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# Attention orienting and inhibitory control across the different mood states in bipolar disorder: An emotional antisaccade task

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ABSTRACT

An antisaccade experiment, using happy, sad, and neutral faces, was conducted to examine the effect of mood-congruent information on inhibitory control (antisaccade task) and attentional orienting (prosaccade task) during the different episodes of bipolar disorder (BD) – manic ($n = 22$), depressive ($n = 25$), and euthymic ($n = 24$). A group of 28 healthy controls was also included. Results revealed that symptomatic patients committed more antisaccade errors than healthy individuals, especially with mood-congruent faces. The manic group committed more antisaccade errors in response to happy faces, while the depressed group tended to commit more antisaccade errors in response to sad faces. Additionally, antisaccade latencies were slower in BD patients than in healthy individuals, whereas prosaccade latencies were slower in symptomatic patients. Taken together, these findings revealed the following: (a) slow inhibitory control in BD patients, regardless of their episode (i.e., a trait), and (b) impaired inhibitory control restricted to symptomatic patients (i.e., a state)



## 1. Introduction

Bipolar disorder (BD) is characterized by succeeding episodes of mania, depression, and euthymia that entail impaired mood regulation even during asymptomatic periods (Goodwin & Jamison, 2007). BD patients exhibit forms of psychological vulnerability such as an impairment interaction between cognitive and emotional networks in the brain (Strakowski, DelBello, & Adler, 2005) that yield deficits in the processing of emotional information and executive functioning. To understand the psychological vulnerability of BD patients, it is crucial to examine in detail the difficulties in cognitive functioning that can result in emotion dysregulation. In the present paper, we examined the interplay between mood symptoms and cognition in BD by assessing the inhibitory control of attention along the different episodes of the disorder (i.e., mania, depression, and euthymia). We do so by presenting emotional information (i.e., happy, sad, neutral) to which participants have to respond (e.g., see García-Blanco, Perea, & Livianos, 2013, for recent behavioral evidence with emotion words in BD patients).

Impaired attention control is an important vulnerability factor for mental disorders, supporting the hypothesis that (abnormal) emotional attention brain processes cause considerable impairment during information processing (see Berggren & Derakshan, 2013). At the theoretical level, cognitive models propose that mood disorders are characterized by impairment in overriding dominant responses and inhibiting the processing of irrelevant information that attracts attention (see Beck, 1976). Indeed, a growing body of research has associated this dysfunction for inhibiting mood-congruent stimuli with the biased processing of new information (see Joormann, Yoon, & Zetsche, 2007). Importantly, negative biases in depression or positive biases in mania may evoke extreme emotional responses that require more effortful inhibitory control, and may represent an important component of emotion dysregulation in BD (see Phillips, Ladouceur, & Drevets, 2008).

An excellent strategy for assessing inhibitory attention control is the antisaccade task (Hallett, 1978). In each trial, while the participant is fixating on a central point, a sudden-onset peripheral visual stimulus appears either to the left or right of the central point. In separate experimental blocks, participants are required to make one of two eye movements: either an eye movement toward the stimulus (prosaccade) or an eye movement away from the stimulus (antisaccade) (see Mueller et al., 2010, for review). The prosaccade task requires participants to orient their attention toward the peripheral stimulus, while the antisaccade task requires participants to inhibit the automatic prosaccade toward a target and voluntarily generate an antisaccade to the mirror position. Thus, a prosaccade involves an automatic orientation response, whereas an antisaccade involves a controlled inhibition response. Importantly, the antisaccade task provides a precise assessment of top–down cognitive processes that influence attention allocation (e.g., beliefs, mood, etc.), which is particularly relevant in individuals with

