Race (2)		1	-.31(.56)	0.31	0.73	0.24-2.21
Set Shifting Time		1	.02(.04)	0.24	1.02	0.94-1.11
Overall χ^2	25.20***	4				
<i>N</i>	403					

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count Time Score.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A16. *Offspring EF Predicting to Maternal Lifetime History of MDD Controlling for Child Axis I Disorder*

	χ^2	df	B(SE)	Wald	OR	95% CI
<u>Predictor</u>						
Step 1	26.08***	4				
Child Diagnosis		1	.63(.30)	4.42	1.88*	1.04-3.38
Race (1)		1	-.96(.23)	17.20	0.39***	0.30-0.68
Race (2)		1	-.36(.56)	0.41	0.70	0.20-1.62
Axis I Disorder		1	.37(.23)	2.58	1.44	0.92-2.25
Step 2	1.22	5				
Child Diagnosis		1	.66(.30)	4.78	1.94*	1.07-3.50
Race (1)		1	-.93(.24)	15.77	0.39***	0.25-0.62
Race (2)		1	-.34(.57)	0.37	0.71	0.23-2.15
Axis I Disorder		1	.38(.23)	2.69	1.46†	0.93-2.30
Selective Attn		1	-.01(.04)	0.08	0.99	0.91-1.07
Sustained Attn		1	-.01(.04)	0.03	0.99	0.92-1.07
Divided Attn		1	-.03(.05)	0.38	0.97	0.89-1.06
Set Shifting		1	.02(.04)	0.24	1.02	0.95-1.10
Working Memory		1	.03(.04)	0.64	1.03	0.96-1.11
Overall χ^2	27.30**	9				
<i>N</i>	437					

Note. MDD = DSM OR RDC Major Depressive Disorder. Selective Attn = TEACH Selective Attention Score. Divided Attn = TEACH Decrement Score. Set Shifting = TEACH Creature Count Accuracy Score. Working Memory = WISC. Digit Span. Total Score. † $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A17. *Offspring Set Shifting Time Predicting to Maternal Lifetime History of MDD Controlling for Child Axis I Disorder*

		χ^2	df	B(SE)	Wald	OR	95% CI
	<u>Predictor</u>						
Step 1		33.43***	4				
	Child Diagnosis		1	.69(.30)	5.19	2.00*	1.10-3.62
	Race (1)		1	-1.01(.24)	17.89	0.37***	.23-.58
	Race (2)		1	-.34(.56)	0.37	0.71	.24-2.15
	Axis I Disorder		1	.42(.23)	3.22	1.51 †	.96-2.38
Step 2		0.40	1				
	Child Diagnosis		1	.70(.30)	5.21	2.06*	1.10-3.64
	Race (1)		1	-1.00(.24)	17.27	0.37***	.23-.59
	Race (2)		1	-.37(.56)	0.42	0.69	.23-2.10
	Axis I Disorder		1	.43(.23)	3.37	1.53	.94-1.12
	Set Shifting Time		1	.02(.04)	0.16	1.02	.94-1.11
	Overall χ^2	33.59***	6				
<i>N</i>			403				

Note. CDI = Children's Depression Inventory Scores. Set Shifting Time = TEACH Creature Count Time Score. †p < .10, *p < .05, **p < .01, ***p < .001.