Published in final edited form as:

Anxiety Stress Coping. 2015 January ; 28(1): 31–49. doi:10.1080/10615806.2014.925545.

Executive Function Moderates the Relation between Coping and Depressive Symptoms

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Abstract

Background and Objectives—Identifying risk factors early in the course of depression has important implications for prevention, given that the likelihood of recurrence increases with each successive episode.

Design—This study examined relations among coping, executive functioning, and depressive symptom trajectories in a sample of remitted-depressed ($n = 32$) and never-depressed ($n = 36$) young adults (ages 18 to 31).

Methods—Participants completed a clinical interview, a measure of coping, and tasks assessing two components of executive function -- inhibition and cognitive flexibility. Participants were re-assessed regarding the timing and severity of depressive symptoms that had occurred during the interval period (mean = 35.16 weeks, $SD = 9.03$).

Results—Among never-depressed individuals, less primary control coping (e.g., problem-solving) and greater disengagement coping (e.g., avoidance) predicted increases in depressive symptoms. Greater secondary control coping (e.g., acceptance) predicted decreases in depressive symptoms and was unrelated to depression history. Higher inhibition scores predicted less increase in depressive symptoms for individuals reporting less primary control coping or more disengagement coping. Higher cognitive flexibility scores predicted less increase in depressive symptoms among individuals reporting less secondary control coping.

Conclusions—Interventions aiming to enhance either coping strategies or executive functions may reduce risk for depression recurrence.

Keywords

depression; coping; executive function; stress

Major depressive disorder (MDD) is associated with alterations in both the stress response and stress regulation systems. For instance, adults with MDD exhibit increased

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hypothalamic-pituitary-adrenal (HPA) activity (Holsboer, 2000), impairments in neurocognitive functioning (Snyder, 2013), and changes in behavioral and cognitive coping responses to stress (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). Certain alterations in these systems persist into remission from a major depressive episode (MDE; McClintock, Husain, Greer, & Cullum, 2010; Snyder, 2013), although their relation to recurrence is unclear. Identifying risk factors early in the course of depression has important implications for prevention, given that the likelihood of recurrence increases with each successive MDE (Solomon et al., 2000).

The association between life stress and risk for depression is well established (e.g., Kendler, Karkowski, & Prescott, 1999). How coping processes are linked to this stress-depression relation over time is less well understood (e.g., Compas et al., 2001; Connor-Smith & Compas, 2004). Coping has been defined as “conscious volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances” (Compas et al., 2001, p. 89), and is distinguished from responses to stress that are involuntary and automatic. The present study examined a well-validated model of coping that involves either engagement or disengagement from a stressor or stress-related emotional reactions (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000). According to this model, primary control engagement coping is characterized by attempts to change the stressor or one’s emotional responses to it (i.e., problem-solving, emotional expression, emotional modulation); secondary control engagement coping involves adapting to a stressful situation by regulating attention and cognitions (i.e., acceptance, cognitive restructuring, positive thinking, distraction); and disengagement coping involves withdrawing from the stressor or emotional response (i.e., avoidance, denial, wishful thinking). Greater use of primary control engagement coping and secondary control engagement coping has been found to be associated with lower levels of internalizing symptoms (Compas et al., 2001), better adjustment to stressful situations (Connor-Smith and Compas, 2004), and lower levels of depressive symptoms (Weisz, Francis, & Bearman, 2010). In contrast, greater use of disengagement coping has been linked with higher levels of internalizing problems (Compas et al., 2001). Depressive coping style (e.g., being overwhelmed by problems) also has been associated with increased risk for recurrent depressive episodes (Ormel, Oldehinkel, & Vollebergh, 2004). The bulk of this research on the relation between coping and adjustment has been cross-sectional, which can be useful in identifying correlates of coping but cannot address the temporal links among these constructs.

The experience of depression has been likened to a “neurotoxic trauma” that may have a long-term impact on cognitive functions (Grant, Thase, & Sweeney, 2001). Cognitive theories of depression have focused on features of thought content and information processing (e.g., Beck, 1976; Bower, 1981; Ingram, 1984; Teasdale, 1988). Three primary mechanisms have been proposed to account for the association between processing biases and emotion dysregulation in depression: inhibitory processes, working memory deficits, and ruminative responses (see Gotlib & Joorman, 2010, for a review). Deficits in executive functions, which include a set of interrelated, higher-order cognitive functions, also characterize some depressed individuals and can affect emotion regulation (McClintock et al., 2010; Snyder, 2013).

Executive function allows information to be maintained and manipulated, while impulses are controlled, for the purposes of goal-directed activity (Miyake et al., 2000). Executive function includes such cognitive processes as cognitive flexibility, planning, inhibitory control, error detection and correction, working memory, and fluency. Confirmatory factor analyses reveal that executive function can be divided into three separable, but related, factors: *updating*, *cognitive flexibility*, and *inhibition* (Miyake et al., 2000). Updating involves active manipulation of information in working memory. Cognitive flexibility, which also is referred to as “shifting” or “task switching,” involves transitioning between tasks, operations, or mental sets (Monsell, 1996). Inhibition involves the deliberate, controlled suppression of dominant, automatic, or prepotent responses (Miyake et al., 2000).

