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ORIGINAL PAPER

Altered Oscillatory Responses to Feedback in Borderline Personality Disorder are Linked to Symptom Severity

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Abstract

Several studies using electroencephalography (EEG) demonstrate that the processing of feedback in patients suffering from borderline personality disorder (BPD) is altered in comparison to healthy controls. Differences occur in the theta (ca. 5 Hz) and high-beta frequency-ranges (ca. 20 Hz) of oscillations in response to negative and positive feedback, respectively. However, alpha (ca. 10 Hz) and low-beta (ca. 15 Hz) oscillations have also been shown to be involved in feedback processing. We hypothesized that additional alterations might occur in these frequency ranges in BPD. Eighteen patients with BPD and twenty-two healthy controls performed a gambling task while 64-channel-EEG was recorded. Induced oscillatory responses to positive (i.e. gain) and negative (i.e. loss) feedback in the alpha and low-beta frequency range were investigated. No significant differences were found in the alpha frequency range. Regarding the low-beta frequency range a significant Group (i.e. BPD vs. healthy controls) × Valence (i.e. gain vs. loss) interaction in the time frame between 600 and 700 milliseconds after feedback was found. This effect showed a significant correlation with symptom severity (assessed with the BSL-23). The results indicate that feedback processing in BPD could be more heavily altered than previously expected, with more severe symptomatology being linked to stronger alterations in oscillatory responses to feedback in the low-beta range.

Keywords Feedback processing · Borderline personality disorder · Beta oscillations · Symptom severity · EEG · sLORETA

Introduction

The ability to recognize contingencies and to adapt behaviour accordingly is a key function in our everyday life. In this context, reward and loss are two of the most important indicators in guiding human behaviour and learning mechanisms (Marin 1991; Schultz 2006). The reward network consists of several core brain areas. Among these are the ventral striatum, the orbitofrontal and prefrontal cortex as well as the anterior cingulate cortex (Delgado et al. 2000; Knutson et al. 2001; Nieuwenhuis et al. 2005). Deficiencies of the reward network have been attributed a leading role in psychiatric disorders, especially in substance use disorders (Taber et al. 2012) and certain personality disorders such as borderline personality disorder (Bandelow et al. 2010), the main object of this paper.

Borderline personality disorder (BPD) affects large sections of the population with a 12-month prevalence of 1.4% (Lenzenweger et al. 2007) and life-time prevalence correspondingly higher with 5.9% (Grant et al. 2008). Patients affected by BPD suffer from instability in affect-regulation, interpersonal relationships, self-perception and

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Paul Alexander Schauer and Jonas Rauh share first authorship, whilst Christina Andreou and Christoph Mulert share last authorship.

Paul Alexander Schauer and Jonas Rauh have contributed equally to this work. Christina Andreou and Christoph Mulert have contributed equally to this work.

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impulse control (Bohus et al. 2007). This instability leads to severe impairments in everyday life and mortality rates four times higher than in the general population (Kjaer et al. 2015). The societal costs for patients suffering from BPD are considered to be substantial (van Asselt et al. 2007). Despite several efforts to determine the neurobiological origins of the severely dysfunctional behaviour in BPD no clear consensus could yet be reached.

Studies using electroencephalography (EEG) have identified certain electrophysiological response markers for the processing of feedback information which seem to be altered in BPD. One such marker is an event-related potential (ERP) called feedback error-related negativity (fERN; Holroyd and Coles 2002). The fERN is marked by a negative deflection around 250–300 ms following loss feedback (Hajihosseini and Holroyd 2013) and has been shown to be reduced in BPD patients (Schuermann et al. 2011). Apart from differences in ERPs, additional changes were observed in event-related oscillations (EROs; e.g. Vega et al. 2013): In healthy populations negative feedback in gambling tasks has been associated with a frontally distributed increase in theta-band power (Leicht et al. 2013; Marco-Pallares et al. 2008). In contrast, positive feedback in such tasks is associated with an increase of high-beta and low-gamma oscillatory activity (Andreou et al. 2017; HajiHosseini et al. 2012; Leicht et al. 2013; Marco-Pallares et al. 2008). Significant reduction in theta oscillatory power after negative feedback has been shown in patients with BPD (Vega et al. 2013), while processing of positive feedback has generally been found to be unimpaired. This seems to support the notion of two distinct pathways for the processing of positive and negative feedback (cf. Andreou et al. 2017), with only the latter being affected in patients with BPD.

