**Event-related Potential Correlates of Affective Response Inhibition in Bipolar I Disorder:
Comparison with Schizophrenia**

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Abstract

Background: Individuals with bipolar I disorder (BD) have difficulty inhibiting context-inappropriate responses. However, the neural mechanisms contributing to these difficulties, especially in emotional contexts, are little understood. This study aimed to inform mechanisms of impaired impulsivity control in response to emotion in BD, and whether response inhibition indices are altered to a similar degree in schizophrenia spectrum disorders (SZ). We examined alterations to behavioral performance and event-related potentials (ERPs) during inhibition to affective stimuli in BD, relative to healthy control participants (HC) and SZ.

Methods: Sixty-six participants with BD, 32 participants with SZ, and 48 HC completed a Go/No-Go task with emotional face stimuli while electroencephalography was recorded. Behavioral signal detection metrics (perceptual sensitivity, response bias) and ERPs (N200, P300) were compared across groups.

Results: Relative to HC, participants with BD showed reduced (1) discrimination of Go vs. No-Go stimuli (i.e., emotional vs. neutral faces), and (2) P300 amplitudes elicited by emotional faces. Results similarly extended to SZ: BD and SZ groups did not differ on behavioral performance nor ERP amplitudes.

Limitations: Aspects of the Go/No-Go task design may have limited findings, and medication effects on ERP amplitudes in patient samples cannot be fully ruled out.

Conclusions: Findings suggest the difficulty participants with BD and SZ experienced on the current affective response inhibition task lied largely in discriminating between facial expressions. Difficulties with discriminating emotional from neutral expressions may contribute to difficulties with appropriate behavioral responding in social-affective contexts for individuals with BD and SZ.

Keywords: *bipolar disorder; schizophrenia; response inhibition; event-related potential; signal detection*

1. Introduction

Cognitive control processes allow for flexible human behavior and adaptive capacity to achieve goals in dynamic and novel environments. Impairments to cognitive control processes, as indexed by traditional neuropsychological tests of executive function, are predictive of functional outcomes in bipolar disorder (BD).^{1,2} Response inhibition is an aspect of cognitive control that involves suppression of a prepotent but context-inappropriate response tendency.³ Difficulties with response inhibition are thought to contribute to impulsive behavior, and impulsivity in the context of heightened emotion is linked to risk for BD and adverse illness outcomes.^{4,5}

Difficulties with behavioral response inhibition, however, are not specific to BD.⁶ A growing body of research supports overlapping neurobiological and cognitive correlates of BD and schizophrenia spectrum disorders (SZ).^{7,8} Individuals with SZ show modestly more impairment on neuropsychological tests of executive function relative to BD⁹; however, tests specifically probing inhibitory control indicate a similar degree of impairment across both illnesses.¹⁰ Gotra et al. (2020) observed that while behavioral inhibition was impaired to a similar extent across individuals with BD and SZ, only those with BD showed such impairment that was not accounted for by the generalized cognitive deficit—suggesting distinct mechanisms may give rise to the observed behavioral impairments. However, neural correlates of inhibition impairments in individuals with BD and whether these alterations are similar in extent to SZ are unclear. Thus, examining neural indices in addition to behavioral metrics in BD and SZ groups can inform whether altered inhibitory control mechanisms are common across these related psychiatric disorders.

Behavioral response inhibition can be assessed with a Go/No-Go task, in which participants respond as quickly as possible to frequent target stimulus (Go) and withhold (i.e. inhibit) responding to an infrequent distractor stimulus (No-Go). Prior studies supporting behavioral inhibition impairments in BD have typically used accuracy or commission errors (false alarms) as primary outcome measures,⁶ with only a few exceptions analyzing signal detection metrics (e.g. Gopin et al., 2011). Signal detection metrics use information from both rate of correct response execution (Go accuracy) and rate of failing to inhibit (false alarm rate) to separate out sensitivity (ability to distinguish between target and non-target stimuli) and response bias (tendency to respond)—which are confounded in accuracy rates.¹²

Neural activity associated with inhibitory control can be examined using event-related potentials (ERPs) elicited during such a Go/No-Go task.¹³ The N200 elicited during an inhibitory control task is a negative-going deflection in the ERP waveform maximal over frontocentral regions, proposed to index monitoring of response conflict.¹⁴ The P300 is a parietally maximal positive-going deflection, thought to index attention and memory processes, and is impacted by perceptual discrimination difficulty.¹⁵ By directly measuring neural activity with high temporal resolution, ERPs allow for assessment of alterations in rapidly-occurring cognitive processes, including in the absence of a behavioral response (e.g. while correctly inhibiting responses on No-Go trials).¹⁶

