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Happy and less inhibited? Effects of positive mood on inhibitory control during an antisaccade task revealed using topographic evoked potential mapping

Naomi Vanlessen, Rudi De Raedt, Sven C. Mueller, Valentina Rossi & Gilles Pourtois

Psychopathology & Affective Neuroscience Laboratory, Department of Experimental, Clinical and Health Psychology, Ghent University, Belgium

Correspondence :

Naomi Vanlessen

Department of Experimental-Clinical and Health Psychology

Ghent University

Henri Dunantlaan 2

9000 Gent, Belgium

Phone: +32 9 264 6472

Email: Naomi.vanlessen@ugent.be

ABSTRACT

Affective states might influence inhibitory control, a cognitive process fundamental for goal adaptive behavior. Here, we recorded high-density EEG while participants performed an antisaccade task, after the induction of a happy (n=20) or neutral (n=20) mood, to compare the same inhibition-related processes across these two affective contexts. Topographical evoked potential mapping methods were used to characterize changes in the electric field depending on mood and saccade type (pro vs. anti) concurrently. Results showed that prior to target onset, the CNV component was enhanced for anti- compared to prosaccades, selectively in the neutral mood group. Following target onset, the topography of the N2 was more strongly expressed in the happy mood group, and was also altered by saccade type. The subsequent P3 components were not modulated by mood. We discuss these new findings in light of recent neurobiological and neuropsychological models that posit that positive affect dynamically changes cognitive control.

Keywords: inhibition; positive mood; ERP; CNV; N2; cognitive control

INTRODUCTION

By virtue of cognitive control abilities, humans can flexibly adapt their behavior to comply with changes in context or task demands. Cognitive control is best exemplified in situations in which reflexive actions have to be voluntarily suppressed and overwritten by other, less automatic, behaviors. Inhibition is usually defined as a central regulatory mechanism for preventing the processing of task-irrelevant information or execution of prepotent responses (Friedman & Miyake, 2004). Interestingly, our capacity to exert cognitive control in general (e.g., inhibition, conflict monitoring, and selective attention) fluctuates with changes in our internal state, such as variations in motivation (e.g., triggered by reward context; Padmala & Pessoa, 2010) and positive mood (Dreisbach & Goschke, 2004; Martin & Kerns, 2011).

Although reward and positive mood share overlapping neurobiological grounds (Funahashi, 2011; Kringelbach, 2010), they seem to have opposite effects on various measures of cognitive control, with performance-contingent reward enhancing, and positive mood diminishing control (see Chiew & Braver, 2011; Fröber & Dreisbach, 2014 for reviews). For example, positive affect has been found to reduce proactive control, as shown by a reduced ability to maintain task goals (Dreisbach, 2006) and a decreased use of predictive cues (Fröber & Dreisbach, 2012). At the electrophysiological level, Yuan et al. (2011) interpreted enhanced brain expressions of cognitive control during positive mood as a compensatory mechanism, in order to maintain behavioral performance at a similar level as when participants were in a negative or neutral mood. This paradoxical observation, with benefits from reward but reduced cognitive control during positive affect, can likely be attributed to fundamental differences between the two systems. For example, while reward is thought to be phasic, positive mood

might have more tonic effects on cognitive functions. Along the same lines, reward is usually bound to specific responses or stimuli, while positive mood is often manipulated independently from the task (i.e., contextual effect), hence not directly biasing motivation towards one specific stimulus type or condition.

Given these structural differences between reward and positive mood, a similar pattern of opposing influences of these two factors can be hypothesized for inhibitory control specifically. However, while several studies show that reward can enhance inhibitory control (e.g., Boehler, Hopf, Stoppel, & Krebs, 2012; Mueller, et al., 2010), it remains an open question how positive mood influences this process. Indirect evidence for an impairment of inhibitory control by positive mood comes from studies on the effects of mood on attentional processes (e.g. Moriya & Nittono, 2011; Vanlessen, Rossi, De Raedt, & Pourtois, 2013). In these studies, the authors pointed to *diminished* inhibitory control as a possible underlying mechanism explaining their findings in the attention domain (as suggested by Rowe, Hirsh, & Anderson, 2007). In addition, enhanced susceptibility for distraction was found in positive mood (Biss & Hasher, 2011; Biss, Hasher, & Thomas, 2010; Dreisbach, 2006; Dreisbach & Goschke, 2004; Rowe, et al., 2007), which was taken as an indication of disturbed prefrontal inhibitory control (Chao & Knight, 1997). However, these earlier studies on positive mood measured resistance to distraction, conflict monitoring or other facets of cognitive control, rather than inhibition of a prepotent response tendency *per se*.

