Modulating Speed-Accuracy Strategies in Major Depression

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Abstract

**Background:** Depression is associated with deficits in cognitive flexibility. The role of general slowing in modulating more specific cognitive deficits is however unclear. **Aim:** We assessed how depression affects the capacity to strategically adapt behavior between harsh and prudent response modalities and how general and specific processes may contribute to performance deficits.

**Methods:** Patients suffering from major depression and age- and education-matched healthy controls were asked to randomly stress either speed or accuracy during perceptual decision-making.

**Results:** Diffusion models showed that patients with depression kept using a less conservative strategy after a trial with speed vs. accuracy instructions. Additionally, the depression group showed a slower rate of evidence accumulation as indicated by a generally lower drift rate.

**Conclusions:** These data demonstrate that less efficient strategic regulation of behavior in depression is due not only to general slowing, but also to more specific deficits, such as a rigid dependence on past contextual instructions. Future studies should investigate the neuro-anatomical basis of this deficit.

Keywords: flexibility, executive functions, depression, speed-accuracy trade off, perceptual decision-making, diffusion models.
Introduction

Depression is a major psychiatric disorder, which is usually accompanied by deficits in cognitive functioning, including impairments in cognitive flexibility (Airaksinen et al., 2004; Meiran et al., 2011; Whitmer and Banich, 2007). In particular, Whitmer and Banich (2007), by using a task-switching paradigm, showed that depressive rumination is associated with deficits in inhibiting previous mental sets. Moreover, patients with major depression show significant deficits when performing the Wisconsin Card Sorting Test (WCST), a test of cognitive flexibility, including problems in shifting cognitive sets when appropriate (e.g., Franke et al., 1993; Merriam et al., 1999). Perseverative responses and other deficits in the WCST are predicted by the severity of depressive symptoms independently of general intellectual abilities (Martin et al., 1991).

Cognitive flexibility deficits are possibly mediated by prefrontal serotonin deficiency (Clarke et al., 2004). From the functional-anatomical point of view, the dorsolateral prefrontal cortex (especially on the left hemisphere), which is reliably activated during tasks tapping cognitive flexibility (Kim et al., 2011; Vallesi, 2012), has been shown to be hypo-metabolic in depression (Bench et al., 1992; Davidson et al., 2002; Drevets, 2000; Mayberg et al., 1999), although many fMRI studies have shown that this region may be inefficiently hyper-active during task execution (see Graham et al., 2013, for a recent meta-analysis). Depression is also accompanied by an abnormal pattern of activation of medial prefrontal structures such as the anterior cingulate cortex (Bench et al., 1992; Diener et al., 2012; Drevets et al., 1992; Kennedy et al., 2001), a region implicated in energization, drive, and in the effortful allocation of cognitive and motor control (Paus, 2001; Shenhav et al., 2013; Stuss, 2011; Vallesi, 2012).

The prediction follows that patients suffering from major depression will show impairment in flexible regulation of behavior, especially in tasks recruiting the dorsolateral prefrontal cortex and the anterior cingulate cortex. Cognitive flexibility is for instance required when trading off speed and accuracy. This capacity is important in everyday life because it allows us to flexibly adapt to different and quickly changing environmental and endogenous demands. It has been shown that
switching from speed to accuracy by adopting stricter criteria for decision-making involves the left dorsolateral prefrontal cortex (Vallesi et al., 2012). Patients suffering from depression are thus expected to be impaired in this condition. A second prediction concerns possible deficits in the energization process based on anterior cingulate cortex, which should produce generally slower responding (Stuss et al., 2005).

Although a neuroimaging study should be set up to directly test the link between the involvement of these key regions and cognitive flexibility problems in depression, the present study aimed at finding behavioral evidence for deficits in speed-accuracy strategy regulations in major depression. A perceptual decision-making task was adopted in which speed and accuracy instructions were manipulated on a trial-by-trial basis to understand whether depression is associated with cognitive flexibility deficits in trials that require a switch in response strategy, and in slow response patterns especially in trial sequences with high time pressure. To gain deep insights on the possible mechanisms of depression-related deficits in speed-accuracy trade off regulation, we adopted a diffusion model analysis (e.g., Ratcliff, 1978; Voss and Voss, 2007) of the performance data (i.e., response times and accuracy), which allowed us to estimate more informative decisional and non-decisional sub-processes.