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psychopathology (see Hutton & Ettinger, 2006, for a review) – note that prosaccades may be instead more influenced by bottom–up processes (i.e., stimulus-driven attention; see Egeth & Yantis, 1997). Many studies have used the antisaccade task in BD using neutral stimuli (e.g., a small white light) (see Gooding & Tallent, 2001; Gooding, Mohapatra, & Shea, 2004; Katsanis, Kortenkamp, Iacono, & Grove, 1997). However, the majority of these experiments focused on studying inhibitory control in schizophrenia. The BD group was included merely to examine whether the deficit in the antisaccade task was an inherent feature of schizophrenia or a shared feature with other mental disorders. These studies did not report affective symptoms in BD patients. Several studies reported that BD patients, regardless of whether they were recruited from outpatient or inpatient units, committed more antisaccade errors and were slower in correct antisaccades than healthy individuals. No differences were found across groups in the prosaccade task (e.g., see Katsanis et al., 1997, for in-patients; Gooding & Tallent, 2001, for outpatients). In addition, Gooding et al. (2004) evaluated antisaccade performance at two time points (with an average interval of 33 months) in BD patients. Unlike the healthy controls, BD patients showed temporal instability in accuracy and speed in the antisaccade task, thus suggesting that this deficit in inhibitory control may be a state rather than a trait marker of BD. However, the patients' affective symptoms were not indicated. We believe that potential confounds such as mixed-mood states or residual symptoms or heterogeneous criteria for saccade definition may have affected previous studies. In fairness to these studies, the focus was on schizophrenia, not on BD.

Of particular interest here is that the antisaccade task can be modified by the substitution of the neutral peripheral target with an emotional stimulus (e.g., a sad or a happy face) (see Derakshan, Salt, & Koster, 2009, for evidence with dysphoric individuals; see also Hardin et al., 2009, for evidence with anxious adolescents). Specifically, Derakshan et al. (2009) used facial expression (angry, happy, and neutral) in anti- and prosaccade tasks in order to examine the effects of subclinical depression (dysphoria) on attentional processing. Participants had to look toward the face (prosaccade task) or look away from the face (antisaccade task). Dysphoric individuals committed more antisaccade errors in response to emotional faces than to neutral faces (18.3% vs. 12.3%, respectively), while this effect did not occur in healthy controls (11.0% vs. 10.6%). No differences were found in the latency data for any of the groups. Derakshan et al. concluded that there is impaired attentional control in response to emotional faces in dysphoria.

We believe that it is important to examine the performance in the antisaccade task in BD patients during their distinct moods (mania, euthymia, and depression) when the peripheral stimuli are emotional images (facial expressions: neutral, happy, and sad). This manipulation allows us to examine the effects of mood on emotional information processing. In this respect, the mood-congruency hypothesis (see Bower, 1981) postulates that positive moods should facilitate orienting toward positive stimuli and hinder their inhibition, and negative moods should facilitate orienting toward negative stimuli and hinder their inhibition (see García-Blanco et al., 2013, for behavioral evidence of a mood-congruency effect with emotional words in BD patients). In addition, the present manipulation also sheds light on the question of neuropathological specificity being a state (e.g., as revealed by differences between symptomatic vs. asymptomatic BD patients) or a trait (e.g., as revealed by differences between BD patients vs. controls).

To our knowledge, the present (emotion-modified) antisaccade experiment is the first that examines the effect of mood-congruent information on inhibitory control (antisaccade task) and attention orienting (prosaccade task) among the different episodes in BD. The present experiment had two specific goals. The first goal was to assess the presence of mood-congruent biases on two different attentional processes (orientation [prosaccades] vs. inhibition [antisaccades]) in BD. If antisaccades reflect voluntary responses subject to inhibitory control that are influenced by top–down processes such as the participants' mood state (see Hutton & Ettinger, 2006), we would expect a mood-congruent effect in symptomatic patients (i.e., *antisaccade errors* should be particularly pronounced in response to happy faces for manic patients or to sad faces for depressed patients). In addition, if prosaccades reflect an automatic orientation response that is mainly influenced by bottom-up processes (see Egeth & Yantis, 1997), we expect that the latencies/errors on prosaccades would be modulated by the stimulus valence (i.e., stimulus-driven attention) rather than by the participants' mood state. The second goal was to examine whether difficulties in inhibitory control in BD patients are a trait (i.e., BD patients, regardless of their episode, should show general impaired inhibitory control reflected as slow [and error-prone] antisaccades relative to healthy individuals; see Gooding & Tallent, 2001; Katsanis et al., 1997) or a state (i.e., BD patients in depressive and manic episodes should commit more antisaccade errors and have slower antisaccades than healthy controls, while there would be differences between asymptomatic patients and healthy individuals; see Gooding et al., 2004).