Investigations of whether impairment in executive function domains persists after recovery from an MDE have yielded inconsistent findings. Whereas some studies have reported lingering impairment in certain cognitive functions (e.g., Gualtieri, Johnson, & Benedict, 2006; Hammar, Lund, & Hugdahl, 2003; Paelecke-Habermann, Pohl, & Lepow, 2005), others have not found persistent neurocognitive impairment (e.g., Biringier et al., 2005) in persons whose depression has remitted. Factors that may contribute to these heterogeneous findings include depression severity, depression history, and time since last MDE (Hamman & Ardal, 2009; McClintock et al., 2010). The first aim of the current study was to compare individuals with and without a prior history of MDE with regard to executive function components and coping strategies. We examined inhibition and cognitive flexibility in particular because impairments in these executive function domains have been shown to persist even after remission from a MDE (McClintock et al., 2010; Snyder, 2013). Few longitudinal studies, however, have explored the association between cognitive functions and depression history; whether cognitive impairment predicts recurrence of depression (Hammar & Ardal, 2009), or whether the relations between coping strategies or executive function domains and depressive symptom trajectories vary as a function of depression history.

Coping may recruit multiple executive function components, particularly working memory, planning, sequencing, cognitive flexibility and inhibitory control (Compas, 2006). For example, coping effectively with a stressor may involve holding prior encounters with similar stressors in mind (i.e., working memory) while evaluating coping alternatives and adjusting cognitive and behavioral responses (i.e., cognitive flexibility) (Compas, 2009). Hence, an individual’s capacity to select and implement coping strategies that forestall the onset of depression may be affected by the executive function components that subserve these strategies.

In a study of youth who had survived acute lymphocytic leukemia, Campbell and colleagues (2009) showed that good executive functioning (i.e., working memory, cognitive flexibility, self-monitoring) was positively associated with primary and secondary control coping and negatively associated with disengagement coping. Positive associations between secondary control coping and working memory also have been found in a sample of young adults (Andreotti et al., 2013), and cognitive inflexibility has been linked to repetitive thoughts and rumination (Davis & Nolen-Hoeksema, 2000). Additionally, a connection between cognitive functions and coping in depressed individuals has been found in studies of emotion

regulation. In particular, the effectiveness of a strategy such as reappraisal has been associated with memory biases, impaired inhibition, and difficulty disengaging attention from negative stimuli (Gotlib & Joorman, 2010).

The final aim of the current study was to test whether the prospective relation between coping and changes in depressive symptoms over time varied by executive function components -- inhibition and cognitive flexibility. Relative strengths or weaknesses in particular executive functions may either enhance or interfere with the selection and effective implementation of coping strategies, and thereby impact psychological adjustment (Compas & Boyer, 2001; Wadsworth, Raviv, Compas, & Connor-Smith, 2005). That is, these executive function components could facilitate adaptive coping, exacerbate maladaptive coping, or mitigate against the harmful effects of maladaptive coping. For example, Oldehinkel and colleagues (2007) demonstrated that adolescents with greater internalizing problems had a combination of cognitive deficits (low effortful control) and poor stress regulation (high negative emotionality). In a sample of individuals with posttraumatic stress disorder, Aupperle and colleagues (2012) found that difficulty inhibiting automatic stress responses was associated with a greater use of maladaptive coping strategies, such as avoidance.

Mediation models assume change and examine mechanisms through which one variable (e.g., stress) affects another variable (e.g., psychopathology). In contrast, moderator models test differences in the relations between two variables (e.g., stress and psychopathology) as a function of some pre-existing and presumably stable other characteristic(s) (Rose, Holmbeck, Coakley & Franks, 2004). Some researchers have argued that mediational models of coping are particularly appropriate during childhood, when coping strategies are undergoing change shaped by developmental factors and by stressors themselves (Campos, Campos & Barrett, 1989). That is, stressors can undermine a child's ability to effectively implement a coping strategy, which in turn may alter the developmental trajectories of regulatory processes (Skinner & Zimmer-Gembeck, 2007) and increase risk for psychopathology. By adulthood, coping has become relatively more stable and trait-like (Hewitt & Flett, 1996), and may be best considered a moderator of the relation between stress and psychopathology (Wadsworth et al., 2005). According to this perspective, stressors are more likely to predict psychopathology for adults who employ maladaptive coping strategies (for a review of how coping can serve as a mediator or moderator, see Rose et al., 2004).

In summary, the present study addressed the following research questions: (1) To what extent do the executive function components of inhibition and cognitive flexibility and coping strategies (i.e., primary control, secondary control, disengagement) characterize individuals with and without a prior history of a MDE? (2) Do the relations of (a) these executive function components and (b) coping strategies with changes in weekly depressive symptoms over the follow-up period differ by depression history (i.e. remitted vs. never depressed)? (3) Do the relations between each type of coping and changes in depressive symptoms vary by executive function -- inhibition and cognitive flexibility?

