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Executive function deficits associated with current and past major depressive symptoms

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Abstract

Background—Although there has been extensive research showing that depression is associated with executive function (EF) deficits, the nature of these deficits is not clearly delineated. Specifically, previous reviews on this topic have yielded different conclusions about the particular domains of EF that are disrupted in depressed individuals. Further, research on whether these deficits persist after depressed mood has remitted is less prevalent and not consistent.

Methods—In two independent samples of college students, we examined associations between clinical ratings of current and past symptoms of a Major Depressive Episode (MDE) and difficulties in two domains of EF: inhibition and shifting. In Study 1 (n=162), EF was measured using behavioral tasks shown to index these two domains. In Study 2 (n=95), EF was measured using a self-report questionnaire believed to capture EF difficulties experienced in daily life.

Results—In both studies, past MDE symptoms were associated with worse shifting. In contrast, current MDE symptoms were associated with worse inhibition, though only on the behavioral measure (in Study 1).

Limitations—Both studies used college samples and retrospective assessments of past symptoms. Further, only two domains of EF were examined, and the EF measures employed in each study have their own unique methodological limitations.

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K. Bredemeier and S.L. Warren developed the study concept and design, with Study 1 conceived and executed under the primary mentorship of H. Berenbaum and Study 2 drawing on a larger project conceived and executed by W. Heller, G.A. Miller, and others not directly involved with this manuscript. Bredemeier performed the literature search and data analyses. Bredemeier drafted the initial version of the manuscript, which was revised by all the authors. All authors approved the final version of the manuscript for submission.

Conclusions—Findings suggest that inhibition deficits vary as a function of current symptoms and thus may be a by-product of distress rather than a causal contributor. In contrast, shifting deficits associated with depression appear to be more enduring, suggesting that they could contribute to risk for depression.

Keywords

depression; executive function; inhibition; shifting; mood

Major depressive disorder (MDD) is among the most common mental disorders; the lifetime prevalence of MDD is approximately 17% (Kessler et al., 2005). Depression is associated with poor quality of life, which in turn is associated with poor work performance and social adjustment (Goldberg & Harrow, 2005; Rapoport et al., 2005). In fact, major depression has been deemed a leading cause of disability worldwide (e.g., based on years lived with severe impairment; Lopez & Murray, 1998).

Cognitive deficits may play a key role in understanding the impairment associated with depression, and possibly its etiology. Difficulties with concentration and/or decision-making are diagnostic features of MDD (APA, 2013), and research has shown that people experiencing depression display a wide range of deficits in cognitive performance, including difficulties with attention, memory, and problem-solving (see Levin et al., 2007, Hammar & Ardal, 2009, and Rock et al., 2014, for reviews). Given the breadth of these deficits, some have argued that depression is characterized by a general depletion in cognitive resources (e.g., Mathews & MacLeod, 1994). However, the results of numerous studies suggest that depressed individuals have sufficient resources but have difficulty initiating efficient cognitive strategies (e.g., Hertel & Gerstle, 2003; Marx et al., 1992; see Hertel, 1994) and/or appropriately allocating these resources (e.g., Levens et al., 2009; Yee & Miller, 1994; see Ellis & Ashbrook, 1989).

Executive function (EF) involves the effortful guidance of behavior towards a goal state. EF regulates other, non-executive cognitive processes (e.g., perception, motor responses), is particularly important in non-routine situations, and relies heavily on prefrontal cortex (Banich, 2009). In light of evidence for structural and functional abnormalities in prefrontal cortex associated with depression (e.g., Davidson et al., 2002), coupled with findings inconsistent with the general resource depletion hypothesis (e.g., Hertel & Gerstle, 2003), some have argued that the broad range of cognitive deficits observed in depressed individuals could be driven by deficits in EF (e.g., Levin et al., 2007). In line with this proposal, there is now ample evidence that depressed individuals show impaired performance on tasks that require EF (see Austin et al., 2001, Fossati et al., 2002, Ottowitz et al., 2002, Rogers et al., 2004, and Snyder, 2013, for reviews). However, research suggests that EF is multi-dimensional (e.g., Burgess et al., 1998; Fisk & Sharp, 2004; Miyake et al., 2000). In the context of such findings, the nature of the EF deficits associated with depression remains unclear. Some reviews conclude that these impairments might be specific to the domain of shifting (i.e., switching between tasks or mental “sets”; Austin et al., 2001), some suggest they are unique to inhibition (i.e., avoiding/suppressing habitual or “pre-potent” responses; Fossati et al., 2002), and still others conclude that EF is broadly

impaired in depressed individuals (e.g., Rogers et al., 2004; Snyder, 2013; see also Snyder et al., 2015).

