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Reduced P300 in depression: Evidence from a flanker task and impact on ERN, CRN, and Pe

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Abstract

Individuals with current depression show reduced amplitude of the P300 component of the stimulus-locked event-related potential (ERP)—an effect most often examined in oddball tasks. Although imperative stimuli in response-monitoring paradigms (e.g., the flanker task), also elicit a P300, it is unclear whether a blunted P300 can be observed in depression in these tasks. Moreover, the P300 overlaps with the correct-response negativity (CRN) and error-related negativity (ERN), and is similar to the error positivity (Pe)—response-locked ERPs frequently examined in flanker tasks. The current study examined the stimulus-locked P300 and response-monitoring ERPs on error (i.e., ERN, Pe) and correct responses (i.e., CRN) during an arrowhead flanker task in 72 individuals with a current depressive disorder and 42 never depressed healthy individuals. Consistent with findings from oddball tasks, P300 amplitude was reduced among participants with depression. Further, results indicated increased ERN and CRN, and decreased Pe, in depression. However, when the blunted P300 was included in analyses, group differences in response-monitoring ERPs were no longer evident. Accordingly, P300 amplitudes were correlated negatively with the ERN/CRN and positively with Pe in both groups. A blunted P300 in depression can be observed in speeded response tasks, and can produce apparent increases in ERN and CRN due to ERP component overlap. Further, reduced Pe in participants with depression may reflect a reduced P300 to error commission. These data highlight the central role of reduced P300 in clinical depression, and demonstrate that this effect can be observed across both stimulus- and response-locked ERPs in speeded response tasks.

KEYWORDS

CRN, depression, ERN, ERPs, flanker task, MDD, P300, Pe

1 | INTRODUCTION

Depressive disorders are among the most frequent and severe mental health problems worldwide (Kessler et al., 2003; Kessler & Bromet, 2013) and are associated with significant impairment and socioeconomic costs (Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015; Whiteford et al., 2013). Although depression is defined by loss of pleasure and changes in mood, it is also commonly associated with deficits in a range of executive functions, including memory and attention (Rock, Roiser, Riedel, & Blackwell, 2014). The neuropsychological dysfunctions have been shown to persist after remission of depressive symptoms, suggesting that cognitive impairment in depression may not be directly linked to low mood (Rock et al., 2014). Moreover, these cognitive impairments are associated with poorer treatment responses

(Roiser, Elliott, & Sahakian, 2012) and reduced psychosocial functioning (Rock et al., 2014). Consistent with these neuropsychological data, psychophysiological research on depression using event-related potentials (ERPs) has reliably found a decrease in the P300 component in individuals with current clinical depression when compared to healthy individuals (Bruder, Kayser, & Tenke, 2012). Functionally, the P300 has been associated with attentional allocation, evaluative processing, context updating, as well as inhibitory control (Polich, 2012, for review). Thus, the reduced P300 in depression is consistent with the reported attentional and cognitive difficulties that are part of the diagnostic criteria for the disorder (American Psychological Association, 2013).

Several studies have reported a reduction in P300 amplitudes to infrequent target stimuli in individuals with current depressive disorders (Bruder et al., 2009; Gangadhar, Ancy, Janakiramaiah, & Umapathy, 1993; Roschke & Wagner, 2003; Urretavizcaya et al., 2003). There is some indication that this P300 effect might be modulated by depression severity (Gangadhar et al., 1993; Nan et al., 2018), increased suicidality (Hansenne, Pitchot, Gonzalez Moreno, Zaldua, & Ansseau, 1996), or specific subtypes of depression, such as the melancholic subtype (Ancy, Gangadhar, & Janakiramaiah, 1996; Gangadhar et al., 1993; Urretavizcaya et al., 2003).

The studies described above on the P300 in depression most often employ variations of the oddball task as the experimental paradigm. In oddball tasks, participants are required to count or otherwise respond to one stimulus (i.e., the target), while ignoring another stimulus (i.e., the standard). The amplitude of the P300 in oddball tasks is highly sensitive to target frequency (Donchin, 1981). Thus, it is possible that the reduced P300 in depression during oddball tasks reflects deficits in working memory and the degree to which the target frequency is accurately monitored. Alternatively, the reduced P300 in depression might be indicative of broader stimulus-processing deficits that would be apparent in other speeded reaction time tasks as well.

Indeed, many other speeded response tasks require participants to make an imperative response *on each trial*. For instance, the Eriksen flanker task (Eriksen & Eriksen, 1974) is a speeded response paradigm that requires participants to respond on each trial to either congruent and incongruent stimuli, thus introducing different levels of response conflict. Stimulus-locked ERPs in this type of conflict tasks have typically been examined to understand individual differences in processing response conflict (e.g., the N200; Cavanagh, Meyer, & Hajcak, 2017; Riesel, Klawohn, Kathmann, & Endrass, 2017). However, imperative stimuli in this task also elicit a pronounced P300, although this has much less often been examined as an individual difference measure. One primary goal of the current study was to determine whether depression is associated with decreased P300 to imperative stimuli in a Flankers task—and if this deficit relates to more specific clinical characteristics within the group of participants with current depression.