Method

Participants

Participants were 68 individuals, age range 18 to 31 years (mean age = 23.39, $SD = 3.88$), recruited from undergraduate and graduate programs at a mid-size university in the southeastern United States and through a medical center listserv email; participants were part of a larger study of stress and depression. All participants were screened for current and prior MDEs. Individuals had either (a) a history of MDE but were not currently in a depressive episode (i.e., remitted depressed), or (b) no current or prior MDE (i.e., never depressed). The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996) was administered to evaluate clinical diagnoses according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition (DSM-IV-TR; American Psychiatric Association, 2000) criteria. Full remission was defined as an absence of significant symptoms of depression for at least two months according to the criteria outlined by Frank and colleagues (1991). Exclusion criteria were current MDE, lifetime bipolar disorder or posttraumatic stress disorder (PTSD), or health conditions (e.g., Cushing's disease, Addison's disease, diabetes) or medications (e.g., corticosteroids, amphetamines) known to affect stress response systems. This resulted in one person being excluded due to pregnancy and another for use of mirtazapine; these individuals were excluded after the screening process.

Of 32 remitted-depressed participants, 13 had experienced one prior MDE, 10 had had two episodes, and 9 had experienced 3 episodes. The never-depressed group consisted of 36 individuals. Participants from the larger study ($n = 102$) who expressed interest in the follow-up assessment were re-contacted to schedule a telephone interview (mean weeks in study = 35.16, $SD = 9.03$); all those who agreed to participate ($n = 68$) completed the assessment. Participants who completed the follow-up assessment did not differ from those who only completed the baseline assessment with regard to their age ($t = 1.58, p = .12$), SES ($t = 1.07, p = .29$), sex ($X^2 = .02, p = .88$), depressive symptoms ($t = .15, p = .88$), inhibition ($t = .41, p = .69$), primary control coping ($t = .48, p = .64$), secondary control coping ($t = 1.11, p = .27$), or disengagement coping ($t = .28, p = .78$). Remitted-depressed participants, however, comprised a larger proportion of those who did not express interest in the follow-up assessment (10 never-depressed individuals, 24 remitted-depressed individuals; $X^2 = 5.07, p = .02$). In addition, cognitive flexibility was higher in individuals who did the follow-up ($t = 2.29, p = .03$).

Written informed consent was obtained at baseline (Time 1); for follow-up assessments (Time 2), consent forms were mailed to participants and telephone interviews were conducted after the signed forms were returned. Participants received either 6 course credits or \$30 for the T1 assessment and \$10 for the follow-up assessment (T2). All procedures were approved by the University Institutional Review Board.

Measures at Time 1

Socioeconomic status (SES) was calculated using the Hollingshead (1975) four-factor index based on employment status, occupation, highest level of education, and marital status.

Depression—The SCID-I was used to assess current and lifetime diagnoses of a subset of Axis I disorders (i.e., MDD, bipolar disorder, PTSD). Detailed information about the total number of previous MDEs was obtained. All interviews were audio-taped; a random 20% were re-rated for reliability by an independent evaluator. Inter-rater reliability for history of depression yielded a kappa = 1.00.

The 21-item *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) was used to assess current level of depressive symptoms. The BDI-II has good reliability and validity (Beck, Steer, Ball, & Ranieri, 1996); coefficient alpha in this sample was .85.

Coping—The 57-item young adult peer stress version of the Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000) was used to assess coping responses. Items described ways in which individuals might respond to stressful peer interactions (e.g., “*Being left out or rejected.*” “*Having problems with a friend.*”). Participants rated how often they responded in the manner described, using a Likert scale (1 = not at all; 2 = a little; 3 = some; 4 = a lot). Proportional scoring methods were used to adjust for base rate differences in endorsement of coping items; each subscale score (i.e., the total score of all items endorsed for either primary control coping, secondary control coping, or disengagement coping) was divided by the total number of items endorsed for all coping methods by each individual on the RSQ (Connor-Smith et al., 2000). Hence, scores reflect the degree to which individuals used a particular subscale relative to their use of all other coping and involuntary stress response approaches. The coping strategies examined in the current study were: primary control engagement coping ($\alpha = .81$), secondary control engagement coping ($\alpha = .83$), and disengagement coping ($\alpha = .64$).

Executive Function -- Inhibition—The Stroop Color and Word Test (Golden, 1978) yields three scores (i.e., word reading, color-naming, color-word) based on three stimulus sheets, takes approximately five minutes to complete, and was administered to participants by the first author (MCM). This measure can be used to compute an interference score that reflects the ability to inhibit an over-learned response (i.e., word reading) by correctly identifying the printed color of a word when that color does not correspond to the word itself. Consistent with recommendations by Lansbergen and colleagues (2007), an interference score (I_R) was computed as the ratio of the raw score for the color-word condition to the raw score of the color-naming condition.

Executive Function -- Cognitive flexibility—The Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993) computer version was used to assess participant’s ability to develop and maintain an appropriate problem-solving strategy across changing stimulus conditions to achieve a goal. This measure is not timed. Participants are instructed to match cards according to three categories (color, shape, number of objects) to four reference cards that remain constant throughout the study; the sequence of categories was repeated once (i.e., each participant completed up to 6 categories). Feedback (“right” or “wrong”) is provided by the computer after each match allowing participants to learn the classification rule. The rule then changes after a fixed number of correct matches without notifying participants, which thereby requires them to adjust their sorting method. The learning-to-learn index was calculated from the percentage

of errors on the first presentation of the three categories; positive scores reflect improvements in processing efficiency across categories.