## 2. Method

### 2.1. Participants

The participants were 71 BD patients from the Psychiatry Department (42 from in-patient wards and 29 from the outpatient Bipolar Disorders Unit) at the Hospital Universitario y Politécnico La Fe (Valencia, Spain) and 28 healthy individuals recruited through advertising in the community. Patients fulfilled the DSM-IV-TR criteria for BD and were included in the manic ($n = 22$), depressed ($n = 25$), or euthymic ($n = 24$) group at the time of assessment. Four patients in manic episodes refused to cooperate. This study was authorized by the ethics committee at the Health Research Institute La Fe. Demographic and clinical details are presented in Table 1.

No participant reported neurological history, major medical disorders, use of nonpsychotropic medication that could influence cognition (e.g., treatment with corticosteroids), or difficulty in obtaining stable eye tracking (e.g., eye diseases, interference from glasses, or frequent crying). No healthy control reported any kind of psychiatric history. Additional exclusion criteria for patients were (a) other psychiatric diagnoses based on DSM-IV criteria (American Psychiatric Association [APA], 2000) and (b) having received electroconvulsive therapy within the previous 3 months.

All patients were referred by psychiatrists in the department. DSM-IV-TR diagnoses were established with a clinical interview and case note review. Every patient had to present at least one manic episode. The responsible psychiatrist of the unit and a postgraduate clinical psychology intern corroborated the diagnosis. The Beck depression inventory-II (BDI-II; Beck, Steer, & Brown, 1996) and Young mania rating scale (YMRS; Young, Biggs, Ziegler, & Meyer, 1978) were used to exclude mixed states as well as the absence of affective symptoms in euthymic patients and healthy participants (BDI-II scores < 9, except in the depressed group > 18; YMRS scores < 6, except in the manic group > 20). Additionally, every participant filled out (a) the Beck anxiety inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) to measure anxiety and (b) the social adaptation self-evaluation scale (SASS; Bosc, Dubini, & Polin, 1997) to measure social functioning. Eighteen of the 117 participants in the original sample (89 patients, 28 healthy controls) were excluded based on these criteria, resulting in a final sample of 99 participants.

### 2.2. Eye-tracking paradigm

The stimuli were 90 faces (half female) depicting sad, happy, and neutral facial expressions (30 of each valence) taken from FACES (Ebner, Riediger, & Lindenberger, 2010). Nonfacial features were removed, and the faces were resized to 50 mm × 77 mm. The experiment entailed two blocks (prosaccade, antisaccade) each comprising 60 trials, totaling 120 trials. Each block of 60 trials included 20 sad, 20 happy, and 20 neutral trials. The order of the antisaccade and prosaccade tasks was counterbalanced. The intertrial interval was 600 ms. Each trial began with a central fixation point (12 mm × 12 mm) for 1600 ms. A face then appeared for 1600 ms with equal probability to the left or right side of the screen at 13.1° away from the fixation point. The number of trials and the stimulus presentation time were chosen in order to adapt the task to the characteristics of our sample – pilot testing showed that symptomatic patients had difficulties completing longer versions of the experiment and when the stimulus presentations were shorter. Participants looked at the fixation point. As soon as the face appeared, the participants had to direct their

**Table 1**
Demographic and clinical data from control group, depressed, euthymic and manic patients. Data shown are averages and standard deviations.

| | Control ($N$ = 28) | Euthymic ($N$ = 24) | Depressed ($N$ = 22) | Manic ($N$ = 25) | $p$ |
|---|---|---|---|---|---|
| % Female | 46.4 | 37.5 | 45.5 | 44.0 | .92 |
| Age | 42.1 (12.4) | 40.6 (11.4) | 49.1 (10.7) | 42.5 (11.4) | .06 |
| SASS | 43.8 (6.0) | 40.1 (5.3) | 40.8 (6.8) | 39.5 (6.2) | .07 |
| # Of episodes | – | 5.9 (5.4) | 7.7 (4.8) | 6.8 (5.7) | .53 |
| BAI | 11.2 (6.9) | 5.8 (3.6) | 23.9 (8.3) | 10.6 (6.0) | .00 |
| BDI | 6.4 (6.3) | 3.0 (3.5) | 25.7 (7.2) | 4.9 (3.4) | .00 |
| YMRS | – | 1.1 (2.2) | 1.9 (2.6) | 24.4 (5.6) | .00 |
| Medication (% of patients) | | | | | |
| Lithium (%) | – | 87.5 | 63.3 | 76.0 | .17 |
| Antiepileptic (%) | – | 45.8 | 72.7 | 40.0 | .06 |
| Antipsychotic (%) | – | 37.5 | 54.5 | 100.0 | .00 |
| Antidepressive (%) | – | 8.3 | 59.1 | 0.0 | .00 |
| Anxiolytic (%) | – | 41.7 | 81.8 | 92.0 | .00 |