In contrast to theta and high-beta oscillations, the role of alpha and low-beta frequency bands has generally been neglected in the past. In recent years, several studies have started to focus on these lower frequency bands (e.g. Hauser et al. 2015; Leicht et al. 2013; Pornpattananangkul and Nusslock 2016; Yaple et al. 2018). These studies sparked a new interest in these frequencies in relation to feedback processing, especially low-beta oscillatory responses to feedback in healthy controls (Yaple et al. 2018). Moreover, in a study by Leicht et al. (2013), low-beta frequency oscillations were linked to sensation seeking, a trait which is closely linked to BPD (Peters et al. 2013). Based on the above, two questions have arisen for us: First, whether the processing of positive feedback is really unaffected in BPD especially considering that the alpha and low-beta frequency ranges (in particular low-beta oscillations in response to positive feedback) have not yet been investigated in previous studies. Second, whether alterations in these frequency ranges might be linked to BPD-specific symptoms.

The present study aimed to investigate alpha and low-beta oscillations in response to feedback in BPD and their correlations with symptom severity in patients. To this end, we re-analysed data from a previous EEG-study in BPD patients (Andreou et al. 2015).

Materials and Methods

Participants

The total sample for the present study consisted of 21 patients with BPD and 23 healthy controls. For reasons explained below 3 patients and 1 healthy control had to be excluded. Included in the final sample used in all analyses were 18 patients and 22 healthy controls (see Table 1). Patients were recruited from the in- and outpatient clinics of the Department for Psychiatry and Psychotherapy, University Hospital Hamburg-Eppendorf, whereas healthy controls were recruited through advertisements and word-of-mouth. The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee of the Medical Council of Hamburg. All participants provided written informed consent prior to inclusion in the study.

Patients were required to fulfill criteria of BPD according to DSM-IV. The Mini International Neuropsychiatric Interview (Sheehan et al. 1998) and the Structured Clinical Interview for DSM-IV Axis II (Wittchen et al. 1997) were used to establish the diagnosis of BPD and assess Axis I comorbidities in patients. In order to minimize the effects of comorbid disorders associated with reward system dysfunction, patients were excluded from the study if they presented a current depressive episode or a score of 20 or higher on the Montgomery–Asberg Depression Rating Scale (MADRS; Montgomery and Åsberg 1979), alcohol or drug dependence, or alcohol or drug abuse, in the past year, or a lifetime diagnosis of psychotic or bipolar disorder. One female patient had to be excluded because of a MADRS score of 20 or higher. Further exclusion criteria for all subjects were neurological and developmental disorders, and the presence of uncorrected visual problems or hearing loss. In healthy controls, additional exclusion criteria were a family history of psychotic disorders or a personal history of any psychiatric disorder or treatment.

Symptom severity in patients was assessed with the Borderline Symptom List—Short Form (BSL-23; Bohus et al. 2009). The BSL-23 consists of 23 items answered on a fivepoint Likert scale and has been shown to have good internal consistency and validity.

Table 1 Sociodemographic characteristics of the two participant groups

	Healthy controls ($N=22$)		BPD ($N=18$)		χ^2/t	p
Gender (m/f)	4/18		1/17		1.44	0.35
	Mean	SD	Mean	SD		
Age	26.27	4.9	28.28	5.8	1.18	0.25
Education (years)	12.18	1.4	11.4	1.6	1.56	0.13

Paradigm

Participants performed a computerized two-choice gambling task adapted from Gehring and Willoughby (2002) and used in previous studies by our group and others (Marco-Pallares et al. 2008; Leicht et al. 2013; Vega et al. 2013). Each trial began with the presentation of a fixation square for 3 s, followed by two numbers (5 and 25) on a computer screen (randomized left–right order) for 1 s. Within this time, participants were required to select one of the two numbers per mouse-click. After a delay of 700 ms, one of the two numbers randomly turned green and the other red (feedback stimulus). If the selected number turned green, the participant gained the corresponding amount of points. A color change to red indicated a respective loss of points. Thus, the feedback stimulus varied along two dimensions, valence (positive vs. negative feedback) and magnitude (5 vs. 25 points). The feedback stimulus was displayed on the screen for 700 ms followed by a display of the current account status for 2000 ms (see Fig. 1 for a graphic depiction of the procedure).