A landmark paradigm to investigate inhibitory control is provided by the antisaccade task (Hallett, 1978). This task requires participants to execute a saccade towards (prosaccade) or away from (antisaccade) a unilateral visual target, appearing at a peripheral location along the

horizontal axis relative to fixation, following a cue that indicates if a pro- or an antisaccade is required in that trial. Antisaccade trials typically elicit more errors and slower latencies compared to prosaccades. This presumably reflects enhanced top-down control necessary to inhibit the execution of reflexive saccades towards the target and to generate an eye movement in the opposite direction (for a review, see Munoz & Everling, 2004), although prosaccades also require some form of cognitive control (Hutton, 2008). Studies specifically focusing on the influence of positive mood on inhibition are scarce. A previous study investigated the effect of positive mood on inhibition in an antisaccade task, and reported that participants made less errors in the positive compared to the neutral mood condition (Van der Stigchel, Imants, & Ridderinkhof, 2011). Surprisingly, this improvement was confined to "express saccades" that are fundamentally different from normal latency saccades. Express saccades are defined as reflexive saccades with very short latencies that occur in conditions in which the activity of fixation neurons in the superior colliculus (SC) decrease (Klein & Fischer, 2005; Klein, Rauh, & Biscaldi, 2010). This is further supported by different underlying neural networks, which attribute a strong role of the SC in express saccades, but more cortical involvement during regular saccades (see McDowell, Dyckman, Austin, & Clementz, 2008). As a result, performance of regular latency saccades are likely to be more informative about higher order cognitive functions, such as executive functions (Klein et al., 2010). Another study investigated the effects of imagining neutral, happy or pride-related events on performance in a inhibition task, in which participants had to indicate the direction of an arrow while distracting images were shown at the opposite side of the screen (Katzir, Eyal, Meiran, & Kessler, 2010). The authors found that imagining fun moments decreased inhibitory control. In a third study, Event-Related Potentials (ERPs) were

used to investigate the inhibition of prepotent responses in participants who performed a Go/Nogo task while emotional stimuli were shown in the background (Albert, López-Martín, & Carretié, 2010). Larger inhibition-related activation, at the level of the P300 component, was elicited when participants had to suppress a response while positive (as compared to negative) images were shown in the background, suggesting that a greater effort was necessary to withhold a prepotent response in this condition (i.e., enhanced distraction by positive content). However, none of these earlier studies actually manipulated the felt emotional experience of the participants, but rather aimed at activating short-term (fun) or long-term goals (pride; Katzir, et al., 2010), or installing a specific emotional context (Albert, et al., 2010). Mood on the other hand can be conceptualized as a general, long-lasting, mild affective state, that is not elicited by specific external emotional stimuli (Beedie, Terry, & Lane, 2005; Larsen, 2000). Hence, whether or not positive mood can influence inhibition remains an open question.

At the ERP level, antisaccades usually elicit an increased frontocentral negativity (N2), and a decreased later parietal positivity (P300), as compared to prosaccade trials. This "N2/P3 complex" has been proposed as a valid neurophysiological marker of inhibition in the antisaccade task (Mueller, Swainson, & Jackson, 2009). In addition, differential preparatory activity in this task is often captured by a negative wave ramping up prior to target onset (Klein, Heinks, Andresen, Berg, & Moritz, 2000; Reuter, Herzog, Endrass, & Kathmann, 2006; Richards, 2013; Klein et al., 2010), consistent with the Contingent Negative Variation (CNV; Walter, Cooper, Aldridge, McCallum, & Winter, 1964). This preparatory activity, typically triggered by a cue preceding the target of the saccade, is generally more negative in anti- compared to

prosaccades (Ansari & Derakshan, 2011; Klein, et al., 2000; Richards, 2003), in line with the assumption of a higher preparatory control in the former compared to the latter trials.

Current study

The main goal of this study was to assess whether positive mood could influence inhibitory processes, and if confirmed, to better characterize at which level during stimulus processing (either before or after target onset, or maybe both). To this aim, we recorded high-density EEG while healthy adults carried out a standard antisaccade task, after receiving either a positive or a neutral mood induction (between-subjects variable), previously validated in our laboratory (see also Bakic, Jepma, De Raedt, & Pourtois, 2014; Vanlessen, et al., 2013; Vanlessen, Rossi, De Raedt, & Pourtois, 2014). In light of the evidence reviewed above, we predicted that positive mood would be associated with a less efficient inhibition, compared to a control condition where neutral mood was used.