*Note:* the $p$ values correspond to the omnibus test for all groups.

gaze as quickly as possible away from the face to its mirror position on the screen – antisaccade block – or toward the face – prosaccade block (see Fig. 1).

### 2.3. Apparatus

Eye movements were tracked using a remote eye-tracking system (SMI RED250). The system allows the participant free head movements across a wide range. The gaze-point position was estimated at 250 Hz.

### 2.4. Procedure

After signing an informed consent form, all participants responded to a demographic interview and the SSAS, BAI, and BDI-II rating scales. Additionally, patients completed a clinical interview and the YMRS. In the second session, participants completed the experiment individually in a dimly lit room. They were seated approximately 60 cm in front of the monitor in a height-adjustable chair. The experimental session began once the eye-tracker was successfully calibrated (i.e., average error was less than 1.5° of the visual angle for each calibration point). Nine practice trials and calibration of the eye-tracker proceeded each block. The experimenter was located in the room and monitored the stimulus presentation and eye tracking throughout each trial.

### 2.5. Data analyses

Similar to Mueller et al. (2010), the saccade threshold criterion was set at 30°/s, and anticipatory saccades with latencies less than 70 ms or late saccades more than 700 ms were discarded. Additionally, we considered other potentially relevant metrics to exclude eye movements unlikely to have been generated in response to the task. We removed those trials in which the initial fixation was not within 3.1° from the fixation point. In addition, short saccades of less than 6.3° in the $x$-axis were not included in the analysis. To assess the hypotheses, we computed several eye-movement measures: (a) *errors* in the prosaccade and antisaccade tasks and (b) *mean correct saccade latencies* in the prosaccade and antisaccade tasks. For each participant, the number of erroneous saccades on images with the same valence was summed up to generate the percentage of errors for each category. Correct saccade latencies on images with the same valence were averaged over the trials to generate the mean correct saccade latencies for each valence.

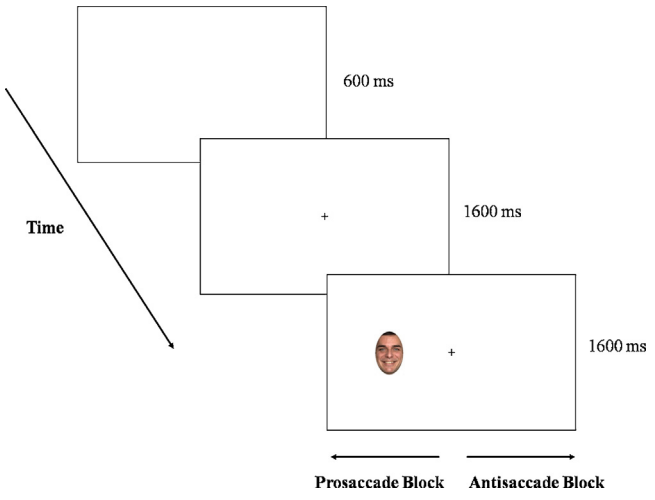


**Fig. 1.** Example of an antisaccade trial or a prosaccade trial.

## 3. Results

Each task was entered separately into a two-way repeated measures analysis of variance (ANOVA) with Group (control, euthymic, depressed, manic) as the between-subjects factor and Valence (neutral, happy, sad) as the within-subject factor. The dependent variables were the error rates and the correct saccade latencies in the prosaccade and antisaccade tasks. The averages and the standard deviations for each condition are presented in Fig. 2 and Table 2. When the effects were significant, we conducted a series of tests that controlled for type-I error: (a) Dunnett tests were used to compare each group of BD relative to the appropriate control group (i.e., between-group comparisons; see Miller, 1981). (b) Bonferroni post hoc tests were used to analyze the effect of Valence (i.e., within-group comparisons).