The Presentation software (Version 14) was used for stimulus presentation. Participants were instructed in a standardized way to choose freely between the two presented numbers (5 or 25) in every trial and to gain as many points as possible during each block. Thus, participants were not required to choose 5 and 25 in an equal ratio (relevant for statistical analyses, see below). The instruction stated that participants would receive €10 for study participation, plus an additional amount of money depending on the total points won. The paradigm comprised a short practice block and four experimental blocks of 108 trials each. Participants started each block with 1000 points on their account. The occurrence of loss and gain events was maintained at equal probability (50% each).

EEG Recording and Pre-processing

Recordings took place in a sound-attenuated and electrically shielded room. Participants were seated in a slightly reclined chair with a head rest, at a distance of 1 m from a 19″ computer screen. Electro-encephalographic activity was recorded at a sampling rate of 1000 Hz with 64 Ag/AgCl electrodes mounted on an elastic cap (ActiCaps, Brain Products, Munich, Germany), using the Brain Vision Recorder software version 1 (Brain Products, Munich, Germany). Electrodes were arranged according to a modified 10/10 system without electrodes at positions FPz, F9, F10, T9, T10, CP3, CP4, P9, P10, PO7, PO8, and with two additional electrodes at positions PO9 and PO10. Eye movements were recorded with four EOG channels (positioned at the outer canthi bilaterally and infra- and supraorbitally on the right). An electrode at the FCz position was used as the reference, while the electrode at position AFz served as ground.

Offline preprocessing was performed with Analyzer 2 (Brain Products GmbH). After band-pass filtering (0.1–100 Hz, Butterworth zero-phase filter 24 dB/octave), prominent non-stereotyped artefacts such as movement artefacts and channel drifts were removed by visual inspection. Independent component analysis (ICA) was applied to remove blink and eye movement artefacts. A restricted Infomax algorithm was used for ICA; components representing ocular artefacts were identified and removed based on their topography, power spectrum and time course. Subsequently, the continuous EEG was segmented into 3-s epochs starting 1800 ms prior to the feedback stimulus. Segments including amplitudes exceeding ± 95 µV, voltage steps higher than 50 µV between sampling points, a difference higher than 200 µV between the highest and lowest value within a segment or activity below 0.5 µV were automatically rejected. After re-referencing to the common average reference, baseline correction (using the 200 ms pre-stimulus interval) was applied. Only subjects with a minimum number of 20 artefact-free trials per condition were considered in further analyses; one female patient had to be excluded based on this criterion. Two further subjects (one female patient and one male control subject) did not complete all blocks of the paradigm. Thus, the final sample consisted of 18 patients and 22 healthy controls.

Time–Frequency Analysis

Time–frequency information was extracted for EEG activity at electrode Fz using complex Morlet wavelet convolution for the frequencies from 10 to 30 Hz (formula:

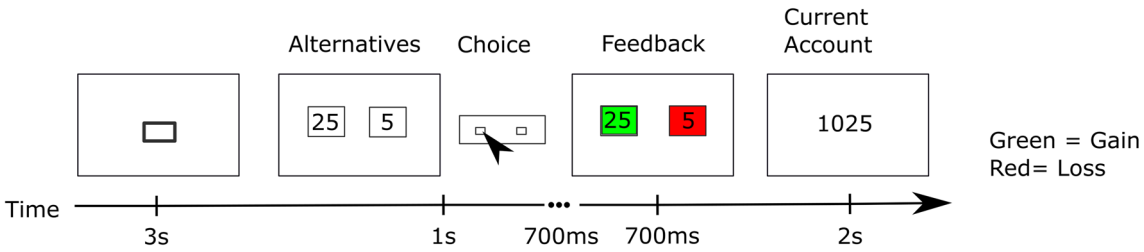

Fig. 1 Procedure of the two-choice gambling task adapted by us from Gehring and Willoughby (2002)