At the behavioral level, we expected that the classic decrease in performance (slower latencies and increased error rate) in anti- compared to prosaccades would be exacerbated in the positive relative to the neutral mood group. At the electrophysiological level, we expected that the topographical and ERP correlates of inhibition (i.e., at the level of the CNV and N2/P3 complex) would be less strongly expressed in the positive (relative to neutral) mood group. We used advanced topographical evoked potential mapping methods to characterize electrophysiological differences between the two groups during the generation of pro- and antisaccades. Such topographical analysis allows to differentiate between different brain topographies or "maps" (sometimes referred to as "microstates") over time, that reflect

activations of different neural networks and thus different cognitive or affective processes, taking the whole electrical field into account (see Murray, Brunet, & Michel, 2008; Pourtois, Delplanque, Michel, & Vuilleumier, 2008). Preceding target onset, we focused on the CNV. We hypothesized that antisaccades would elicit a larger CNV than prosaccades, and that preparation in the positive mood group would be less efficient, specifically for antisaccades (in line with Ansari & Derakshan, 2011; Klein, et al., 2000). Following target onset, we assessed whether positive mood would influence the N2, P3a and P3b, in such a way that these components would be expressed less strongly in the positive mood group, in addition to a modulation by saccade type.

METHODS

Participants

Forty-one undergraduate students at Ghent University participated in the study (age: *M* = 22; *SD* = 2; 7 male participants). All participants reported to be right-handed, to have normal or corrected-to-normal vision and no history of psychiatric or neurological disorders. Participants gave written informed consent prior to participation, and received 30 euros compensation. One participant from the positive mood group was excluded because reported levels of happiness decreased more than 50% after the positive mood induction, relative to the baseline. Hence, the statistical analyses were performed on 40 participants, 20 in each mood group. The study protocol was conducted in accordance with the Declaration of Helsinki and approved by the local ethics committee.

Mood Induction Procedure (MIP)

The MIP was identical to the one used in previous studies (see Bakic, et al., 2014; Vanlessen, et al., 2013, 2014). In this MIP, participants were first trained to vividly imagine to experience situations from their own perspective (i.e., field perspective; see Holmes, Coughtrey, & Connor, 2008; McIsaac & Eich, 2002). Next, they were instructed to recall a positive (in the positive mood group) or a neutral event from episodic memory and briefly report the episode[1]. Then participants were instructed to vividly imagine that they were reliving this experience for 30 seconds, followed by questions about their sensory experiences during visual imagery (i.e., what they can see, feel, hear, taste, smell), in order to facilitate imagining the situation from their own perspective (Watkins & Moberly, 2009; based on Holmes, et al., 2008). Next, participants continued imagining the same episode for another 30 seconds. Participants rated their mood on three Visual Analogue Scales (VAS; see Vanlessen, et al., 2013, 2014), the Positive Affect and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988) and the Self-Assessment Manikin (SAM; Bradley & Lang, 1994) for Arousal.

Stimuli and task

A white fixation cross (1.7° in diameter) was presented in the center of the screen and turned either green or red (i.e., cue), indicating whether a prosaccade or an antisaccade had to be executed, respectively. The target consisted of a white asterisk (2° in diameter) presented at 10.5 degrees of visual angle on the right or left side of the fixation cross, on the same horizontal

[1] Examples of episodic memories that could be used during the positive MIP are memories about passing exams, partying with friends or holidays; examples for the neutral MIP are events such as overhearing conversations in public transport, doing groceries and watching television.

axis. In a prosaccade trial, participants were instructed to perform a saccade towards the target as fast as possible. In an antisaccade trial, an eye movement was required towards the mirror position of the target along the same horizontal axis in the opposite visual field (see Fig. 1A). Participants were instructed to fixate the target position for the entire duration of the target presentation. All stimuli were presented against a uniform black background. The task was programmed using E-Prime Version 2 (Psychology Software Tools, Inc., 2001).

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Each experimental block consisted of 40 trials each, comprising an equal number of pro- and antisaccade trials in random order. Half of the targets appeared in the left visual field and the other half in the right visual field (relative to fixation) in random order. Each trial started with a white fixation cross presented for 500 ms, followed by either a green or a red fixation cross (for a jittered duration between 1000 and 1500 ms). This color cue indicated the specific eye movement (relative to the target) to be executed: prosaccade for green cue and antisaccade for red cue. Then, the unilateral target appeared for 1000 ms, with no concurrent fixation cross shown in the center of the screen. Next, the target disappeared and the white fixation cross was presented again for an average duration of 2100 ms (i.e., the interval varied randomly between 1000 and 3200 ms; see Fig. 1A). Hence, the duration of the trial varied from 3500 to 6200 ms.