The P300 amplitude is reduced in both BD and SZ, relative to healthy individuals.^{17–20} Although less studied, N200 reductions are also reported in both BD and SZ—though some exceptions have reported reduced N200 was unique to either only BD or SZ, respectively.^{17,21–23} However, only a handful of studies have employed tasks requiring inhibition to examine both disorders,^{21,22} and ERPs during inhibition in *affective contexts* in BD and SZ have not yet been

studied. Given evidence that impulsive behavior in response to heightened emotions is particularly relevant to BD risk and illness outcomes,²⁴ examining neural activity during behavioral inhibition to affective stimuli may better inform mechanisms contributing to impaired control of impulsive responding that is characteristic of BD. Thus, the current study utilized an affective task to characterize alterations to behavioral performance and ERPs during response inhibition in BD, and examine whether these alterations similarly extend to SZ.

1.2. Present study aims

Our primary aims were to test whether behavioral performance and N200 and P300 ERP amplitudes during affective response inhibition differ between individuals with BD, SZ, and healthy control participants (HC).

We hypothesized both behavioral performance and ERP amplitudes to be altered in BD and SZ, relative to HCs. Specifically, we expected decreased perceptual sensitivity, greater bias to respond to emotional stimuli, and decreased N200 and P300 amplitudes—particularly on No-Go trials. We also hypothesized that degree of alterations to behavioral and ERP indices would be similar in BD and SZ, based on prior behavioral evidence of a similar degree of inhibitory control impairment.^{10,25} We also explored 1) effects of different emotions on ERP amplitudes and behavioral performance, and 2) behavioral and symptom correlates of ERP amplitudes during affective response inhibition, across all participants.

2. Methods

2.1. Participants

Data from 146 participants, consisting of 66 individuals diagnosed with BD, 32 with SZ (19 schizophrenia, 13 schizoaffective), and 48 HC were included in the analyses of the current report. Participants aged 18—65 were recruited from the community via advertisements, local mental health clinics, and referrals by other researchers. Exclusion criteria included substance abuse/dependence in the past 6 months, history of closed head injury, neurological illness, or medical conditions with neurological sequelae (e.g., encephalitis, seizure disorders). All participants had normal or corrected-to-normal vision (i.e., 20/30 or above according to a Snellen chart). Additional exclusion criteria for HCs included personal lifetime history of a DSM-IV axis-I disorder and a first-degree relative with a history of psychotic or bipolar disorder.

All participants were administered a diagnostic interview based on DSM-IV criteria to either confirm psychiatric diagnosis or rule out psychiatric diagnosis for HCs. The BD sample and 29 of the HCs were drawn from the Prechter Longitudinal Study of Bipolar Disorder,²⁶ and their diagnoses were established with the Diagnostic Interview for Genetic Studies²⁷ and confirmed through a best estimate process in which at least two MD/PhD level clinicians reached consensus. Diagnoses for SZ participants and 21 HC were established with the Structured Clinical Interview for DSM-IV-TR.²⁸ Demographic and clinical participant characteristics are presented in **Table 1**.

One BD, 6 HC, and 3 SZ participants were excluded from analyses due to technical errors. Two BD and 2 SZ participants were excluded due to poor data quality (>50% trials removed due to artifact during offline processing), and one additional BD participant was excluded due to extreme values (>3 SD of sample's mean amplitude) along with visual inspection confirming poor data quality. This yielded the analytic sample of 146 participants.

This study was approved by the University of Michigan Medical School Institutional Review Board. Informed consent was obtained from each participant and compensation was provided.

2.2. Additional assessments

The current analytic dataset was compiled by integrating EEG data collected over multiple studies with differing assessment protocols. Thus, the same clinical measures were not consistently available across participants. Mood symptoms were assessed via self-report using the Beck Depression Inventory (BDI-II)²⁹ and the Altman Self-rating Mania Rating Scale (ASRM)³⁰ in 97% and 81% of the sample, respectively. The clinician-rated 17-item Hamilton Depression Rating Scale (HAM-D-17)³¹ and Young Mania Rating Scale (YMRS)³² were also administered to a smaller subset of participants. For SZ participants, clinical symptoms were also assessed with the Scale for the Assessment of Positive Symptoms (SAPS)³³ and Scale for the Assessment of Negative Symptoms (SANS).³⁴ Details on scoring and availability of symptom scales are presented in **Table 1**.

2.3. Affective Go/No-Go task

The affective Go/No-Go task (adapted from Schulz et al., 2007), which was programmed using Eprime software (Psychology Software Tools), is depicted in **Figure 1** in full detail. The task consisted of 32 grey-scaled facial image stimuli with emotional and neutral expressions from the NimStim set.³⁶ Each task block consisted of one emotion face (happy, sad, or angry) as the target (Go stimulus) with neutral faces as No-Go stimuli or vice-versa. Participants were instructed to respond as quickly as possible to the target face (Go) and withhold responding to non-target faces (No-Go). Stimuli order was pseudorandomized to not have two consecutive No-Go stimuli. Go/No-Go pairs consisted of happy go/neutral no-go, angry go/neutral no-go, sad

go/neutral no-go, neutral go/happy no-go, neutral go/sad no-go, and neutral go/angry no-go.

