Major depressive disorder skews the recognition of emotional prosody☆

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1. Introduction

Major depressive disorder (MDD) is associated with abnormalities in the way emotional stimuli are perceived, responded to, and stored in memory. It is thought to underlie problems of interpersonal behavior and contribute to the onset and maintenance of mood disorders (Leppanen, 2006). Understanding the specific nature of these emotion processing abnormalities and their attendant mechanisms may therefore prove helpful in the diagnosis, treatment, and prevention of mood disorders.

Objective: Major depressive disorder (MDD) is associated with abnormalities in the recognition of emotional stimuli. MDD patients ascribe more negative emotion but also less positive emotion to facial expressions, suggesting blunted responsiveness to positive emotional stimuli. To ascertain whether these emotional biases are modality-specific, we examined the effects of MDD on the recognition of emotions from voices using a paradigm designed to capture subtle effects of biases.

Methods: Twenty-one MDD patients and 21 healthy controls (HC) underwent clinical and neuropsychological assessments, followed by a paradigm featuring pseudowords spoken by actors in five types of emotional prosody, rated on continuous scales.

Results: Overall, MDD patients performed more poorly than HC, displaying significantly impaired recognition of fear, happiness and sadness. Compared with HC, they rated fear significantly more highly when listening to angry stimuli. They also displayed a bias toward surprise, rating it far higher when they heard sad or fearful utterances. Furthermore, for happiness stimuli, MDD patients gave higher ratings for negative emotions (fear and sadness). A multiple regression model on recognition of emotional prosody in MDD patients showed that the best fit was achieved using the executive functioning (categorical fluency, number of errors in the MCST, and TMT B-A) and the total score of the Montgomery–Asberg Depression Rating Scale.

Conclusions: Impaired recognition of emotions would appear not to be specific to the visual modality but to be present also when emotions are expressed vocally, this impairment being related to depression severity and dysexecutive syndrome. MDD seems to skew the recognition of emotional prosody toward negative emotional stimuli and the blunting of positive emotion appears not to be restricted to the visual modality.
(Drevets, 2001), superior parietal cortex, temporal gyrus and orbitofrontal cortex (Chan et al., 2008), and insula (Anand et al., 2005).

Nevertheless, we do not yet know whether these emotional biases in MDD are modality-specific or supramodal. Whereas the majority of the above-mentioned studies used visual material, such as facial expressions and category recognition, the present study therefore examined the effects of depression on the recognition of emotions conveyed by the human voice (i.e., emotional prosody).

Emotional prosody is defined as modifications in segmental and supra-segmental speech parameters during an emotional episode (Grandjean et al., 2006). Explorations of the perception of emotional prosody in FMRI and patient studies have allowed researchers to delineate a distributed neural network involved in the identification and recognition of emotional prosody. In addition to primary and secondary auditory regions, modulation of neuronal activity within the superior temporal sulcus and gyrus has been reported in response to exposure to emotional prosodic stimuli (Ethofer et al., 2006b; Grandjean et al., 2005; Sander et al., 2005). Increased activity within the amygdala has also been observed, in response not only to emotional prosody (Ethofer et al., 2006a; Grandjean et al., 2005; Sander et al., 2005) but also to emotional animal vocalizations (Belin et al., 2008; Fecteau et al., 2007).

Modulations in activity within anterior regions such as the orbitofrontal cortex and inferior frontal areas have also been recorded, especially when participants pay attention to the emotional auditory stimulus or are asked to identify emotional prosody (Ethofer et al., 2006a; Sander et al., 2005; Wildgruber et al., 2004). Recently, a study of neglect patients confirmed that the superior temporal sulcus, superior temporal gyrus and orbitofrontal cortex are important for detecting emotional voices in the environment (Grandjean et al., 2008). Beyond these regions, the involvement of the basal ganglia in the processing of emotional prosody, particularly the caudate nucleus and putamen, has been revealed by fMRI and patient studies (Bach et al., 2008; Grandjean et al., 2005; Kotz et al., 2003; Morris et al., 1999).