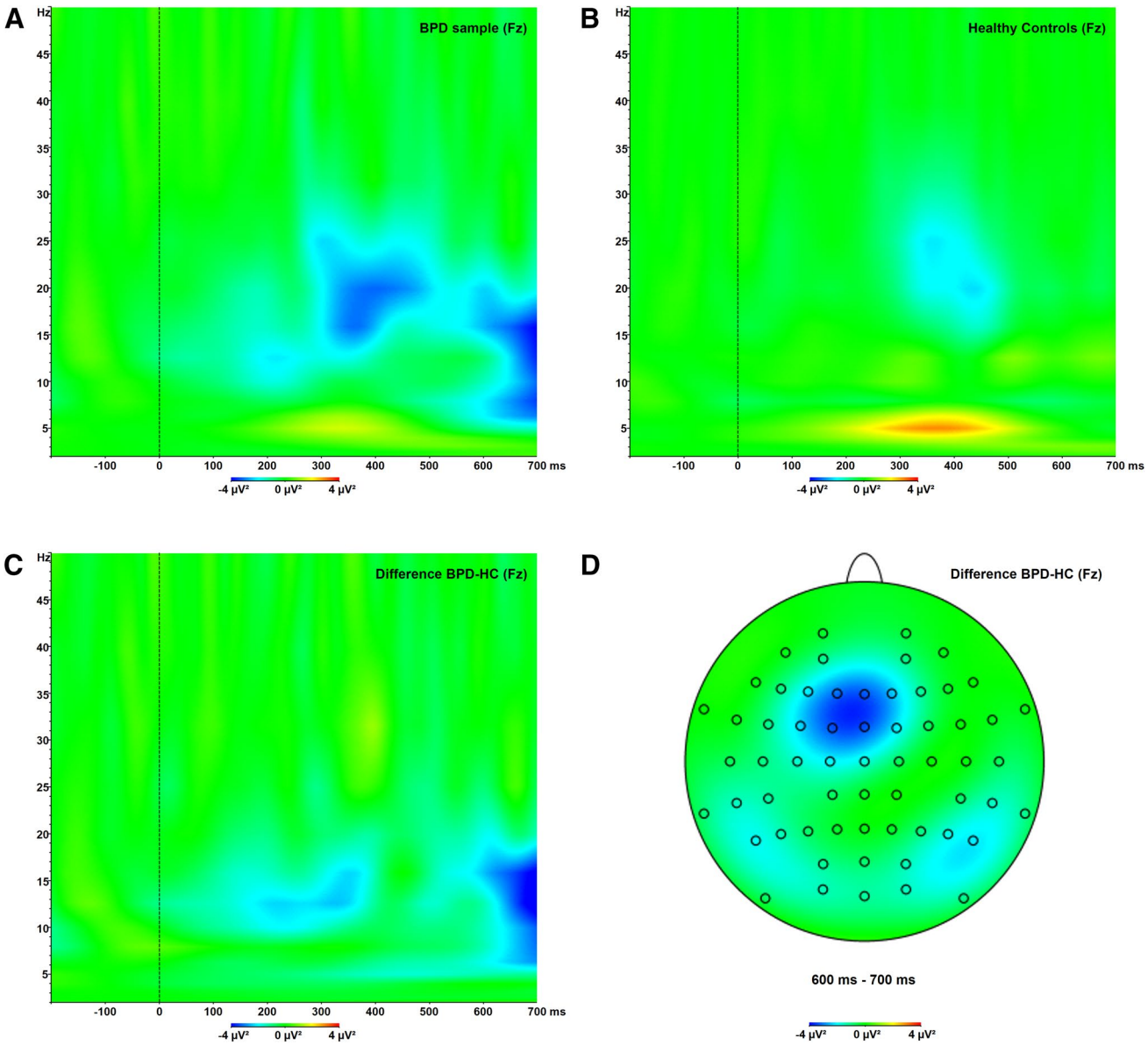

Fig. 2 Difference between gain and loss conditions in BPD patients (**a**) and healthy controls (**b**) in induced oscillatory responses. **c** depicts the difference between the two groups. To obtain time–frequency plots a complex Morlet wavelet convolution for the frequencies from 2 to 50 Hz (15 frequency steps distributed on a logarithmic scale, Morlet parameter $c=5$, Gabor Normalization) was applied. **d** Scalp distribution map of the difference between the two groups of low-beta power (15 Hz) in the timeframe of 600 and 700 ms

$\Psi(t,f) = Ae^{-\frac{-t}{2\sigma_t}} e^{i2\pi ft}$, 5 frequency steps distributed on a linear scale, Gabor Normalization, see Fig. 2). The Morlet parameter that describes the ratio of the standard deviation of frequency and the time relative to the frequency expressed by the formulas $c = \frac{f}{\sigma_f}$ and $c = 2\pi\sigma_t f$ was set to $c = 5$. Following Andreou et al. (2015) the calculation of induced power the ERP was subtracted from the single-trial series and wavelet transformation was applied at the single-trial level prior to averaging. In order to assess alpha and low-beta activity across conditions, we extracted wavelet layers with central frequencies of 10 Hz (alpha) and 15 Hz (low-beta), similar to a previous study by our group (Leicht et al. 2013). Based on the methods used in Leicht et al. (2013) and Andreou et al. (2015) the peak amplitude of activity for low-beta defined as the highest value was readout within the timeframe 600–700 ms post-stimulus. For alpha-activity we extracted the mean amplitude in the timeframe between 500 and 600 ms post-stimulus according to Leicht et al. (2013).