In addition to examination of stimulus processing, variations of the flanker task have been used extensively to study ERPs related to response monitoring—such as the error-related negativity (ERN), the correct response negativity (CRN), and the error positivity (Pe). The ERN and CRN are both fronto-central negativities that peak within 50 ms following the execution of correct and incorrect responses, respectively. The ERN has been linked to both error and conflict detection (Gehring, Goss, Coles, Meyer, & Donchin, 2018) and seems to represent a type of alarm signal that indicates the need for subsequent adjustment (Ullsperger, Danielmeier, & Jocham, 2014) and that varies with error salience due to situational or personality factors (Proudfit, Inzlicht, & Mennin, 2013). The CRN occurs as a smaller negative deflection shortly after the correct responses and is quite similar to the ERN in appearance and time course—and is thought to reflect a basic response monitoring process (Klawohn, Riesel, Grützmann, Kathmann, & Endrass, 2014; Roger, Benar, Vidal, Hasbroucq, & Burle, 2010). The Pe follows the ERN on error trials, and is evident as a positive deflection with centro-parietal maximum from 200 to 400 ms window after incorrect response onset. Variation in magnitude of the Pe has been linked to error-awareness and motivational significance of errors (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Steinhauser & Yeung, 2010).

Although there is consistent evidence that anxiety disorders are associated with an increased ERN (Hajcak, Klawohn, & Meyer, 2019; Meyer, 2017), findings regarding the ERN and CRN in depression are quite mixed. Some studies have suggested that depression is characterized by significantly more negative ERN/CRN amplitudes (Aarts, Vanderhasselt, Otte, Baeken, & Pourtois, 2013; Chiu & Deldin, 2007; Tang et al., 2013). In contrast, other studies find a significant reduction in ERN or CRN amplitudes in individuals with depression (Ladouceur et al., 2012; Schrijvers et al., 2008) or no differences in CRN/ERN between the healthy and depression groups (Olvet, Klein, & Hajcak, 2010; Ruchsow et al., 2004). Similarly, findings on the Pe in depression are discrepant. Blunted Pe (Aarts et al., 2013; Olvet et al., 2010; Schrijvers et al., 2008, 2009), heightened Pe (Schoenberg, 2014), as well as no significant differences in Pe amplitude (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008) have all been reported in relation to depression.

Complicating matters further, it has been demonstrated that the CRN and ERN likely overlap and summate with stimulus-locked ERPs such as the P300 (Coles, Scheffers, & Holroyd, 2001; Hajcak, Vidal, & Simons, 2004). If depression impacts the stimulus-locked P300 in flanker tasks, then it is possible that blunted stimulus-related ERPs could impact the amplitude of these response-locked ERPs. Furthermore, the Pe has timing and scalp distribution

quite similar to the P300, covaries with the P300, and shares functional similarities with the P300 (Ridderinkhof, Ramautar, & Wijnen, 2009). This raises the possibility that depression-related reductions of P300 amplitude would similarly be associated with a reduction in Pe amplitude. A second goal of the current study was to examine the possibility that a reduced P300 in depression might cause apparent differences in the response-locked CRN and ERN; finally, we sought to evaluate whether reductions in the stimulus-locked P300 would be associated with blunted Pe in depression—and if these deficits would explain unique or overlapping variance in depression.

Overall then, the current study first examined whether adults with current clinical depression would be characterized by a reduced P300 on correct trials during an arrowhead version of the flanker task. In the case that depression-related differences in P300 were found, we sought to further examine whether these would be modulated by clinical characteristics within the participants with current depression, such as the presence of a diagnosis of persistent depressive disorder (PDD), comorbidity with other psychiatric disorders, current psychotropic medication, suicidality, or presence of melancholic depression. We also analyzed the response-locked CRN/ERN and Pe to determine if these ERPs related to response monitoring were abnormal among participants with depression; finally, we sought to determine whether P300 abnormalities in depression might explain potential group differences in the CRN/ERN, and related to deficits in the Pe. By investigating neural correlates of both stimulus-related and error-related processing and their respective overlap, the current study aimed at further elucidating the respective involvement of these cognitive functions in the pathophysiology of depression.