Follow-up Measures

Depression—The Longitudinal Interval Follow-Up Evaluation (LIFE; Keller et al., 1987), which parallels the SCID-I, was administered at the follow-up assessment, by the first author and a trained research assistant, to measure depressive symptoms and disorders during the follow-up interval. Participants were provided with chronological ‘anchors’ (e.g., salient events, holidays) to increase recall. The LIFE yields a depression symptom rating score from 1 to 6, reflecting the extent of depressive symptoms and impairment for each week of the follow-up interval. A score of 3 indicates fewer symptoms (e.g., two to three symptoms) than full *DSM-IV-TR* criteria with mild or moderate impairment; a score of 4 indicates four symptoms with moderate to marked impairment; and 5 indicates an MDE according to *DSM-IV-TR* criteria, and significant impairment. Inter-rater reliability for depression symptom rating scores over the follow-up yielded a kappa of 1.00.

Data Analytic Plan

All variables were examined for distributional properties and cases were screened for univariate and multivariate outliers. One participant was removed from analyses of cognitive flexibility, due to having a score that was four standard deviations higher than the mean. To address hypotheses regarding within- and between-individual change simultaneously over the follow-up interval, we specified a series of multilevel models using HLM6 (Raudenbush, Bryk, & Congdon, 2004) consisting of a within-person (i.e., level-1) sub-model describing how each individual’s weekly depressive symptoms changed over time, and a between-person (i.e., level-2) sub-model describing how these changes in weekly depressive symptoms varied across individuals (Bryk & Raudenbush, 1992; Singer & Willett, 2003). All multilevel models utilized the maximum number of weekly data points available for each participant, which was influenced by the timing of their follow-up interview.

Multilevel modeling was used to first examine whether relations between coping scales or executive function measures (inhibition, cognitive flexibility) and changes in weekly depressive symptoms over follow-up differed by depression history. These models included the following variables: level-2 covariates [person mean of depressive symptoms over follow-up, baseline depressive symptoms (BDI-II)]; level-1 predictor (time); level-2 predictors [depression history, coping (or executive function)]; two-way interactions [depression history X coping (or executive function), depression history X time, coping (or executive function) X time]; three-way interaction of depression history, coping (or executive function), and time.

Next, multilevel modeling was used to examine whether relations between coping scales and changes in weekly depressive symptoms varied by executive function measures. These models included the following variables: level-2 covariates [person mean of depressive symptoms over follow-up, baseline depressive symptoms (BDI-II), depression history]; level-1 predictor (time); level-2 predictors (coping, executive function); two-way

interactions (coping X executive function; coping X time; executive function X time); three-way interaction of coping, executive function, and time.

All participants (i.e., both remitted- and never-depressed individuals) were included in analyses examining executive function X coping interactions so as to have variability in the key constructs (i.e., coping, depressive symptoms) and thereby reduce potential problems with restriction of range. Covariates and predictors included in all interactions were centered. Simple slope analyses and slope difference tests were conducted on all significant three-way interactions (Aiken & West, 1991; Dawson & Richter, 2006). Figures depicting interactions present time from baseline to the mean follow-up week (i.e., weeks 1 to 35). Hereafter when describing results of simple slopes analyses, “high” or “higher” refers to 1 standard deviation above the mean and “low” or “lower” refers to 1 standard deviation below the mean. To adjust for multiple tests for each coping scale, a Bonferroni correction procedure was used (corrected $\alpha = .025$).

Results

Table 1 presents the means and standard deviations of all T1 study variables for remitted-depressed and never-depressed individuals. Remitted-depressed participants reported significantly less use of primary and secondary control coping compared to never-depressed participants; the groups did not differ on disengagement coping, inhibition, or cognitive flexibility. Group status (remitted- versus never-depressed) and baseline depressive symptoms (BDI-II) were included as covariates in subsequent analyses. SES did not differ significantly between remitted- and never-depressed individuals, and therefore was not included in subsequent data analyses. Table 2 presents correlations among depression history, coping, and executive function variables at T1.

What were the Relations between Coping Strategies or Executive Function Domains and Depressive Symptom Trajectories over Follow-up, and did Depression History Moderate these Relations?

Coping—Greater use of primary control coping predicted decreases in depressive symptoms over the follow-up ($B = -.003$, $t(2,390) = 2.72$, $p = .007$); depression history moderated this relation ($B = .005$, $t(2,386) = 2.14$, $p = .032$). Simple slope analyses revealed that depressive symptoms increased for the remitted-depressed group at both higher ($B = .016$, $t = 4.31$, $p < .0001$) and lower ($B = .014$, $t = 4.89$, $p < .0001$) use of primary control coping. In the never-depressed group, lower use of primary control coping predicted significant increases in depressive symptoms ($B = .012$, $t = 3.79$, $p = .0002$), whereas higher use of primary control coping did not predict significant change in depressive symptoms ($B = .004$, $t = 1.63$, $p = .103$). Slope difference tests revealed that among individuals reporting high primary coping, depressive symptoms increased more rapidly for remitted-depressed individuals than never-depressed individuals ($t = 3.67$, $p = .001$). For secondary control coping, lower use significantly predicted increases in depressive symptoms ($B = -.009$, $t(2,390) = 8.68$, $p < .0001$); depression history did not moderate this relation ($B = -.0004$, $t(2,386) = 0.17$, $p = .869$).