sLORETA Analysis

Intracortical sources of brain electrical activity were localized using standardized low-resolution electro-magnetic tomography (sLORETA; Pascual-Marqui 2002). sLORETA belongs to a group of three-dimensional, distributed, linear minimum-norm inverse solutions that have been extensively used and cross-validated (Mobascher et al. 2009; Mulert et al. 2004; Olbrich et al. 2009). The sLORETA software (http://www.uzh.ch/keyinst/sloreta.htm) was used to calculate time-varying cross-spectra from single trials after subtracting the average ERP to calculate induced activity. This transformation applied a sliding Gaussian Window function with a centre frequency of 15 Hz (window length 0.5 s) for the low-beta frequency range. sLORETA computations were made in a real-istic head model (Fuchs et al. 2002), using the MNI152 template (Mazziotta et al. 2001). The source space (6239 voxels at a spatial resolution of 5 mm) was restricted to cortical gray matter and hippocampi, as determined by the probabilistic Talairach atlas (Lancaster et al. 2000).

The time frame for current source density computations was defined at 600–700 ms following the feedback stimulus, based on the grand average of the time–frequency plots at electrode Fz in patients and controls. Using the in-built statistics function of sLORETA we calculated voxel-wise whole brain-analysis with between subjects factor group (i.e. BPD vs. healthy controls) and within subject factor valence (i.e. gain vs. loss) resulting in a 2 × 2 mixed design.

Statistical Analyses

Statistical analyses were performed using SPSS 24. The pattern of "high-gain, high-risk" behaviour during the gambling task, i.e., the percentage of trials, in which participants selected the higher number, was contrasted between groups with an independent *t*-test. Differences between groups in the number of artefact-free trials were assessed with a 2 (magnitude) × 2 (valence) × 2 (group) repeated-measures ANOVA.

The above analyses revealed significant differences between groups regarding the mean number of trials included in the averages for each condition, due to the fact that patients with BPD chose the higher number (25) much more often than healthy controls (see below). Therefore all further analyses were conducted with linear mixed models, including the number of valid trials in each condition as a covariate. For the analysis of the event-related oscillations in the alpha- and low-beta-frequency-ranges the induced power within the predetermined time windows was used as dependent variable. Group was defined as a between-subjects fixed-effect factor; valence and magnitude were used as repeated-measures fixed-effect factors. In all cases, participant ID was included as a random-effects factor, and the full factorial model was assessed. A diagonal covariance structure assuming heterogeneous variances and zero correlations between elements was used. Only main effects and interactions involving the factor group are presented below.

Difference scores between gain and loss conditions (averaged over both magnitudes) were constructed for effects that emerged as significant from the above analysis and their correlation to the symptom severity as assessed by the BSL-23 (Bohus et al. 2009) was explored with Spearman's rho. Two participants (both female) with extreme outlier difference scores (> 3 standard deviations from the group mean) were excluded from these analyses.

Results

As previously reported elsewhere (Andreou et al. 2015), patients with BPD placed significantly more often a "higher bet", i.e., selected significantly more often 25 compared to healthy controls [55.2% vs. 45.9%, $t(38) = 2.220$, $p = 0.03$]. Regarding the number of artefact-free trials, the main effect of group was not significant [$F(1,39) = 0.961$, $p = 0.33$]. However, there was a significant group × magnitude interaction [$F(1,39) = 10.423$, $p = 0.003$]; there were significantly more trials in the high-stake than in the low-stake condition in patients [$F(1,18) = 22.995$, $p = 0.001$], whereas controls had an equal number of trials in the two conditions [$F(1,21) = 0.488$, $p = 0.49$].

Induced Event-Related Responses

For low-beta induced power at electrode Fz, a significant group × valence interaction was found [$F(1,83.45) = 6.820$, $p = 0.011$], which increased more in patients than in controls following gain in comparison to loss feedback (see Fig. 3). No significant effects involving the factor group were found for induced alpha power at electrode Fz.