2 | METHODS

2.1 | Participants

A total of 88 individuals with a current diagnosis of depression and 49 healthy control participants were recruited from the general population in the Tallahassee area via locally targeted online advertisement (i.e., on facebook.com), through the psychology clinic at FSU, community postings, and word of mouth. Potential participants first underwent a phone screening regarding general inclusion criteria (normal or corrected vision, absence of neurological disorder and lifetime severe head trauma, and 18 to 60 years of age). Potentially suitable participants were then invited to the lab for a full interview to determine final eligibility with a clinical interview (see below). All participants in the depression group (DEP) met current diagnostic criteria for either a major depressive episode (MDE), PDD, or both. Exclusionary criteria for both groups were: current substance or alcohol use disorder, lifetime diagnosis of any psychotic disorder or bipolar disorder. Further exclusionary criteria for the healthy participant group (HC) were: presence of a lifetime mood disorder or any current psychiatric disorder. Data were collected during a single visit of a 2-visit protocol, and the results from other assessed measures (e.g., eye-tracking, fMRI) will be analyzed and presented separately. From the original sample, 23 participants (HC: $n = 7$; DEP: $n = 16$) were excluded either due to insufficient error numbers needed to obtain reliable ERN and Pe quantification (i.e., <6 errors; Olvet & Hajcak, 2009) during the flanker task (HC: $n = 6$, DEP: $n = 11$), or poor EEG data quality as determined by visual inspection of grand average waveforms (HC: $n = 1$, DEP: $n = 5$). The final sample for this study thus encompassed 114 participants (DEP: $n = 72$, HC: $n = 42$).

2.2 | Measures

2.2.1 | Clinical interview

All diagnostic criteria for the mood disorders were assessed with the Structured Clinical Interview for DSM-5-Research Version (SCID-5-RV; First, Williams, Karg, & Spitzer, 2015), while other forms of psychopathology were assessed with the Mini-International Neuropsychiatric Interview 7.0.2 for DSM-5 (MINI; Sheehan et al., 1998) by two PhD-level clinical psychologists. Using the clinical interview, further clinical measures were determined within the group of participants with depression, including current presence of a diagnosis of PDD, current comorbidity with other psychiatric disorders, current treatment with psychotropic medication (i.e., present/absent), presence of current symptoms of suicidality (i.e., endorsement of increased thoughts about own death, suicidal ideation, specific suicide plans, and/or suicide attempts), as well as presence of diagnostic criteria for the melancholic subtype of depression.

2.2.2 | Beck depression inventory–II

Participants in both groups further rated the current severity of depressive symptoms with the Beck Depression Inventory–II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a well-validated measure of depressive symptom severity with good psychometric properties. The total score derived from 21 items ranges from 0 to 63. In the current sample, internal consistency of the BDI-II was good (Cronbach's alpha; MDD: $\alpha = .88$; HC: $\alpha = .80$).

2.3 | Procedures

2.3.1 | Flanker task

While EEG was being recorded, participants completed an arrowhead version of the flanker task. The participants were seated about 31 cm away from the computer screen while performing the task. The flanker task was administered using Presentation software (Neurobehavioral Systems, Albany, California). Each trial consisted of five horizontal arrow heads presented for 200 ms, with an ITI varying from 2,300 to 2,800 ms. Half of the trials were congruent (<<<<<, >>>>>) and the other half were incongruent trials (<<><<, >><>>), and the order was randomly generated. Participants were instructed to respond as quickly and accurately as possible in response to the direction of the center arrow using the left and right buttons on the computer mouse. In order to assure competence at the task, all participants completed 10 practice trials before starting the actual task; this was repeated until accuracy was above 80%. The subsequent task consisted of 11 blocks of 30 trials (330 trials in total). Participants received performance-based feedback at the end of each block. If their accuracy was 75% or lower during a block they would receive the message "Please try to be more accurate". If their score was 90% or higher on a block they would receive the message "Please try to respond faster", else they received a message reading "You're doing a great job".

2.3.2 | EEG recording & quantification

EEG was recorded during the flanker task using an actiCHamp system (Brain Products GmbH, Gilching, Germany) with 32 (Ag/AgCl) active ActiCap slim electrodes placed in accordance with the international 10/20 system, with Cz serving as the online recording reference. Two electrodes were placed on left and right mastoids. The electrooculogram (EOG) was recorded with four additional electrodes were placed above and below the left eye, and to the sides of the eyes near the outer canthus. Data was digitized at a sampling rate of 1,000 Hz utilizing a low-pass online filter set at 100 Hz.

EEG analyses were performed using Brain Vision Analyzer (version 2.1; Brain Products, Gilching, Germany). First, EEG data was referenced offline to averaged mastoids, then filtered with low and high filter cutoffs set at 0.1 and 30 Hz, respectively. Ocular corrections were made using the Gratton, Coles, and Donchin (1983) procedure. Epochs with a voltage greater than 50 μV, a voltage difference of 175 μV within a 600 ms interval, or a maximum voltage difference of less than 0.50 μV within 100 ms intervals were discarded on a channel-specific basis using automatic artifact rejection.