We posit that there are two prominent reasons for this lack of clarity. First, most of the existing work on this topic uses classic but problematic EF tasks (e.g., Wisconsin Card Sorting, Tower of London). Although these measures are generally well-validated, they typically require the use of multiple aspects of EF, as well as other, non-executive abilities (for a detailed discussion of this "task impurity" problem, see Miyake et al., 2000). As a result, impaired performance is difficult to interpret. An alternative approach is to use tasks that have been shown to measure specific dimensions of EF (e.g., inhibition, shifting). Second, most of the existing work in this area has focused on current symptoms. However, depression is typically an episodic phenomenon (see Kessler et al., 1997). Thus, it is important to consider whether participants have experienced depression in the past as well, as this may be indicative of vulnerability to depression. In fact, there is some evidence to suggest that EF deficits associated with depression can persist even after depressed mood has remitted (e.g. Clark et al., 2005; Paelecke-Habermann et al., 2005). However, relatively few studies have explored this, and there have been mixed findings amongst those that have (see Hasselbalch, Knorr, & Kessing, 2011, and Rock et al., 2014).

The goal of the present research was to test contrasting hypotheses about EF deficits in depression. Clarifying the nature and time-course of EF deficits associated with depression may have important practical and clinical implications. For example, if EF deficits contribute to depression risk, interventions aimed at remediating these deficits could be used in effort to treat (or even prevent) depression. In contrast, if EF deficits in depressed individuals are simply a byproduct of their distress, there is reason to assume that they will resolve when their symptoms improve, either naturally or through treatment. Further, conclusive evidence about whether these deficits are unique to (or simply stronger in) a particular domain of EF would be helpful for honing initiatives to address EF in clinical applications. For example, such evidence might provide a clearer sense of the daily tasks on which depressed individuals are likely to struggle and what to target in remediation efforts.

Study 1

To test contrasting hypotheses from reviews regarding the nature of EF deficits associated with depression, we administered performance tasks that have been shown to measure the EF dimensions of inhibition and shifting (Miyake et al., 2000). This approach tested whether depression is associated with deficits that are specific to shifting or inhibition (as suggested by Austin et al., 2001 and Fossati et al., 2002, respectively) or a general EF deficit (evidenced by impaired performance on both tasks, consistent with the conclusions of Rogers et al., 2004, and Snyder, 2013). We did not plan to examine the EF dimension of updating/working memory, because reviews have not concluded that depression is uniquely associated with deficits in this domain and because working memory data from this sample are presented in Bredemeier & Berenbaum (2013). Motor and processing speed was also measured, to ensure that impaired performance on the EF tasks is not due to psychomotor retardation (a symptom of MDD documented on behavioral tasks; see APA, 2013, and Snyder, 2013).

To address the episodic nature of depression, we assessed participants' past as well as current symptoms. This approach should prove helpful in identifying deficits that could confer vulnerability to depression, as opposed to those that are simply a by-product of the individual's current symptoms/distress. In order to provide an even stronger test of the latter possibility, measures of mood at the time of study participation were also administered.

Methods

Participants—One hundred and sixty-two college students (57% female), ranging in age from 18 to 26 years (M=19.7), participated in the study[1]. The sample was predominantly European-American and Asian American. Of these individuals, 128 (79%) were recruited through the University of Illinois Psychology participant pool and received course credit. The remaining 34 were recruited using flyers targeting individuals who had experienced problems with depression and/or anxiety (either recently or in the past) and were paid $10/hour. The latter recruitment strategy was used to obtain better representation of individuals with elevated levels of distress in the sample. Given the nature of the performance tasks (see below), only individuals who reported normal or corrected-to-normal vision and no significant hearing problems were eligible. The same sample was used in other papers (Bredemeier & Berenbaum, 2013, and Study 2 in Bredemeier & Simons, 2012), but the questions addressed in those papers are distinct from those addressed in this one, and the data and analyses reported here were not part of those papers.