In sLORETA whole-brain analyses using cluster-corrected non-parametric permutation tests, the group x valence interaction for low-beta reached marginal significance in a cluster situated in the medial frontal cortex (see Fig. 4) and consisting mainly of Brodmann area 6 and parts of Brodmann areas 24 and 31 [$t = 3.036$, $p = 0.052$].The same pattern of a stronger activation following gain in comparison to loss feedback in patients emerged with no such effect for controls (see Fig. 5).

Correlations with Symptom Severity

In the patient group we found a significant positive correlation between the valence difference score (i.e. gain minus loss) for induced low-beta and the BSL-23 score (Spearman's rho = 0.522, $P = 0.038$). Indicating higher induced low-beta responses to gain compared to loss feedback in patients with higher BSL-23 scores. Further exploratory analyses using the individual item scores in place of the total BSL-23 score were conducted to further investigate this significant correlation. Both item 2 and item 3 of the BSL-23 showed significant correlations with the difference score (rho = 0.637, $P = 0.008$ and rho = 0.597, $P = 0.015$ for items 2 and 3 respectively). Item 2 codes helplessness ("During the last week I experienced myself as being helpless") whilst item 3 codes dissociative experiences ("During the last week I was mentally absent and I do not remember exactly what I was doing"). Since no BSL-23 data were available for healthy controls there was no possibility of calculating similar correlations for this group.

Discussion

The aim of the present study was to investigate alpha and low-beta frequency band oscillatory responses associated with reward feedback in patients with borderline personality disorder (BPD) compared to healthy controls. Although we found no significant alterations in the alpha frequency range, we found significant impairments in the low-beta range. BPD patients showed a pattern of increased low-beta power following gain trials in comparison to loss trials with no comparable effect in the control group. Furthermore, we found a significant correlation between the low-beta valence difference score and BPD symptom severity assessed by the BSL-23. The observed increase in frontal low-beta power following positive feedback in patients with BPD attests, to the best of our knowledge for the first time, to a deficit not just in negative, but also in positive feedback processing in patients.

Adding to previous reports on altered responses to feedback in the theta range (Andreou et al. 2015; Vega et al.

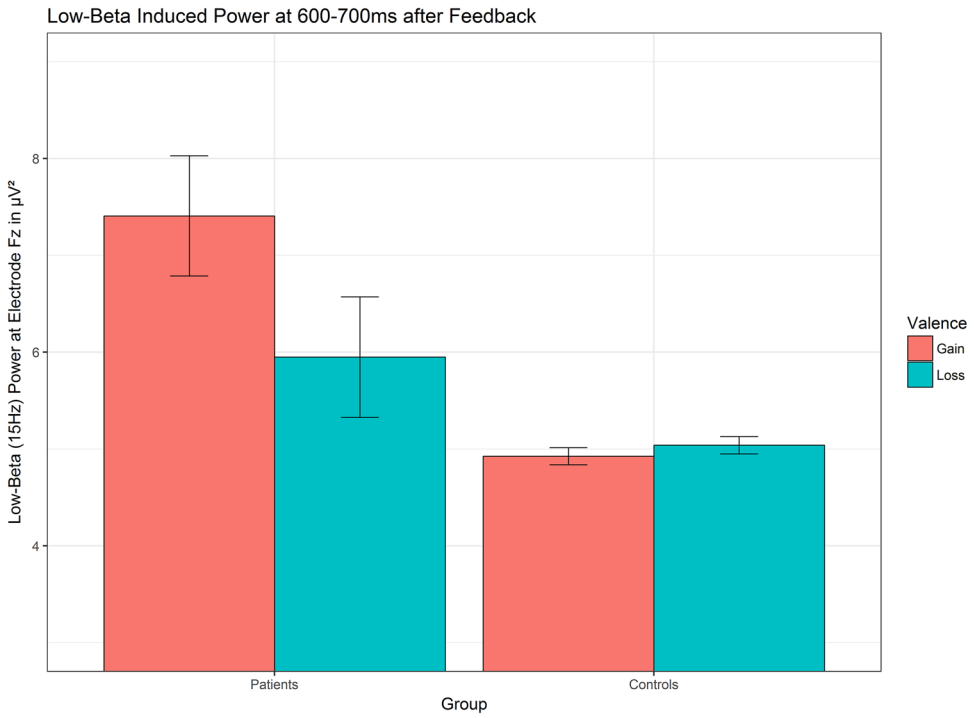

Fig. 3 Low-Beta (15 Hz) power at electrode Fz. Errorbars represent the standard error of the mean for within-subject factors, which were calculated by the method of Morey (2008)