Methods

Participants

Twenty patients with a current or previous diagnosis of Major Depression (mean age: 47 years, range: 23-72; mean education years: 13 years, range: 5-18; 5 males; mean score for the Hamilton Depression Rating Scale for Depression (HDRS) at the time of testing: 6.8, range: 0-18) and 28 healthy volunteers (mean age: 48 years, range: 23-73; mean education years: 14.6 years, range: 5-19; 13 males) took part in the experiment. According to t-test analysis, the two groups were matched for age [t(46)=.16, p=.87] and years of education [t(46)=1.37, p=.18]. All the patients with
depression apart from two were under different anti-depressant treatments at the time of testing, while all of them had previously been under pharmacological treatment.

Patients with major depression met the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) criteria and were recruited from the out-patient Psychiatric Clinic of the University Hospital of Udine, Italy, as diagnosed with Structured Clinical Interview for Axis I DSM-IV Disorders (SCID-I). Diagnoses were confirmed by the clinical consensus of two staff psychiatrists. All the patients in the depression group did not have any other comorbid Axis-I diagnosis, including history of substance or alcohol abuse. They had been administered various (mainly serotonergic) drugs for variable life-time periods. Sixteen patients were treated with antidepressants: 8 took selective serotonin reuptake inhibitors (SSRI), 1 SSRI and Tricyclic Antidepressant (TCA), 4 selective norepinephrine reuptake inhibitor (SNRI), 1 TCA, 1 selective noradrenaline reuptake inhibitor (NARI) and 1 agomelatina.

Table 1 reports the type of antidepressant and dosage, length of disease, and HDRS scores, number of past episodes, age at depression onset and time in remission. Additionally, Table 1 also reports scores of an adapted Antidepressant Treatment History Form rating scale (see Sackeim, 2001) on a 4 point-scale (0=no drug, 3= highest drug load). This procedure was slightly adapted since some antidepressants were not present in the original study, and we assimilated those drugs with drugs with similar profile among those reported in Sackeim’s (2001) study (e.g., venlafaxina and duloxetina, escitalopram and citalopram).

The study was approved in advance by the ethics committee: “IRCCS E. Medea Associazione La Nostra Famiglia”. The authors assert that all procedures contributing to this work comply with the Helsinki Declaration of 1975, as revised in 2008.

Materials and task

---Insert Table 1 about here---
The basic task was to judge whether the predominant color in the target square was orange or green by responding with the index and middle fingers of the right hand (keys “B” and “N” of the laptop keyboard, appropriately covered with orange and green labels, respectively). The association between prevailing color and response button was counterbalanced between-subjects.

Visual stimuli were squares of 100 mm\(^2\) presented centrally against a constantly grey background. Lighter and darker grey pixels randomly dispersed in the square frame (50% each) were used to form the fixation space during the presentation of cue stimuli. Cues were triplets of capital letters (VEL, for speed, that is “velocità” in Italian, or ACC for accuracy) appearing on the top of the grey square frame at the beginning of the trial and disappearing with the target offset. Orange and green pixels were randomly dispersed in the square in various ratios (44/56, 47/53, 53/47, 56/44) to form target stimuli (adapted from Vallesi et al., 2012). Cues appeared on the top of the screen 1000 ms before the presentation of the target and disappeared with the target offset. The target lasted 2000 ms, followed by a blank screen of 500 ms. Response deadline was 2500 ms. Moreover, participants received a feedback which was different according to whether they fulfilled the speed-accuracy instructions or not (see below). The feedback was presented at the center of the screen for 1500 ms.

A first familiarization run with no speed-accuracy instructions or feedback (32 trials) was performed before the beginning of the real test. Two experimental runs with feedback were subsequently performed (64 trials each). In each run, the 4 orange/green proportions were presented pseudo-randomly and equiprobably. In the runs with cue (all but the first familiarization run), the combination of 2 cue type (accuracy vs. speed) and 2 previous cue type (accuracy vs. speed) factors was also presented randomly and with the same probability.