Questionnaires

Participants completed the Resilience scale (Rs; Portzky, Wagnild, De Bacquer, & Audenaert, 2010), the BIS/BAS scales (Carver & White, 1994) and the Beck Depression Inventory (BDI; Beck, Steer, Ball, & Ranieri, 1996), in order to ascertain that the two groups did not differ on these measures, given that they relate in important ways to positive affect. More precisely, positive emotions are positively related to high resilience (Ong, Bergeman, Bisconti, & Wallace, 2006) and might be paired with a relative stronger activation of the approach system (Yan & Dillard, 2010), while high levels of depressive symptoms might block the possibility of a person to experience positive affect (Pizzagalli, 2014).

Procedure

After preparation for EEG recording, participants completed eight practice trials, followed by the first positive or neutral MIP. Next, participants completed six blocks of the task while they were seated 57 cm in front of a 19" CRT screen, with their head movements restrained by a chinrest and the center of the screen aligned to their eyes position. The MIP was shortly repeated every two blocks using the same episodic memory (see Fig. 1B), in order to maintain the desired mood throughout the experiment. Participants indicated their mood levels at the beginning of the experiment (baseline measure), after each MIP (repetition) and at the end of the experiment. After completion of the experimental task, participants filled out the three questionnaires.

Analysis of affective response

First, we averaged together the mood scores obtained after each MIP (i.e., the first MIP and the two repetitions), separately for the three VASs, the PA and NA scales of the PANAS and the SAM. Next, we compared these post-MIP average scores with the respective baseline measures by performing a mixed model ANOVA for each scale separately, with time (baseline vs. post-MIP) as within-subjects and mood (positive vs. neutral) as between-subjects factors. Independent samples t-tests were used to follow-up significant interactions, as well as to compare mean scores for the trait-related questionnaires between mood groups. Two participants assigned to the positive mood group did not complete all arousal SAMs and the questionnaire data from one participant from the neutral mood group were not saved properly.

Effect sizes were reported for all analyses. We calculated partial eta squared for the results of the ANOVAs and Cohens' *d* based on the observed means and standard deviations for both the independent and paired t-tests (see Lakens, 2013).

Acquisition and analysis of ocular data

We recorded the EEG continuously using a Biosemi Active Two System, from 128 Ag/AgCl electrodes distributed evenly over the scalp and attached to an elastic cap. The signal was referenced online to the CMS-DRL ground and digitized at 512 Hz. Both vertical and horizontal electro-oculograms (EOG) were recorded through bipolar electrodes placed above and below the left eye, and on the outer canthi of both eyes, respectively. Additionally, bipolar electrodes were placed on the right and left mastoids for offline referencing.

Using Brain Vision Analyzer 2.0 (Brain Products GmbH, Munich, Germany), the two horizontal EOG channels were subtracted from each other to create a single monopolar channel, showing positive deflections for eye movements to the right and negative deflections for eye movements to the left (see Fig. 1C). For each trial, the accuracy and latency of the saccade were calculated based on this monopolar channel (see Mueller, et al., 2009). Saccades were semi-automatically defined as a sharp deflection from baseline on the EOG channel with an amplitude exceeding ±70 microvolts. Correct and incorrect trials were identified based on the polarity of the first deflection following target onset, detected on this channel. Note that when erroneous eye movements were corrected afterwards (partial errors), these trials were considered incorrect (see Mueller, et al., 2009). The saccade response time was defined as the latency between target onset and saccade onset (semi-automatically defined as the moment when the EOG channel exceeded the ±70 microvolts criterion). Outliers were defined as latencies exceeding ±2,5 standard deviations from the mean of each individual and were excluded (separately for anti/pro and left/right).

We used a 2 x 2 mixed design with saccade (pro vs. anti) as within-subjects factor, and mood as between-subjects factor. Accuracy (in percentage) and response times were calculated for each mood group and each saccade type separately, and entered in mixed ANOVA's with saccade and mood as factors.

Acquisition and reduction of EEG data

Scalp EEG signals were referenced offline to the linked mastoids. We segmented the ocular and EEG data separately for components preceding (starting 700 ms prior to and ending at target

onset) and following target onset (from -300 ms prior to until 1000 ms following this event). A spherical splines procedure was used for interpolating noisy channels. The individual epochs were baseline-corrected, using the first 50 ms of the epochs for the pre-target ERP activity (from -700 to -650ms), and the entire pre-target interval for the post-target epochs. The standard Gratton algorithm was applied on the EEG data to automatically correct artifacts caused by vertical and horizontal eye movements (Gratton, Coles, & Donchin, 1983). Remaining artifacts were rejected semi-automatically using an absolute voltage criterion of ±100 μV. With this criterion, 86.58 % and 88.64 % of the epochs were included in the averages for the positive and the neutral mood group, respectively (with no significant group difference, $t(38) = 0.71$, $p = .48$). Individual averages were calculated separately for the pre- and post-target components, and separately for the pro- and antisaccade trials. Only correct trials were included in these averages, before grand average ERP waveforms were calculated.