2.3.3 | ERP scoring

For analysis of the P300, EEG data epochs of 1,000 ms were extracted starting from 200 ms prior to stimulus onset on correct trials and then averaged combining compatible and incompatible stimuli. The 200 ms pre-stimulus interval served as the baseline. The P300 was quantified as the mean amplitude between 300 and 600 ms at electrode site Pz. For analyses of response-related ERPs (i.e., ERN, CRN, and Pe) data was first segmented starting 500 ms before the response continuing for 1,000 ms after the response, separately for error and correct trials. Error and correct response segments were baseline-corrected using the interval from 500 to 300 ms before the response. The ERN and CRN were both scored as the mean amplitude from 0 to 100 ms at electrode FCz. The Pe was scored at electrode site Pz as the mean amplitude from 200 to 400 ms after errors, the respective mean amplitude after correct responses was analyzed as the Pc.

In accordance with previous studies (Levinson, Speed, Infantolino, & Hajcak, 2017), the internal consistency of ERP measures (i.e., P300, ERN,CRN, Pe, and Pc) was examined with a split-half approach: the correlation between averages of odd and even numbered trials was corrected using the Spearman–Brown prophecy formula (Nunnally, Bernstein, & Berge, 1967). Results indicated good to excellent internal consistency in both experimental groups for the P300 (DEP: $r = .981$; HC: $r = .982$), the ERN (DEP: $r = .839$; HC: $r = .751$), the CRN (DEP: $r = .981$; HC: $r = .978$), the Pe after errors (DEP: $r = .847$, HC: $r = .835$), and the Pc scored on correct trials (DEP: $r = .955$, HC: $r = .947$).

2.3.4 | Statistical analyses

All data analyses were performed using SPSS Statistics software (Version 23.0; IBM, Armonk, N.Y., USA). Group differences in demographics, self-report scores, and task performance (i.e., reaction times, error rates) were examined using independent samples t tests or χ^2-tests. Error rates were calculated as the percentage of errors in relation to all responses for each participant. Pearson's correlation coefficient r was used to evaluate the relationship between P300, ERN/CRN, and Pe/Pc amplitudes in the overall sample, and to assess the relationship between P300 and BDI-II scores in the DEP group. A one-way ANOVA was used to assess for a difference in P300 amplitude between the HC and DEP groups. In addition, subgroups within participants with depression were compared with regards to P300 amplitudes using one-way ANOVAs with the factors: presence of PDD, comorbidity, medication, suicidality, and melancholic depression. Further, ERN/CRN and Pe/Pc amplitudes were compared between both groups with two separate mixed-model ANOVAs with response type

(error, correct) as within-subjects factor and group (DEP, HC) as between-subjects factor. Repeated measures ANCOVAs that included P300 as a covariate were utilized to determine whether CRN/ERN and Pe/Pc differences between groups remained after controlling for the P300—and to assess potential interactions between the P300 and response-locked ERPs. Significant interactions were followed-up with *t* tests. Effect sizes for significant results were reported as partial eta squared (η_p^2) and Cohen's *d*. All statistical tests were two-tailed with an alpha-level of .05.

3 | RESULTS

3.1 | Demographic and Behavioral Results

Demographic and clinical measures for the DEP and HC groups are presented in Table 1. There were no significant differences between groups with respect to age, $t(112) = -1.52$, $p = .130$, or gender, $\chi^2(1) < 0.001$, $p = .987$. Participants in the DEP group met diagnostic criteria for either a current MDE ($n = 42$, 58.3%), current PDD ($n = 4$, 5.6%), or both ($n = 26$, 36.1%). Half of the participants in the DEP group ($n = 36$; 50%) were currently treated with psychotropic medication, such as antidepressants ($n = 30$), stimulants ($n = 6$), anxiolytics ($n = 13$), or other ($n = 9$). Within the DEP group, current comorbidity with other mental health disorders was present in 38 individuals (53%), and included the following: generalized anxiety disorder ($n = 17$), panic disorder ($n = 16$), agoraphobia ($n = 10$), social anxiety ($n = 12$), obsessive-compulsive disorder ($n = 5$), posttraumatic stress disorder ($n = 4$), eating disorders ($n = 6$), and specific phobia ($n = 2$). Further, 30 participants with depression reported current suicidality, whereas 42 did not. Finally, within the DEP group, 39 out of 72 individuals met diagnostic criteria for the melancholic subtype of depression.

