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Motivational enhancement of cognitive control depends on depressive symptoms

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Abstract

Performance feedback can motivate improvements in executive function (Ravizza et al., 2012). The present study examines whether the enhancement of task switching with performance feedback is modulated by the level of depressive symptoms. Depressive symptoms have been linked to deficits in processing affective information inherent to such feedback (Henriques, 1994; Pizzagalli, et al., 2005). Task switching speed was assessed when performance feedback about accuracy was present or absent in a group of participants with minimal to moderate levels of depression. A significant positive correlation was observed between depressive symptoms and feedback effects on executive function indicating that those with lower depressive symptoms were more likely to show improvements in switching speed when performance feedback was present. These results suggest a novel link between executive function deficits and depression symptoms; namely, that greater levels of depressive symptoms are linked to diminished executive functioning via deficits in processing the affective component of performance feedback.

Keywords

depression; task switching; executive function; motivation

Motivational deficits are a hallmark of depression and are evidenced by the association of depressive symptoms with reduced reward sensitivity (Eshel & Roiser, 2010; Henriques, 1994; Pizzagalli, et al., 2005). In addition to motivational deficits, major depression is marked by cognitive impairments, most notably in the realm of executive function (see Snyder, 2012 for review). The causal link between executive function and depression, however, is currently unknown (Snyder, 2012). One possibility is that the lower hedonic pleasure experienced by rewarding stimuli affects the ability to improve executive function by use of performance feedback, which carries an inherent positive value. For example, both speed and accuracy of task switching improve when performance is rewarded (Capa et al., 2012; Muller et al., 2007). Interestingly, individuals scoring lower on trait measures of reward sensitivity are less able to use performance feedback to improve cognitive control (Jimura et al., 2010). In this paper, we investigate whether depressive symptoms are related to the ability to use positive performance feedback to enhance executive functions.

In non-depressed individuals, improvements in executive function are observed after positive performance feedback (see Pessoa, 2009 for a review), irrespective of whether the feedback is monetary (Aarts et al., 2012; Gilbert & Fiez, 2004; Locke & Braver, 2008) or informative (Ravizza et al., 2012). Studies of cognitive control deficits and performance feedback in depression have been scarce, however. One study found that depressed patients were less aware than controls when performance feedback did not reflect their behavior (i.e., false feedback) in the Tower of Hanoi task suggesting that they were processing this feedback in a different way than controls (Elliott et al., 1998). Another study found that depressive symptoms were related to the use of performance feedback on task switching speed in older adults and those with Parkinson's disease; that is, higher levels of depressive symptoms was associated with less improvement in task switching speed when performance feedback was presented (Ravizza et al., 2012). Thus, there is some evidence that depressive symptoms may compromise the ability to use performance feedback to enhance cognitive control in older adults.

It is possible that depressive symptoms observed in younger adults may not interact with cognitive control in the same manner as older adults, however (Ravizza et al., 2012). Late-onset depression is associated with differences in symptomatology and in the degree of cognitive impairment compared to early-onset depression (Gottfries, 1998). Impairments of cognitive control, for instance, are more characteristic of late-onset depression than early-onset depression (Hermann, Goodwin, & Ebmeier, 2007). Neural abnormalities associated with late-onset depression may also differ; for example, late-onset depression is associated with more extensive bilateral hippocampal atrophy (Lord et al., 2004) and a higher rate of periventricular hyperintensities (Delaloye et al., 2010) than early-onset depression. In fact, it is currently unknown whether depressive symptoms in young adults modulate the ability to use performance feedback in cognitive control.

The current study focuses on depressive symptoms and their relationship to cognitive control abilities in young adults attending college. Episodes of depression are relatively frequent in college with 14% of students reporting symptoms in a 2-week period (Eisenberg et al., 2007). At the same time, academic performance is related to cognitive control abilities (Best, Miller, & Naglieri, 2011; Rohde & Thompson, 2007). Understanding how depressive symptoms relate to the ability to use performance feedback in cognitive control may provide insight into the mechanism underlying the relationship between depressive symptoms and lower academic performance (Haines, Norris, & Kashy, 1996).

