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Performance monitoring and empathy during active and observational learning in patients with major depression

Patrizia Thoma[a,*], Christine Norra[b], Georg Juckel[b], Boris Suchan[a], Christian Bellebaum[c]

[a] Department of Neuropsychology, Institute of Cognitive Neuroscience, Faculty of Psychology, Ruhr University Bochum, Bochum, Germany
[b] Department of Psychiatry, Ruhr-University of Bochum, LWL University Hospital, Alexandrinenstraße 1, 44791 Bochum, Germany
[c] Institute for Experimental Psychology, Heinrich Heine University Düsseldorf, Universitätsstraße 1, 40225 Düsseldorf, Germany

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ABSTRACT

Previous literature established a link between major depressive disorder (MDD) and altered reward processing as well as between empathy and (observational) reward learning. The aim of the present study was to assess the effects of MDD on the electrophysiological correlates – the feedback-related negativity (FRN) and the P300 – of active and observational reward processing and to relate them to trait cognitive and affective empathy. Eighteen patients with MDD and 16 healthy controls performed an active and an observational probabilistic reward-learning task while event- related potentials were recorded. Also, participants were assessed with regard to self-reported cognitive and affective trait empathy. Relative to healthy controls, patients with MDD showed overall impaired learning and attenuated FRN amplitudes, irrespective of feedback valence and learning type (active vs. observational), but comparable P300 amplitudes. In the patient group, but not in controls, higher trait perspective taking scores were significantly correlated with reduced FRN amplitudes. The pattern of results suggests impaired prediction error processing and a negative effect of higher trait empathy on feedback-based learning in patients with MDD.

1. Introduction

Disrupted reward processing contributes to cardinal depressive symptoms such as anhedonia (Nestler & Carlezon, 2006; Russo & Nestler, 2013). While healthy individuals perceive a range of stimuli as rewarding (e.g., Aron et al., 2004; Bellebaum, Jokisch, Gizewski, Forsting, & Daum, 2012; Kampe, Frith, Dolan, & Frith, 2001; Rademacher et al., 2010), participants with subclinical depression and those with major depressive disorder (MDD) show lower reward sensitivity and motivationally reduced reward-seeking (Brinkmann & Franzen, 2013; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008), a diminished capacity to sustain reward-maximizing behavior (Liu et al., 2011) and poor reward-based decision making (Kunisato et al., 2012; Must et al., 2006).

The reward system involves projections from the midbrain to the striatum and frontal cortex, particularly the medial orbitofrontal cortex (OFC) and anterior cingulate cortex (ACC) (Berger, Gaspar, & Verney, 1991; Haber & Fudge, 1997; Williams & Goldman-Rakic, 1993), with dopamine (DA) as the primary neurotransmitter (Schultz, Dayan, & Montague, 1997). MDD has been related to reduced volumes of the ACC and OFC (Bora, Fornito, Pantelis, & Yucel, 2012a; Bora, Harrison, Davey, Yucel, & Pantelis, 2012b; Lai, 2013) and to blunted DA responses to rewards (Epstein et al., 2006; Nestler & Carlezon, 2006). In functional magnetic resonance imaging (fMRI) studies, altered activation patterns to reward were observed in reward-sensitive regions in patients with acute MDD (Zhang, Chang, Guo, Zhang, & Wang, 2013), remitted MDD (Dichter, Kozink, McClernon, & Smoski, 2012; Schiller, Minkel, Smoski, & Dichter, 2013) and in individuals with an increased risk for depression (Gotlib et al., 2010; Macoveanu et al., 2013; McCabe, Woffindale, Harmer, & Cowen, 2012).