TABLE 1 Demographic, clinical, behavioral, and ERP measures in the groups of participants with current depressive disorder (DEP) and the healthy participants group (HC)

	DEP ($n = 72$)	HC ($n = 42$)	*p*
Demographics & clinical			
Age (Years)	38.19 (12.14)	34.42 (13.74)	.130
Gender (% female)	76.1	76.2	.987
Caucasian (%)	95.8	88.1	.124
Education (Years)	16.06 (2.04)	16.29 (1.70)	.539
BDI-II	27.92 (10.74)	2.21 (3.04)	<.001
Behavioral data			
Correct RT (all trials; ms)	456.24 (91.80)	409.12 (52.83)	.001
Error RT (all trials; ms)	345.25 (70.22)	323.74 (50.19)	.088
Error rate (%)	9.42 (4.62)	8.53 (4.03)	.304
ERP measures			
P300 at Pz (μV)	7.32 (3.55)	8.89 (3.84)	.028
ERN at FCz (μV)	−1.01 (6.18)	0.45 (5.50)	.207
CRN at FCz (μV)	5.58 (4.85)	8.04 (4.71)	.009
Pe (error) at Pz (μV)	9.63 (6.03)	12.30 (5.34)	.020
Pe (correct) at Pz (μV)	2.60 (3.00)	2.88 (2.23)	.584

Note: Means are presented, standard deviations in parentheses. Behavioral data is missing for 2 participants, DEP: $n = 71$, HC: $n = 41$.

Abbreviations: BDI-II, Beck Depression Inventory II; RT, reaction time.

3.2 | Behavioral results

Behavioral results are presented in Table 1. Behavioral data from two participants was missing, resulting in 71 DEP and 41 HC for behavioral analyses. The DEP group had significantly slower reaction times as compared to the HC group for correct responses, $t(110) = -3.45$, $p = .001$, $d = .66$, with a similar trend on error trials, $t(110) = -1.72$, $p = .088$. On average, participants in the DEP group made 30.8 errors ($SD = 14.7$; range: 7–76) and participants in the HC group had an average of 28.7 errors ($SD = 13.3$; range 6–58), there was no significant difference in error rate between groups, $t(110) = -1.03$, $p = .304$.

3.3 | ERP results

Stimulus- and response-locked ERP waveforms are presented in Figure 1, values for all ERPs are presented in Table 1. With regards to the stimulus-locked P300, results of a one-way ANOVA indicated that participants in the DEP group had significantly smaller P300 compared to participants in the healthy control group, $F(1, 112) = 4.93$, $p = .028$, $d = .42$. Within the DEP group, no differences in P300 were evident when comparing DEP participants with and without a diagnosis of PDD, $F(1, 70) = 0.91$, $p = .344$, with and without current comorbidity, $F(1, 70) = 0.95$, $p = .334$, current symptoms of suicidality, $F(1, 70) = 1.34$, $p = .251$, or current melancholic subtype of depression, $F(1, 70) = 0.57$, $p = .453$. Participants with depression currently treated with psychotropic medication had a numerically (though not statistically) larger P300 ($M = 8.00$, $SD = 2.98$) than those not currently taking medication ($M = 6.64$, $SD = 3.97$), $F(1, 70) = 2.72$, $p = .104$. Within the DEP group, P300 amplitude and depression severity measured with the BDI-II were not correlated, $r(72) = -.091$, $p = .447$.