Each of these six pairs was repeated with the reverse order of stimulus presentation to yield 12 experimental conditions (600 trials) in total.

2.4. Behavioral metrics

The primary behavioral outcomes of interest were signal detection measures of d' -prime (d') and criterion (β), both calculated using formulas provided by Schulz et al. (2007). All participants' number of hits, false alarms, Go trials, and No-Go trials were adjusted so that it was mathematically possible to perform the z-transformation and compute d' and β for those with hit rates or false alarm rates of 100% or 0%.¹² D' provides a measure of perceptual sensitivity to differing stimuli (in this case, emotional vs. neutral faces), with larger d' indicating greater ability to distinguish stimuli. β provides a measure of response bias, with negative β values indicating a stronger tendency to respond.¹² D' and β were calculated separately for experimental blocks with emotional Go/neutral No-Go stimuli and neutral Go/emotional No-Go stimuli (collapsed across happy, sad, and angry), to examine sensitivity and bias to respond to emotional cues relative to neutral. These experimental block types will henceforth be called *emotion predominant blocks* and *neutral predominant blocks*, respectively.

2.5. ERP Data Acquisition and Processing

Participants' electroencephalogram (EEG) was recorded using a 32-channel cap with Ag/AgCl electrodes using the International 10–20 system (Brain Products, GmbH, Munich, Germany) while participants completed the affective Go/No-Go task. Data was processed offline using custom MATLAB R2020b scripts based on EEGLAB v2020.0 functions.³⁷ Full detail on data acquisition and processing is available in the **Supplement**.

Following processing, EEG data only from behaviorally correct trials (when participants either correctly responded on Go trials or withheld responses on No-Go trials) were averaged to produce ERP waveforms for subsequent analyses. Correct Go trials were only included when preceded by another Go trial, to match Go and No-Go trials on the preceding trial type. For ERP amplitudes, only data on emotional experimental conditions (angry go, happy go, sad go, angry no-go, happy no-go, and sad no-go) were included, to examine inhibitory processes in affective contexts relative to neutral. Mean ERP amplitudes were computed separately for Go trials (that followed Go trials) and No-Go trials (collapsed across happy, sad, and angry for both trial types). Mean amplitudes were calculated for N200 between 230—300 ms following stimulus onset at the average of Fz, Cz, FC1, and FC2 electrodes and for P300 between 300—500 ms at the Pz electrode, based on prior literature³⁸ in combination with visual inspection of averaged waveforms and scalp topography collapsed across groups and experimental conditions from this sample.³⁹ Given the N200 is negative-going, *smaller* amplitudes will refer to *less negative* N200 mean voltage, for clarity.

2.6. Data Analysis

All statistical analyses were conducted in RStudio 1.3.959. Differences in demographic characteristics between diagnostic groups (HC, BD, SZ) were tested using one-way ANOVA, followed by post-hoc Tukey HSD tests.

Primary behavioral and ERP analyses. Effects of diagnostic group, experimental condition (block type or Go/No-Go trial type for examining behavioral and ERP outcomes, respectively), and their interactions on behavioral performance (d' , β) and ERP amplitudes (N200, P300) were analyzed within linear mixed-effects models framework using the lme4 package⁴⁰ and lmerTest package extension to extract test statistics.⁴¹ Linear mixed-effects

models were estimated using restricted maximum likelihood and compound symmetry. For both behavioral and ERP outcomes, non-significant ($p > .05$) interaction effects were removed from the final models, and main effect results presented below come from these final models. For final models for both behavioral and ERP outcomes, significant effects were followed up with False Discover Rate-adjusted pairwise contrasts within each model. Cohen's d effect size metrics were computed to illustrate magnitudes of group differences on behavioral and ERP variables.

To analyze behavioral metrics, we specified block type (emotion predominant blocks [emotion Go/neutral No-Go]; neutral predominant blocks [neutral Go/emotion No-Go]) as a within-subject measure, group as a between-subjects factor (HC, BD, SZ), and their interaction as independent variables to predict d' and β in separate models. To analyze ERP amplitudes, we specified Go/No-Go trial type (Go, No-Go) as a within-subject measure, group as a between-subjects factor (HC, BD, SZ), and their interaction as independent variables to predict N200 and P300 mean amplitudes as dependent variables in separate models.