In order to assess this relationship in young university students, feedback effects were measured on a well-practiced task switching paradigm in which non-monetary feedback has been shown to improve performance (Ravizza et al., 2012). Specifically, the speed of switching between tasks improved when positive performance feedback about accuracy was presented. Thus, positive feedback about accuracy was able to motivate performance despite having little instructive value regarding the speed of switching. We expect that, similar to older adults, the severity of depressive symptoms will be related to the motivational effects of performance feedback on cognitive control.

Methods

Participants

146 undergraduates (mean age = 19.23; 38M) at Michigan State University participated in this experiment for course credit. Age and sex were not acquired for seven participants. All participants provided informed consent.

Stimuli

The switching task contained two sets: color (red and blue) and shape (triangle and square). On each trial, participants saw one of the colors inside one of the shapes (Figure 1).

Procedure

Participants were required to switch between identifying colors or shapes that were presented in the compound figure. A cue was presented simultaneously with the compound figure and instructed the participant as to the relevant feature. Once the color or shape of the relevant feature was identified, participants were asked to press the left (red, triangle) or right (blue, square) key to make a response. The cue and compound stimulus remained on the screen until the participant responded. The probability of a switch in the task set was .5

Feedback condition was blocked and the order was counterbalanced across participants (ABBA or BAAB). In the performance feedback condition, participants received positive or negative information about accuracy for that trial 500 ms after the response. In the no feedback condition, the word “Ready” was presented after each trial. The start of the next trial occurred 500 ms after the presentation of feedback.

Participants practiced the individual color and shape mappings in blocks of 24 trials with feedback until 90% accuracy rate was achieved. After practicing the stimulus-response mappings, task switching was assessed with four blocks of 80 trials using the compound stimuli. All participants completed the Beck Depression Inventory II (BDI-II) at the end of the session to assess their level of depressive symptoms.

Congruent trials were included in the study to increase the number of unique stimuli presented to participants; however, these trials were not analyzed because accuracy for these trials was ambiguous given that both color and shape stimuli afforded the same response. Reaction times (RTs) slower than 3 seconds (less than 4% of trials on average) and incorrect trials (5% of trials on average) were excluded from the analyses leaving a minimum of 20 trials per condition. RTs were then log transformed to create a more normal distribution.

Data from thirteen participants were discarded due to poor performance (<80% correct) on the task switching paradigm. Additionally, two participants were excluded because they did not fill out the back page of the BDI. For the correlation, a preliminary leverage analysis was performed on BDI-II scores and the shift cost in the feedback and no feedback conditions. Centered leverage values (h_i) were calculated and participants with six times the average leverage of the other participants ($6 \cdot p/n$) were eliminated from the analysis. This is a conservative estimate of influence as an estimate of 2 times the average leverage is noteworthy (Faraway, 2009). The average leverage value for a pool of 131 participants is .

0076 ($p/n = 1/131$) and participants were excluded if their leverage value indicated extreme values for BDI ($n=3$), shift cost in the feedback condition ($n=2$), or shift cost in the no feedback condition ($n=4$) was above .045 (.0076*6). Correlations run with the whole group showed a similar pattern of results as reported below; namely, improvement in the shift cost was negatively related to depressive symptoms, however, the relationship was weaker.

Results

A 2 (feedback) $\times$ 2 (shift) repeated-measures ANOVA was performed on the log-transformed RTs using the BDI-II scores as a covariate. A main effect of shift on RT ($F(1,120) = 120.67$, $p < .05$, partial $\eta^2 = .5$) confirmed that our task switching paradigm was successful in producing a shift cost in both feedback conditions (feedback repeat: 1088 ms [SEM = 16 ms; 95% CI = 1061 ms, 1129 ms]; feedback shift: 1239 ms [SEM = 19 ms; 95% CI = 1197 ms, 1274 ms]; no feedback repeat: 1100 ms [SEM = 16 ms; 95% CI = 1070 ms, 1139 ms]; no feedback shift: 1272 ms [SEM = 20 ms; 95% CI = 1230 ms, 1309 ms]). No main effect of feedback was observed ($F(1,120) = .05$, $p = .82$, partial $\eta^2 = 0$), but the interaction of shifting $\times$ feedback was significant ($F(1,120) = 8.78$, $p < .05$, partial $\eta^2 = .07$). Post-hoc analyses of the interaction effect indicated that RT in shift trials ($t(121) = 2.33$, $p < .05$) benefitted more from performance feedback than repeat trials ($t(130) = .31$, $p = .75$).