Greater use of disengagement coping predicted increases in depressive symptoms ($B = .004$, $t(2,390) = 3.97$, $p < .0001$); depression history significantly moderated this relation ($B = -.011$, $t(2,386) = 5.28$, $p < .0001$). Simple slope analyses revealed that depressive symptoms increased for remitted-depressed individuals with both higher ($B = .014$, $t = 6.10$, $p < .0001$) and lower ($B = .016$, $t = 6.16$, $p < .0001$) use of disengagement coping. For the never-depressed group, higher disengagement coping significantly predicted increases in depressive symptoms ($B = .018$, $t = 6.07$, $p < .0001$), whereas lower disengagement coping was not significantly related to changes in depressive symptoms ($B = .002$, $t = 0.75$, $p = .456$). Slope difference tests revealed that depressive symptoms increased more rapidly for remitted-depressed individuals reporting higher disengagement coping ($t = 4.11$, $p < .001$), remitted-depressed individuals reporting lower disengagement coping ($t = 4.81$, $p < .001$), and for never-depressed individuals reporting higher disengagement coping ($t = 5.16$, $p < .001$) relative to never-depressed individuals reporting low disengagement coping.

Executive Function—Multilevel modeling was used to examine prospective, within-person relations of executive function domains to depressive symptoms over the follow-up and whether depression history moderated these relations. Results indicated that inhibition was not significantly associated with change in depressive symptoms over time ($B = -.002$, $t(2,389) = 1.54$, $p = .122$), and depression history did not moderate this relation ($B = .004$, $t(2,386) = 1.73$, $p = .084$). Similarly, cognitive flexibility was not associated with changes in depressive symptoms over time ($B = -.004$, $t(2,214) = 1.27$, $p = .206$), and this relation was not significantly moderated by depression history ($B = -.012$, $t(2,211) = 1.92$, $p = .055$).

Does the Relation between Primary Control Coping and Changes in Depressive Symptoms vary by Executive Function?

Inhibition—Multilevel modeling analyses revealed that the relation between primary control coping and changes in depressive symptoms over follow-up varied significantly by inhibition ($B = .003$, $t(2,385) = 2.39$, $p = .017$). Simple slope analyses revealed that depressive symptoms increased significantly for individuals who reported lower use of primary control coping and poor performance in the inhibition domain ($B = .03$, $t = 6.67$, $p < .0001$). Depressive symptoms did not change significantly over follow-up for individuals reporting lower use of primary coping for those who had good performance in the inhibition domain ($B = -.006$, $t = 1.40$, $p = .162$) or for individuals reporting higher primary coping use who had either low ($B = .008$, $t = 1.78$, $p = .076$) or high inhibition ($B = .008$, $t = 1.74$, $p = .082$) scores. Slope difference tests revealed that depressive symptoms increased more rapidly for individuals who were low in inhibition and lower in primary control coping use compared to individuals who were high in inhibition and high in primary control coping use ($t = 3.45$, $p = .001$), low in inhibition and high in primary control coping use ($t = 3.88$, $p < .001$), or high in inhibition and low in primary control coping use ($t = 3.37$, $p = .001$).

Cognitive Flexibility—Multilevel modeling analyses revealed that the primary control coping X cognitive flexibility interaction did not significantly predict changes in depressive symptoms over follow-up ($B = .004$, $t(2,210) = 1.56$, $p = .120$).

Does the Relation between Secondary Control Coping and Changes in Depressive Symptoms vary by Executive Function?

Inhibition—Multilevel modeling analyses revealed that the secondary control coping X inhibition interaction did not significantly predict changes in depressive symptoms over follow-up ($B = -.002$, $t(2,385) = 1.45$, $p = .149$).

Cognitive Flexibility—Multilevel modeling analyses indicated that the secondary control coping X cognitive flexibility interaction ($B = .011$, $t(2,210) = 4.24$, $p < .0001$) significantly predicted changes in depressive symptoms over follow-up (Figure 2). Simple slope analyses revealed that low secondary control coping predicted significant increases in depressive symptoms over the follow-up interval for individuals who were low in cognitive flexibility ($B = .027$, $t = 3.99$, $p = .0001$). Low secondary control coping did not predict significant changes in depressive symptoms for those with high cognitive flexibility ($B = -.003$, $t = 0.49$, $p = .625$). The relation between high secondary control coping and depressive symptoms over the follow-up was not significant for those with either high ($B = .011$, $t = 1.70$, $p = .090$) or low ($B = -.003$, $t = 0.42$, $p = .676$) cognitive flexibility. Slope difference tests revealed that depressive symptoms increased more rapidly for individuals low in flexibility reporting lower secondary coping use compared to individuals who were low in flexibility and higher in secondary control coping use ($t = 6.41$, $p < .001$), high in flexibility and higher in secondary control coping use ($t = 3.19$, $p = .002$), or high in flexibility and lower in secondary control coping use ($t = 3.38$, $p = .001$). In addition, in individuals reporting higher secondary control coping use, depressive symptoms increased more rapidly for those with high as compared to low flexibility ($t = 2.17$, $p = .034$).