An event-related potentials (ERPs) component, the feedback-related negativity (FRN), occurring between 200 and 300 ms after presentation of the feedback stimulus, has been associated with the processing of monetary punishment and negative performance feedback (Bellebaum & Daum, 2008; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003; Miltner, Braun, & Coles, 1997). It is regarded as a DA-driven teaching signal in feedback-based learning (Holroyd & Coles, 2002). As the response-locked error-related negativity (ERN) or error negativity (Ne) (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993), which also relies on DA signalling (Holroyd & Coles, 2002), the FRN is gener-

* Corresponding author at: Institute of Cognitive Neuroscience, Department of Neuropsychology, Ruhr University Bochum Universitaetsstrasse 150, 44780 Bochum, Germany. Fax: +49 234 32 14622.
E-mail address: Patrizia.Thoma@rub.de (P. Thoma).

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ated in the ACC (Gehring & Willoughby, 2002), but the striatum was also identified as a likely neural generator (Foti, Weinberg, Dien, & Hajcak, 2011b). The FRN is thought to reflect the use of reward signals by the ACC for representations of stimulus-outcome contingencies (Bismark, Hajcak, Whitworth, & Allen, 2013). Attenuated FRN amplitudes have been related to decreased reward sensitivity (Bress & Hajcak, 2013).

Another prominent ERP component, the P300, has rarely been addressed in the context of feedback processing, with unclear results. The P300, a positive ERP peaking about 300 ms after stimulus onset at parietal electrode sites, is generally known to be sensitive to violations of expectations (e.g., Courchesne, Hillyard, & Courchesne, 1977; Johnson & Donchin, 1980). Feedback valence either did not modulate P300 amplitudes (Sato et al., 2005; Yeung & Sanfey, 2004), or there was P300 enhancement for positive feedback, unexpected and larger outcomes, lower stimulus occurrence probability and greater motivational salience (Carrillo-de-la-Pena & Cadaveira, 2000; Hajcak et al., 2005; Leng & Zhou, 2010; Yeung & Sanfey, 2004). To our knowledge, only Foti and Hajcak (2009) investigated the P300 in association with feedback processing and depressive symptoms. They reported attenuated amplitudes in response to positive and negative feedback in healthy participants with elevated depression scores.

Findings relating depressive symptoms to the ERN/FRN are inconsistent: In children, depressive symptoms were associated with blunted FRN amplitudes to positive feedback (Bress, Smith, Foti, Klein, & Hajcak, 2012), but state negative affect was related to an increased FRN to negative feedback in adults (Gu, Ge, Jiang, & Luo, 2010; Santesso et al., 2011). This might reflect developmental differences as FRN attenuation to monetary gains was also related to greater sadness following mood induction in girls with increased risk for depression (Foti, Kotov, Klein, & Hajcak, 2011a) and prospectively predicted the development of MDD in never-depressed adolescent girls (Bress, Foti, Kotov, Klein, & Hajcak, 2013). In (largely) unmedicated MDD samples, ERN amplitudes were either inconspicuous (Olvet, Klein, & Hajcak, 2010) or the ERN (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008; Tang et al., 2013) and FRN (Mies et al., 2011) were enhanced. In medicated patients, both the ERN and FRN were reduced (Ruchsow et al., 2004, 2006). Finally, patients with remitted MDD showed ERN (Georgiadi, Liotti, Nixon, & Liddle, 2011) and FRN (Santesso et al., 2008) enhancement. Taken together, in unmedicated acute or remitted MDD, increased ERN/FRN amplitudes might suggest hyperactive performance monitoring. FRN attenuation may represent an early vulnerability marker and later reflect combined effects of acute depression and medication.