Exploratory analyses. We tested emotion condition effects and diagnostic group by emotion interactions on ERP amplitudes and behavior, to explore whether group effects differed by emotion conditions. Briefly, analyses were conducted similarly to those of the primary analyses, with the addition of a within-subjects factor of emotion (happy, sad, angry) and its interaction with group. More details on analyses of emotion condition effects are available in the **Supplement**.

We additionally explored whether ERP indices related to task performance and clinical symptoms. We tested correlations between ERP amplitudes and 1) behavioral signal detection metrics (d' , β) and 2) self-report mood symptoms (depressive [BDI-II], manic [ASRM]). Only ERPs that were found to differ between groups in primary analyses were correlated with

behavioral performance, to explore whether degree of alteration of neural and behavioral indices corresponded. However, mood symptoms were correlated with all four ERP variables of interest (N200 Go, N200 No-Go, P300 Go, P300 No-Go), given the relevance of mood disturbance for BD illness.

Finally, we explored possible effects of antipsychotic, lithium, and benzodiazepine medications on ERPs across the BD and SZ groups. N200 and P300 amplitudes (Go and No-Go amplitudes averaged together for each ERP) were compared between users versus non-users of each of the three medication classes in separate one-way ANOVAs.

3. Results

The three groups were matched on age, gender, and parental education (See **Table 1**).

3.1. Behavioral task performance

Descriptive statistics for behavioral performance are depicted in **Table 2** and **Figure 2**. There was no group difference in the effect of block type on either behavioral outcome (group X block type interaction non-significant for d' ($F[2, 143] = 1.377, p = .256$) and β ($F[2, 143] = 1.521, p = .222$). Thus, these interaction terms were removed from the final models presented below.

Perceptual sensitivity (d'). Sensitivity was higher for emotion predominant blocks than neutral predominant blocks, indicating that participants were more able to discriminate emotional from neutral faces when emotional faces were the predominant stimuli, $F(1, 145) = 65.184, p < .0001$. Groups differed in perceptual sensitivity, $F(2, 143) = 3.639, p = .029$. Post-hoc analyses indicated both BD and SZ groups showed reduced discrimination of Go vs. No-Go stimuli,

relative to HCs (HC vs. BD: $t[143] = -2.328, p = 0.033$, HC vs. SZ: $t[143] = 2.316, p = .033$); BD and SZ groups did not differ from each other in sensitivity ($t[143] = 0.404, p = .687$).

Response bias (β). Response bias was more negative for emotion predominant blocks than for neutral predominant blocks, indicating a stronger bias to respond (i.e., more disinhibited) when emotional faces were predominant across all participants, $F(1, 145) = 45.173, p < .0001$. Groups did not differ in response bias, $F(2, 143) = 0.132, p = .877$.

3.2. Effects of diagnostic group and Go/No-Go trial type on ERP amplitudes

Descriptive statistics of ERP amplitudes and number of trials averaged to produce ERP waveforms are shown in **Table 2**. ERP waveforms are displayed in **Figure 3**.

There was no group difference in the effect of Go/No-Go trial type on either ERP amplitude: group X trial type was non-significant for N200 ($F[2, 143] = 2.527, p = .083$) and P300 ($F[2, 143] = 1.558, p = .214$). Thus, these interaction terms were removed from the models presented below.

N200. The N200 mean amplitude was larger on No-Go trials than Go trials, $F(1, 145) = 8.316, p = .005$. Groups did not differ in N200 amplitudes, group $F(2, 143) = 2.380, p = .096$.

P300. The P300 mean amplitude was larger on Go trials than No-Go trials, $F(1, 145) = 26.255, p < .0001$. Groups also showed differential P300 amplitudes, group, $F(2, 143) = 4.842, p = .009$. Post-hoc contrasts revealed that both BD and SZ groups showed reduced P300 amplitudes, relative to HCs, whereas BD and SZ groups did not differ from each other (BD vs. HC: $t(143) = 2.770, p = .017$, SZ vs. HC: $t(143) = 2.575, p = .017$; BD vs. SZ: $t(143) = 0.289, p = 0.773$).

Cohen's d effect sizes illustrating the magnitude of difference between groups on ERP amplitudes and behavioral performance metrics are shown in **Table 3**.

3.3. Effects of emotion condition on behavior and ERP amplitudes

Briefly, we observed that behavioral performance and ERP amplitudes differed by emotion condition across the sample, but did not observe significant interactions between diagnostic group and emotion on ERP amplitudes. Descriptive statistics for behavioral performance and ERP amplitudes broken down by emotion are available in **Supplemental Tables 1 and 3**; analytic results regarding effects of emotion are presented in **Supplemental Tables 2 and 4**.