Of primary interest was the relationship of depressive symptoms to switching with and without performance feedback. A significant 3-way interaction of shifting, feedback and BDI-II scores indicated that depressive symptoms were related to task switching and feedback use ($F(1,120) = 4.48$, $p < .05$, partial $\eta^2 = .04$) (Figure 2). To assess the magnitude and direction of this effect, shift cost in the feedback condition was subtracted from the shift cost in the no feedback condition. Depressive symptoms were related to the shift cost improvement with feedback ($r(121) = -.19$, $p < .05$); that is, greater symptoms were associated with a diminished effect of feedback in reducing the shift cost. Shift cost was positively related to depressive symptoms when shifting with performance feedback ($r(121) = .18$, $p < .05$) (Supplemental Figure 1). In contrast, depressive symptoms were not significantly related to switch cost when performance feedback was absent ($r(121) = -.011$, $p = .9$). These correlations were significantly different from each other ($z = 2.03$, $p < .05$) using a test comparing dependent correlations (Meng, Rosenthal, & Rubin, 1992). Thus, greater depressive symptoms were related to task switching when feedback was present but not when it was absent.

The BDI-II assesses several components of depression including two that are relevant in the present study – anhedonia and melancholia (Pizzagalli, Jahn, O'Shea, 2005). Anhedonia is constructed from questions: 4) loss of pleasure 12) loss of interest 15) loss of energy and 21) loss of interest in sex. The melancholia subscale includes anhedonic symptoms but also psychomotor disturbances (Pizzagalli et al., 2004). It is constructed from questions: 4) loss of pleasure, 5) guilty feelings 11) agitation 12) loss of interest, 16b) early morning awakening, and 21) loss of interest in sex. Improvement in shift cost was significantly related to melancholia scores ($r = -.21$, $p < .05$), but was not reliably related to anhedonia ($r = -.13$, $p = .14$).

The same analysis was used on accuracy and only a main effect of shift was observed ($F(1,120) = 36.92, p < .05$, partial $\eta^2 = .24$) and was not moderated by BDI-II scores. Accuracy was at ceiling with an average of 95% collapsed across conditions.

Performance feedback affected task switching speed even though this feedback informed participants about their accuracy. This suggests that the improvement in speed was mostly likely due to the motivational value of performance feedback rather than to its instructional content. However, it is possible that negative feedback on even a small amount of error trials could be instructive as well as motivational. For example, participants' knowledge of an error often causes them to slow down on the following trial so that they do not sacrifice accuracy for speed. To see whether negative feedback was influencing the correlation, we added accuracy in the feedback condition as a nuisance variable in a partial correlation between depressive symptoms and shift cost. The correlation between depressive symptoms and shift cost was still significant and of a similar magnitude as previously observed ($r(117) = .2, p < .05$).

Discussion

In this experiment, we found that the benefits of performance feedback for task switching were modulated by the level of self-reported depressive symptoms. These results suggest that even minimal to moderate depressive symptoms can influence the ability of performance feedback to motivate cognitive control. This finding is consistent with our study of older adults (Ravizza et al., 2012) and suggests that geriatric depression and depression in younger adults are similarly related to the ability to use feedback. Moreover, performance effects were observed despite the relatively weak hedonic nature of this feedback compared to feedback which is associated with monetary reinforcers (Delgado et al., 2004). These results suggest that executive function impairments in depression are related to deficits in using performance feedback.