To our knowledge, very few studies have investigated the recognition of emotional prosody in MDD. In one early study, Murphy and Cutting (1990) examined MDD patients (n = 15) who had to judge sentences read out by the experimenter. These were supposed to express neutral emotion, sadness, surprise, or anger. Results showed that MDD patients performed poorly in comparison to matched HC for all four emotions and made more errors when they had to detect sadness. Emerson et al. (1999) compared a population of school-aged boys with depressive syndrome and a matched control group on recognition of declarative sentences expressing happiness, sadness, anger, and neutral emotion. In half the trials, the emotional prosody of the sentences was congruent with the semantic content, while in the remaining trials, the emotional content and semantic content were incongruent. The depressed group was found to have difficulty recognizing all the emotions, whatever the experimental condition (congruent or incongruent). For their part, Kan et al. (2004) presented semantically neutral sentences and short nonsense sentences supposed to express sadness, anger, fear, disgust, or happiness to MDD patients (n = 16) and matched HC. The authors observed that the MDD patients included in their study tended to judge surprise stimuli as negative, whereas HC judged them as neutral, but reported no difference between the two groups for the other emotions. They interpreted their results as reflecting a bias toward negative emotions. Finally, Uekermann et al. (2008) sought to assess the relationship between executive functioning and the perception of emotional prosody in MDD patients (n = 29) using a design comparable to that implemented by Emerson et al. (1999), insofar as they manipulated the congruency factor. The authors hypothesized that the incongruent condition would require executive functioning, whereas the congruent condition would not, and the MDD patients would thus be impaired in the former but not in the latter, due to their dysexecutive syndrome. In accordance with their hypothesis, results showed that MDD patients performed poorly in the incongruent condition but normally in the congruent one. Finally, Bach et al. (2009) examined MDD patients on the recognition of emotional prosody (stimuli taken from Banse and Scherer, 1996), but did not compare the MDD group with HC because they had chosen to focus their analyses on the second pathological group included in their study, namely schizophrenia patients. It is noteworthy that the results of the aforementioned studies were rather different from those reported for the facial modality, in that they failed to observe enhanced recognition of sadness. This divergence can be interpreted in two ways. First, it could reflect a functional difference between the two modalities (facial vs. vocal). Second, it could be linked to the experimental designs used by the authors, in that they all opted for categorical judgments which are ill-suited to capturing subtle effects of biases in information processing and could induce categorization biases (Scherer and Ekman, 2008). It should be noted that this methodological limitation is shared by the vast majority of the previously mentioned studies in the facial modality.

Overall, these findings highlight the overlap between the neuroanatomical substrates of the recognition of emotional prosody and the neuroanatomical circuit that depression is thought to modify, particularly the ventral striatum and the orbitofrontal cortex areas (Savitz and Drevets, 2009). Accordingly, in the present study, we postulated that the emotional bias observed in MDD is not specific to the visual modality, but is also present when emotions are expressed by the human voice. To test this notion, we explored the recognition of emotions in the vocal modality, using an original emotional prosody paradigm specially designed to study emotional bias (Pérón et al., 2010b). The following hypotheses were tested:

i) MDD patients have impaired recognition of emotional prosody compared with HC;

ii) MDD patients display a negative emotional bias in the vocal modality just as they do in the facial one, that is, they rate positive stimuli more negatively (or less positively) than HC.

2. Participants and methods

2.1. Participants (Table 1)

One group of MDD patients and one group of HC took part in the study. The clinical group included 21 patients with MDD (6 men, 15 women), all born in France and native French speakers. Their mean (±SE) age was 49.3 (±2.28, range = 26–64) years. Nineteen were right-handed and two were left-handed, according to the criteria of the Edinburgh Handedness Inventory (Oldfield, 1971). Their mean (±SE) education level was 13.3 (±0.50, range = 11–17) years. MDD was diagnosed by the treating clinician and confirmed by a clinically trained member of the study group (S.E.T.) on the basis of the entire Mini International Neuropsychiatric Interview (MINI, Sheehan et al., 1998) and according to the criteria of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV, American Psychiatric Association, 1994). All patients scored 23 or above on the Montgomery–Asberg Depression Rating Scale (MADRS, Montgomery and Asberg, 1979) (M = 30.2, SE = 1.0, range = 23–37). Participants with MDD were inpatients from the psychiatry departments of hospitals and clinics in Rennes, France. As far as comorbidities were concerned (according to the MINI), none of the patients in the sample presented atypical, psychotic, or catatonic features, although two patients presented melancholic features in addition to their depressive symptomatology. In addition, there was no report of either panic disorder or generalized anxiety disorder, except for one depressed patient who had presented with generalized anxiety disorder some three years before the study (for that reason, it was not regarded as a co morbidity). Seven patients had...
had no previous episode (single), while 14 patients had had one previous episode (recurrent). The mean (±SE) length of time since the first symptoms of the current episode was 3 (±0.4, range = 0.75–6) months (Duration 1). The mean (±SE) length of time since the first symptoms of the first episode was 118 (±34.1, range = 0.75–528) months. Medication consisted of selective serotonin and noradrenaline reuptake inhibitors (citalopram, escitalopram, paroxetine, venlafaxine; n = 15) and tricyclic antidepressants (amitriptyline, doxepin, clomipramine; n = 6). Regarding the current episode, the MDD patients had been on antidepressant treatment for two weeks.

