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Application of attentional bias modification training to modulate hyperactive error-monitoring in OCD☆

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ABSTRACT

Because obsessive-compulsive disorder (OCD) is a highly impairing and often chronic psychiatric disorder, there is high interest in novel add-on or alternative intervention approaches. The error-related negativity (ERN), a response-related ERP occurring shortly after incorrect responses, might provide a promising target for novel interventions. Increases in the ERN have been reliably shown in OCD and are viewed as an important biomarker for the disorder. The ERN has been functionally associated with a heightened response to errors as negative, potentially harmful events. Attention Bias Modification (ABM) may be one promising strategy to reduce the ERN. Thus, in the current study, we used ABM training with the aim to reduce the threat value of errors and thus the ERN in OCD. Participants with OCD (n = 23) and healthy participants (n = 24) performed a 20-minute probe detection task in a condition that trained to attend to neutral and away from negative stimuli, while another group of healthy participants (n = 24) performed a sham version of the training. Results indicated a significant reduction of initially increased ERN amplitudes in the OCD group after the training, whereas in both healthy subgroups no significant changes in ERN were observed, resulting in non-significant group differences after ABM. These results indicate that ABM training may be a viable intervention to reduce ERN in participants with increased error-signaling, as in OCD. The current study serves as a clinical pilot investigation for future studies needed to elucidate underlying mechanisms, clinical effects and long-term outcome.

Obsessive-compulsive disorder is a highly debilitating psychiatric disorder that often follows a chronic course. Individuals with obsessive-compulsive disorder (OCD) are mostly treated with pharmacotherapy or cognitive-behavioral therapy (CBT), or a combination of both (Foa et al., 2005; Koran et al., 2007). These treatments are effective in reducing OCD symptoms for most, but not all, patients: of those receiving CBT with exposure and response prevention, 20% of patients drop out prematurely and another 20% do not adequately respond (Foa, 2010). Thus, the development of additional innovative treatments as supplementary or even alternative interventions is of crucial importance. Biological markers, such as event-related potentials (ERPs) associated with a disorder could function as targets of such novel intervention approaches (Hajcak et al., 2019).

One biomarker of interest in this respect is the error-related negativity (ERN; Gehring et al., 1993), a response-related ERP occurring shortly after incorrect responses. The ERN is often contrasted with a similar, but smaller component on correct trials, called correct-response negativity (Ford, 1999). Functionally, the ERN has been interpreted as a neurocognitive alarm signal in the case of errors that is sensitive to the motivational salience of errors (Falkenstein et al., 2000; Klawohn et al., 2014). Amplitude of the ERN is modulated by context and interindividual difference characteristics. It has been shown to be increased in situations when response correctness is evaluated or reinforced (Endrass et al., 2010; Grützmann et al., 2014; Hajcak et al., 2005; Riesel et al., 2012) and among individuals with high apprehensive traits characterized by motivation to avoid errors (Proudfit et al., 2013; Weinberg et al., 2016) or increased error-sensitivity (Chong and Meyer, 2019). In line with this, increased magnitude of the ERN has

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been linked to OCD (Riesel, 2019) as well as several anxiety disorders (Cavanagh and Shackman, 2014; Weinberg et al., 2015) both in adults and children (Meyer, 2017) with high robustness (Klawohn et al., 2020). Moreover, increased amplitudes of the ERN seem to indicate higher risk for anxiety disorders and OCD, as indicated by both family studies (Olvet and Hajcak, 2008; Riesel et al., 2011; Riesel, Klawohn, Grutzmann, Kaufmann, Heinzel, Bey et al., 2019) and prospective investigations (Meyer et al., 2015; Meyer et al., 2018). Importantly, it is possible that reduction in the ERN could lead to reduction in risk for the disorder as well (Kendler and Neale, 2010) – a notion that is consistent with recent findings suggesting elevated ERN as a mediator of anxiety symptoms (Chong and Meyer, 2019). Therefore, the ERN was proposed as a target biomarker for novel interventions (Hajcak et al., 2019; Proudfit et al., 2013), since reduction in ERN amplitudes might ultimately result in reduced symptoms of or risk for OCD and anxiety.

Importantly, treatment studies have demonstrated elevated ERN amplitudes after conventional cognitive-behavioral therapy, despite successful reduction of OCD or anxiety symptoms (Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). This may indicate that relevant aspects of dysfunctional performance monitoring in these patients are not targeted by psychotherapy in its current design, like increased worries about performance (Ladouceur et al., 2018) or increased sensitivity for errors. In the context of findings suggesting that the ERN is a prospective indicator of risk for anxiety disorders and OCD, this could indicate that leaving increased ERN amplitudes unaltered by intervention might yet be associated with a subsequently heightened risk for these disorders, and thus for relapse. This possibility further emphasizes the need to develop and investigate interventions directly targeted at decreasing the ERN as biomarker of overactive error monitoring in OCD and anxiety (Hajcak et al., 2019). Recently, a brief computerized intervention that psychologically targets error sensitivity proved effective in reducing ERN amplitude, especially in those with higher initial ERN (Meyer et al., 2019). With respect to clinical populations, heightened ERN amplitude has been shown to be susceptible to short-term modulation in patients with OCD under conditions of limited cognitive resources and attentional reallocation (Klawohn et al., 2016). Taken together, these studies represent starting points in the search for novel interventions to induce a sustainable reduction error-monitoring in clinical samples.