Materials

Mood measures: Current and past symptoms of a Major Depressive Episode (MDE) were assessed using the mood module of the Structured Clinical Interview for DSM-IV Disorders, Nonpatient Edition (SCID-NP; First et al., 2002). Interviews were conducted by the lead author, who has extensive experience conducting diagnostic assessments. In line with SCID guidelines, "current" was operationalized as symptoms experienced in the past month, each diagnostic criterion was rated on a three-point scale (0=absent, 1=subthreshold, 2=threshold), and additional MDE symptoms (e.g., sleep disturbance) were not assessed for participants who denied experiencing depressed mood or anhedonia (i.e., were rated as 0 for both).

Based on evidence that depression exists on a continuum of severity (see Haslam, Holland, & Kuppens, 2012), we computed dimensional symptom scores by summing ratings for all substantive MDE diagnostic criteria. To examine inter-rater reliability, secondary raters (graduate students experienced with the SCID) listened to 25 randomly selected interviews, and intraclass correlations were computed by treating raters as random effects and the individual rater as the unit of reliability (Shrout & Fleiss, 1979). The intraclass correlations for current and past MDE symptom scores were .91 and .92, respectively.

State positive affect (PA) and negative affect (NA) were measured using the Positive Affect and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PA scale

[1]Initially, 173 students participated in the study. However, 11 participants reported that they were taking anti-depressants and/or stimulant medications. In light of evidence that these types of medication can, for better or worse, influence cognitive performance (e.g., Kempton et al., 1999; McClintock et al., 2010), these participants were excluded from analysis.

consists of 10 pleasant emotion words (e.g., active, strong, proud), and the NA scale consists of 10 unpleasant emotion words (e.g., jittery, guilty, ashamed). Both scales were supplemented with 5 additional low-arousal emotion words (e.g., content, proud, bored, ashamed) to provide better coverage of the full range of positive and negative affect states. State mood was assessed by asking participants to rate each item based upon how they felt "at the moment." Because the testing session was long, the PANAS was administered twice, and (standardized) scores were averaged. Research has shown that the PANAS is a reliable and valid measure of state mood in undergraduate and community samples (e.g., Crawford & Henry, 2004; Watson et al., 1988a). Average internal consistencies (Cronbach's alpha) for PA and NA scales in the present sample were .87 and .84, respectively.

Behavioral tasks

Stop-signal task: To measure inhibition, participants completed the STOP-IT task (Verbruggen et al., 2008), a novel variant of the classic stop-signal paradigm (Logan, 1994). In this task, participants must categorize shapes as quickly and accurately as possible. On some trials (25%), an auditory beep occurs after the shape appears on the screen. This sound serves as the "stop-signal", and participants are told to try not to respond (i.e., inhibit their response) when they hear it. The task consisted of a practice block, followed by three experimental blocks of 64 trials each. After each block, performance feedback was provided. The primary dependent measure on this task is stop-signal reaction time (RT), which is determined using a tracking method. Each time the participant successfully inhibits their response, the stop signal (beep) occurs 50 ms later on the next stop trial; otherwise, the beep occurs 50 ms earlier on the next stop trial. Stop-signal RT is considered to reflect the estimated time that it takes the "stopping process" to finish – thus, higher scores suggest worse inhibition. Mean RT on no-signal trials was also examined as an index of processing speed (higher scores reflect slower processing).

Plus-minus task: To measure shifting, participants completed the plus-minus task (Jersild, 1927; Spector & Biederman, 1976). This task consists of three lists of 30 two-digit numbers (pre-randomized without replacement) on separate sheets of paper. For the first list, participants must add three to each number and write down the answer. For the second list, participants must subtract three from each number. For the third list, participants must alternate between adding and subtracting three (i.e., add three to the first number, subtract three from the second, and so on). Participants are instructed to complete each list as quickly and accurately as possible. The dependent measure for this task is shifting costs, computed as the difference between the time each participant took to complete the third list and their average time for the first two. Higher scores reflect larger switch costs and thus worse shifting. Errors were also analyzed to examine possible speed-accuracy tradeoffs.