The sociodemographic and illness-related characteristics of each patient in the MDD group are presented in Table 1.

The second group included 21 healthy individuals (6 men, 15 women) who were recruited from the general population and were given no reward for their participation. They were all born in France and were all native French speakers. Mean (±SE) age was 53.7 (±2.35, range = 35–81) years. Nineteen HC participants were right-handed and two were left-handed. Their mean (±SE) education level was 14.8 (±0.58, range = 11–19) years. Their health status was assessed by a clinically trained member of the study group (S.E.T.) on the basis of the MINI and according to DSM-IV criteria.

Exclusion criteria for all participants (MDD and HC) were the wearing of hearing aids or a history of tinnitus or a hearing impairment, a history of neurological disorders, head trauma, anoxia, stroke and major cognitive deterioration, as attested by their score on the Mattis Dementia Rating Scale (Mattis, 1988). None of the participants scored above 130 on the Mattis scale, displayed addictive or psychiatric disorders (Axis I of the DSM-IV) (except for depression in the MDD group), or had undergone electroconvulsive therapy.

The two groups were comparable for gender, age, F(1, 40) = 1.79, p = .2, education level, F(1, 40) = 3.53, p = .07, and handedness.

The study was approved by the Ethics Committee of Rennes University Hospital. After a complete description of the study, written informed consent was obtained from each participant, and the study was conducted in accordance with the Declaration of Helsinki.

### Table 1

<table>
<thead>
<tr>
<th>MDD patients</th>
<th>Gender (M/F)</th>
<th>Age (years)</th>
<th>Education level (years)</th>
<th>Handedness (right/left)</th>
<th>Duration 1 (months)</th>
<th>Duration 2 (months)</th>
<th>Type (single/recurrent)</th>
<th>MADRS (max. 60)</th>
<th>Drug treatment</th>
<th>Dose (mg/day)</th>
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<td>F</td>
<td>62</td>
<td>13</td>
<td>R</td>
<td>6</td>
<td>6.00</td>
<td>Single</td>
<td>33</td>
<td>Citalopram</td>
<td>60</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>54</td>
<td>11</td>
<td>R</td>
<td>6</td>
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<td>Single</td>
<td>34</td>
<td>Venlafaxine</td>
<td>75</td>
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<tr>
<td>3</td>
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<td>51</td>
<td>13</td>
<td>R</td>
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<td>Recurrent</td>
<td>23</td>
<td>Amitriptyline</td>
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<td>12</td>
<td>R</td>
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<td>Single</td>
<td>32</td>
<td>Venlafaxine</td>
<td>75</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>26</td>
<td>17</td>
<td>R</td>
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<td>Recurrent</td>
<td>34</td>
<td>Excitopram</td>
<td>20</td>
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<tr>
<td>6</td>
<td>F</td>
<td>46</td>
<td>12</td>
<td>R</td>
<td>3</td>
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<td>R</td>
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<tr>
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<td>12</td>
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<td>Single</td>
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<td>Citalopram</td>
<td>40</td>
</tr>
<tr>
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<td>15</td>
<td>R</td>
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<tr>
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<tr>
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<td>11</td>
<td>R</td>
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<td>R</td>
<td>5</td>
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<td>Clomipramine</td>
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<tr>
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<td>F</td>
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<td>L</td>
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<td>4.00</td>
<td>Single</td>
<td>32</td>
<td>Clomipramine</td>
<td>50</td>
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<tr>
<td>17</td>
<td>F</td>
<td>64</td>
<td>12</td>
<td>R</td>
<td>3</td>
<td>3.00</td>
<td>Single</td>
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<td>Doxipinin</td>
<td>50</td>
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<tr>
<td>18</td>
<td>F</td>
<td>46</td>
<td>12</td>
<td>R</td>
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<td>24.00</td>
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<td>Citalopram</td>
<td>40</td>
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<tr>
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<td>58</td>
<td>16</td>
<td>R</td>
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<td>24.00</td>
<td>Recurrent</td>
<td>30</td>
<td>Clomipramine</td>
<td>75</td>
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<tr>
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<td>F</td>
<td>40</td>
<td>14</td>
<td>L</td>
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<td>Recurrent</td>
<td>31</td>
<td>Venlafaxine</td>
<td>75</td>
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</table>

MDD = Major Depressive Disorder; Duration 1 = Length of time since the first symptoms of the current episode (in months); Duration 2 = Length of time since the first symptoms of the first episode (in months); Type = Type of MDD, single or recurrent; MADRS = Montgomery–Asberg Depression Rating Scale.

### 2.2. Methods

All the participants (MDD and HC) underwent psychiatric, neuropsychological and emotional assessments. For obvious ethical reasons, all the depressed patients remained on medication.