In the first familiarization run, participants were asked to simply perform the color estimation task (i.e., “are there more green or orange pixels”). During the next 2 training runs with feedback, participants were required to stress either speed or accuracy according to the nature of the cue at the beginning of the trial. Visual feedback was displayed as a sentence (font: Courier New, black color)
for 1000 ms after each trial, followed by a 500 ms blank screen. The provided feedback could be one of the following:

- “Sbagliato, attenzione!” (Wrong, be careful! in English): if participants failed to obey the accuracy instruction by making a mistake.
- “Sii più veloce” (Be quicker!): if RT was < average RT + 1 SD of the familiarization run during speed trials.
- “Tempo scaduto”! (Time expired!): if no response was collected before deadline.
- “Bene così” (Well done!): in any other case.

During the last run, which was a test run, participants performed the same task as during the training runs with feedback but now no feedback was provided at the end of every trial.

Data Analysis

The 32 trials from the familiarization run, trials with no response, trials with RTs < 200 ms, and the first trial of each run were excluded from further analyses. Both RTs and accuracy data were submitted to a 2x2x2x2 mixed ANOVA, with phase (training with feedback, test), preceding cue (accuracy, speed) and current cue (accuracy, speed) as the within-subject factors, and group (Depressed, Controls) as the between-subjects factor. Tukey’s HSD test was used as the post-hoc test to detect the sources underlying significant effects.

RT and accuracy data were also combined together in an additional analysis based on diffusion models (Ratcliff, 1978; Voss and Voss, 2007), as this model is specifically suited to analyse differences in speed-accuracy trade-off. The analysis of two-choice RT data by means of the diffusion-model assumes that information is continuously accumulated until one of two thresholds is passed. The analysis is based on the distributions of both correct and erroneous responses. From these distributions a set of parameters is estimated that allows drawing conclusions about non-decisional and decisional processes. Specifically, the model parameter t0 represents the duration of non-decisional processes such as stimulus encoding and response execution. The model parameter
‘v’ (drift rate) indicates the strength of the systematic influence that drifts the decision process from a starting point (parameter z) to one of two response thresholds. When either response threshold is reached, a response is initiated. Moreover, the distance between response thresholds is indicated by the parameter ‘a’. This parameter thus indicates how much information is required before either response is initiated. This is a measure of conservativeness/liberality. The z/a measure is instead used as a measure of bias towards one of the two responses (range 0-1), with 0.5 indicating no decisional bias.

We used the fast-dm free software (http://www.psychologie.uni-heidelberg.de/ae/meth/fast-dm/index-en.html) to estimate the parameters of the diffusion model (Spaniol et al., 2006; Voss and Voss, 2007; Voss et al., 2004). A multi-dimensional optimization method (i.e., Simplex downhill search) was used to optimize the fitting between predicted and empirical distributions and the Kolmogorov–Smirnov test was used to statistically assess this fitting. We allowed ‘z’, ‘a’ and ‘v’ to vary with each of the four conditions (2 preceding cue x 2 current cue), while other parameters of the diffusion model were assumed to be constant for all conditions. Each of the three varied parameters was treated as the dependent variable of a 2x2x2 mixed ANOVA, with preceding and current cue type as the within-subject factors and group (Depression vs. Control) as the between-subjects factor. The effect of group on the constant parameter t0, which indicates the duration of non-decisional processes, was also assessed through a two-sample t-test.

For each significant effect involving the group factor, within the depression group we performed (i) Pearson’s correlations between the relevant dependent variable and the HDRS, and (ii) Spearman Rank Order correlations with the Antidepressant Treatment History form Rating scores (cf., Sackeim, 2001), since the latter were distributed on a scale of just 4 points. We only reported correlations in the text if they reached the significance level (p<.05).

**Results**
All the behavioral data are shown in Table 2.

**Accuracy.** In the ANOVA concerning the speed-accuracy experimental runs, there was a strong trend for a cue main effect \([F(1, 46)=3.73, p=.059]\), with participants being more accurate for accuracy (90%) than for speed cues (88.7%). Depressed patients were on average less accurate than their controls \([86.8\% \text{ vs. } 91.8\%; F(1, 46)=10.13, p=.0026]\), although they were still able to perform the task with a reasonable accuracy level. Accuracy improved from the training phase with feedback (87.2%) to the test phase (91.4%) in both groups \([F(1, 46)=27.2, p<.0001]\). An interaction between phase and group \([F(1, 46)=5.33, p=.025]\) was better qualified by a 3-way interaction with the preceding cue \([F(1, 46)=7.55, p=.008]\). Post-hoc Tukey’s tests revealed that this interaction was mainly due to differential sequential effects for the two groups in the test phase. Specifically, the accuracy level in the Depression group depended on the preceding cue type when no feedback was provided during the test phase, with worse accuracy when the current trial followed speed (88%) vs. accuracy (91.7%) instructions in the preceding trial \((p=.004)\). There was no modulation by the preceding cue in the control group \((93.4 \text{ vs. } 92.5\%; p=.97)\). This was further confirmed with separate ANOVAs for each phase, which revealed a significant preceding cue by group interaction for the test phase \([F(1, 46)=9.58, p=.0033]\), but not for the training one \((p=.55)\). Correlation analyses performed on the depression group showed that, for the test phase, the difference between the two preceding cues (accuracy - speed) in terms of accuracy level was negatively correlated with the antidepressant rating scale \((\text{Spearman R}=-.52, p=.019)\), that is, the higher the antidepressant load, the smaller the differential carry-over effects from the preceding cue type. No other effect was significant.