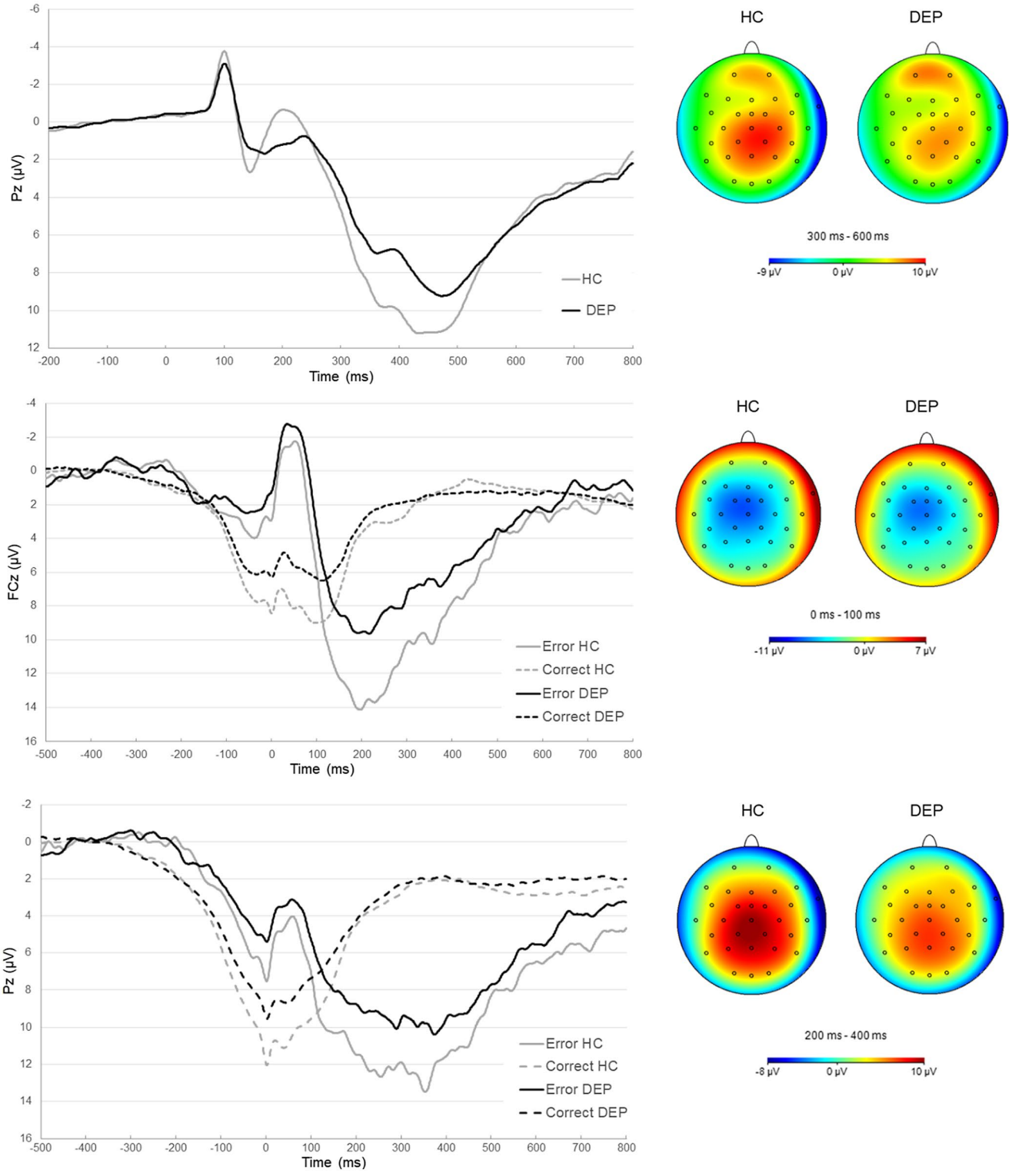

FIGURE 1 Grand average waveforms for correct trials relative to stimulus onset at electrode Pz and headmaps with scalp distribution during the interval from 300 to 600 ms after stimulus onset (upper panel); grand average waveforms for error and correct response trials relative to response onset at electrode FCz and headmaps for error-correct scalp distribution during the interval from 0 to 100 ms after response onset (middle panel), grand average waveforms for error and correct response trials relative to response onset at electrode Pz and headmaps for error-correct scalp distribution during the interval from 200 to 400 ms after response onset (lower panel), in the groups of participants with DEP and the HC

The repeated measures ANOVA for ERN/CRN indicated a main effect of response type, $F(1, 112) = 190.93$, $p < .001$, $\eta_p^2 = .63$ and of group, $F(1, 112) = 4.61$, $p = .034$, $\eta_p^2 = .04$, but no interaction, $F(1, 112) = 0.952$, $p = .331$; these results are consistent with the impression from Figure 1, such that the ERN was overall more negative than the CRN, and that

both the ERN and CRN were increased (i.e., more negative) in the DEP compared to the HC group.

Results of the repeated measures ANOVA for Pe/Pc amplitudes indicated a significant main effect of response type, $F(1, 112) = 260.87$, $p < .001$, $\eta_p^2 = .70$ and group $F(1, 112) = 4.26$, $p = .041$, $\eta_p^2 = .04$, further specified by a significant interaction of group and response type, $F(1, 112) = 5.41$, $p = .022$, $\eta_p^2 = .05$. Follow-up tests confirmed the impression from Figure 1 that DEP and HC groups differed in Pe amplitude after error responses ($t(112) = 2.37$, $p = .020$, $d = .49$), but not Pc after correct responses ($t(112) = .546$, $p = .584$).

When including P300 as a covariate in the ANCOVA on ERN/CRN amplitudes, the response type effect remained significant, $F(1, 111) = 30.55$, $p < .001$, $\eta_p^2 = .22$, and there was no interaction between response type and group, $F(1, 111) = 0.797$, $p = .374$. Moreover, the group effect was no longer significant, $F(1, 111) = 0.938$, $p = .335$, whereas the P300 emerged as a significant covariate, $F(1, 111) = 66.84$, $p < .001$, $\eta_p^2 = .38$. No significant interaction between P300 and response type was observed, $F(1, 111) = 0.081$, $p = .777$. Correlational analyses further confirmed a larger P300 was related to smaller (i.e., more positive) ERN (DEP: $r(72) = .365$, $p = .002$, HC: $r(42) = .676$, $p < .001$) and CRN (DEP: $r(72) = .562$, $p < .001$, HC: $r(42) = .708$, $p < .001$).