Finger-tapping test: The Halstead-Reitan finger-tapping test (Halstead, 1947; Reitan, 1979) was administered to measure motor speed. In this task, participants place their dominant hand on a table, palm down and fingers extended, with their index finger resting on a lever attached to a counting device. Participants are instructed to tap their finger as quickly as possible for 10 secs, keeping their hand and arm stationary. Each participant completed four

trials, timed using a stopwatch. The dependent measure is the average number of taps across the four trials, with higher scores reflecting greater motor speed.

Procedures & Statistical Analyses—Participants were tested individually. The orders of the tasks and questionnaires were randomized across participants, while the clinical interview was always administered at the end of the session.

As expected, the dimensional symptom variables were negatively skewed (as scores of zero were common), as were state NA scores and errors from the plus-minus task. Thus, non-parametric (Spearman) correlations were used for these analyses (Pearson correlations were used for the remaining analyses). Correlations coefficients were statistically compared using methods described by Steiger (1980), with Spearman correlations converted following Rupinski and Dunlap (1996). In order to better differentiate current and past major depressive symptoms, participants who met full diagnostic criteria for a current MDE were excluded from analyses involving past MDE dimensional scores. However, the correlation between the current and past symptom variables in this sample was minimal (r_S=.10; p=.23), and the results were quite consistent when the entire sample was included in analyses of past MDE symptoms.

Results & Discussion

Descriptive statistics for the study measures are provided in the top panel of Table 1. Five participants (3%) met full diagnostic criteria for a current MDE, and 29 (18%) met full criteria for a past MDE, comparable to rates reported in epidemiological studies of depression in older adolescents (e.g., Lewinsohn et al., 1998). An additional 23 participants (17% total) reported experiencing some current symptoms (current MDE symptom scores>0), whereas an additional 43 reported some past symptoms (46% total). No participants met full diagnostic criteria for a DSM-IV bipolar disorder. A wide range of scores was represented on the state PA and NA scales (see Table 1). Sixteen participants stopped significantly less or more than 50% of the time on stop trials for the stop-signal task and were excluded from analyses involving the performance indices for that task (see Logan, 1994, and Verbruggen et al., 2008) – two of these participants reported current MDE symptoms, but none met full diagnostic criteria for a current MDE (three reported past symptoms, and one met full criteria for a past MDE). Five participants did not follow instructions properly on the plus-minus task and were excluded from those analyses (two reported some past MDE symptoms, but none reported current MDE symptoms or ever met full MDE criteria). One participant was missing data for each of these tasks. Importantly, the primary outcome variables from the stop-signal and plus-minus tasks were not significantly correlated (r=.11, p=.21), supporting the contention that these tasks index distinct dimensions of EF.

Table 2 presents correlations between the mood/symptom measures and task performance, which suggest a double dissociation between domains of EF and past vs. current MDE symptoms. Specifically, elevated past (not current) MDE symptoms were associated with shifting difficulties (switch cost from the plus-minus task) and not with inhibition difficulties (stop-signal RT). Notably, these correlations coefficients significantly differed from one

another (Z=2.0, p<.05). In contrast, current (not past) MDE symptoms were associated with inhibition difficulties (but not switch cost; Z=2.2, p<.05). When comparing past vs. current symptoms, the correlations differed for both shifting (Z=1.7, p<.05) and inhibition (Z=2.5, p<.01).

State PA was also associated with stop-signal RT. Although current MDE symptoms were not associated with switch costs, they were associated with math errors on the plus-minus task (as was state NA), suggesting that those experiencing current symptoms/distress exhibited a speed-accuracy tradeoff. None of the variables were associated with processing speed (mean RT on no-signal trials in the stop-signal task), and only state PA was associated with motor speed (from the finger-tapping task), suggesting that the associations between EF task performance and MDE symptoms cannot be accounted for by psychomotor slowing[2].