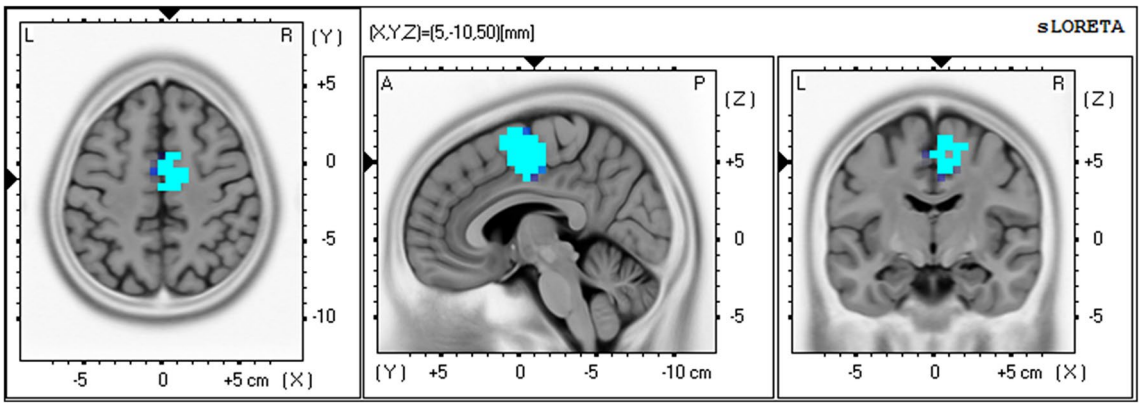

Fig. 4 Graphic depiction of cluster activation in medial frontal cortex as identified by sLORETA analysis

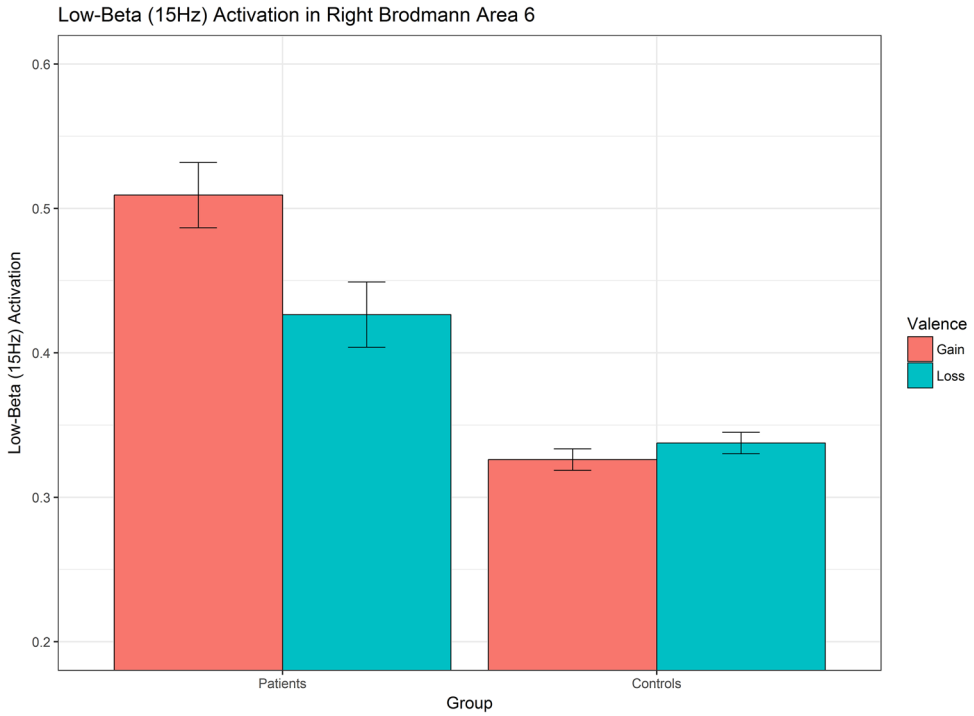

Fig. 5 Low-Beta (15 Hz) activation in a cluster consisting of parts of Brodmann areas 6, 24 and 31. Errorbars represent the standard error of the mean for within-subject factors, which were calculated by the method of Morey (2008)