Attentional bias modification (ABM) training might represent one promising approach for interventions to modulate both ERN and symptoms of OCD or anxiety. These trainings aim at reducing negative attentional bias, which is the tendency of individuals to selectively allocate attention to negative, threat-related information (MacLeod et al., 1986). Negative attentional biases have been suggested to underlie a range of internalizing disorders such as depressive disorders, various anxiety disorders, and also OCD (Bar-Haim et al., 2007; Cisler and Koster, 2010; Gotlib et al., 2004; Hezel and McNally, 2016). Based on dot-probe paradigms used to assess these biases, ABM trainings have been developed (Amir et al., 2009; MacLeod et al., 2002). ABM trainings generally make use of a contingency between one stimulus type/valence category and the probe, e.g. the probe might appear more often in the previous location of a positive/neutral than a negative stimulus.

Several reports on symptom reduction in anxiety disorders suggest clinical efficacy of attentional bias modification interventions (Linetzky et al., 2015; MacLeod and Mathews, 2012; Mogoase et al., 2014; Price et al., 2016). For instance, using a dot-probe based multi-session ABM intervention Schmidt et al. (2009) demonstrated a reduction in social anxiety symptoms comparable to traditional cognitive-behavioral therapy. Importantly, Amir et al. (2015) were able to show that a spatial cueing form of ABM was able to significantly enhance an exposure and response prevention treatment in participants with clinical OCD. Moreover, evidence for ecologically valid transfer effects of ABM interventions were shown both with respect to socially anxious participants (Amir et al., 2008), as well as in participants with contamination fears (Najmi and Amir, 2010). However, inconsistencies and non-replications of findings regarding symptom reductions through ABM interventions have given rise to concerns about the validity and measurement quality of reaction time based measures of attentional bias (Boettcher et al., 2013; Carlbring et al., 2012; Heeren et al., 2015a). Specifically, insufficient psychometric properties, including poor reliability and internal consistency have been pointed out (Kappenman et al., 2014; Rodebaugh et al., 2016; Schmukle, 2005; Waechter et al., 2014) and validity has been questioned (Thigpen et al., 2018). In line with this, recent accounts of the literature draw a more balanced picture, as they represent the inconsistencies and describe effect sizes of attentional bias modification as rather small, yet significant (e.g., Mogoaşe et al., 2014). It has been suggested that further research is needed to advance understanding of mechanisms and that new research approaches should be developed, including the use of new paradigms and measures (Clarke et al., 2014; Mogg and Bradley, 2018).

Effects of ABM training on ERP measures associated with anxiety/OCD have not been studied extensively yet. To date, two studies investigated the effects of ABM training on error monitoring in healthy participants and showed significantly reduced ERN amplitudes after single-session application of ABM training (Nelson et al., 2015; Nelson et al., 2017). Existing EEG studies so far have been limited to healthy samples, and further research is needed to elucidate the effects of ABM on overactive performance monitoring in clinical groups. With the current study we aimed to examine the impact of ABM on the ERN in both healthy individuals and patients with a diagnosis of OCD. Using a within-subject design, we implemented an active dot-probe based ABM training (i.e., trained attention away from negative stimuli) in a group of participants with a current diagnosis of obsessive-compulsive disorder, as well as a healthy participant group without current psychiatric diagnosis and no history of OCD. Additionally, we implemented a sham training condition in another group of healthy participants to assess potential unspecific training effects. In a within-subject design, ERN and CRN were assessed before and after a single-session (ca. 20 min) ABM training. We expected to observe heightened error-related ERPs in the OCD relative to the two HC groups before the intervention. Further, we hypothesized that decreases in ERN relative to CRN amplitudes would occur in both active training groups, but not the sham training group. Finally, we also aimed to test whether heightened negative attentional bias would be measurable in the OCD group and whether decreases in negative attentional bias would be present in both active training, but not the sham training groups.

1. Methods

1.1. Participants

The full study sample consisted of 32 individuals with OCD and 55 healthy comparison participants. Data were excluded from analyses either due to insufficient error numbers to compute the ERN (i.e., < 5) in at least one of the two flanker task assessments (OCD: $n = 8$, HC: $n = 7$), or due to insufficient EEG data quality (OCD: $n = 1$). The final samples then comprised 23 individuals with OCD, and 48 participants without current psychiatric disorder who were randomized to an active training group ($n = 24$) or to a sham training condition ($n = 24$). All participants received verbal and written explanation of the aims and procedures of the study and gave written informed consent before participation. The study was conducted in accordance with ethical standards, as approved by the local institutional review board at Humboldt-Universität zu Berlin (HU). Participants with a diagnosis of OCD were recruited from the OCD outpatient clinic at HU. Healthy comparison participants were recruited from the general population via public advertisements. All participants were reimbursed for their participation with 10 € per hour. The following exclusionary criteria applied to participants of the OCD group: lifetime diagnosis of psychotic, bipolar, or substance use disorder; neuroleptic medication in the past four weeks, or benzodiazepine medication in the past two weeks.