That worse inhibition performance was associated with current but not past major depressive symptoms is consistent with research showing a link between depression and impaired inhibition (see Fossati et al., 2002, and Snyder, 2013). This, coupled with the present finding that state PA was also associated with stop-signal RT, suggests that inhibition deficits may vary as a function of symptoms/distress. In contrast, worse shifting performance was specifically associated with past major depressive symptoms. This is consistent with other evidence for a unique link between depression and deficits in shifting (e.g., Austin et al., 2001; De Lissnyder et al., 2010, 2012), and that deficits in this domain of EF persist even when depressive symptoms have remitted (e.g., Clark et al., 2005; see Hasselbalch et al., 2011). In line with previous work (see Snyder, 2013), these links between depression and EF did not seem to due to psychomotor retardation.

Study 2

Study 1 yielded intriguing evidence that current and past major depressive symptoms are differentially associated with deficits in distinguishable domains of EF, which in turn may help explain inconsistencies from previous research on this topic (given imprecise/incomplete measurement approaches often used). In Study 2, we attempted to replicate these findings in an independent sample of college students. Further, we explored whether these findings would extend to different measures of EF and thus are not accounted for by method variance unique to the tasks used in Study 1. Given the limitations of laboratory EF tasks (e.g., questionable generalizability outside the lab, potential impact of state/contextual factors), Study 2 utilized a self-report questionnaire designed to assess EF in the everyday life and thus potentially capture more pervasive EF difficulties (which may be more predictive of functional impairment; see Barkley & Fischer, 2011).

Methods

Participants—Ninety-five undergraduate students (58% female) ranging in age from 18 to 22 years (M=19.0), with varying levels of anxiety and depression, participated in the study.

[2]In further support of these conclusions, post hoc linear regression analyses revealed that current MDE symptoms remained a significant predictor of inhibition; β=.22, p<.05; $R^2\Delta$=.04, and past MDE symptoms remained a significant predictor of shifting; β=.19, p<.05; $R^2\Delta$=.04, when the control performance measures (motor speed, processing speed, and math errors) and both symptom variables were entered together as predictors.

Like Study 1, this sample was predominantly European-American and Asian American. These participants were recruited for a larger neuroimaging study examining relationships among cognition, emotion, and psychopathology (see Warren et al., 2013, for details). Previously published articles overlapping with the present sample were neuroimaging work examining different research questions and utilizing different measures (Engels et al., 2010; Silton et al., 2010, Silton et al., 2011; Spielberg et al., 2011; Spielberg et al., 2012a; Spielberg et al., 2012b; Spielberg et al., 2013; Warren et al., 2013). All participants were right-handed, native English speakers, with self-reported normal color vision and hearing and no neurological disorders/impairments. Participants were excluded if they had ever experienced loss of consciousness > 10 minutes or reported current and clinically significant substance abuse/dependence, mania, or psychosis on the SCID-NP. Information about psychotropic medications use was not systematically collected.

Measures—As in Study 1, current and past MDE symptoms were assessed using the mood module of the SCID-NP. Interviews were conducted by advanced clinical psychology graduate students, including the lead author. Again, each diagnostic criterion was rated on a three-point scale, and dimensional symptom scores were computed for both current and past MDE. For additional details (including inter-rater reliability), see Study 1 of Bredemeier et al. (2010).

Inhibition and shifting were measured using subscales of the Behavior Rating Inventory of Executive Function, Self-Report version (BRIEF-SR; Roth et al., 2005). Participants were asked to rate how accurately each item describes them in general during the last six months on a three-point scale (1=never; 2=sometimes; 3=often), with higher scores reflecting greater difficulties. The inhibit subscale consists of 13 items, such as “I act too wild or ‘out of control’” and “I am impulsive.” The shift subscale consists of 10 items, such as “I have trouble changing from one activity to another” and “I get disturbed by an unexpected change.” Both subscales have been shown to have good test-retest reliability as well as convergent and discriminant validity in diverse community and clinical samples (e.g., Loyo et al., 2013; Rabin et al., 2006; Roth et al., 2005; Toplak et al., 2008). Internal consistencies for the inhibit and shift subscales in the present sample were .80 and .86, respectively.