**Response Times (RTs).** RTs were shorter in the test run than in the training runs with feedback \([887 \text{ vs. } 974 \text{ ms}; F(1, 46)=40.57, p<.00001]\). They were shorter for preceding speed cues than for preceding accuracy cues \([920 \text{ vs. } 940 \text{ ms}; F(1, 46)=13.25, p=.0007]\). Moreover, RTs were shorter for current speed cues than accuracy cues \([893 \text{ vs. } 967 \text{ ms}; F(1, 46)=31.37, p<.00001]\), this
difference being more accentuated in the training runs than in the test run [cue x phase interaction: \( F(1, 46)=10.46, p=.002 \)]. A preceding x current cue interaction \( F(1, 46)=27.3, p=.00001 \) was mainly due to the fact that RTs in speed trials preceded by speed trials were shorter than speed trials preceded by accuracy trials (872 vs. 915 ms, \( p=0.0002 \)), while the preceding cue did not modulate RTs for current accuracy trials (966 vs. 969 ms; \( p=.98 \)). No other effect was significant.

Diffusion model analysis.

Decisional Bias ‘z/a’. The decisional bias was higher (i.e., closer to the correct response criterion) for current speed vs. accuracy instructions \( F(1, 46)=4.36, p=.042 \). There was also a preceding cue by group interaction \( F(1, 46)=5.28, p=.026 \), which suggested that the two groups modulated the decisional bias differently depending on the preceding cue. However, no comparison survived the Tukey’s HSD post-hoc test (all \( ps>.21 \)). No other effect was significant.

Distance between response criteria ‘a’. This parameter, which measures the conservativeness of the adopted response criteria, was generally higher in the training runs with feedback than in the test run \( F(1, 46)=13.31, p=.0007 \). The ‘a’ value was also higher for preceding accuracy cues than for speed ones \( F(1, 46)=10.46, p=.0023 \). Moreover, this value was higher for current accuracy cues than for speed ones \( F(1, 46)=21.78, p=.00003 \). A preceding cue x group interaction \( F(1, 46)=4.3, p=.038 \) indicated that only the patients with depression modulated the parameter ‘a’ as a function of preceding cue (\( p<.006 \)), being it lower for preceding speed than for preceding accuracy trials (see Figure 1 and table 2), while the control group did not show any modulation of this parameter (\( p=.81 \)).
Drift rate ‘v’. There was a phase main effect [F(1, 46)=25.9, p=.00001], which showed that this parameter was higher for the test run than for the training runs, and a group main effect [F(1, 46)=10.49, p=.0022], with the drift rate being generally higher in controls than in the depressed patients. Correlation analyses showed that the average drift rate in the depressed patient group was negatively correlated with the HDRS (Pearson’s r=-.52, p=.018), that is, the higher the depression level as measured with the Hamilton scale, the lower the drift rate. No other effect was significant for the ANOVA.

Non-decisional processes ‘t0’. There was no group difference in the constant parameter t0 (p=-.72).

Discussion

The present study investigated how depression affects the flexible regulation of cognitive strategies. In particular, the capacity to switch between speed and accuracy in perceptual decision-making according to the task demands was investigated. Patients with depression had difficulties in overcoming the instructions presented in the previous trial, as indicated by the presence of sequential effects in the percentage of errors, especially in the test phase with no feedback. In particular, they were 4% less accurate when they had already traded off accuracy for speed in the previous trial (i.e., preceding speed cues), independently of the current instructions. This sequential effect was negatively correlated with a comprehensive measure of antidepressant treatment load, suggesting that the dependence on the previous trial instructions ameliorated with a higher medication load. The control group did not show these sequential effects.