Does the Relation between Disengagement Coping and Changes in Depressive Symptoms vary by Executive Function?

Inhibition—Multilevel modeling analyses revealed that the disengagement coping X inhibition interaction ($B = -.006$, $t(2,385) = 5.41$, $p < .0001$) significantly predicted changes in depressive symptoms over the follow-up (Figure 3). Simple slope analyses revealed that high disengagement coping predicted significant increases in depressive symptoms over the follow-up interval for those low in inhibition ($B = .020$, $t = 3.64$, $p < .001$). Change in depressive symptoms was not significant for individuals with high disengagement coping and high inhibition ($B = .004$, $t = 0.70$, $p = .484$), or for those low in disengagement coping and either high ($B = .010$, $t = 1.81$, $p = .071$) or low inhibition ($B = .002$, $t = 0.36$, $p = .718$). Slope difference tests revealed that depressive symptoms increased more rapidly for individuals low in inhibition and higher in disengagement coping use than for those reporting lower disengagement coping use ($t = 2.03$, $p = .047$).

Cognitive Flexibility—Multilevel modeling analyses revealed that the disengagement coping X cognitive flexibility interaction ($B = -.006$, $t(2,210) = 1.94$, $p = .052$) predicted changes in depressive symptoms over follow-up at the level of a non-significant trend (Figure 4). Exploratory simple slope analyses were conducted to examine the pattern of this interaction. High disengagement coping predicted significant increases in depressive symptoms over the follow-up period for those with low cognitive flexibility ($B = .030$, $t = 3.31$, $p = .0009$). Change in depressive symptoms was not significant for individuals with

high disengagement coping and high cognitive flexibility ($B = -.010, t = 1.15, p = .251$), or for those low in disengagement coping and with either high ($B = .012, t = 1.55, p = .122$) or low cognitive flexibility ($B = .004, t = 0.45, p = .650$). Slope difference tests revealed that depressive symptoms increased more rapidly for individuals low in flexibility and higher in disengagement coping than individuals high in flexibility and higher in disengagement coping use ($t = 2.28, p = .026$), and for individuals low in flexibility who reported lower disengagement coping use ($t = 2.11, p = .039$).

Discussion

The present study assessed coping, executive function (inhibition, cognitive flexibility), and depressive symptom trajectories over a follow-up period (mean duration of 35 weeks) in individuals with and without a history of major depressive episodes. Models examining depression history as a moderator of the relations between coping and depressive symptom trajectories revealed a similar pattern for primary control coping and disengagement coping. Among never-depressed individuals, less use of primary control coping and greater use of disengagement coping predicted increases in depressive symptoms; for remitted-depressed individuals, depressive symptoms increased over time, but were not related to their use of primary control or disengagement coping. Greater use of secondary control coping predicted decreases in depressive symptoms over time and was unrelated to depression history. Thus, secondary control coping, which involves adapting to stressful conditions by modifying interpretations of events, accepting present circumstances, discovering “silver linings,” and engaging in distractions that interrupt ruminative processes, may be especially helpful in reducing the likelihood of increases in depressive symptoms.

Impairment in cognitive functioning may precede the first onset of a depressive episode, emerge during a MDE and show delayed normalization following episode recovery, or may predict increased risk for recurrence of depression (Hammar & Ardal, 2009). The present study, however, found that executive function was not related to depression history and did not predict change in depressive symptoms over time. Hammar and Ardal (2009) suggested that heterogeneity in depression characteristics (e.g., depression severity and chronicity) and treatment factors (e.g., medication use and hospitalization) may contribute to inconsistencies in this literature. The present study, therefore, controlled for baseline depression severity and history of depression; including these covariates did not affect the pattern of findings. It is possible that executive function impairments had recovered among individuals whose depression had remitted, or that there was not sufficient variability on executive function measures in this primarily college student sample.

Inhibition and cognitive flexibility were found to moderate the relations between coping and depressive symptoms. Lower inhibitory control combined with less use of primary control coping was associated with lower depressive symptoms at baseline but predicted increases in depressive symptoms over the follow-up. Inhibitory control deficits, particularly in the capacity to disengage attention from negative stimuli, have been found to interfere with recovery from negative affect and may be a risk for depression (Gotlib & Joorman, 2010). We also found that among individuals who used less primary control coping, higher scores on inhibition predicted lower depression scores over time, and could function as a protective

factor. Finally, for individuals who used more primary control coping, lower scores on inhibition was not related to increases in depression. Thus, use of primary control coping (e.g., problem-solving) may overcome the possible negative effects of low inhibitory control.

In addition, cognitive flexibility moderated the relation between secondary control coping and depressive symptom trajectories. When secondary control coping was low, higher cognitive flexibility predicted lesser increase in depressive symptoms over the follow-up, whereas when use of secondary control coping was higher, lower cognitive flexibility did not significantly predict changes in depression. Low cognitive flexibility, as indexed by poorer performance on the WCST, has been linked to current depression (Grant et al., 2001; Snyder, 2013) and rumination (Davis & Nolen-Hoeksema, 2000). The current results indicate that the relation between cognitive flexibility and depressive symptoms may be further modified by the coping strategies used, particularly secondary control coping.