Similarly, when the P300 was included into the ANCOVA on Pe/Pc amplitudes, there was still a significant effect of response type, $F(1, 111) = 9.42$, $p = .003$, $\eta_p^2 = .08$ whereas neither the between-group effect, $F(1, 111) = 1.39$, $p = .242$, nor the interaction between response type and group were significant, $F(1, 111) = 2.26$, $p = .135$. The P300 again was a significant covariate in the ANCOVA, $F(1, 111) = 26.83$, $p < .001$, $\eta_p^2 = .20$, and there was a significant interaction between P300 and response type, $F(1, 111) = 23.10$, $p < .001$, $\eta_p^2 = .17$. In line with the interaction of the covariate and response type, the P300 was associated with Pe amplitudes following errors (DEP: $r(72) = .338$, $p = .004$, HC: $r(42) = .711$, $p < .001$), but not with Pc amplitudes following correct responses (DEP: $r(72) = .168$, $p = .159$, HC: $r(42) = .268$, $p = .086$).

3.4 | Control analyses

Identical results for ERN/CRN analyses were found when a more response-proximal baseline-correction period (i.e., from −200 to −50 ms before response) was used: there was a significant main effect of response type, $F(1, 112)$ 122.46, $p < .001$, and group, $F(1, 112) = 4.38$, $p = .039$, with no interaction between response type and group, $F(1, 112) = 0.49$, $p = .487$. When P300 amplitude was included as a covariate, P300 amplitude emerged as a significant covariate, $F(1, 111) = 33.78$, $p < .001$. The response type effect remained, $F(1, 111) = 40.31$, $p < .001$, whereas the initial group effect was no longer significant, $F(1, 111) = 1.29$, $p = .259$. There was no significant interaction between response type and group, $F(1, 111) = 1.13$, $p = .290$.

Further, results of the ANCOVA analyses remained when both P300 amplitude and mean reaction time (RT) were included as covariates. Regarding analyses of covariance for ERN/CRN, there was no group difference, $F(1, 108) = 0.249$, $p = .619$, and P300 was the only significant covariate, $F(1, 108) = 55.94$, $p < .001$; there was trend toward RT as an additional covariate, $F(1, 108) = 3.46$, $p = .065$. For the Pe/Pc analyses of covariance, both P300 and mean RT emerge as significant covariates, $F(1, 108) = 20.22$, $p < .001$ and $F(1, 108) = 15.52$, $p < .001$, respectively. Further, the initially observed interaction of response type and group was no longer significant, $F(1, 108) = 0.80$, $p = .375$, and there was similarly no longer a main effect of group, $F(1, 108) = 0.08$, $p = .778$.

Within the DEP group, no differences in ERN were evident when comparing participants with and without a current comorbid diagnosis of any anxiety (i.e., GAD, social anxiety, panic disorder, and OCD), $F(1, 70) = 0.77$, $p = .382$.

In addition to analysis of the P300, the stimulus-locked N200 was analyzed. Mean amplitudes were quantified separately for compatible and incompatible stimuli types on correct responses at electrode FCz from 200 to 350 ms after stimulus onset. Results from a 2 (group) × 2 (trial type) mixed model ANOVA indicated a main effect of stimulus type, $F(1, 112) = 26.00$, $p < .001$, such that the N200 was more negative on incompatible than compatible trials; however, there was no main effect of group, $F(1, 112) = 0.03$, $p = .872$, and no interaction between group and stimulus type, $F(1, 112) = 1.11$, $p = .294$.

4 | DISCUSSION

In the current study, a significantly decreased stimulus-locked P300 to flanker stimuli was found in a group of individuals with current depression compared to never depressed individuals. This finding is in line with past reports of depression characterized by diminished P300 amplitude in auditory oddball tasks (Bruder et al., 2009; Gangadhar et al., 1993; Nan et al., 2018; Roschke & Wagner, 2003; Urretavizcaya et al., 2003). Findings on P300 in depression using Go/No-Go tasks are much more mixed: some studies fail to find significant differences in P300 between participants with depression and healthy participants (Kaiser et al., 2003; Ruchsow et al., 2008), even with relatively large sample sizes (Quinn, Harris, & Kemp, 2012). The current study extends previous work to demonstrate that a reduced P300 can also be obtained on correct trials in speeded response tasks typically used to examine response conflict and response monitoring ERPs.