ERP topographical mapping was performed with the Cartool software (brainmapping.unige.ch/cartool). A K-means spatial cluster analysis was used to identify the different dominant scalp topographies during a 600 ms time window starting at target onset, and encompassing therefore the N2 and P3 components, for the pro- and the antisaccades separately. This analysis showed the presence of three different topographical components, corresponding to the timing and scalp distributions characteristic of the N2, P3a and P3b. We isolated the dominant voltage maps for the N2 (140-160 ms post-target onset), the P3a (205-229 ms) and the P3b (234-547 ms post-target onset) components. The dominant topographical maps were determined objectively, using both cross-validation (Pascual-Marqui, Michel, & Lehmann, 1995) and Krzanowski-Lai criteria (Pascual-Marqui, et al., 1995; Tibshirani, Walther, &

Hastie, 2001). The following parameters were used during clustering: number of random trials: 100; smoothing strength (Besag factor) of 10; smoothing half window size of 3; merging of clusters correlating above 0.92; rejection of segments less or equal to 5 time-frames; no sequentializing. Next, these dominant topographies were fitted back to the individual ERP data using spatial fitting procedures in order to quantify their expression across subjects and conditions, for each component separately. We focused on variations in the global field power (GFP, see Lehmann & Skrandies, 1980) of these dominant topographies, depending on saccade type and mood group. GFP is a reference-free measure of the activation strength, taking all electrodes into account. For each of these components, we submitted the GFP values resulting from the fitting to a mixed model ANOVA with saccade as well as map configuration as within-subjects factors, and mood (positive vs. neutral) as between-subjects factor. Significant interaction effects were followed up by two-tailed t-tests. When normality assumption was violated, corrected p-values are reported.

The same procedure with the same parameters was applied to identify the dominant topographical maps during the 700 ms interval preceding target onset. Also the fitting procedure to extract the GFP values of the dominant map during the -200 to 0 ms interval preceding target onset (i.e., during the CNV) was identical to the post-target interval. However, given that the GFP did not yield a clear peak for the CNV, in line with the specific electrophysiological properties of this component characterized by a slow and steady negative amplitude increase following cue onset (Brunia & Damen, 1988; van Boxtel & Brunia, 1994), we also measured the mean amplitude of this negative ERP component. Based on the topographical properties of this component, we focused on six centro-parietal electrodes (A2/CCPz, A3/CPz,

A4/CPPz, A5/P1, A19/Pz and A32/P2), where the CNV reached its maximum amplitude just prior to target onset in our study (see Keil, et al., 2014). The CNV was defined as the mean ERP activity recorded at these six leads during the last 200 ms preceding target onset (Kropp, Kiewitt, Göbel, Vetter, & Gerber, 2000; Travis, Tecce, Arenander, & Wallace, 2002; Travis, Tecce, & Guttman, 2000). The mean amplitude values were then submitted to a mixed model ANOVA with saccade as within-subjects factor and mood as between-subjects factor.

RESULTS

Affective response results

The ANOVA carried out on the mean VAS scores showed a significant interaction effect between time and mood for happiness ($F(1,38) = 15.53$, $p = .001$; see Fig. 2A), and pleasantness ($F(1,38) = 15.83$, $p < .001$; see Fig. 2B), but not for sadness ($F(1,38) = 0.21$, $p = .65$). In addition, we also found a time and mood interaction for the PA scales ($F(1,38) = 10.15$, $p = .003$) and NA scales ($F(1,38) = 4.35$, $p = .044$) of the PANAS, as well as for the arousal scores ($F(1,36) = 8.19$, $p = .007$). The follow-up t-tests showed a significant difference between the positive and neutral mood group for both feelings of happiness and pleasantness, the PA, and arousal scores following the MIP, but not at baseline (see Table 1). These results confirmed that positive mood increased following the MIP in the positive mood group.

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Trait questionnaires

The scores obtained on the three questionnaires did not differ between the two groups for the BDI ($t(37)= 0.14$, $p = .89$), the BIS/BAS (BIS: $t(37)= 0.20$, $p = .85$, BAS Drive $t(37) = 0.78$, $p = .44$, BAS Fun Seeking: $t(37) = 0.46$, $p = .65$, BAS Reward Responsiveness: $t(37) = 0.12$, $p = .91$, $d = 0.04$), or the Rs ($t(37) = 0.47$, $p = .64$), suggesting that the two mood groups were matched along these traits.