3.5. Correlates of ERP amplitudes

Only significant correlations ($p < .05$) are reported below. Full correlational results are available in **Supplemental Table 5**. Significant correlations in the full sample were followed up in the patient-only sample, collapsing across BD and SZ groups (due to null differences observed between patient groups in the primary analyses).

Behavioral Correlates. Across all participants, increased perceptual sensitivity on neutral predominant blocks was associated with larger P300 amplitudes (Go: $r[144] = .166, p = .045$; No-Go: $r[144] = 0.188, p = .023$). When restricting the sample to BD and SZ participants only, the correlations between increased perceptual sensitivity on neutral predominant blocks and larger P300 amplitudes were no longer significant, although the effect sizes were similar in magnitude to those obtained with all participants (Go: $r[96] = .143, p = .16$; No-Go: $r[96] = .140, p = .17$).

Mood symptoms. Across the full sample, increased depressive symptoms (BDI-II) was associated with smaller P300 amplitudes (Go: $r[140] = -.325, p < .001$; No-Go: $r[140] = -.286, p < .001$). Increased manic symptoms (ASRM) was associated with smaller N200 amplitudes during No-Go trials ($r[116] = .246, p = .007$). When restricting the sample to BD and SZ groups

only, the correlation between greater depressive symptoms and smaller P300 amplitudes remained significant (Go: $r[92] = -.249, p = .016$; No-Go: $r[92] = -.259, p = .012$). Although the magnitude of the correlation between manic symptoms and N200 No-Go amplitude was similar to that obtained with all participants ($r[76] = .211$), it did not reach statistical significance in the patient-only subsample ($p = .064$).

3.6. Effects of medication on ERP amplitudes

Collapsed across BD and SZ groups, neither P300 amplitude nor N200 amplitude (averaged across Go and No-Go) differed by antipsychotic, lithium, or benzodiazepine use. Full medication analyses results are available in the **Supplemental Table 6**.

4. Discussion

The present study was the first to use a Go/No-Go task with emotional face stimuli to characterize alterations to behavioral and ERP correlates of response inhibition in BD and SZ. We aimed to inform whether alterations to indices of response inhibition in affective contexts are unique to BD or common across these related psychiatric illnesses. We found behavioral and ERP evidence of impaired discrimination of facial expressions in BD, which was similar in degree to SZ.

We observed greater behavioral difficulty discriminating emotional from neutral faces in both BD and SZ groups, relative to HCs, consistent with prior behavioral studies indicating difficulties with facial emotion recognition in BD and SZ.⁴² In the absence of any group differences in response bias, this suggests the difficulty participants with BD and SZ experienced on this task was in discriminating facial expressions. Given prior evidence that individuals with BD and SZ show the greatest emotion recognition impairment for neutral faces,⁴³ the current task

may have been particularly challenging by requiring discriminating a given emotion from neutral faces in each experimental block.

As was hypothesized, individuals with BD and SZ showed reduced overall P300 amplitude relative to HC. This is consistent with the bulk of prior findings of illness-related P300 reductions during auditory or visual oddball tasks involving distinguishing a rare from frequent stimulus^{17–19,23} and tasks involving response inhibition to simple non-emotional stimuli, such as letters,^{21,44} with a few exceptions (e.g. Morsel et al., 2018). This finding of illness-related P300 reductions aligns with our behavioral finding of reduced perceptual sensitivity, providing further support that BD and SZ patients had difficulty discriminating and categorizing emotional faces as cues to respond (Go) or withhold their response (No-Go). Exploratory correlational analyses also indicated that P300 amplitude increased with greater perceptual sensitivity (when neutral faces were the predominant stimuli, which was more difficult than when emotional faces were predominant). This suggests that neurophysiological activity as indexed by the P300 (even when based on trials in which the individual responds correctly) has relevance for behavioral discrimination difficulty.

As hypothesized, the degree of alteration on behavioral performance (d') and neurophysiological activity (P300) observed in individuals with BD was similar to that of SZ. Given prior findings of more severe facial emotion recognition impairment in SZ than BD,⁴³ one would expect greater perceptual sensitivity impairments in SZ than BD. However, the current task was more complex than simple emotion recognition and also required inhibitory processes. The present findings of similar behavioral and ERP impairments between the two groups aligns with prior behavioral studies showing comparable error rates on response inhibition tasks between BD and SZ groups.²⁵

Contrary to our expectation, we did not observe greater ERP alterations (reduced N200 or P300 amplitudes) in BD on No-Go than Go trials (to indicate more impairment during inhibition). This may be because Go/No-Go tasks require ongoing inhibition processes on both trial types, given the uncertainty of each upcoming trial and the need to withhold an immediate response while gathering perceptual information and determining the appropriate response.⁴⁶ Rather than indicating a No-Go specific impairment, our P300 results are in line with overall impairments in BD and SZ to cognitive processes that are engaged during both execution and withholding of responses while facing this ongoing uncertainty. Exploratory correlational analysis also supported that altered ERP amplitudes across both Go and No-Go trials (rather than just during No-Go trials) were related to prominent phenomena in BD and SZ (poorer perceptual sensitivity and increased depressive symptoms).