Results of the current study further indicated that the reduced P300 in the DEP was unrelated to a range of clinical

characteristics: presence of a diagnosis of PDD, current comorbidity, suicidality, medication, or melancholic subtype did not impact the P300. Further, severity of depression also did not relate to variation in P300 amplitude within the depression group. Thus, our findings indicate a blunted P300 in depression occurring independently of clinical subtypes and symptom severity levels. While this is at odds with some previous findings (Ancy et al., 1996; Gangadhar et al., 1993; Hansenne et al., 1996; Nan et al., 2018; Urretavizcaya et al., 2003), it is worth noting that most of these studies varied considerably in the clinical characteristics that were linked to P300 and were based on rather small clinical sample sizes, that is, $n < 25$ in all but the Nan et al. ($n = 45$) and Urretavizcaya et al. ($n = 50$) studies. Since the P300 amplitude has been functionally associated with attentional allocation and processing of motivationally relevant stimuli, the generic reduction in this component is consistent with the broad impairment of neuropsychological functioning that is observed in depression (Rock et al., 2014; Snyder, 2013). Evidence shows that attention and executive function deficits persist following depressive symptom remission (Bhalla et al., 2006). Further, since cognitive dysfunctions have detrimental effects on psychosocial functioning and treatment response, further cognitive interventions might be needed (Rock et al., 2014), and the P300 could be explored as a potential biological target (Hajcak et al., 2019). Research on the persistence or recovery of depression-related P300 alterations after symptom remission is scarce (Karaaslan, Gonul, Oguz, Erdinc, & Esel, 2003). Therefore, future research is needed to further examine how reduced P300 in the flanker task relates to measures of neuropsychiatric functioning in depression, as well as changes in depressive symptoms and syndromes over time—including course of illness or response to treatment.

With regard to response monitoring ERPs, we initially observed a more negative ERN and CRN, as well as a significantly blunted Pe in the depression group. However, when individual differences in the P300 were included in analyses, both the ERN/CRN and Pe no longer varied between participants with and without depression. Insofar the ERN and CRN overlap with the positive deflection of the P300 (Hajcak et al., 2004), group differences in the ERN/CRN could likely result directly from ERP component overlap: a smaller P300 could produce apparently larger (i.e., more negative) response-locked ERPs. In line with this possibility, P300 amplitudes were strongly correlated with both ERN and CRN amplitudes in both groups. Collectively, our findings suggest that the ERN and CRN were relatively normal in the depression group, but were instead modulated by overlap with reduced stimulus-locked P300s.

In a previous article, we demonstrated how variability in the P300 interacted with anxiety disorder status to impact the ERN (Meyer, Lerner, De Los Reyes, Laird, & Hajcak, 2017): a significantly increased P300 amplitude in anxious participants explained an apparent association between CRN and clinical anxiety. In the current study, we observed the opposite effect related to the same ERP overlap: blunted P300 amplitudes were observed in the group of participants with depression, which explained an apparent increase in ERN/CRN amplitudes in the response-locked ERPs. Thus, without controlling for the group difference in P300 amplitudes, results of this study would have indicated altered response-related ERPs in participants with depression as compared to healthy individuals. More broadly, differences in stimulus-related processing, (e.g., due to task or sample characteristics) and overlap with response-locked ERPs might explain some of the inconsistent findings in the literature on ERN and CRN in depression.

With regards to the Pe, a similar pattern emerged, insofar as initial differences in Pe between groups were explained by the P300. Given the timing of the Pe with respect to average reaction times, it seems unlikely that the P300 and Pe overlap physically with one another. More likely, the Pe reflects a distinct P300 response to infrequent errors (Ridderinkhof et al., 2009). Thus, the reduction in the P300 and Pe in depression could reflect two manifestations of the same underlying abnormality.

In sum, we found significant differences in in the P300 between individuals with depression and never depressed individuals on correct trials in the flanker task. Thus, the current investigation demonstrates that the flanker task is a viable paradigm to study the P300 in depression. The current results did not find evidence that variability in the P300 related to clinical characteristics within this rather large sample of adults with current clinical depression, pointing toward a broad impairment in basic attentional allocation to task-relevant stimuli, independent of specific symptom subtypes. Further, the inclusion of stimulus-related differences helped to gain a more comprehensive—and potentially more accurate—perspective on response-monitoring processes in the individuals with current depression. Namely, the decrease in P300 accounted for seemingly increased ERN/CRN components in the group of participants with depression. Furthermore, the current study found a reduction in Pe amplitude in depression that was associated with the P300—and both accounted for the same variance in depression status. Thus, reduced P300 in depression can be observed time-locked to imperative stimuli in the flanker task and following the commission of rare errors. These data collectively suggest a common impairment in depression: reduced attentional allocation to salient events (Foti & Hajcak, under review); moreover, this deficit does not appear to depend on event frequency (i.e., as in the oddball task). Finally, the current data suggests that depression is characterized by relatively

normative early response-monitoring processes (i.e., CRN and ERN). Future studies might further examine P300 and Pe in relation to changes in depressive symptoms over time to examine whether these ERP deficits follow similar patterns after symptom remission—and if they can be used to predict the course of depressive symptoms and syndromes.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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