Additional exclusionary criteria were applied for healthy comparison participants: any psychoactive medication in the past three months, any current psychiatric disorder, or a lifetime diagnosis of OCD. All participants were required to have normal or corrected-to-normal vision, no history of head trauma or neurological condition, and to be aged between 18 and 65 years. Participants of all three experimental groups were parallelized for age, gender, and level of education.

1.2. Measures

All participants were assessed with regard to current or past psychopathology by trained masters- and PhD-level clinical psychologists using the German version of the Structured Clinical Interview (SCID) for DSM-IV (First et al., 1996; Wittchen et al., 1997). In participants diagnosed with OCD, severity of obsessive–compulsive symptoms was further assessed using the Yale–Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989). The German version (Hand and Büttner-Westphal, 1991) has been shown to have high interrater-reliability and internal consistency (Jacobsen et al., 2003). Total scores are derived from 10 items and range from 10 to 40. Further, obsessive-compulsive symptoms were assessed in all participants with the German version of the Obsessive-Compulsive Inventory-Revised (OCI-R, Foa et al., 2002; Gonner et al., 2008). The OCI-R is a self-report measure with excellent psychometric properties and can be used as a measure of symptom severity (Foa et al., 2002; Gonner et al., 2008). Additionally, participants of both groups rated depressive symptoms with the Beck Depression Inventory–II (BDI-II; Beck et al., 1996). The German version (Hautzinger et al., 2006) has been shown to have good psychometric properties (Kuehner et al., 2006).

1.3. Task & experimental procedures

All participants performed an arrow-version of a flanker task, presented using Presentation (Neurobehavioral Systems, Albany, California, USA). On each trial, five vertically aligned arrows were presented and participants were asked to respond with their right or left index finger using response buttons in accordance with the direction of the central arrow. Half of the stimuli were compatible (i.e., all arrows pointing in the same direction), the other half were incompatible (i.e., flanking stimuli pointing in the opposite direction than the center arrow), presented in pseudo-randomized order. The stimuli were presented for 100 ms, followed by a 1000 ms response window and an intertrial interval that varied randomly between 200 and 1200 ms during which a fixation cross was presented. The task encompassed 480 trials, administered in 6 blocks with short breaks in between.

After the flanker task, all participants performed a dot probe task in three experimental blocks (pre bias assessment, ABM training, post bias assessment). In all blocks, each trial began with the presentation of a fixation cross for 200 to 800 ms (mean = 500 ms). Then, a vertically aligned word pair of one negative and one neutral word was displayed for 500 ms. The word pair was immediately followed by the target stimulus (the letter 'E' or 'F') at the former location of either the neutral or the negative word, presented until participants responded or for up to 1500 ms. Participants were instructed to respond by pressing a response button with their left index finger if the letter was F, or their right index finger if it was an E. The combinations of location of the negative word (above or below), target stimulus type (E or F), and sequence (target replacing negative or neutral word) were balanced and pseudo-randomized across trials. All words used for the paradigm were selected from a normed sample (Berlin Affective Word List, BAWL; Võ et al., 2009) and matched for number of syllables. There was an initial block of 64 trials for a bias assessment before the ABM training and an analogous post-training block; both used one of two matched word lists of 8 pairs in counterbalanced order. After the initial bias assessment, participants completed the ABM training part of the paradigm, which consisted of 400 trials divided into 4 blocks and had an approximate total duration of 20 min. All participants of the OCD group performed an active ABM training, whereas healthy participant were assigned to either the active ABM (HC-train) or a sham ABM training (HC-sham) according to a pre-defined randomization that both the participant and the experimenter were blind to. For the ABM training, another set of 25 word pairs was used. In the active training condition, target letters always followed in the location of neutral words. The sham training was identical to the initial bias measurement in terms of stimulus presentation; that is, targets appeared at the location of neutral or negative word with equal probability. After the ABM training, the flanker task was administered again to re-assess the ERN after the ABM training. During debriefing, participants were asked whether they noticed any contingency in the presentation of word pairs and target stimuli. One participant reported the correct contingency after the experiment, all other participants either were not aware of any contingency ($n = 50$) or assumed an incorrect pattern ($n = 19$).