2013) we were able to show additional altered responses in the low-beta range. Midbrain dopaminergic signaling also includes lower-frequency and higher-frequency modes, which have been assumed to reflect tonic and phasic dopamine responses, respectively (Volkow et al. 2011). Thus, our findings might be related to complex dopaminergic abnormalities. Therefore, our results are in line with theories emphasizing the role of the reward system in explaining the symptoms of patients suffering from BPD (e.g. Bandelow et al. 2010). Regarding the specific role of low-beta oscillations in reward processing a recent study by Yaple et al. (2018) in healthy participants showed an increase in low-beta power during omission of rewards. The authors speculate that the low-beta response might reflect a learning mechanism using the feedback information. Interestingly, a study by Paret et al. (2016) was able to show that individual symptoms observed in patients with BPD, namely emotional arousal, dissociation and aversive tension, were associated with impaired reward learning in patients suffering from BPD. They also reported an influence of symptom severity (assessed via the BSL-23) on reward learning in their study.

Another psychiatric disorder known for displaying a disturbed theta/beta ratio is the attention deficit hyperactivity disorder (ADHD; e.g. Heinrich et al. 2014). In ADHD the information on this specific deficit is even used to administer neurofeedback training (Bluschke et al. 2016). A uniting factor between BPD and ADHD could lie in attention deficits and associated mechanisms. But whilst ADHD is associated

with reduced low-beta responses, the opposite seems to be true for patients suffering from BPD. This is quite surprising considering the similarity of some of the symptoms of these two disorders (Philipsen et al. 2009). A possible explanation might be that in ADHD reduced attention could lead to reduced processing of reward and other stimuli, which might in turn result in a reduced low-beta response. In BPD the opposite might be true, with patients showing a stronger response to reward due to a higher tension and higher emotional arousal leading to attention deficits.

In the light of the findings of Paret et al. (2016) we conducted an exploratory analysis on the individual items of the BSL-23 (Bohus et al. 2009). In our exploratory analysis two items representing learned helplessness (item 2) and dissociative symptoms (item 3) showed moderate to strong correlations with the low-beta valence difference score. Even though these results are exploratory, this might be an indication that dissociative symptoms, which have also been shown to be correlated to aversive tension by Paret et al. (2016), might indeed be linked to reward processing in patients with BPD and more specifically the altered low-beta response we observed. Further support for the association of low-beta oscillations and attention symptoms might come from a study by Lee et al. (2017), who were able to show that resting state low-beta power was associated with inattention symptoms after childhood-trauma. This is particularly interesting, considering that BPD patients are known to suffer from high trauma exposure during childhood (e.g. Ball and Links 2009; Herman et al. 1989) and associated dissociative symptoms (e.g. Herman et al. 1989; Stiglmayr et al. 2001) resulting in inattention. A cue as to the association with helplessness might be provided by studies showing that lower levels of resting state beta activity are associated with greater motor-action preparation and approach tendencies (e.g. Threadgill and Gable 2018). This could be a possible explanation why BPD patients, who showed increased low-beta responses to feedback in our study, might suffer from increased feelings of helplessness. However, our interpretations are still speculative; future studies are needed to replicate our finding of increased low-beta power following gain trials in comparison to loss trials in BPD patients and its associations with specific symptoms.

An interesting discrepancy to the previous study by Leicht et al. (2013), which used a similar paradigm as the present study, is the lack of a significant increase in alpha and low-beta power following loss feedback in healthy participants. A possible explanation for this might lie in sample differences between the two studies. One important factor is the ratio between female and male participants, with Leicht et al. (2013) having a higher percentage of male participants. This is particularly important considering the known differences between men and women in sensation seeking (e.g. Cross et al. 2013), a trait which in turn has been associated with both the alpha and low-beta power in feedback processing by Leicht et al. (2013).

It should be stressed that inherent in the use of EEG is a trade-off between a good temporal resolution on the one hand and limitations in its spatial resolution especially concerning deeper brain regions such as the ventral striatum or the midbrain. Both of these structures are often reported as core areas of the reward network. A goal for future research could thus be to use fMRI or even a combination of EEG and fMRI to be able to localise the differences between BPD patients and controls with more precision.

In conclusion, our findings suggest that patients with BPD may exhibit more extensive impairments in the processing of feedback than previously expected, especially in regard to the processing of positive feedback. In addition, these impairments seem to be linked to symptom severity in patients with BPD.

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