### 3.1. Errors in the antisaccade task

The ANOVA revealed a main effect of Group, $F(3,95) = 19.41$, $p < .001$, $\eta^2 = .38$. The Dunnett tests revealed that patients in manic and depressive episodes committed more antisaccade errors than healthy individuals (32.5% for depression and 47.7% for mania vs. 19.3% for the control group, $p = .012$ and $p < .001$, respectively), whereas there were no significant differences between the healthy individuals and the euthymic patients (13.3%, $p = .98$). Patients in manic and depressive episodes committed more errors than euthymic patients (all $ps < .001$). The main effect of Valence was not significant, $F(2,190) = 1.08$, $p = .34$. Importantly, there was a significant Valence × Group interaction, $F(6,190) = 3.47$, $p = .003$, $\eta^2 = .10$. This interaction revealed that, for the patients in a manic episode, the effect of valence was significant, $F(2,48) = 5.43$, $p = .007$, $\eta^2 = .19$.

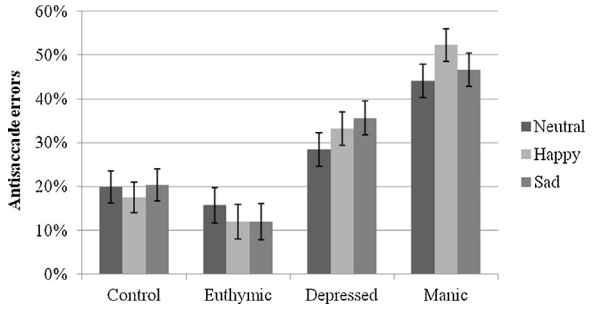


**Fig. 2.** Percentage of antisaccade errors for valence and group.

**Table 2**
Mean (standard deviation) for percentage of error in prosaccade task and mean response latency in anti and prosaccade task for each stimulus category for control (C), euthymic (E), depressed (D), and manic (M) groups.

| Stimulus category | Antisaccade task | | | | Prosaccade task | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Mean latency (ms) | | | | Number errors (% of total) | | | | Mean latency (ms) | | | |
| | C | E | D | M | C | E | D | M | C | E | D | M |
| Neutral | 366 (47) | 411 (59) | 470 (54) | 460 (66) | 2.1 (3.5) | 1.5 (3.7) | 2.7 (5.3) | 5.7 (6.8) | 284 (35) | 299 (42) | 332 (58) | 319 (37) |
| Happy | 370 (44) | 412 (55) | 471 (73) | 478 (65) | 1.6 (2.5) | 1.9 (3.7) | 7.3 (9.5) | 6.2 (6.9) | 286 (29) | 297 (37) | 327 (65) | 311 (43) |
| Sad | 385 (52) | 413 (57) | 465 (60) | 473 (58) | 1.6 (3.4) | 2.4 (3.8) | 5.1 (7.1) | 6.4 (7.9) | 294 (33) | 307 (41) | 339 (58) | 323 (37) |

The Bonferroni comparison tests revealed that these patients committed more antisaccade errors with happy faces than with neutral and sad faces ($p = .027$ and $p = .022$, respectively). There were no signs of a difference in the responses to neutral and sad faces ($p > .9$). Furthermore, for the patients in a depressive episode, Valence also had an effect, $F(2,42) = 3.36$, $p = .044$, $\eta^2 = .14$. The Bonferroni comparison tests revealed that these patients committed more antisaccade errors with sad faces than with neutral faces (the difference approached significance, $p = .070$). The other comparisons did not approach significance (all $ps > .34$). Finally, neither the control group nor the euthymic group revealed an effect of Valence ($p = .48$ and $p = .18$, respectively). Taken together, these data reflect a mood-congruency effect (see Fig. 2).

Additionally, post hoc correlational analyses were conducted in order to examine the relationship between the magnitude of affective symptomatology and antisaccade effect toward mood-congruent faces. The differences between sad–neutral and happy–neutral scores were calculated and then correlated with the BDI and YMRS scores, respectively, in BD patients. The Pearson coefficient was significant for sad–neutral scores and BDI scores ($r = .389$, $p = .001$). The Pearson coefficient was also significant for happy–neutral scores and YMRS scores ($r = .275$, $p = .020$). Thus, the larger the BDI/YMRS score, the larger the mood-congruency effect.