Behavioral results

Participants had a high accuracy in the prosaccade condition ($M = 93.36$, $SD = 5.94$), while their performance decreased in the antisaccade condition ($M = 75.63$, $SD = 16.50$), as shown by a main effect of saccade ($F(1,38) = 61.78$, $p < .001$, 95% CI [13.23, 22.23], $\eta_p^2 = 0.62$; see Fig. 2C). There was no significant main effect of mood, nor a significant interaction effect between saccade and mood (all ps > .64). The same ANOVA on the latencies showed faster response times in prosaccade ($M = 202.38$, $SD = 25.27$) compared to antisaccade trials ($M = 280.60$, $SD = 41.84$, $F(1,38) = 271.20$, 95% CI [68.71, 87.72], $p < .001$, $\eta_p^2 = 0.88$; see Fig. 2D), while the main effect of mood and the interaction effect between saccade and mood were not significant (all ps > .32).

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Electrophysiological results

Pre-target activity

During the 700 ms window preceding target onset, the K-means spatial cluster analysis showed a solution with three dominant maps that explained 95% of the variance.

CNV. A single topographical map compatible with the electrophysiological properties of the CNV was present during the last 200 ms prior to target onset. The mixed model ANOVA showed a significant main effect of saccade ($F(1,38) = 21.55$, $p < .001$, 95% CI [.67 ,.26], $\eta_p^2 = 0.36$). The GFP was larger for anti- ($M = 2.60$, $SD = 0.72$) compared to prosaccades ($M = 2.13$, $SD = 0.52$). No significant interaction effect between saccade and mood was found ($F(1,38) = 0.55$, $p = .46$) for the GFP values, nor a main effect of mood ($F(1,38) = 0.10$, $p = .75$).

In addition, we also analyzed this CNV activity during this time window when measured at six representative scalp electrodes (see Methods section). The mixed model ANOVA performed on the mean CNV amplitude showed a significant main effect of saccade ($F(2,38) = 14.85$, $p < .001$, 95% CI [.60, 2.19], $\eta_p^2 = 0.28$). The amplitude of the CNV was larger for antisaccade ($M = -6.00$, $SD = 2.74$) compared to prosaccade trials ($M = -4.61$, $SD = 2.38$). Moreover, we found a significant interaction effect between saccade and mood ($F(2,38) = 7.95$, $p = .008$; see Fig. 3A and B), showing that the preparatory activity differed clearly between pro – and antisaccades in the neutral mood group (pro: $M = -4.05$, $SD = 2.25$, anti: $M = -6.46$, $SD = 2.87$; $t(19) = 3.85$, $p = .001$, 95% CI [1.10 ,3.73], $d = 0.94$), but not in the positive mood group (pro: $M = -5.17$, $SD = 2.42$, anti: $M = -5.54$, $SD = 2.61$; $t(19) = 1.03$, $p = .31$). In this latter group, the CNV amplitude was equally large for pro- and antisaccades.

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Post-target activity

The K-means spatial cluster analysis showed that a solution with six dominant maps during the 600 ms following target onset explained 92% of the variance.

N2. The topographic analysis showed two different map configurations during the N2 interval (see Fig. 3C for the ERP), one related to prosaccade trials ("prosaccade map") and the other one to antisaccade trials ("antisaccade map"; see Fig. 4). In line with these observations, the mixed model ANOVA revealed a significant two-way interaction between saccade and map configuration ($F(1,38) = 35.06$, $p < .001$), showing that one map was more expressed during prosaccades than antisaccades (prosaccade: $M = 1.41$; $SD = 1.08$; antisaccade: $M = 0.45$, $SD = 0.77$, $t(39) = 5.01$, $p < .001$, , 95% CI [.57 ,1.35], $d = 1.02$), while the other map showed the opposite effect (prosaccade: $M = 0.90$; $SD = 1.04$; antisaccade: $M = 1.74$, $SD = 1.02$, $t(39) = 5.28$, $p < .001$, 95% CI [1.16 ,.52], $d = 0.81$). Mood influenced the strength of the N2 topographies: there was a significant main effect of mood ($F(1,38) = 4.42$, $p = .042$, 95% CI [0.01 ,.54], $\eta_p^2 = 0.10$), showing a stronger GFP in the positive ($M = 1.26$, $SD = 0.49$) compared to the neutral mood group ($M = 0.99$, $SD = 0.32$), irrespective of saccade type.