Procedures & Statistical Analyses—Participants were given a laboratory tour, informed of study procedures, screened for contraindications for MRI participation, and then completed questionnaires. Following the tour and questionnaire sessions, participants completed the clinical interview. In later sessions, participants completed MRI and EEG procedures (results not reported here).

As in Study 1, non-parametric correlations were computed and compared, and participants who met full criteria for a current MDE were excluded from analyses of past MDE dimensional scores (correlation between the symptom variables: r_s=.21; $p<.05$). Despite directional hypotheses (based on results from Study 1), two-tailed p-values are presented.

Results & Discussion

Descriptive statistics for the study measures are provided in the bottom panel of Table 1. Three participants met full diagnostic criteria for a current MDE (3%), and 15 met full

diagnostic criteria for a past MDE (16%). An additional 6 participants (9% total) reported some current symptoms, and an additional 17 reported some past symptoms (34% total). As in Study 1, none met full diagnostic criteria for a DSM-IV bipolar disorder. Scores from the BRIEF inhibit and shifting scales were significantly correlated with one another; r=.35, p<.01.

Consistent with Study 1, there was a positive association between past MDE symptoms and self-reported shifting difficulties; r_s=.20, p=.05. In contrast, current MDE symptom were not significantly associated with shifting; r_s=.16, p=.11. Also, neither current nor past MDE symptoms were associated with self-reported inhibition difficulties (r_s=.04 and .12, respectively). None of these correlation coefficients significantly differed from one another (ps>.15) [3].

The findings from Study 2 are consistent with the interpretations of the findings from Study 1. Specifically, past major depressive symptoms were again associated with difficulties reported in the domain of shifting, further evidence that deficits in this domain of EF persist even when symptoms improve (see also Clark et al., 2005, and Hasselbalch et al., 2011). Although inhibition was not significantly associated with major depressive symptoms in Study 2, this could be a result of using an EF measure that indexes more stable, trait-like difficulties – thus, if deficits in this domain of EF vary as a function of symptoms/distress, a reliable association with depressive symptoms would not be expected. Also, some have argued that self-report measures of EF provide less discrimination amongst EF dimensions (e.g., Snyder et al., 2015), which could help explain why the correlations did not significantly differ from one another in this sample, despite the consistent pattern. In line with this proposal, the inhibition and shifting measures were significantly correlated in Study 2, but not Study 1. Notably, other publications from this sample have found evidence for functional abnormalities in inhibitory processes associated with current symptoms of distress (e.g., anhedonic depression, worry; Engels et al., 2010; Silton et al., 2011; Warren et al., 2013). Also of note, mean MDE symptom scores were somewhat lower in this sample, likely due to the use of different recruitment procedures. This presumably decreased statistical power, which was already lower than in Study 1 due to the smaller sample size.

General Discussion

Although there has been extensive research showing that depression is associated with deficits in EF, the nature of these deficits has not been clearly delineated. In two independent samples of college students, we examined relations between current and past major depressive symptoms (measured using a structured clinical interview) and deficits in two domains of EF: inhibition and shifting (measured using performance tasks in Study 1 and a self-report inventory in Study 2). Results showed that current and past major depressive symptoms were differentially associated with these two domains of EF. If further replicated

[3]As in Study 1, the results from the correlational analyses for past MDE symptoms remained consistent when all participants were included (regardless of current MDE status). Also, post hoc regression analyses showed further support for a link between shifting difficulties and past MDE symptoms; β=.18, p=.08, $R^2\Delta$=.03, but not current MDE symptoms; β=.11, p=.29, $R^2\Delta$=.01, when both variables were entered together.

(particularly in more diverse and distressed samples), these findings may provide important new insights into the nature of the relationship between depression and EF.

