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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.
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1. 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

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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.

2. Methods

2.1. 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 [$t(46) = 0.16$, $p = .87$] and years of education [$t(46) = 1.37$, $p = .18$]. All the patients with depression apart from two were under different antidepressant 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 serotoninergic) 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.

2.2. Materials and task

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

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.

Patient number	Type and dosages of antidepressants	Length of disease in months	HDRS scores	N° past depressive episodes	Age at illness onset	Time in remission in months	Antidepressant treatment history form rating scale
1	Amitriptilina 10 mg/die	17	8	1	49	10	1
2	Fluoxetina 20 mg; amitriptilina 20 mg/die	96	8	1	42	80	3
3	Paroxetina 20 mg/die	12	18	0	22	0	3
4	Duloxetina 60 mg/die	21	1	2	41	12	2
5	None	24	8	1	28	18	0
6	Duloxetina 60 mg/die	456	6	3	14	444	2
7	Paroxetina 20 mg/die	12	0	0	40	4	3
8	Venlafaxina 75 mg/die	42	5	1	68	33	2
9	None	72	2	1	41	61	0
10	Duloxetina 60 mg/die	96	0	2	39	83	2
11	Sertralina 50 mg/die	48	9	1	27	34	2
12	Citalopram 20 mg/die	17	8	1	49	9	3
13	None	6	0	2	24	1	0
14	Agomelatina 25 mg/die	72	13	1	44	66	1
15	None	89	6	1	36	77	0
16	Escitalopram 10 mg/die	26	5	1	38	15	2
17	Bupropione 300 mg/die	720	11	6	28	677	3
18	Escitalopram 10 mg/die	84	13	2	56	68	2
19	Citalopram 20 mg/die	36	8	1	40	22	3
20	Fluoxetina 20 mg/die	276	5	5	68	48	3

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