Although the bulk of behavioral studies of response inhibition in BD have focused on accuracy and error rates as outcomes, a few exceptions have analyzed signal detection metrics¹¹—which leverage both response execution accuracy and false alarm rate to separate response bias (the tendency to respond “Go” or withhold response) from perceptual sensitivity.¹² While our current perceptual sensitivity findings are in line with Gopin et al.’s (2011) report of reduced discrimination of affective word Go/No-Go stimuli in both BD and SZ groups; we did not find illness-related alterations in bias to respond to specific emotional stimuli, as Gopin et al. (2011) did. We expected poor inhibition of prepotent responses would be reflected in greater response tendency in BD. However, the difficulty patient participants experienced discriminating emotional from neutral faces in the current study may have limited their opportunity to learn the Go/No-Go ratio and establish the prepotent tendency of Go cues, relative to HCs. Though group differences did not reach statistical significance, the HC group showed numerically the strongest

response tendency, suggesting a stronger prepotency effect was established among HCs. This is in line with prior findings that individuals with SZ do not learn as strong of a prepotent response bias as HCs in Go/No-Go tasks.⁴⁷ Further research with higher Go to No-Go ratios (e.g., 4:1 ratio instead of the approximately 2:1 ratio in this study) and lower task demands (e.g., affective words or scenes instead of emotional faces) may be able to establish a stronger prepotency effect of Go trials to properly probe response bias and disinhibition in patient groups.

Extant literature has showed that tasks with a rare No-Go and frequent Go stimulus category elicit a larger N200 and P300 on the infrequent No-Go stimuli.¹⁶ Our N200 finding of a larger negative deflection in response to No-Go than Go stimuli was consistent with this literature—suggesting the current task successfully elicited some degree of conflict between Go and No-Go stimuli.¹⁴ However, our finding of larger P300 to Go than No-Go stimuli is in contradiction with these traditional findings. This may be due to the task design that consistently paired neutral faces with each emotional face (emotion Go with neutral No-Go or vice versa—neutral faces appeared in all experimental blocks. Despite being relatively infrequent within each emotion predominant block, neutral faces were the most frequent stimulus type over the course of the task (accounting for 50% of all stimuli presented). This might have weakened the prepotency effect of emotional faces and confounded the Go/No-Go effect. Additionally, the complex affective images (emotional faces) likely elicited a late positive potential, which is typically maximal over midline centroparietal areas and visible by 300 ms following emotional stimulus onset.⁴⁸ This likely overlaps with the P300 time window, which may have contributed to the unusual Go/No-Go finding. This unusual Go vs. No-Go P300 pattern was observed across all three groups, suggesting this was driven by experimental design factors rather than illness factors.

Contrary to our hypotheses of reduced conflict monitoring in BD (particularly during trials requiring inhibition) we did not find evidence supporting N200 ERP amplitude alterations in either illness. It is possible the inclusion of only trials with correct behavioral responses contributed to this null group difference, as incorrect No-Go trials (indicating failure to inhibit) may better capture the neural correlates of impaired cognitive control in BD. However, prior evidence for reduced N200 amplitudes in BD on correctly inhibited No-Go trials in a Cued Continuous Performance Test⁴⁴ and on successful Stop trials in a Stop Signal Task²² suggest that altered neural conflict monitoring occurs in BD *even during successful behavioral inhibition*, under particular methodological conditions. For example, the Stop Signal Task may more directly tap inhibition of an already prepared motor response, and thus have strengths for isolating cognitive control processes. Analytic approach may have also contributed to discrepant findings, as Michelini et al. (2016) analyzed the No-Go N200 alone (in contrast to our approach of testing the interaction between diagnostic group and Go/No-Go trial type, which can more rigorously test whether N200 deficits were specific to No-Go trials).

The inclusion of correct trials only for our ERP analyses is conventional and intends to exclude trials in which participants did not attend to the task. However, we cannot ascertain that all correct No-Go trials reflected successful inhibition, per se. Given the perceptual difficulties within the patient groups in the current study, it is possible that some “correct” No-Go trials resulted from failure to discriminate stimuli and thus not respond. Though examining neural activity indexing failures to inhibit would be of interest, these trials in our current data would be too few in number to compute reliable ERPs.