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P3a. A main topographical map was found during the transition of the N2 to the P3 (see Fig. 3C for the ERP and Fig. 4 for the map). This P3a map was characterized by a positivity with a

fronto-central scalp distribution, and was stronger for prosaccades (M = 2.79, SD = 0.91) compared to antisaccades (M = 2.44, SD = 0.88, F(1,38) = 6.013, p = .019, 95% CI [.06 ,.64], η_p^2 = 0.14). At the level of the P3a, the interaction effect between saccade and mood was not significant (F(1,38) = 1.75, p = .19), nor was the main effect of mood (F(1,38) = 1.10, p = .30).

P3b. The topographic analysis showed that two distinct topographical maps were present during the P3b interval (see Fig. 3C for the ERP and Fig. 4 for the map). The mixed ANOVA performed on the mean GFP values showed a significant main effect of map (F(1,38) = 27.27, p < .001, 95% CI [.85 ,1.90], η_p^2 = 0.42), showing that one map (M = 2.38, SD = 1.36) was less strongly expressed than the other one (M = 3.75, SD = 0.82), regardless of saccade type (F(1,38) = 3.50, p = 0.07; see Fig. 4). Mood did not influence this pattern of results (all p > .24).

DISCUSSION

In this study, we used high-density EEG in a mixed antisaccade task to characterize how positive mood influenced the electrophysiological correlates of motor inhibition. As expected, at the behavioral level, results showed that participants were less accurate and slower for anti- compared to prosaccades. However, although previous studies suggested that positive mood could be associated with a decreased inhibitory control (Biss & Hasher, 2011; Biss, et al., 2010; Rowe, et al., 2007; Vanlessen, et al., 2013), our behavioral results do not lend support to this interpretation. The similar accuracy and latencies in the two groups suggest that positive mood did not interfere with inhibitory control. Nonetheless, at the electrophysiological level, inhibitory processes were reliably changed by positive mood, at two non-overlapping epochs around target onset. First, a main CNV component was apparent prior to target onset, followed

by a sequence of three ERP topographical activities (the N2, P3a and P3b components). Importantly, positive mood reduced the amplitude difference between pro- and antisaccades at the level of the CNV, while it increased target processing at the N2 level, regardless of the specific saccade type requested. We discuss the implications of these results in greater detail below.

Blunted preparatory inhibitory control in positive mood

Positive mood altered the sustained negative ERP activity arising during the interval between cue and target, sharing many electrophysiological similarities with the CNV (Brunia & Damen, 1988). Within the antisaccade paradigm, this CNV-compatible activity is thought to reflect proactive preparatory inhibition processes (Ansari & Derakshan, 2011) or, alternatively, working memory load (Klein, Rockstroh, Cohen, & Berg, 1996), anticipatory attention, or motor preparation (Brunia, Hackley, van Boxtel, Kotani, & Ohgami, 2011; Brunia & van Boxtel, 2001). At the topographical level, the CNV was stronger for anti- compared to prosaccades, indicating more cognitive control exerted in the former compared to the latter trials, likely reflecting the need to prevent the generation of a reflexive prosaccade towards the target in antisaccade trials (Aron, 2011). Although we did not find a differentiation between the two mood groups at the level of the GFP in the topographical analysis, the CNV measured at representative electrode positions showed a clear interaction effect between mood and saccade type. More specifically, we found that the amplitude of this preparatory component was enhanced in anti- compared to prosaccades in the neutral mood group, while both saccade types elicited equally strong preparatory activity in the positive mood group. Such a lack of differentiation between pro- and

antisaccades during the pre-target interval has previously been observed in populations thought to have deficient prefrontal control functions, including trait high anxious individuals (Ansari & Derakshan, 2011) and people with schizophrenia (Klein, et al., 2000), and it has been interpreted as a less efficient proactive preparation. Accordingly, positive mood seems to decrease this proactive control process associated with the implementation of motor inhibition during the antisaccade task.

Alternatively, one might argue that the CNV results for the positive mood group indicate an overall stronger (albeit undifferentiated) cognitive control, because the amplitudes for both pro- and antisaccades seemed higher compared to the CNV preceding prosaccades in the neutral mood group. To scrutinize the proactive control processes at work in our study in more detail, we performed an additional analysis to distinguish the CNV amplitudes in "switch" trials (i.e., prosaccade trials following antisaccade trials, and vice versa) and "repeat" trials (i.e., prosaccade trials following prosaccade trials, and antisaccade trials following antisaccade trials). Results of this auxiliary analysis showed that switch trials elicited stronger activation compared to repeat trials in the neutral mood group only (see Fig. 5A). In contrast, in the positive mood group, very similar CNV amplitudes were observed in all trial types, regardless of saccade type or switch/repeat factors, thus not showing a normal inter-trial adaptation in this group (see Fig. 5B; full analyses are available as supplementary material). Hence, two landmark correlates of efficient cognitive control at the CNV level were absent in this mood group. In sum, the CNV results obtained for both pro- and antisaccades and the additional analysis on repeat and switch trials unequivocally indicated that participants in the positive mood group showed an intermediate and undifferentiated cue-related preparatory activity prior to target onset,