### 3.2. Latencies of correct responses in the antisaccade task

The ANOVA revealed only Group had a significant effect, $F(3,95) = 24.208$, $p < .001$, $\eta^2 = .43$. The Dunnett tests revealed that the antisaccade latencies of BD participants in depressive, manic, and euthymic states were higher than the antisaccade latencies in the healthy controls (all $ps < .009$). Neither the effect of Valence nor the interaction between the two factors approached significance (both $ps > .32$).

### 3.3. Errors in the prosaccade task

The ANOVA revealed Group had a main effect, $F(3,95) = 6.62$, $p < .001$, $\eta^2 = .44$. The Dunnett tests indicated that participants in depressive and manic episodes committed more prosaccade errors than the healthy controls ($p = .011$ and $p = .001$, respectively), but not the euthymic patients ($p = .71$). In addition, patients in depressive or manic episodes committed more prosaccade errors than euthymic patients ($p = .020$ and $p = .001$, respectively. Neither the effect of Valence nor the Group × Valence interaction was significant (both $ps > .14$).

### 3.4. Latencies of correct responses in the prosaccade task

The ANOVA revealed Valence had a significant main effect, $F(2,190) = 6.42$, $p = .002$, $\eta^2 = .06$, and Group, $F(3,95) = 5.76$, $p = .001$, $\eta^2 = .15$, while the Valence × Group interaction was not significant ($F < 1$). In the analysis of the effect of Valence, the Bonferroni tests revealed that the latency for sad faces was slower than for happy faces ($p = .001$), and for neutral faces although the difference did not reach the conventional criterion of significance ($p = .07$). No other significant differences were found ($ps > .83$). In the analysis of the effect of Group, the Dunnett tests revealed that the patients in depressive and manic episodes revealed higher latencies than the healthy controls ($p < .001$ and $p = .012$, respectively), but not the euthymic group ($p = .27$).

## 4. Discussion

To the best of our knowledge, this is the first eye-movement experiment that examined inhibitory control when emotional faces (happy, sad, neutral) are processed by BD patients during different episodes (i.e., manic, depressive, and euthymic). The main findings can be summarized as follows. First, patients in manic episodes committed more antisaccade errors in response to happy facial expressions than to sad/neutral facial expressions, while patients in depressive episodes tended to have more antisaccade errors with sad faces than with neutral faces. Indeed, the larger the BDI/YMRS score, the larger the mood-congruency effect on the number of antisaccade errors. Second, patients in the manic and depressed groups committed more antisaccade errors than the healthy controls. This difference was absent in the patients in euthymic episodes, thus suggesting that deficient inhibitory control in BD patients in an acute episode (mania and depression) is a state rather than a trait. Third, the antisaccade latencies were slower for BD patients (regardless of their mood) than for healthy individuals, and prosaccade latencies were slower for symptomatic patients (but not euthymic) than for healthy controls. This finding suggests that slow inhibitory control is a trait in BD. As we discuss, these findings allow characterizing how emotional faces are processed in BD patients.

First, the presence of a mood modulation on antisaccade error rates in BD patients depending on their episodes (mania and depression) offers empirical support to the mood-congruency hypothesis (Bower, 1981). Similarly to response time tasks with emotional words (García-Blanco et al., 2013), we found impairment in inhibiting the processing of irrelevant information that attracts attention. Patients with bipolar depression had problems inhibiting the impact of sad information, whereas patients in manic episodes had more difficulty inhibiting happy stimuli. This impaired inhibition for mood-congruent information could be involved in hindering the adjustment of emotional responses to changing situations (Beck, 1976) and in a ruminative cognitive style, which may play an important role in the maintenance of this mood disorder (see Joormann & Gotlib, 2008). The errors in attentional orienting (i.e., prosaccade errors) did not reveal any signs of a mood-congruent effect. The explanation for this dissociation is straightforward: although attentional orienting does not require voluntary control, top–down attentional control is needed to effectively inhibit reflexive prosaccades toward the emotional face in the antisaccade task (see Hutton & Ettinger, 2006). Thus, an influence of mood would be expected in a controlled rather than in an automatic task.