#### 2.2.1. Psychiatric and neuropsychological assessment

As indicated above, the MDD patients’ intensity of depression was assessed on the MADRS.

Given that performance on measures of executive functioning tend to be impaired in depressed patients (for a review, see Marazziti et al., 2011) and in order to study the relationship between emotional prosody recognition and executive functions, a short neuropsychological battery was administered to all participants prior to the vocal emotion recognition session. This battery included the Mattis scale and a series of tests assessing frontal executive functions: Nelson’s modified version of the Wisconsin Card Sorting Test (MCST, Nelson, 1982), the Trail Making Test (MTT, Reitan, 1958), the Categorical and Literal Fluency Test (Cardebat et al., 1990), the Action (Verb) Fluency test (Woods et al., 2005), and the Stroop test (Stroop, 1935). Finally, to ensure that participants had no auditory impairment, they all underwent the Montreal-Toulouse auditory agnosia battery (PEGA, Agniel et al., 1992). None of the participants included in the study presented any impairment on the latter.

#### 2.2.2. Vocal emotion recognition

A set of vocal stimuli consisting of meaningless speech (pseudowords) was played to all participants. This paradigm has already been used – and described – in a study conducted by our group to assess the impact of subthalamic nucleus deep-brain stimulation on the recognition of emotional prosody in Parkinson’s disease patients (Péron et al., 2010b).

##### 2.2.2.1. Pseudoword stimuli

The vocal stimuli were extracted from the database developed by Banse and Scherer (1996) and validated in their study. These items, which have already been described elsewhere (Péron et al., 2010b), consisted of short segments of
meaningless speech (pseudowords), obtained by concatenating different syllables found in Indo-European languages so that they would be perceived of as natural utterances, with emotional intonation (shared across different cultures) but no semantic content. Four different categories of emotional prosody (anger, fear, happiness and sadness), together with a neutral condition, were used in the present study. The mean duration of the stimuli was 2044 ms (range: 1205–5236 ms). An ANOVA failed to reveal any significant difference in duration between the different prosodic categories (neutral, anger, fear, happiness and sadness), \( F(4, 156) = 1.43, p > .1 \), and there was no significant difference in mean acoustic energy expended, \( F(4, 156) = 1.86, p > .10 \) (none of the systematic pairwise comparisons between the neutral condition and the emotional prosodies was significant). Likewise, there was no significant difference between categories for the standard deviation of the mean energy of the sounds, \( F(4, 156) = 1.9, p > .10 \).

We selected 60 pseudowords pronounced by 12 different actors (6 women and 6 men) each in one of five different prosodies (anger, fear, happiness, neutral and sadness).

### 2.2.2.2 Vocal emotion recognition procedure

All the stimuli were played through stereo headphones using an Authorware program designed especially for this study. Participants sat comfortably in a quiet room, in front of the computer, and gazed at a fixation cross while listening to the stimuli. They were told that they would hear meaningless speech uttered by male/female actors and that these actors would express emotions through their utterances. After listening to a stimulus, participants rated its emotional content on a set of scales displayed simultaneously on the computer screen. More specifically, they were instructed to judge the extent to which the different emotions were expressed on visual analogue scales ranging from “not at all” to “very much”. There were six scales: one scale for each tested emotion (anger, fear, happiness and sadness) and one for neutral utterances, plus a scale to rate the “surprise” emotion, in order to find out whether the fear emotion expressed by the human voice is confused with surprise, as is the case with facial expressions (Ekman, 2003; Scherer and Ellgring, 2007). Participants were told that they could listen again to each stimulus as many as six times, by clicking on a button on the computer interface (click count). Participants were played two examples in order to familiarize them with the task. An example of the computer interface used for the recognition of emotional prosody task is provided in Appendix A.

The entire protocol was completed within a single session lasting approximately 3 hours, with several short breaks.

### 2.2.3. Statistical analysis

For the sociodemographic, psychiatric, and neuropsychological data, comparisons between the two groups (HC and MDD) were performed using a single-factor ANOVA. For the vocal emotion recognition data, we performed two levels of analysis.

First, we compared the performances of the two groups on categorical judgments according to percentages of correct responses using the chi-square \( (\chi^2) \) test. Second, we compared the performances of the two groups on the continuous rating scales for each prosodic category (plus neutral) and for each scale on the basis of a) overall performances, b) target scales and c) non-target scales. To do so, we conducted a repeated-measures ANOVA with two within-participants factors – emotion (5 levels) and scale (6 levels) – and one between-participants factor: group (MDD and HC; 2 levels). In order to investigate the effects in more detail, contrasts were performed between the two groups for each prosodic category and each rating scale (‘HC vs. MDD’).