Despite group differences in N200 not reaching statistical significance, exploratory analyses indicated associations between reduced N200 on No-Go trials and higher manic

symptoms. Correlational results regarding both N200 and P300, taken together, suggest relevance of neural activity elicited by emotional faces (even when based on trials in which individuals manage to execute the “correct” behavioral response) for mood symptom severity. Given the exploratory nature of these findings, replication is needed to inform whether these ERP indices could serve as neural markers of mood symptoms and treatment response for BD and SZ.

4.1. Limitations

Findings of the present study should be interpreted in light of multiple limitations, in addition to those already discussed related to experimental task design. We cannot completely rule out medication effects on ERP amplitudes in the patient samples. However, we did not observe differences in ERP amplitudes according to whether participants in BD and SZ groups were taking antipsychotics, lithium, or benzodiazepines. Additionally, the lack of a consistent, comprehensive assessment battery (due to integrating data collected over multiple studies) limited our ability to explore other meaningful clinical or functional correlates of ERPs, and limited us from examining whether current results depend on mood state. Future research specifically examining participants in symptom remission and investigating effects of mood/psychosis symptoms on these ERP measures would be informative.

4.2. Conclusions

Reduced perceptual sensitivity and P300 amplitudes in individuals with BD, relative to HC participants, indicated impaired discrimination between emotional and neutral faces during the affective response inhibition task. These findings similarly extended to individuals with SZ, in that BD and SZ groups did not differ from each other on behavioral performance or ERP amplitudes. These impairments may contribute to difficulty responding appropriately in social

affective contexts and relate to mood symptoms, and therefore may be a treatment target in both illnesses.

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Data Availability Statement:

Research data are not shared because participants did not give prior permission to share their data.

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Table 1*Demographic and clinical characteristics of the sample*

	Healthy Control N = 48		Bipolar I Disorder N = 66		Schizophrenia Spectrum N = 32			
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>F</i> or χ^2	Tukey HSD
Demographic								
Age	40.48	14.25	41.50	11.23	42.31	12.79	0.21	
Gender (% F)	43.8%		51.5%		34.4%		2.62	
Race (% white)	73.2%		89.1%		82.4%			
Education (yrs)	16.00	2.57	15.34	2.30	14.33	1.92	4.95**	HC>SZ
Parental edu (yrs)	15.00	3.84	15.00	3.11	15.25	3.32	0.06	
WRAT-R	51.50	4.27	49.19	6.05	50.77	4.30	1.40	
Clinical								
BDI-II	1.08	1.53	10.94	8.93	8.94	7.21	29.10***	HC<BD=SZ
ASRM	1.85	2.66	3.69	4.05	5.31	4.66	5.29**	HC<BD=SZ
HAMD-17			9.32	8.60	-			
YMRS			4.94	6.29	3.31	2.14	0.83	
SAPS			-		5.34	3.16		
SANS			-		5.28	3.33		
Duration of Illness (yrs)	-		24.80	12.77	23.13	12.52	0.38	
History of psychosis (%)	-		54.0%		100%			
Antipsychotic (%)	-		39.4%		81.3%			
Lithium (%)	-		34.8%		3.1%			
Anticonvulsant (%)	-		45.5%		21.9%			
Antidepressant (%)	-		48.5%		43.8%			
Benzodiazepine (%)	-		21.2%		25.0%			

* $p < .05$ ** $p < .01$ *** $p < .001$

Note. ASRM=Altman Self Rating Mania Scale (possible scores range 0—20); BDI-II = Beck Depression Inventory (0—63); HAMD-17=Hamilton Depression Rating Scale (0—52); YMRS = Young Mania Rating Scale (0—60); SAPS = Scale for the Assessment of Positive Symptoms (0—25); SANS = Scale for the Assessment of Negative Symptoms (0—20). SAPS and SANS scores were computed by summing the 5 and 4 global item scores, respectively (SAPS: Hallucinations, Delusions, Bizarre Behavior, Formal Thought Disorder, Inappropriate Affect; SANS: Affective flattening, Alogia, Avolition-apathy, and Anhedonia-asociality). BDI-II was available for 48 HC, 63 BD, and 31 SZ; ASRM was available for 40 HC, 65 BD, and 13 SZ participants. HAMD-17 was available for 31 BD. YMRS was available for 33 BD and 13 SZ. Race available for 41 HC, 64 BD, and 17 SZ participants. Personal education available for 47 HC, 65 BD, and all 32 SZ participants. Parental education was available for 42 HC, 48 BD, and all 32 SZ participants. WRAT-R=Wide Range Achievement Test, reading subtest. WRAT-R was available for 20 HC, 31 BD, and 26 SZ participants.