In both studies, past major depressive symptoms were associated with deficits in shifting. This suggests that deficits in this domain of EF persist even after active symptoms remit (see also Hasselbalch et al., 2011). Thus, shifting deficits may represent an enduring vulnerability to depression. In line with this proposal, dopaminergic dysfunction has been implicated in shifting deficits (O'Reilly, 2006) as well as the etiology of depression (Nestler & Carlezon, 2006). Also, this formulation is in line with evidence for impaired shifting in unaffected first-degree relatives of individuals with mood disorders (e.g., Clark et al., 2005). That said, the correlational design and retrospective measures used in the present research do not permit causal inferences. In fact, one could also argue that experiencing depression in the past might cause shifting problems, in line with the "scar hypothesis" (Lewinsohn et al., 1981; see Just et al., 2001). Evidence to explore these contrasting ideas about temporal precedence can be obtained by conducting prospective (longitudinal) studies[4]. Also, the association between shifting difficulties and past depressive symptoms could be explained by other variables that were not measured in the present research. For example, the association between past symptoms and shifting might be driven by trait rumination, which has been linked with both depression risk (see Nolen-Hoeksema, 2000) and shifting impairments (e.g., Davis & Nolen-Hoeksema, 2000; De Lissnyder et al., 2012).

In contrast, evidence for a link between depression and difficulties with inhibition only emerged when using a behavioral task (in Study 1) and examining current symptoms. Further, state mood (measured using the PANAS in Study 1) was also associated with performance on the behavioral measure of inhibition, and depressive symptoms were not significantly correlated with a self-report measure of (persistent) inhibition difficulties (in Study 2). Collectively, these findings (along with previous publications of neuroimaging data from the sample reported on in Study 2) suggest that inhibition difficulties linked with depression are a by-product of current distress/symptoms, and in turn should resolve when symptoms remit. That said, stronger evidence for this proposal could be obtained by experimentally manipulating participants' mood and examining how this impacts their ability to inhibit. Further, given the limited number of participants with prominent current symptoms of depression in both of samples (but particularly Study 2), it is important to consider that null relationships observed could be due to insufficient statistical power. Interestingly, the association between inhibition and state mood suggests that inhibition deficits may not be unique to depression, as diminished positive affect has also been linked to other forms of psychopathology (Berenbaum et al., 2003; Clark, 2005). In fact, inhibition difficulties have been documented in numerous other psychological disorders (see Nigg, 2000). Still, examining other diagnoses and comorbidity within the same sample in future research would strengthen this argument (e.g., Engels et al., 2010; Silton et al., 2011), as

[4]Letkiewicz and colleagues (2014) examined the relationship between EF (measured using the BRIEF) and depression prospectively and did not find that self-reported shifting difficulties reliably predicted changes in depressive symptoms over time (though working memory scores did). However, some methodological details from that study deserve consideration when contrasting those results with ours, most notably that Letkiewicz et al. (2014) examined: 1) anhedonic depression rather than MDE symptoms; and 2) changes over 3 months (thus the findings might be more relevant to understanding short-term, rather than long-term, risk).

would examining both performance and self-report measures together (e.g., Warren et al., 2013).

Present findings may help clarify some inconsistencies from past research and reviews on EF and depression. In particular, these findings highlight the importance of considering past symptoms when examining relations between EF and depression. If participants without current symptoms are all treated the same regardless of previous history, important EF deficits might be overlooked (in shifting, according to both studies). Conversely, work contrasting individuals who are currently depressed and those who have never been depressed inherently confounds present symptoms and depression history, and thus it becomes impossible to disentangle them.

Findings from the present studies also highlight the importance of considering the multidimensional nature of EF, and accordingly using measures designed to capture separable dimensions of EF. Previous conclusions that depression is associated with broad impairments in EF (e.g., Rogers et al., 2004; Snyder, 2013) might simply reflect the widespread use of “impure” measures of EF (see Snyder et al., 2015, for more discussion of this possibility). We found that distinguishable deficits in the domains of inhibition and shifting are associated with current and past major depressive symptoms respectively, perhaps explaining why others have suggested that EF deficits in depression might be unique to each of these domains (see Austin et al., 2001, and Fossati et al., 2002). Similarly, present findings might explain inconsistent findings from past research about whether EF deficits persist after remission (see Hasselbalch et al., 2011, and Rock et al., 2014), as they suggest that some do (in shifting) whereas others do not (in inhibition).