To assess the relationship between the psychiatric, clinical, neuropsychological and sociodemographic variables and recognition of emotional prosody variables, they were entered into a multiple regression model using the stepwise method (Darlington, 1990; Draper and Smith, 1981). The regression analyses were only carried out for the MDD group and only for the variables that had previously been found to differ significantly between the MDD and HC groups.

The p-value was significant if less than 0.05. Statistical analysis was performed using Statistica 9.0.

### 3. Results

#### 3.1. Neuropsychological assessments (Table 2)

As shown in Table 2, there was no significant difference between the two groups, either on the Mattis score or on the PEGA.

As far as executive functions are concerned, the MDD group performed significantly worse than the HC group on the interference score of the Stroop test, all the variables of the TMT, the numbers of errors and categories completed in the MCST, categorical fluency and Action (Verb) Fluency. However, there was no significant difference

### Table 2

Neuropsychological background data for the depressed patient group and the healthy control (HC) group.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Depressed (( n = 21 ))</th>
<th>HC (( n = 21 ))</th>
<th>Stat. val.</th>
<th>df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mattis (max. = 144)</td>
<td>Mean ± SE</td>
<td>Mean ± SE</td>
<td>( F )</td>
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<td>.0001</td>
</tr>
<tr>
<td>Stroop</td>
<td>Interference</td>
<td>-4.3* 1.0</td>
<td>2.7 1.5</td>
<td>15.12</td>
<td>1.40</td>
</tr>
<tr>
<td>TMT</td>
<td>A (seconds)</td>
<td>52.4 4.9</td>
<td>36.7 3.7</td>
<td>6.52</td>
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</tr>
<tr>
<td>B (seconds)</td>
<td>131.1 15.8</td>
<td>84.6 9.3</td>
<td>6.42</td>
<td>1.40</td>
<td>.01</td>
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<tr>
<td>Verbal fluency</td>
<td>A (seconds)</td>
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<td>48.0 7.1</td>
<td>4.19</td>
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<td>B-A (seconds)</td>
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<td>35.0 1.9</td>
<td>4.19</td>
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<tr>
<td>Categorical (2 min.)</td>
<td>19.7 2.5</td>
<td>23.8 1.5</td>
<td>1.85</td>
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<tr>
<td>Action (Verb) (1 min.)</td>
<td>12.1 1.3</td>
<td>19.1 2.1</td>
<td>7.69</td>
<td>1.40</td>
<td>.01</td>
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<td>MCST</td>
<td>Categories (max. = 6)</td>
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<td>5.9 0.5</td>
<td>6.04</td>
<td>1.40</td>
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<td>2.9 0.5</td>
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<td>1.40</td>
<td>.03</td>
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<td>0.6 0.2</td>
<td>1.33</td>
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<td>9.1</td>
<td>1.34</td>
<td>1.40</td>
</tr>
</tbody>
</table>

Statistical values (stat. val.), degrees of freedom (df) and p-values between depressed and HC groups are reported (\( t \)-test for two independent groups). SE = Standard error; TMT = Trail Making Test; MCST = Modified version of the Wisconsin Card Sorting Test, max. = maximum score; PEGA = Montreal – Toulouse auditory agnosia battery.

* Significant if p-value less than 0.05.
### Table 3
Percentage of correct responses (and standard errors, SE) on categorical judgments in the emotional prosody task for the depressed patient group and the healthy control (HC) group.

<table>
<thead>
<tr>
<th>Scale</th>
<th>Depressed (n=21)</th>
<th>HC (n=21)</th>
<th>Stat. val.</th>
<th>df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger</td>
<td>Mean ±SE</td>
<td>Mean ±SE</td>
<td>χ² (df)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressed</td>
<td>56.1 ± 13.1</td>
<td>62.3 ± 10.4</td>
<td>5.34</td>
<td>1</td>
<td>.066</td>
</tr>
<tr>
<td>HC</td>
<td>52.8 ± 14.1</td>
<td>57.5 ± 12.3</td>
<td>3.69</td>
<td>1</td>
<td>.053</td>
</tr>
</tbody>
</table>

#### Statistical values (stat. val.)
- Degrees of freedom (df) and p-values between the two groups.

* Significant if p-value below 0.05.

### Table 4
Means (and standard errors, SE) of rating scales in the emotional prosody task for the depressed patient group and the healthy control (HC) group.

<table>
<thead>
<tr>
<th>Scale</th>
<th>Depressed (n=21)</th>
<th>HC (n=21)</th>
<th>Stat. val.</th>
<th>df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Happiness</td>
<td>Mean ±SE</td>
<td>Mean ±SE</td>
<td>χ² (df)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressed</td>
<td>2.92 ± 0.6</td>
<td>3.56 ± 0.8</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HC</td>
<td>3.48 ± 0.7</td>
<td>4.02 ± 0.9</td>
<td>1.52</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Significant if p-value below 0.05 and ** significant if p-value below 0.01 in comparison to HC.
3.2.2.3. Sadness. For overall performances, when the stimulus was “Sadness”, contrasts revealed no significant difference between the MDD and HC groups, F(1, 40) = 3.57, p = .06.