1.4. Psychophysiological recordings

Continuous EEG was recorded from 64 Ag/AgCl-sintered electrodes, positioned on the scalp using an electrode cap with equidistant electrode locations (EASYCAP GmbH, Herrsching, Germany), with Cz serving as recording reference. External electrodes were placed below the left and right eye, on the nasion, and below T1 (ground). All impedances were kept below 5 kΩ. The EEG was digitized at a sampling rate of 1000 Hz and amplified with BrainAmp amplifiers (Brain Products, Gilching, Germany), using a band pass filter of 0.01–100 Hz. All offline analyses were performed using BrainVision Analyzer software (version 2.1.2, Brain Products, Gilching, Germany). Off-line, data were filtered with a bandpass from 0.1 to 30 Hz (4th order Butterworth) and then re-referenced to averaged mastoids. Response-locked epochs with a duration of 1500 ms including a 500 ms pre-response interval were extracted. Eye movement artifacts were corrected using the Gratton et al. (1983) procedure as implemented in Brain Vision Analyzer. Then, segments with artifacts were excluded from further analyses on a channel-specific basis using automatic artifact rejection using the following criteria: a voltage step of > 50 μV between consecutive sample points, absolute voltage difference > 200 μV within the epoch, or low activity < 0.5 μV over 100 ms. Further semiautomatic artifact rejection was applied based on visual inspection. Response-locked averaged were computed and baseline-corrected using the -500 to -300 ms pre-response interval as baseline. ERN and CRN amplitudes were quantified at electrode FCz as the mean amplitude from 0 to 100 ms after response onset, ΔERN was determined as the difference ERN minus CRN.

1.5. Statistical analyses

All statistical analyses were conducted using SPSS Statistics version 25 software, using an α-level of 0.05.

1.5.1. Behavioral data

To test whether group differences or intervention effects were present in behavioral data, accuracy rates and response times in the flanker task were analyzed with mixed-effects analysis of variance (ANOVA) including the within-subjects factor intervention (pre, post) and the between subjects factor group (OCD-train, HC-train, HC-sham).

1.5.2. Attentional bias data

For the analysis of attentional bias in the dot-probe paradigm, only trials with correct responses to the probes were included. Attentional bias was quantified as the difference in median response times for correct identification of targets following a neutral versus a negative word, with negative bias values indicating an attentional bias towards negative stimuli. Group differences in negative attentional bias before and after the ABM training were analyzed with a mixed-effects ANOVA

including the within-subjects factor intervention (pre, post) and the between-subjects factor group (OCD-train, HC-train, HC-sham).

1.5.3. Event-related potentials

Response-related ERPs (i.e., ERN and CRN amplitudes) were first tested for pre-intervention group differences in ERN, CRN, and ΔERN amplitudes using univariate ANOVAs comparing the OCD and both HC groups and according *t*-tests. Further, to investigate effects of the ABM intervention with respect to groups, response-related ERPs were submitted to a mixed-effects ANOVA including the within-subject factors intervention (pre, post) and response type (error, correct) and the between-subjects factor group (OCD-train, HC-train, HC-sham). All significant interactions or main-effects were followed-up with Bonferroni-corrected comparisons as implemented in SPSS and corrected *p*-values were reported for all post-hoc tests. To obtain the corrected p-value, the p-value is multiplied by the number of comparisons that are needed to follow-up a given effect. Finally, an analogous ANCOVA additionally including the pre-and post-ABM response error rates was conducted as control analysis.

2. Results

2.1. Sample characteristics

Demographic and clinical characteristics of participant groups are presented in Table 1. In the OCD group, 11 participants (47.8%) were currently taking psychotropic medication (serotonin reuptake inhibitors: $n = 10$, tricyclic antidepressants: $n = 1$). The majority of participants in the OCD group ($n = 13$; 56.5%) had one to two current comorbid diagnoses, such as major depressive disorder ($n = 6$ current episode), dysthymia ($n = 2$), social phobia ($n = 4$), panic disorder & agoraphobia ($n = 2$), generalized anxiety disorder ($n = 1$), specific phobia ($n = 1$), and somatization disorder ($n = 1$).

2.2. Behavioral results

2.2.1. Reaction times

Reaction times in the flanker tasks before and after ABM are presented in Table 2. The mixed-effects ANOVA regarding reaction times indicated a significant effect of response type, $F(1, 68) = 196.55$, $p < .001$, $\eta_p^2 = 0.74$, with shorter reaction times for errors compared to correct responses. Further, a significant interaction of intervention and response type, $F(1, 68) = 42.55$, $p < .001$, $\eta_p^2 = 0.39$, emerged, while no other main effects or interactions were significant (all $p > .132$). Follow-up tests showed that across groups the reaction time difference between correct and incorrect responses was higher before than after ABM (mean difference 20.7 ms, $SE = 3.23$, $p < .001$).

2.2.2. Error rates

Mean error rates in the flanker task before and after ABM in all groups are presented in Table 2. The ANOVA on error rates yielded a significant main effect of intervention, $F(1, 68) = 8.09$, $p = .006$, $\eta_P^2 = 0.11$, as well as an interaction of intervention and group, $F(2, 68) = 5.64$, $p = .005$, $\eta_P^2 < 0.14$, while the main effect of group did not reach significance, $F(2, 68) = 2.44$, $p = .10$, $\eta_P^2 = 0.07$. As follow-up tests indicated, before the ABM intervention error rates were higher in the HC-sham group than in the HC-train group (mean difference = 2.53%, $SE = 0.87$, $p = .015$) and marginally higher than in the OCD group (mean difference = 2.14%, $SE = 0.88$, $p = .052$). Further, an increase in error rates was present in both the HC-train group (mean difference: 1.23%, $SE = 0.38$, $p = .002$) and the OCD-train group (mean difference = 1.04%, $SE = 0.39$, $p = .008$), but not in the HC sham group (mean difference = 0.41%, $SE = 0.0.38$, $p = .284$).