Table 2*Descriptive statistics of behavioral performance and ERP amplitudes*

	HC (N=48)		BD (N=66)		SZ (N=32)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Behavior: Emotion Predominant Blocks</i>						
Hit Rate	0.926	0.060	0.903	0.081	0.885	0.072
False Alarm Rate	0.133	0.105	0.148	0.126	0.149	0.143
Sensitivity (d')	2.810	0.643	2.614	0.669	2.479	0.671
Response bias (β)	-0.152	0.388	-0.110	0.417	-0.050	0.390
<i>Behavior: Neutral Predominant Blocks</i>						
Hit Rate	0.817	0.179	0.763	0.200	0.791	0.192
False Alarm Rate	0.092	0.062	0.120	0.095	0.146	0.115
Sensitivity (d')	2.502	0.569	2.178	0.691	2.211	0.587
Response bias (β)	0.194	0.497	0.222	0.532	0.103	0.615
<i>Number of Correct Trials</i>						
Go Trials	90.313	12.654	82.864	15.472	83.906	13.412
No-Go Trials	80.729	10.063	79.470	13.510	77.031	14.261
<i>ERP Amplitudes (μV)</i>						
N200 Go	-2.633	3.190	-1.728	3.475	-1.702	3.809
N200 No-Go	-3.882	4.283	-2.453	4.501	-1.484	3.760
P300 Go	9.256	4.107	6.628	5.257	6.228	3.242
P300 No-Go	7.825	4.495	5.797	5.043	5.646	3.894

Note. Hit rate = proportion of correct Go trials; False Alarm rate = proportion of incorrect No-Go trials. Emotion predominant = experimental blocks with emotion Go cues and neutral No-Go cues; Neutral predominant = experimental blocks with neutral Go cues and emotion No-Go cues. Correct trial counts depict the number of trials averaged together to produce ERP waveforms. Correct Go trials were counted only when preceded by another Go trial. Across all participants, number of correct Go (preceded by Go) trials averaged together in ERP waveform ranged from 38—109; correct No-Go trials ranged from 44—108.

Table 3

Cohen's D effect sizes for pairwise comparisons on ERP amplitudes and behavioral performance metrics

	HC vs. BD	HC vs. SZ	BD vs. SZ
<i>Behavior: Emotion Predominant Blocks</i>			
Hit Rate	0.312	0.621	0.228
False Alarm Rate	-0.133	-0.137	-0.007
Sensitivity (d')	0.297	0.505	0.202
Response bias (β)	-0.105	-0.263	-0.146
<i>Behavior: Neutral Predominant Blocks</i>			
Hit Rate	0.283	0.141	-0.143
False Alarm Rate	-0.335	-0.631	-0.264
Sensitivity (d')	0.505	0.506	-0.050
Response bias (β)	-0.055	0.166	0.214
<i>ERP Amplitudes</i>			
N200 Go	-0.270	-0.270	-0.007
N200 No-Go	-0.324	-0.587	-0.227
P300 Go	0.547	0.800	0.085
P300 No-Go	0.421	0.511	0.032

Note. Reference group for each comparison is on the left in that column's header (e.g., for HC vs. BD, HC is reference group). Emotion predominant = experimental blocks with emotion Go cues and neutral No-Go cues; Neutral predominant = experimental blocks with neutral Go cues and emotion No-Go cues.

Figure 1.*Affective Go/No-Go task design.*

Note. A trial of the angry go/neutral no-go block is depicted as an example. Each task block consisted of one emotion face (happy, sad, or angry) as the target (Go stimulus) with neutral faces as No-Go stimuli or vice-versa. Participants were instructed to respond as quickly as possible to the target face (Go) and withhold the response to the distractor face (No-Go). Each block consisted of 34 Go (68%) stimuli and 16 No-Go (32%). The order of stimuli was pseudorandomized in order not to have two consecutive No-Go stimuli. The Go/No-Go pairs consisted of: happy go/neutral no-go, angry go/neutral no-go, sad go/neutral no-go, neutral go/happy no-go, neutral go/sad no-go, and neutral go/angry no-go. Each of these six pairs was repeated with the reverse order of stimulus presentation to yield 12 experimental conditions (600 trials) in total. This task lasted approximately 25 minutes to complete.

Figure 2.

Behavioral performance: signal detection measures across diagnostic groups and experimental block type

Note. Filled black circle indicate the mean; error bars indicate standard deviation of the mean. (A) depicts perceptual sensitivity [d']; (B) depicts response bias [β] across diagnostic groups and experimental block type

Figure 3.*ERP waveforms*

Note. ERP waveforms are depicted each for correct go trials (preceded by another Go trial) and correct No-Go trials for each diagnostic group, collapsed across the 3 emotional conditions (happy, sad, angry). **(A)** depicts ERP waveforms at the average of Fz, Cz, FC1, and FC2 electrode, with the N200 highlighted in gray. **(B)** depicts ERP waveforms at the Pz electrode, with the P300 highlighted in gray.