For the Sadness scale when the stimulus was “Sadness”, contrasts revealed no significant difference between the MDD and HC groups, F<1.

When the stimulus was “Sadness”, contrasts revealed that the MDD rated the Surprise scale significantly higher than the HC did, F(1, 40) = 4.64, p = .04. There was no difference when the scales were Happiness, F<1, Fear, F(1, 40) = 2.25, p = .1, Neutral, F(1, 40) = 2.18, p = .2, or Anger, F<1.

3.2.2.4. Happiness. For overall performances, when the stimulus was “Happiness”, contrasts revealed a significant difference between the MDD and HC groups, F(1, 40) = 6.90, p = .01.

For the Happiness scale when the stimulus was “Happiness”, there was no significant difference between the MDD and HC groups, F<1. When the stimulus was “Happiness”, contrasts revealed that the MDD rated both the Fear scale, F(1, 40) = 684.45, p = .001, and the Sadness scale, F(1, 40) = 4.41, p = .04, significantly higher than the HC did. There was no difference when the scales were Neutral, F<1, Anger, F(1, 40) = 1.48, p = .2, or Surprise, F(1, 40) = 2.32, p = 1.

3.2.2.5. Neutral. For overall performances, when the stimulus was “Neutral”, contrasts did not reveal any difference between the MDD and HC groups, F(1, 40) = 1.95, p = .17.

For the Neutral scale when the stimulus was “Neutral”, there was no significant difference between the MDD and HC groups, F<1. When the stimulus was “Neutral”, there was no difference when the scales were Happiness, F(1, 40) = 3.70, p = .06, Fear, F<1, Sadness, F<1, Anger, F(1, 40) = 1.13, p = .3, or Surprise, F<1.

3.3. Relationship between recognition of emotional prosody and other parameters

A multiple regression model for recognition of emotional prosody in patients with MDD was calculated, including psychiatric, clinical, and neuropsychological variables as predictors. As a reminder, the regression analyses were carried out only for the variables that were specific to the MDD group and for those variables that had previously been found to differ significantly between the MDD and HC groups: 1) the length of time since the first symptoms of the current episode, 2) the length of time since the first symptoms of the first episode, 3) MADRS total score, 4) interference score of the Stroop test, 5), the TMT B-A score, 6) the number of errors, 7) the number of categories completed in the MCST, 8) categorical fluency and 9) Action (Verb) fluency. The p-value was significant if less than 0.05.

When the stimulus was “Anger” and the scale Fear, the best fit was achieved using the TMT B-A score and the MADRS total score as predictors (p = .04 for the TMT B-A score, p = .04 for the MADRS total score, R² = 0.73).

When the stimulus was “Fear” and the scale Surprise, the best fit was achieved using categorical fluency as the predictor (p < .001, R² = 0.69).

When the stimulus was “Happiness” and the scale Fear, the best fit was achieved using categorical fluency as the predictor (p < .001, R² = 0.69).

When the stimulus was “Happiness” and the scale Sadness, the best fit was achieved using categorical fluency as the predictor (p < .001, R² = 0.72).

When the stimulus was “Sadness” and the scale Surprise, the best fit was achieved using the number of errors in the MCST as the predictor (p < .001, R² = 0.54)."

4. Discussion

The aim of the present study was to explore the recognition of emotions expressed in the vocal modality in major depressive disorder (MDD) patients, in order to measure the extent to which the disruptions in emotion recognition observed in MDD are specific to the visual modality or are also present in the auditory one. We compared recognition of emotional prosody performances of an MDD patient group and an HC group (n = 21 in each group) using an original emotional prosody recognition paradigm (Péron et al., 2010b).

The participants’ responses were investigated using two complementary methods adapted to the experimental paradigm. First, we compared the performances of the two groups on categorical judgments, in terms of percentages of correct responses. Second, we compared the performances of the two groups on the continuous judgments for each type of prosody.

At the first level of analysis, the investigation of categorical judgments revealed a significant difference between the two groups on the overall score (Table 3). As a whole, the MDD group performed more poorly than the HC group, displaying significant impairment of recognition of emotional prosody for fear, happiness and sadness. The results of the MDD group replicated previous findings reported in the emotional prosody literature. To date, studies have consistently found that MDD patients display impaired recognition of the emotional meaning of prosodic cues in speech compared with matched HC.