Table 1
Demographical and clinical characterization of the groups of participants with OCD and healthy participants in the active ABM group and the sham training group.

	OCD	HC-train	HC-sham
Sample/*n* female	$n = 23/13$	$n = 24/13$	$n = 24/12$
Age (years)	30.8 (7.8)	29.1 (6.9)	30.8 (8.6)
Y-BOCS	23.13 (4.99)	–	–
OCI-R	27.68 (12.50)[a,b]	7.67 (6.64)[a]	6.08 (4.28)[b]
BDI-II	15.63 (9.56)[a,b]	4.46 (5.21)[a]	5.75 (7.63)[b]

Note. Means are presented, standard deviations in parentheses. BDI-II = Beck Depression Inventory II, RT = reaction time. Shared superscripts indicate a significant group difference in between-group *t*-tests, $p < .05$.

2.2.3. Bias scores

Analysis of bias scores before and after the ABM intervention (presented in Table 2) indicated a significant main effect of group, $F(2, 68) = 3.86$, $p = .026$, $\eta_P^2 = 0.10$, whereas the main effect of intervention, $F(1, 68) = 3.70$, $p = .059$, $\eta_P^2 = 0.05$, and the interaction of group and intervention, $F(2, 68) = 0.09$, $p = .912$, $\eta_P^2 < 0.01$, did not reach significance. As follow-up tests indicated, bias scores in the HC-sham group were generally higher than in the HC-train group (mean difference = 14.68, SE = 5.32, $p = .022$), while none of the other group-comparisons indicated significant differences (both $p > .302$).

2.3. ERP results

Fig. 1 displays grand average waveforms for correct and incorrect responses as well as for the difference waves (error minus correct) for participants in the OCD and both HC groups before and after the ABM intervention. Mean scores for ERN, CRN, and ΔERN (i.e., ERN minus CRN) before and after ABM are presented in Table 1 for all groups.

2.3.1. Pre-intervention group differences

Univariate ANOVAs on the pre-intervention ERPs showed significant initial group differences in ΔERN before the intervention, $F(2, 68) = 3.81$, $p = .027$, $\eta_p^2 = 0.10$, stemming from significant differences in ERN amplitudes before ABM, $F(2, 68) = 5.24$, $p = .008$, $\eta_p^2 = 0.13$, whereas no initial group differences were present in CRN amplitudes, $F(2, 68) = 0.38$, $p = .688$. Follow-up tests showed that ERN amplitudes were significantly larger (i.e., more negative) in the OCD group as compared to both the HC-train group (mean difference = 4.73 μV, $SE = 2.04$, $p = .025$, $d = 0.68$) and the HC-sham group (mean difference = 5.86 μV, $SE = 1.86$, $p = .003$, $d = 0.92$).

2.3.2. Effects of the ABM intervention

Results of the mixed-effects ANOVA on ERN/CRN amplitudes before and after the ABM intervention indicated a significant main effect of response type, $F(1, 68) = 190.09$, $p < .001$, $\eta_p^2 = 0.74$, but no significant main effects of intervention, $F(1, 68) = 1.73$, $p = .193$, $\eta_p^2 = 0.03$, or group, $F(2, 68) = 1.60$, $p = .209$, $\eta_p^2 = 0.05$. The main effect of intervention was further qualified by significant interactions of intervention and group, $F(2, 68) = 4.52$, $p = .014$, $\eta_p^2 = 0.12$, as well as the three-way interaction of intervention, response type, and group, $F(2, 68) = 3.31$, $p = .042$, $\eta_p^2 = 0.09$. The interactions between response type and group, $F(2, 68) = 1.91$, $p = .172$, $\eta_p^2 = 0.03$, and intervention and response type, $F(1, 68) = 2.05$, $p = .137$, $\eta_p^2 = 0.06$, were not significant. In line with the three-way interaction, post-hoc comparisons indicated a group-specific reduction in ΔERN from pre- to post-ABM, which was significant for the OCD group (mean difference = −3.16 μV, $SE = 1.11$, $p = .006$, $d = 0.67$), but not in either healthy group (HC-train: mean difference = 0.54 μV, $SE = 1.08$, $p = .620$, $d = 0.09$; HC-sham: mean difference = 0.01 μV, $SE = 1.08$, $p = .994$, $d < 0.01$; see Fig. 2). As further post-hoc comparisons showed, these group-specific changes in error minus correct differentiation were due to a significant reduction in ERN amplitudes in the OCD group (mean difference = −3.63 μV, $SE = 1.11$, $p = .002$,

Table 2
Behavioral and ERP measures from flanker task before and after attentional bias modification intervention in the groups of participants with OCD and healthy participants in the active ABM group and the sham training group.