Secondary control coping strategies include cognitive restructuring and attempts to reinterpret stressful situations in more balanced or neutral terms. Cognitive flexibility may be especially relevant to such secondary control coping strategies (Campbell et al., 2009) as well as to a wider range of flexible thoughts and behaviors. Individuals who do not utilize secondary coping strategies, however, may still benefit from higher cognitive flexibility because they exhibit other types of flexible responses to stress. Using a greater range of cognitive or behavioral strategies to manage stressful situations has been associated with lower physiological (e.g., cortisol) reactivity to interpersonal stressors (Roubinov, Hagan, & Luecken, 2012).

Currently depressed individuals exhibit cognitive processing biases that are related to problems with emotion regulation and, in turn, sustained negative affect; these cognitive biases are evident during exposure to negative material and include executive control deficits, rumination, and difficulty disengaging (Gotlib & Joorman, 2010). The present study demonstrated that the combination of lower inhibition and greater use of disengagement coping was associated with lower depressive symptoms at baseline but then predicted significant increases in depressive symptoms over follow-up. Individuals with poorer inhibitory control may be particularly unsuccessful in managing their automatic behavioral responses to stress. In contrast, among individuals who were better able to suppress dominant, automatic or prepotent responses on the Stroop Color and Word Test, greater use of maladaptive coping strategies such as avoidance, denial, and wishful thinking did *not* predict increases in depressive symptoms. Overall, these findings are consistent with evidence that cognitive dysfunction is associated with elevated depressive symptoms in individuals who engage in more disengagement (avoidance) coping (Arnett, Higginson, Voss, Randolph, & Grandey, 2002).

Results of the current study should be interpreted in light of its limitations. First, whereas some previous studies have shown that executive function was associated positively with adaptive coping and negatively with maladaptive coping (Campbell et al., 2009), we did not find significant correlations between the coping subscales and the executive function measures (i.e., inhibition or cognitive flexibility). This lack of association might have been

due to a restricted range on executive function scores in this predominantly university student sample who mostly fell within the normal range on the measures of executive function. Future longitudinal studies should address this possible ‘ceiling effect’ by including individuals with greater variability in executive function performance. A related concern is that participants who completed the follow-up assessment had higher scores on the cognitive flexibility measure compared to those who only completed the baseline assessment, which could limit generalizability of the present findings. The absence of a significant relation between executive function domains and depression also could have been a result of low power due to the relatively small sample; therefore larger samples should be used in future investigations.

Second, the possible contribution of other concurrent psychiatric conditions, particularly comorbid anxiety, to the current findings is unclear because participants were only assessed for mood disorders and PTSD. Future studies of the relations among coping, executive function, and depressive symptoms should assess other comorbid disorders. Third, the coping measure was based on self-reports of participants’ responses to specific social stressors. The links between subjective and objective indices of coping and their respective relations to depression need further study (Compas, Jaser, Dunn, & Rodriguez, 2012). Fourth, the disengagement coping subscale of the RSQ had a relatively low coefficient alpha, and therefore results with this measure should be interpreted with some caution. Exploratory analyses revealed that alpha for the never-depressed group was adequate ($\alpha = .70$) but less so for the remitted-depressed group ($\alpha = .56$). Future studies should assess the reliability of the RSQ disengagement coping subscale in previously depressed individuals. Finally, variability in the timing of follow-up interviews could have affected the results because participants contributed somewhat different amounts of data and longer follow-up durations may have increased possible recall bias.

Overall, the current study showed that secondary control coping predicted decreases in depressive symptoms over follow-up regardless of depression history. Therefore, interventions that emphasize secondary control coping strategies should be a focus of depression prevention programs (e.g., see Compas et al., 2012). Greater use of primary control coping and less use of disengagement coping predicted changes in depressive symptoms for never-depressed participants, but not for remitted depressed individuals. If replicated, these results might suggest that interventions targeting different coping approaches may be needed for preventing first onsets of depression as compared to recurrences.

We also found that although remitted depressed individuals did not have impaired inhibition or cognitive flexibility relative to never-depressed individuals, these executive function components significantly interacted with coping to predict increases in depressive symptoms during the 35 week follow-up interval. Interventions that aim to enhance either coping strategies or executive functions may reduce risk for depression recurrence. Some evidence exists that executive function interventions may be helpful adjuncts to standard treatments for depression (Siegle, Ghinassi, & Thase, 2007). Overall, findings from this study can potentially guide the creation of more personalized interventions for depression that target individuals’ coping styles and executive function profiles. Development of such programs is

critical given the prevalence of depression and its impact on disability and health-related quality of life (Moussavi et al., 2007).