Surprise recognition was found to be selectively impaired in Kan et al.’s study (2004), as was neutral emotion, anger, surprise and sadness recognition in Murphy and Cutting’s study (1990), while Emerson and colleagues reported impaired recognition of happiness, anger, neutral emotion and sadness (1999). As far as Bach and colleagues’ findings (2009) are concerned, it is impossible to reach any conclusions about possible impairment within the MDD group, as patients were not compared with HC. In any case, as we pointed out earlier, all these authors opted for categorical judgments, which are ill-suited to capturing subtle effects of biases in information processing.

At the second level of analysis, an investigation of continuous judgments (rating scale means) allowed us to probe our data in greater depth, and to confirm and supplement the categorical results (Table 4). Compared with the HC group, the MDD group rated Fear significantly higher when they listened to “Anger” stimuli. They displayed a similar bias toward Surprise, rating it significantly higher when they listened to sad or fearful utterances. Furthermore, these contrasts revealed that when they listened to “Happiness” stimuli, MDD patients rated negative emotions (i.e., Fear and Sadness) higher than the HC did.

I t is noteworthy that both groups were comparable for age, education and gender, meaning that demographic variables could not account for the observed differences. None of the patients included in the study wore hearing aids or had a history of tinnitus or hearing impairment. In order to assess the influence of neuropsychological and illness-related factors, a multiple regression model was calculated for patients with MDD and for the continuous variables that were significantly different from the HC group. First, the MADRS total score and the TMT B-A score emerged as the best predictors for recognition of emotional prosody (responses on Fear scales when they listened to Anger), rendering all the other illness-related variables redundant and suggesting for the first time that recognition of emotional prosody is related to depression severity and the dysexecutive syndrome. Second, the number of errors in the MCST and categorical fluency emerged as the best predictors for recognition of emotional prosody (responses on Surprise scale when they listened to Fear, responses on Fear scale when they listened to Happiness, responses on Sadness scale when they listened to Happiness, and responses on Surprise scale when they listened to
Sadness), suggesting that recognition of emotional prosody is related to the inability to shift mental set in depression. Even though some authors have found MDD patients to perform normally in many areas of executive functioning (Grant et al., 2001; Landro et al., 2001; Stordal et al., 2005; Vythilingam et al., 2004), impaired executive functioning has frequently been reported in the acute phase of MDD (Hamm and Arndal, 2009; Harvey et al., 2004; Marazziti et al., 2011; Rogers et al., 2004; Stordal et al., 2004) and deficits have been highlighted in tests measuring inhibition (Den Hartog et al., 2003; Gohier et al., 2009; Markela-Lerenc et al., 2006), problem-solving and planning (Naismith et al., 2003), mental flexibility (Airaksinen et al., 2004; Naismith et al., 2003), verbal fluency (Ravnkilde et al., 2002), decision-making (Chamberlain and Sahakian, 2006), working memory (Egeland et al., 2003; Naismith et al., 2003; Rose and Ebmeier, 2006; Taylor Tavares et al., 2007), and attention modulation (Hugdahl et al., 2009). More specifically, it has previously been hypothesized that the most prominent executive functioning impairment in MDD patients is poor set-shifting (Austin et al., 2001), and that this state of cognitive rigidity can prevent patients from coping with life events, thus perpetuating their depressed mood by prolonging stress exposure. These studies, moreover, have underscored the fragility of patients’ subjective responses, the interaction between cognition and emotional response, and the tendency to overreact to the mistakes they make. For example, they respond “catastrophically” to errors and any mistake, even on a simple task of sustained attention, seems to heighten their subjective sense of failure (Farrin et al., 2003). These results have been explained by some recent brain imaging findings, which have shown reduced blood flow, particularly in the medial prefrontal cortex and dorsal anterior cingulate cortex, suggesting that these hyperperfused brain areas may underpin executive function impairments in MDD and highlighting the need to control for executive functioning (Mayberg, 2003).