These findings are consistent with abnormalities in the concentrations of neurotransmitters in regions of the brain important for cognitive control and learning. For example, reductions in glutamate were observed for those with depression in regions of the prefrontal cortex that are associated with cognitive control (see Yuksel & Onger, 2010, for a review). Moreover, reduced dopamine synthesis in the striatum has been linked to depression symptoms and Parkinson's disease (Joutsa et al., 2013), a disorder marked by difficulty in learning from feedback (Shohamy et al., 2004). Thus, there is evidence that neurotransmitters important for learning and feedback processing are abnormal in depression and provide the basis for a neurobiological mechanism for the association of depressive symptoms and the enhancement of cognitive control with feedback.

Performance feedback in this experiment was primarily positive given that participants had practiced the tasks and, thus, were performing at ceiling. Nevertheless, participants switched between tasks faster when they were presented with feedback about their accuracy. This effect is most likely due to the motivational properties of positive feedback rather than its informational content; that is, there was little information in the feedback we presented that would help them to switch between tasks faster. Although participants made a few errors, it

did not appear that the instructive content in these trials was driving the relationship between depressive symptoms and feedback effects. In fact, the correlation between depressive symptoms and switching speed was unaffected by accuracy rates. The most probable explanation is that positive feedback carried some intrinsic rewarding value to participants and motivated them to perform well.

These results provide evidence for a novel link between executive function deficits and depression; namely that deficits in processing the affective component of feedback influences cognitive control. Previous hypotheses about the link between cognitive impairments and depression have emphasized the possible detrimental effects of rumination – the repetitive focusing on the causes and symptoms of the negative affect experienced in depression (Treyner, Gonzalez, Nolen-Hoeksema, 2003). According to this idea, depressive rumination engages mental resources that are then unavailable for other tasks; for example, inhibiting ruminative thoughts might impair the ability to inhibit external distractors. In a previous study of task switching, the level of rumination was related to the ability to inhibit previously relevant task sets (Whitmer & Banich, 2007). It was less related, however, to the ability to switch between tasks. Taken together, these results suggest that motivational deficits and rumination may have different effects on executive functioning in depression.

Several factors may influence the effectiveness of performance feedback including the strength and valence of the reinforcer, the difficulty of the task, and the informative value of the feedback. In this study, we highlight an association between depressive symptoms and the ability of feedback to motivate performance. Importantly, the effect size of this relationship was small ($r = .1-.3$) as defined by Cohen (Cohen, 1988), although comparable to other studies relating cognitive function to BDI scores in young adults (range = $-.22$ to $-.46$ Haines, Norris, & Kashy, 1996; Pizzagalli, Jahn, O'Shea, 2005) and to our previously published work with older adults ($r = -.38$; Ravizza et al., 2012). Further research is necessary to solidify this relationship. One possibility is that the use of more salient and extrinsic performance feedback such as money or primary reinforcers such as food could magnify the effect. In the present study, we chose to use intrinsic reinforcers as motivators that were more akin with the type of feedback received by students in college classes.

Depression is associated with impairments in a broad range of executive functions but the mechanisms underlying this impairment are not well-specified (Snyder, 2012). Our results suggest that the ability to use performance feedback, which carries an intrinsic positive value, to motivate cognitive control may be one explanation for the observed deficits in depression. The results of this study also imply that the ability to process performance feedback may contribute to the relationship between depressive symptoms and lower GPA in university students (Haines, Nolan, & Kashy, 1996). University students are provided with many forms of performance feedback through grades and comments on presentations, exams, and papers. These results suggest that even relatively few depressive symptoms may affect the ability of performance feedback to motivate cognitive control in young adults.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
Example of the task paradigm. Participants switched between making color and shape judgments with or without feedback about accuracy. Performance feedback was blocked across the experiment.

Figure 2.

The relationship of self-reported depression and the improvement in shift cost with feedback (Shift cost in no feedback condition – shift cost in feedback condition). Note that we are presenting shift cost in ms for illustrative purposes although correlations were performed on shift cost between log-transformed RTs.