The contention that deficits in shifting contribute to risk for depression, if supported by additional research, would have important clinical implications. Although EF deficits are generally fairly stable over time (e.g., Biederman et al., 2007), emerging evidence suggests that certain interventions can improve EF (e.g., cognitive remediation, aerobic exercise; see Diamond & Lee, 2011). Such interventions could prove useful for treating or preventing depression (see Mead et al., 2009, for evidence that exercise improves mood/depression). Yet even if EF deficits are a consequence (rather than a cause) of depression, present findings might still have clinical utility. For example, measures of shifting could be used (in conjunction with other factors) to help identify individuals with a history of depression, when full diagnostic interviews are not feasible. Future research could explore these implications directly, for example by testing whether other interventions (e.g., cognitive remediation) that improve EF are effective for treating or preventing depression. Further, it would be valuable to examine the predictive power of EF measures in screening for depression, particularly since the effect sizes observed in the present studies (as well as most previous work on this topic) are in the small to medium range.

Findings from the present research might also have theoretical implications. EF has not been directly addressed in most traditional cognitive theories of depression (e.g., Beck, 2008; Teasdale, 1988). Thus, efforts should be made to incorporate these deficits when developing comprehensive theoretical models. Of course, such efforts are contingent upon understanding the nature of the relations between EF and depression, which the present

research addresses but certainly does not definitively establish. Further, it will be important to explore how EF deficits in euthymic individuals with a history of depression relate to other phenomena observed in this population (e.g., blunted reward responsiveness; Pechtel et al., 2013). Unquestionably, other factors contribute to depression risk, a point supported by the modest effect sizes observed in the present research. In service of this, future research on the topic should incorporate measures of known depression risk factors to include in multivariate statistical models.

In summary, the present research explored relations between depression and facets of EF. Both studies showed that deficits in shifting are associated with past major depressive symptoms, suggesting that these deficits could be a stable risk factor for depression. In contrast, (performance) deficits in inhibition were associated only with current mood, suggesting that these deficits are a by-product of distress and may remit when mood improves. Although some questions remain unanswered regarding links between EF and depression, these findings can help guide future research on this topic, as well as efforts to incorporate EF into new and revised theories of (and possibly interventions for) depression.

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Highlights

- Depression has been linked to impaired executive function (EF).
- The precise nature of the relationship between EF and depression remains unclear.
- Two studies examined types of EF difficulties related to major depressive symptoms.
- Worse inhibition task performance was associated with current depression symptoms.
- Past depression was linked to (behavioral and self-reported) shifting difficulties.

Table 1

Descriptive statistics for all study measures.

	n	M	SD	Min	Max
STUDY 1					
Current MDE sx	162	1.7	4.3	0	18
Past MDE sx	158	5.4	6.6	0	20
State PA	162	42.9	8.8	25	75
State NA	162	22.6	6.5	15	56
Stop-signal RT	146	269.5	40.4	186.0	442.4
Stop-signal mean RT on no-signal trials	146	615.8	154.7	376.1	1013.9
Plus-minus shift cost	156	16.1	11.5	−5.1	56.9
Plus-minus errors	156	2.3	2.9	0	20
Finger tapping	162	45.1	6.3	28.5	58.8
STUDY 2					
Current MDE sx	95	0.9	3.2	0	18
Past MDE sx	95	3.6	5.7	0	16
BRIEF inhibit scale	95	19.4	4.0	13	30
BRIEF shift scale	95	16.6	4.2	10	26

NOTE: sx = symptoms

Table 2

Correlations between symptom/mood indices and performance on the behavioral tasks in Study 1.

	Inhibition: Stop-Signal RT	Shifting: Plus-minus switch cost	Speed-Accuracy Tradeoff: Plus-minus task errors	Motor Speed: Finger tapping	Processing Speed: Mean RT on No-signal trials
Past MDE sx	−.03	.18*	.00	−.05	.07
Current MDE sx	.23**	.00	.22**	.02	.04
State PA	−.20*	.01	.03	.19*	.12
State NA	.08	−.10	.20*	.07	−.13

NOTES:

* = p >.05,

** = p > .01 (two-tailed);

sx = symptoms