	Pre-ABM			Post-ABM		
	OCD	HC-train	HC-sham	OCD	HC-train	HC-sham
Behavioral measures						
Correct RT (ms)	426.56 (48.23)	425.90 (35.16)	429.92 (55.09)	420.02 (55.97)	411.51 (31.35)	422.2 (53.4)
Error RT (ms)	376.79 (49.00)	363.70 (56.81)	373.09 (51.09)	394.05 (66.80)	375.99 (52.78)	377.2 (50.9)
Error rate (%)	3.82 (2.93)[a]	3.43 (2.18)[b]	5.96 (3.72)[a,b]	4.86 (3.08)	4.66 (2.88)	5.55 (3.05)
Bias score (ms)[1]	8.80 (23.67)	−1.00 (27.56)	16.17 (36.23)	0.02 (23.58)	−8.06 (26.45)	4.13 (24.96)
ERP measures						
ERN at FCz (μV)	−6.93 (7.08)[a,b]	−2.19 (6.93)[a]	−1.06 (5.61)[b]	−3.29 (5.92)	−2.44 (5.79)	−1.67 (6.57)
CRN at FCz (μV)	5.11 (4.35)	5.85 (4.26)	6.32 (5.66)	5.59 (4.14)	6.14 (4.28)	5.72 (5.08)
ΔERN at FCz (μV)	−12.04 (7.10)[a,b]	−8.05 (6.18)[a]	−7.38 (5.32)[b]	−8.88 (5.78)	−8.59 (5.64)	−7.39 (5.57)

Note. Means are presented, standard deviations in parentheses.
[1] Bias score indicates the difference in median reaction time to probe following negative words minus following neutral words; more negative values indicate higher attentional bias towards negative. Columns with shared superscripts indicate a significant group difference in independent samples t-tests, $p < .05$.

$d = 0.70$), but not in the HC-train group (mean difference = 0.25 μV, $SE = 1.25$, $p = .821$, $d = 0.04$) or the HC-sham group (mean difference = 0.60 μV, $SE = 0.93$, $p = .580$, $d = 0.13$); there were no significant changes in CRN amplitudes comparing before and after the ABM training in any group (all $p > .224$). In line with this group-specific ERN reduction in OCD, initial group differences in ERN amplitudes were no longer present after ABM intervention, $F(2, 68) = 0.38$, $p = .688$, $\eta_p^2 = 0.01$, such that there were no significant