The studies cited above employed paradigms with deterministic stimulus-response contingencies, such as Flanker, Go/Nogo or Stroop tasks (e.g., Holmes & Pizzagalli, 2008; Ruchsow et al., 2004, 2006) or tasks inducing unclear reward expectations (e.g., time estimation: Mies et al., 2011). Feedback could not be used for the optimization of behavior in any of the studies. However, the FRN is most pronounced when a response strategy can be learned (Holroyd et al., 2009). This gap in the literature is addressed by using a probabilistic learning task in the present study. Furthermore, observational learning has not been assessed in MDD, although it is particularly relevant for interpersonal functioning and for the benefit patients can derive from group therapy. Finally, we take into account that performance monitoring is related to cognitive (e.g., perspective taking) and affective (affective sharing and personal affective responses) empathy. This might be based on overlapping neural circuits, with a key role for the ACC (see Thoma & Bellebaum, 2012 for a review). Positive associations between trait empathy and the ERN were reported for active responding (Larson, Fair, Good, & Baldwin, 2010; Santesso & Segalowitz, 2009), increasing in strength during the observation of another person's behavior (e.g., Newman-Norlund, Ganesh, van Schie, de Bruijn, & Bekkering, 2009). However, when participants had to learn difficult stimulus-reward contingencies by observing others, higher trait empathy was associated with poorer performance, a reduced FRN to positive vs. negative feedback (Kobza, Thoma, Daum, & Bellebaum, 2011) and a smaller P300 (Rak, Bellebaum, & Thoma, 2013). This suggests that a stronger tendency to adopt someone else's emotional perspective might interfere with observational learning. This is particularly relevant for MDD, as depressive symptoms were associated with increased affective (Thoma et al., 2011; Connor, Berry, Weiss, & Gilbert, 2002) and diminished cognitive (Schreiter, Pijnenborg, & Aan Het, 2013) empathy. However, empathy has not been investigated in relation to reward learning in MDD as yet.

Taken together, we assessed feedback processing in adult patients with MDD and healthy controls during active and observational probabilistic reward learning, focusing on the FRN and the P300. We did not specify hypotheses for the P300 due to the scarce and inconsistent previous findings with regard to the role of the P300 in the context of feedback processing. We expected overall FRN reduction in MDD patients. This effect should be more pronounced for observational learning, as this condition ought to be particularly affected by empathy impairment. Based on previous findings (Kobza et al., 2011; Rak et al., 2013), we expected negative associations between reward learning performance/ERPs and trait empathy.

2. Method

2.1. Participants

Twenty-three patients with MDD and 18 healthy control (HC) participants were recruited. Five patients (two because of substance abuse in the past six months, two due to quitting the experiment prematurely, one due to transpiration artifacts in the ERPs) and two controls (one with a neurological disorder, one with a history of MDD) were excluded. Thus, the data of 18 MDD patients and 16HC, matched on age, sex and years of education, were analyzed (see Table 1 for demographic and clinical characteristics). There were no significant group differences regarding age, gender distribution, IQ estimate, as assessed with a multiple-choice vocabulary test (Lehrl, Triebig, & Fischer, 1995), or years of education (all $ps \geq .096$). Patients were recruited from the Department of Psychiatry, LWL Hospital Bochum and diagnosed with MDD according to the DSM IV-TR (American Psychological Association, 2000) by a senior psychiatrist. Eleven patients were hospitalized and seven were treated on an outpatient basis. Demographic data, health status and substance abuse were assessed by self-report. Current or past psychiatric disorders were screened with the M.I.N.I PLUS International Neuropsychiatric Interview (German: Ackenheil, Stotz-Ingenlath, & Dietz-Bauer, 1999). Ten patients suffered from recurrent depressive episodes. A verbal IQ estimate lower than 80, a history of neurological disorders and current or past regular abuse of illicit drugs or alcohol represented general exclusion criteria. Patients were excluded if they presented with any psychiatric disorder other than MDD and controls in the case of any present or past psychiatric disorder or a history of MDD in a first-degree relative. Severity of depressive symptoms was screened with the Hamilton Depression Rating Scale (HAMD) (German: Baumann, 1986). HAMD scores were missing for one MDD and one HC participant. As expected, patients showed a significantly higher total score (range 8–29) than controls (range 0–4) ($t(17) = 10.25$; $p < .001$; see Table 1 for means and standard deviations). The severity of current depressive symptoms was "mild" in seven patients (HAMD-score: 8–13), "moderate" in four (HAMD-score: 14–18), "severe" in another four (HAMD-score: 19–22) and "very severe" ($\geq$23) in two patients. HAMD scores were within