Second, only patients in manic or depressive episodes committed more antisaccade errors than healthy individuals, while euthymic patients had a similar performance as the healthy

controls. Thus, these differences reveal that impaired inhibitory control in bipolar patients may not be specific to BD, but instead may depend on the presence of affective symptomatology. The mood-dependent inhibitory control in BD could explain the temporal instability reported in previous antisaccade experiments that did not control the affective psychopathology (Gooding et al., 2004).

Third, the present antisaccade experiment revealed that the latencies were slower in all groups of patients, including when they were asymptomatic (i.e., euthymic) relative to healthy controls. Although the slow antisaccade latencies in BD patients could be due to anticipation for saccade execution given a fixed and predictable preparation interval (1600 ms), it is unlikely as other studies that employed a non-predictable preparation interval documented similar findings (e.g., Katsanis et al., 1997). Furthermore, this pattern of data is also consistent with response time experiments in BD that reported inhibitory control deficits across mood states, including euthymia (Ryan et al., 2012;). To obtain the whole picture, we should note that in the latencies of the prosaccade task, euthymic patients were not slower than controls. That is, slow latencies in BD (as a trait) do not occur in attentional orienting. This dissociation between orientation and inhibition is consistent with the view that slow inhibitory control is an inherent trait of BD (Gooding & Tallent, 2001; Katsanis et al., 1997). Thus, preattentive judgments of facial stimuli could impede and retard inhibitory control and influence the generation and regulation of affective responses (see Green, Cahill, & Malhi, 2007).

One final finding that deserves consideration is that, unlike antisaccade latencies, prosaccade latencies were influenced by the type of facial expression but not by the participants' features. The orientation of attention, as deduced from the prosaccade latencies, was faster toward happy faces than toward sad/neutral faces, regardless of group (i.e., for healthy controls and BD patients). This is consistent with previous studies that have reported that, during the automatic stages of information processing, happy faces capture attentional orienting more easily than other valences (see Calvo, Nummenmaa, & Hyönä, 2007, for evidence with healthy individuals; see Kellough, Beevers, Ellis, & Wells, 2008, for evidence with individuals with psychopathology). Therefore, differences between the antisaccade and prosaccade tasks in BD could be due to the differences in the automaticity of attentional processes. This would be consistent with Yiend (2010) claim that controlled cognitive processes such as attentional inhibition in antisaccade tasks are more influenced by the individuals' characteristics (e.g., their mood), whereas automatic cognitive processes as attentional orienting in prosaccade task are more influenced by the stimuli's characteristics (e.g., their valence).

The present eye-tracking experiment comes with certain limitations that are typical in studies with patients. At the time of testing, all patients in this study – including those in a euthymic state – were taking psychotropic medication (see Table 1). Regression analyses were conducted with patients on vs. off a particular type of medication and the antisaccade latencies to examine whether medication could cause patients' slow latencies relative to controls. Results revealed that anxiolytic dose significantly predicted the antisaccade latencies ($R^2 = .243$, $p < .001$). However, medication cannot explain differences between the euthymic group and the control group in the antisaccade latencies but not in the prosaccade latencies (only symptomatic patients were slower than healthy individuals). Although medication alone cannot explain the mood congruency effect in the number of errors in the antisaccade task, further research should focus on the effects of medication (and dose) on inhibition control in BD patients.

To conclude, the present emotional antisaccade experiment adds to previous studies on inhibitory control in BD: (a) the examination of the role of emotional stimuli (happy, sad, neutral) in inhibitory processes along the different episodes (mania, depression, euthymia) and (b) the examination of whether inhibitory control in BD, as deduced from the error and latency data in the antisaccade task, is a trait or a state. First, patients in manic and depressive episodes showed impairments in ignoring irrelevant information, predominantly with mood-congruent facial expressions. Second, the latencies in the antisaccade task were longer in BD patients (regardless of the presence of affective psychopathology) than in the healthy controls. This suggests the presence of slow inhibitory control in these patients (i.e., a trait); furthermore, the errors in the antisaccade task revealed a deficient inhibitory control only in symptomatic (manic, depressive) BD patients (i.e., as a state). In sum, determining the components of attentional bias in BD is not only crucial for advancing theoretical models, it is also essential for specifying treatment targets based on training attention. In this context, several recent studies found that lessening bias toward sad stimuli may relieve depression (Wells & Beevers, 2010). Future research should examine if attention modification procedures that improves the inhibitory deficits of negative and positive information may alleviate affective symptomatology in BD.

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