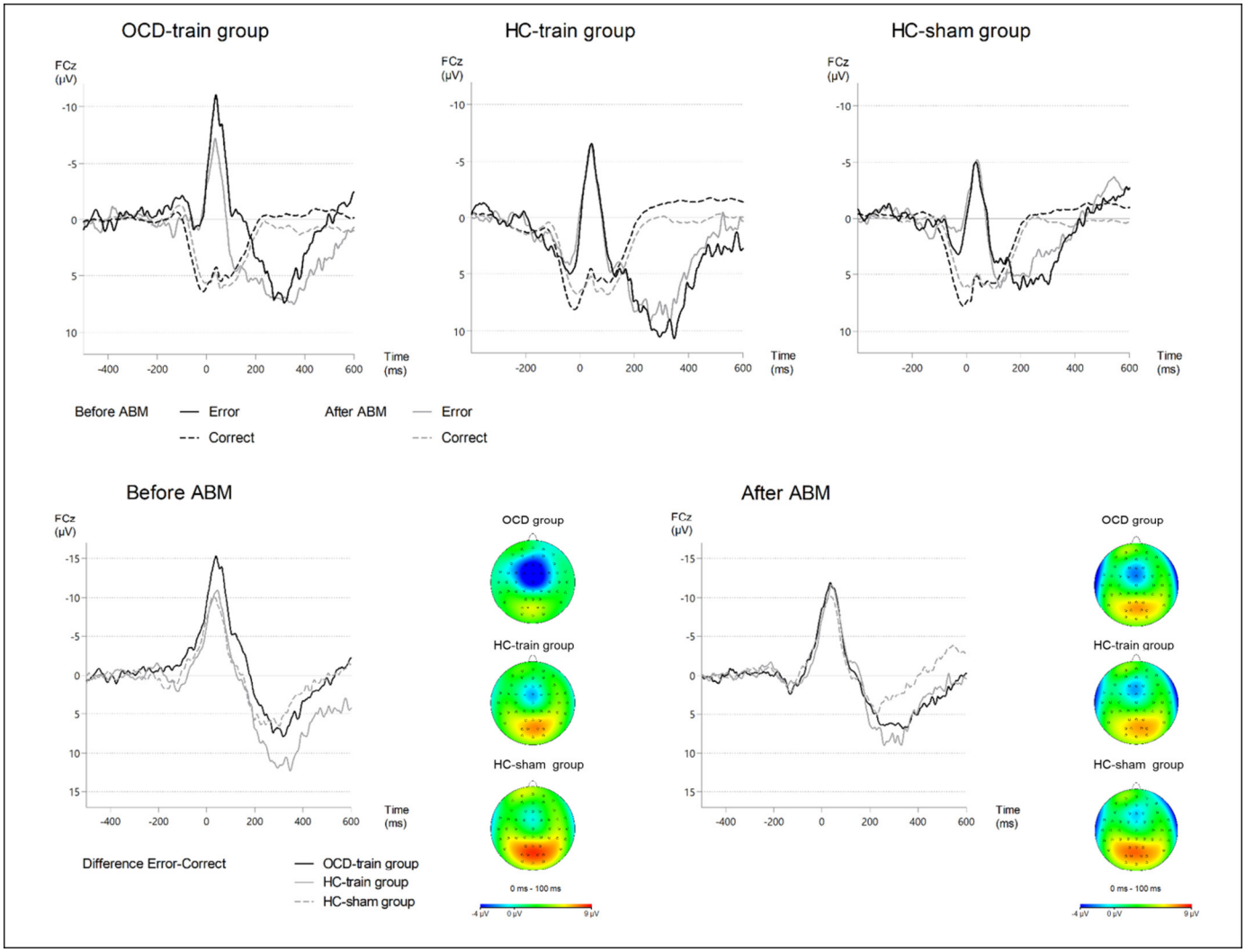

Fig. 1. Grand average waveforms for correct and error response trials relative to response onset at electrode FCz (upper panel); grand average waveforms for difference waves (error minus correct) relative to response onset at electrode FCz and headmaps of scalp distribution for error trials during the interval from 0 to 100 ms after response onset (lower panel).

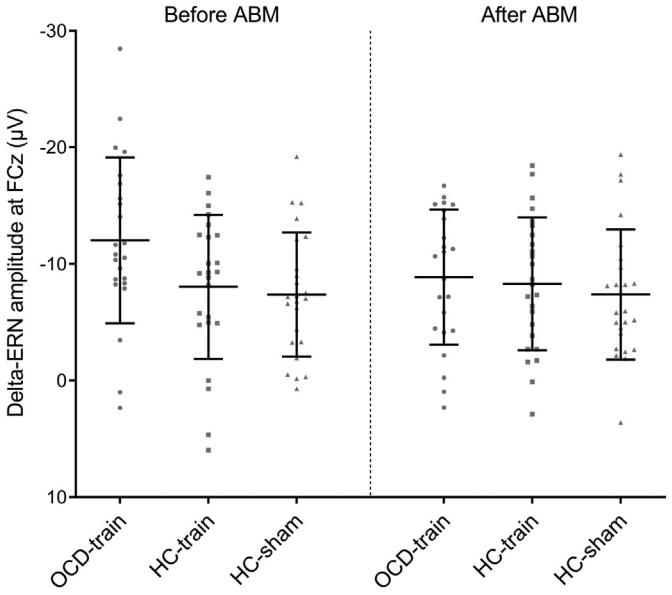

Fig. 2. Scatter plot of Delta-ERN values before and after ABM training in the three experimental groups; horizontal lines represent group means, error bars represent SD.

differences between the OCD group compared to the HC-train group (mean difference = 0.85 μV, SE = 1.71, p = .559, d = 0.17) or to the HC-sham group (mean difference = 1.63 μV, SE = 1.83, p = .418, d = 0.24).

2.3.3. Control analyses

Since behavioral results indicated group differences in error rates that were moderated by an intervention effect, a mixed effects ANCOVA on ERN/CRN amplitudes was conducted using error rates in the flanker task pre- and post-ABM as covariates, respectively. Results remained, with significant effects of response type, $F(1, 66) = 37.48, p < .001$, as well as significant interactions between intervention and group, $F(2, 66) = 4.86, p = .011$, and of intervention, response type and group, $F(2, 66) = 3.61, p = .033$. Neither the error rates before or after ABM emerged as significant covariates in the model (both $p > .179$).

3. Discussion

The current results suggest that a brief attentional bias modification training is effective in reducing hyperactive error-signaling in individuals with OCD: the ERN in participants with a current diagnosis of OCD was significantly reduced after completion of a single-session of dot-probe ABM training. Indeed, initially hyperactive error signals in OCD were no longer apparent following ABM. This ERN reduction after ABM training is generally in line with previous findings in healthy populations (Nelson et al., 2017; Nelson et al., 2015), that similarly showed significantly reduced ERN amplitudes after similar single-session interventions.

The current finding of an ERN attenuation in OCD after ABM intervention is a promising basis for future research, especially since previous treatment studies have failed to impact the increased ERN in anxiety and OCD (Hajcak et al., 2008; Riesel et al., 2015). The current results are also consistent with two previous study that found hyperactive error monitoring in OCD could be reduced by limiting cognitive resources (Klawohn et al., 2016) and, to a lesser extent, through instruction in favor of speed over accuracy in performance (Riesel et al., 2019a). The intervention with attentional bias modification trainings could represent a similarly effective means of modifying hyperactive error signaling, yet potentially with more sustainable effects on both neurocognitive functioning as well as symptoms. Accordingly, future studies are needed to investigate whether ERN reduction induced with ABM in clinical populations can be maintained, especially with longer/more extensive ABM-applications. Moreover, it will be important to examine whether ABM-based reductions in ERN impact symptoms or even course of illness.