Acknowledgments

Matthew C. Morris was supported in part by a Ruth L. Kirschstein Individual National Research Service Award (F31 MH084425), an American Psychological Foundation Elizabeth Munsterberg Koppitz Graduate Student Fellowship, a Vanderbilt Institute for Clinical and Translational Research Resource Request Award (UL1 RR024975/TR000445), an RCTR/MeTRC grant (U54 RR026140/MD007593), and an independent grant (R01 MH068391) and training grant (T32 MH018921) from the National Institute of Mental Health. Lindsay D. Evans was supported in part by National Institutes of Health Grants (RC1 MH08832, R01 MH64735). Uma Rao was supported in part by the grants from the National Institutes of Health (R01 DA017805, R01 MH068391, G12 RR003032/MD007586, UL1 RR024975/TR000445 and U54 RR026140/MD007593), and by the Endowed Chair in Brain and Behavior Research at Meharry Medical College. Judy Garber was supported in part by National Institutes of Health grants R01 MH64735, R01 MH088329, and UL1 RR024975/TR000445 during the completion of this work. These funding agencies had no further role in the study design, data collection, analysis or interpretation of data, writing of the report, or the decision to submit the paper for publication. The authors gratefully acknowledge all individuals who participated in this study. We also thank Bruce Compas for his comments on an earlier version of this article.

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Figure 1.

Interaction of primary control engagement coping and inhibition predicting changes in depressive symptoms from baseline (week 1) to follow-up (week 35) in remitted- and never-depressed individuals. Estimated means for simple slopes are plotted for ± 1 standard deviation. * $p < .0001$.

Figure 2.

Interaction of secondary control engagement coping and cognitive flexibility predicting changes in depressive symptoms from baseline (week 1) to follow-up (week 35) in remitted- and never-depressed individuals. Estimated means for simple slopes are plotted for ± 1 standard deviation. $*p = .0001$.

Figure 3.

Interaction of disengagement coping and inhibition predicting changes in depressive symptoms from baseline (week 1) to follow-up (week 35) in remitted- and never-depressed individuals. Estimated means for simple slopes are plotted for ± 1 standard deviation. * $p < .001$.

Figure 4.

Interaction of disengagement coping and cognitive flexibility predicting changes in depressive symptoms from baseline (week 1) to follow-up (week 35) in remitted- and never-depressed individuals. Estimated means for simple slopes are plotted for ± 1 standard deviation. $*p = .0009$. Note: this interaction predicted change in depressive symptoms at the level of a non-significant trend ($p = .052$).

Table 1

Means and standard deviations of study variables for remitted depressed and never depressed individuals at Time 1.

	Remitted Depressed (<i>n</i> = 32)	Never Depressed (<i>n</i> = 36)	RD vs. ND
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>X</i> ² / <i>t</i>
Age (years)	23.9 (3.9)	23.0 (3.9)	0.98
Education (years)	15.5 (2.7)	15.1 (2.7)	0.62
Socioeconomic Status (SES)	53.3 (11.6)	54.7 (12.7)	0.49
	<i>N</i> (%)	<i>N</i> (%)	<i>X</i> ² / <i>t</i>
Sex			3.60
Male	8 (25.0)	17 (47.2)	
Female	24 (75.0)	19 (52.8)	
Race			0.32
Caucasian	23 (71.9)	28 (77.8)	
Non-Caucasian	9 (28.1)	8 (22.2)	
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>t</i>
Depressive symptoms (BDI-II)	8.72 (6.6)	3.42 (3.4)	4.11 ***
Executive Function			
Inhibition (<i>I</i> _R)	0.64 (0.08)	0.66 (0.08)	0.62
Cognitive flexibility (WCST)	0.57 (2.7)	0.77 (4.3)	0.23
Coping			
Primary control coping	0.19 (0.04)	0.22 (0.04)	3.26 **
Secondary control coping	0.23 (0.06)	0.26 (0.04)	2.52 *
Disengagement coping	0.14 (0.03)	0.13 (0.02)	0.62

* $p < .05$;

** $p < .01$;

*** $p < .001$;

RD = remitted depressed; ND = never depressed; BDI-II = Beck Depression Inventory, second edition; Inhibition (*I*_R) = ratio of Stroop color-word score to color-naming score; Cognitive flexibility (WCST) = Wisconsin Card Sorting Test – learning to learn index.

Table 2

Correlations of Depression, Coping, and Executive Function Measures

Variable	1	2	3	4	5	6	7	8	9
1. Age	--								
2. Sex	-.02	--							
3. MDD history	.08	.14	--						
4. T1 Depressive symptoms (BDI-II)	-.08	.07	.46***	--					
5. Primary Control Coping (RSQ)	-.12	.14	-.32**	-.44***	--				
6. Secondary Control Coping (RSQ)	.03	-.07	-.39***	-.59***	.36***	--			
7. Disengagement Coping (RSQ)	.08	-.09	.07	.29**	-.56***	-.26*	--		
8. Inhibition (Stroop)	-.17	-.01	-.11	.04	-.04	-.02	-.13	--	
9. Cognitive Flexibility (WCST)	.32**	-.07	.03	-.04	-.08	.06	.05	.04	--

* $p < .05$;

** $p < .01$;

*** $p < .001$.

Note: Sex (0 = male, 1 = female); MDD (0 = never depressed; 1 = formerly depressed); BDI-II = Beck Depression Inventory, second edition; RSQ = Responses to Stress Questionnaire; WCST = Wisconsin Card Sorting Test.