There were several limitations to the present study that need to be acknowledged and addressed. First, all the MDD patients had been on their antidepressant treatment for two weeks when we examined them. It is now well documented that even very brief (single day or week) treatment with a serotonergic antidepressant brings about selective changes in emotional information processing, particularly in the recognition of facial expressions of emotions (Bhagwagar et al., 2004; Harmer et al., 2004; Merens et al., 2007), before there have been any changes in mood and symptoms. In this context, the antidepressant treatment could therefore represent a confounding factor. It would be extremely useful to devote a further study to the effects of antidepressant treatment on emotional prosody in MDD patients—something which has never been done to our knowledge. Second, we need to bear in mind that our present data were collected from a relatively small patient sample (n = 21), raising the question of how far our present results can be generalized to other MDD patients. Third, our study did not allow us to distinguish between state-like and trait-like bias. Any future studies should include a personality assessment (e.g., with the SCID-II), in an attempt to answer this question. Fourth and last, it may seem counterintuitive for percent-correct values to differ between MDD and HC for several emotions, when continuous judgment ratings of the target emotion failed to show any group differences. However, such results can be attributed to a categorization bias (Péron et al., 2010a). The categorization score was calculated a posteriori, in that we attributed a dichotomic score of 1 or 0 on the target scale: 1 if this scale was rated higher than the other scales, and 0, if it was not. In this kind of analysis, the responses are therefore polarized between true and false (1 or 0) and the test itself can be likened to a forced-choice procedure, in that participants have to base their recognition of emotional prosody on the suggested alternatives (five in this experiment). This procedure does not necessarily elicit the inferences we are called upon to make (without explicitly listed alternatives) in everyday life and forces participants to make “extreme” choices. On the other hand, the simultaneous use and analysis of different scales for each stimulus allowed us to study responses that were closer to everyday life and to qualify them accordingly.

The biases toward fear with angry expressions and toward surprise with sad and fearful expressions appear to confirm that the disruption of emotion recognition in MDD is not confined to the visual modality (e.g., faces), but is also exhibited when emotions are expressed through the human voice. Although it is still a matter of some debate (Archer et al., 1992; Gaebel and Wolwer, 1992), depressed subjects have been found to recognize expressions both more slowly and less accurately than HC (Feinberg et al., 1986; Persad and Polivy, 1993; Zuroff and Colussi, 1986). However, some studies have only shown recognition deficits for specific types of facial expressions, such as happiness (Mandal and Bhattacharya, 1985) or happiness, interest, and sadness (Rubinow and Post, 1992).

In this context, the performances of the MDD group on this emotional prosody paradigm may thus have been a consequence of a systematic cognitive bias toward negative emotions (Mineka and Sutton, 1992; Wenzel and Finstrom, 2007). As mentioned in the Introduction, the vast majority of studies that have explored the impact of depression on the recognition of facial expressions have examined this emotional processing by asking participants to categorize pictures of facial expressions on the basis of their emotional content (e.g., is the face happy, neutral, or sad?) (Archer et al., 1992; Feinberg et al., 1986; Gaebel and Wolwer, 1992; Mandal and Bhattacharya, 1985; Persad and Polivy, 1993; Zuroff and Colussi, 1986). The present results are thus more comparable to those of studies on the recognition of facial expressions using a methodology similar to ours, that is, by asking MDD patients to rate the intensity of different emotional states displayed in pictures of facial expressions (Bouhuys et al., 1995, 1999, 1997, 1996; Gur et al., 1992; Hale, 1998; Hale et al., 1998). These comparisons are of particular interest when it comes to explaining our present results. For example, Hale (Hale, 1998) found a significant (positive) correlation between judgments of negative emotions in facial expressions and the severity of depressive symptoms. This suggests that depression results in an increased tendency to perceive negative emotional states in others. It has also been shown that depressed patients judge facial expressions to express less positive emotions than HC do (Hale et al., 1998), which is a similar finding to ours, notably concerning the bias found in higher fear and sad ratings for happy expressions. Gur et al. (1992) reported a similar negative bias, in that depressed patients were more likely than controls to incorrectly attribute sadness to neutral faces and a neutral emotional state to happy faces. In this context, our results suggest that the bias for negative emotional stimuli in depression observed in the visual modality (Gotlib et al., 2004a,b) is also present when emotions are conveyed by the human voice. Even though their findings remain subject to debate (Gotlib et al., 2004a), some authors have also suggested that responsiveness to positive emotional stimuli is blunted in MDD. The present results seem to confirm this hypothesis, but also go one step further, by suggesting that the bias toward negative emotional stimuli and the “blunting” of responsiveness to positive ones is not specific to the visual modality. In addition, it is important to stress that the differences we found between groups cannot be accounted for by generalized emotional blunting in the MDD group (i.e., reporting a smaller range of emotional prosody). This seems to be a particular strength of the present results. Finally, our results underline the importance of using continuous scales rather than categorical judgments, as the former enabled us to probe the data in greater depth and uncover these emotional biases.

The present results, particularly the differential impact of major depression on the recognition of negative vocal emotions, now need
to be confirmed with a larger sample of depressed patients. Lastly, further evidence needs to be gathered from depressed patients, in particular using functional neuroimaging, to establish a correlation between deficits in decoding emotional prosody and changes in neuronal activation, particularly in those brain regions that are known to be involved in emotional processing, such as the orbitofrontal cortex and caudate nucleus.

**Conflict of interest**

The authors report no conflicts of interest.

**Contributors**


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**Appendix A. Computer interface for the emotional prosody recognition paradigm**