ABM interventions are very economical in administration, they can be implemented with relatively few sessions (Najmi and Amir, 2010) even with remote, internet-based delivery (See et al., 2009). As the ERN has been shown to indicate risk for the development of OCD or anxiety disorders (Meyer, 2017; Riesel, Klawohn, et al., 2019), and even seems to function as a mediator for anxiety symptom development (Chong and Meyer, 2019), application of ABM trainings in high-risk populations (i.e., those with increased ERN) as a preventative measure targeting aberrant error monitoring might be a viable strategy. Therefore, the present results represent a basis for further larger-scale studies in individuals affected with or at-risk for anxiety disorders or OCD.

In the current study, we did not find a significant reduction in ERN amplitude within the HC group after the ABM training, and accordingly, no differences between the training and sham group with regards to ERN modulation. This is in contrast to previous investigations of ABM effects on the ERN (Nelson et al., 2015; Nelson et al., 2017), where ERN reduction was achieved in healthy individuals. It is worth noting that both previous studies on ABM-induced modulation of the ERN included larger sample sizes than the HC-ABM group of the current study. Potentially more importantly, both previous studies employed an adaptive version of the ABM training (Amir et al., 2016), which might be more effective than the classic dot-probe paradigm employed in the current study. This adaptive version of the ABM training also is based on a spatial cuing paradigm, showing only one instead of two competing targets at a time, as well as uses a more explicit instruction to try and achieve bias changes towards participants. Nonetheless, it is possible that it might be harder to leverage attention bias modification to alter ERN among healthy participants, where attentional mechanisms are normative (Bar-Haim et al., 2007; Clarke et al., 2014). Similarly, modulation of the error-related ERP component might be more effective at the higher end of the ERN distribution—indeed, this was also found by Meyer and colleagues, who tested the only psychological intervention directly targeting ERN reduction to date (Meyer et al., 2019).

A limitation of the current study is the lack of a clinical control-training group, as we submitted all participants of the OCD group to the active training condition. Although the implementation of a within-subjects pre-post-design supports the notion that the observed ERN reduction is induced by the training intervention, this conclusion can only be drawn with caution without a clinical sham training group. It will be especially important to further evaluate ABM specific versus non-specific intervention aspects, and their differential effects on the ERN. Thus, future studies should also incorporate a clinical control-training group, which would not only corroborate a causal link between the ABM intervention and the reduction in ERN, but would also help to further elucidate the mechanisms by which ERN reduction is attained. Another limitation is the use of a short 20-min ABM intervention to examine the impact of ABM on error processing. We conducted the study as an initial attempt to determine the susceptibility of overactive error monitoring in OCD to an ABM intervention. However, in order to achieve clinical change in participants with OCD, a more extensive training would most likely be necessary. Finally, one major limitation of studies applying ABM in clinical populations is the limited understanding of mechanisms underlying clinical change. Meta-analytic evidence suggests a significant small-to medium effect size of ABM interventions (Linetzky et al., 2015; Mogoase et al., 2014; Price et al., 2016). Yet the mechanisms through which this symptom reduction is achieved remain largely unknown or assumed mechanistic models lack reliable proof (Mogg and Bradley, 2018). For instance, most studies fail to provide evidence for reliable attentional bias reduction as the hypothesized mediator of change (Rodebaugh et al., 2016) and in a recent meta-analysis on clinical trials investigating attentional bias

modification, no evidence for initial dot-probe measured attentional bias in clinical anxiety overall could be found (Kruijt et al., 2019). Furthermore, ABM training interventions have often not been able to prove more successful at symptom reduction than sham- or control-training conditions (Heeren et al., 2016; Heeren et al., 2015b; Shechner et al., 2014). Possibly, non-specific aspects of the trainings that function even in absence of a specific emotional stimulus-probe contingency (e.g., repeated exposure to emotional stimuli, generally improved attentional control) as well as specific but thus far not well-assessed processes might play a role in the effectiveness for symptom reductions of these trainings. Similarly, in the current investigation, we did not find an aberrant pre-intervention attentional bias in the OCD group and no systematic changes in attentional bias were observed that could help to explain the training effect on the ERN. Thus, further investigation is needed to elucidate the mechanism through which ABM intervention might lead to a reduction of ERN. For instance, a reduction in error sensitivity, as a form of heightened responsivity to internally generated threat (Meyer et al., 2019), through exposure to threat stimuli would be a plausible mechanism. The use of neurophysiological markers, both as targets as well as process indicators, in future studies investigating ABM effectiveness might help gain a better understanding of the mechanisms involved (Arad et al., 2019; Wieser et al., 2016).

In conclusion, the current findings present novel and promising evidence for the malleability of overactive error monitoring in OCD through attentional bias modification training. Follow-up studies are needed to specify the magnitude and specificity of this effect. Furthermore, the duration of this ERN reduction, and its relationship to beneficial longer-term clinical effects need to be investigated in longitudinal designs. Nonetheless, the fact that it was possible to reduce hyperactive ERN in OCD with a single-session ABM intervention suggests promising avenues for potential applications, and represents a promising basis for further clinical investigation. Multi-session trainings that use adaptive paradigms (Amir et al., 2016) or eye-tracking based trainings (Lazarov et al., 2017) might represent promising avenues for modifying the ERN and should be studied further in clinical samples. As the present study shows, the ERN could provide a promising target for such novel intervention development efforts.

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