A more complete picture was provided by the diffusion model analysis, which showed that this deficit in flexibility was mainly present in the depressed group when considering the distance between response criteria (parameter ‘a’). This distance, which indicates the degree of
conservativeness, was smaller when the previous cue instructed a quick response, independently of the current cue. These effects suggest less flexibility in adapting the response strategy according to the external demands in depression, especially when the previous trial instruction was “speed”. This would be especially problematic when the current instruction cues a switch towards a more accurate strategy, a condition which previous neuroimaging evidence has shown to recruit the left dorsolateral prefrontal cortex (Vallesi et al., 2012). It is worth noting that this prefrontal region shows hypo-metabolism in depression (Davidson et al., 2002; Dougherty and Rauch, 1997; Heller and Nitschke, 1998). No correlation between depressive symptoms or antidepressant level and this measure was found, suggesting that problems with response criterion flexible adjustments could be a stable characteristic of depression.

Another important finding was a generally slower drift rate (i.e., systematic velocity of transmission of information) in patients suffering from depression than in controls, which is compatible with many depressive symptoms such as general psychomotor slowness and loss of drive (Mayberg, 1994; White et al., 1997). However, this finding suggests a more specific slowness in perceptual evidence accumulation than just general slowing, since there was no difference between the group with depression and their controls for the duration of non-decisional processes (t0). The negative correlation with the Hamilton depression rating scale demonstrated that this deficit was specifically linked with the severity of the depression disease, while a comprehensive measure of the amount of drugs taken did not show any role for this factor.

General slowing could be a confounding factor when assessing specific cognitive deficits in depression (White et al., 1997). The present diffusion model approach was therefore useful to isolate different component processes and separately characterize their contribution to impaired performance of the task at hand observed in the depression group. In this respect, diffusion models are superior to standard analyses of raw performance measures, revealing their effectiveness in the evaluation of both specific and general cognitive deficits in depression.
Although the significant results clearly demonstrate that the present study did not suffer from a general lack of power, a possible limitation is represented by the small sample size for the patients with depression. Therefore, replications with future studies including bigger sample sizes and other tasks with SAT manipulation are warranted to corroborate the present findings and make them more generalizable.

Most of the patients with depression tested in the present study were under (mainly serotoninergic) drugs and therefore it is difficult to disentangle the effect of drug from that of depression per se in driving the observed effects. Notwithstanding the difficulty of recruiting drug-free patients with clinically relevant depression, a future study conducted on patients at their first diagnosis could be helpful to better characterize the role of depression in speed-accuracy switching.

In conclusion, the present study shows a depression-related deficit in flexible regulation of speed-accuracy during decision-making, which may be related to frontal lobe dysfunction. Future studies should more directly investigate the neuro-anatomical substrate of this deficit.
References


Table 1. The table shows, for each patient with major depression, arbitrarily numbered from 1 to 20, the type of antidepressant and dosages currently used, the length of disease, HDRS scores at the time of testing, number of past depressive episodes, age at illness onset, time in remission and scores on an antidepressant treatment rating scale.

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<th>Patient number</th>
<th>Type and dosages of antidepressants</th>
<th>Length of disease in months</th>
<th>HDRS scores</th>
<th>N° past depressive episodes</th>
<th>Age at illness onset</th>
<th>Time in remission in months</th>
<th>Antidepressant Treatment History Form Rating Scale</th>
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<td>1</td>
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<td>10</td>
<td>1</td>
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<td>2</td>
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<td>42</td>
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Table 2. Behavioral data shown according to group and task condition. For each variable, mean and standard error of the mean (S.E.M.) are shown. ‘Acc’ and ‘Spd’ stand for Accuracy and Speed current cues, respectively. ‘Prec Acc’ and ‘Prec Spd’ indicate accuracy and speed instructions in the preceding trial, respectively.

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<td>Mean</td>
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<td>Drift rate ‘v’</td>
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</tr>
</tbody>
</table>
Figure Caption

*Figure 1.* Mean values for the diffusion model parameter ‘a’ (and standard errors of the mean), which indicates the distance between response criteria (smaller values denote more conservativeness) are shown according to task conditions and groups. The asterisk indicates significant Tukey’s HSD test at p<.01.