regardless of what eye movement was required (antisaccade, prosaccade, switching or repeating). However, this intermediate preparatory activity probably did not reflect enhanced cognitive control *per se*. Rather, a stronger proactive control would result in an efficient distribution of resources depending on the information given by the cue, where resources are spared during the preparation of prosaccades and a stronger activation is present for antisaccades, as observed in the neutral mood group. Hence, proactive cognitive control processes were qualitatively changed in the positive compared to the neutral mood group. More precisely, in the positive mood group, the cue was probably used as a sign that "a" target was about to come and that they had therefore to prepare for "a" response, apparently without using the cue information to prepare efficiently for the exact eye movement required.

---------- insert Figure 5 about here ----------

Enhanced inhibitory control following target-presentation in positive mood

In addition to these proactive control processes, inhibitory control also includes post-target effects. We found a clear difference in the topography of the N2 for pro- compared to antisaccades, in line with the notion of non-overlapping brain networks for the generation of the two saccade types (McDowell, Dyckman, Austin, & Clementz, 2008). Importantly, the strength of these two topographies was modulated by mood. More precisely, the N2 topographical activity for both pro- and antisaccades was more strongly expressed in the positive compared to the neutral mood group. This result is intriguing, because it suggests that positive mood does not impair inhibitory control *per se*, as we hypothesized, but instead, it

might even sharpen it in certain conditions. One might intuitively expect that such mood-related effect at the level of the N2 component might be largest for antisaccade trials, as opposed to influencing the two saccade types equally. However, control is also needed to correctly execute prosaccades (Hutton, 2008), and this might be even more so in mixed blocks of pro- and antisaccade trials, as used in the current study. Moreover, given the absence of preparatory differentiation between pro- and antisaccades in the positive mood group at the level of the CNV, making the distinction between both saccade types is a necessary process to generate both correct antisaccades and prosaccades at a later stage. Hence, the lack of an interaction effect between saccade type and mood at the N2 level suggests that happy mood likely influenced cognitive control processes implicated in the pro- and antisaccade trials respectively, as opposed to altering conflict detection or resolution processes exclusively.

Interestingly, no further effect of positive mood was evidenced for the subsequent P3 components, suggesting a component-specific effect of positive mood on the earliest expression of post-target inhibitory processes (i.e., the N2 component). This dissociation between the N2 and P3 in terms of mood effects indicates that positive mood might influence inhibitory control via modulations within a specific neural pathway. More precisely, positive mood might stimulate pathways including the dorsolateral prefrontal cortex (activated during the N2 window in inhibition; see Lavric, Pizzagalli, & Forstmeier, 2004), and therefore still be related to the actual inhibition of the reflexive response (Jamadar, Fielding, & Egan, 2013; McDowell, et al., 2008). On the other hand, the P300 component might reflect processes that are related to the evaluation of the inhibitory process (Roche, Garavan, Foxe, & O'Mara, 2005), other post-perceptual response monitoring (Beste, Willemssen, Saft, & Falkenstein, 2010), or perhaps

decision-related processes already (Nieuwenhuis, Aston-Jones, & Cohen, 2005). Accordingly, our new ERP results showing component-specific effects stand in contrast to earlier models that posit that positive mood is processed in an undifferentiated manner in human prefrontal cortex (Ashby, Isen, & Turken, 1999), and point towards the need for more empirical research aimed at delineating the specific loci of mood effects on inhibitory control processes.

Does positive mood change pro- and reactive cognitive control rather than inhibition?

In light of these new results, it is tempting to hypothesize that positive mood changes cognitive control processes rather than inhibition per se. Consistent with this interpretation, we found that positive mood influenced the CNV (proactive control) and the N2 (reactive component) in opposite ways. In the neutral mood group, a stronger proactive effect was evidenced at the level of the CNV, while reactive inhibition was less expressed (N2). By comparison, in the positive mood group, the differentiation between the two saccade types was less clear during the preparation phase, while control was enhanced later, at the level of the N2. To assess if there was a direct linear negative relation between the CNV and the N2 values, we performed a correlation analysis between the difference amplitudes (antisaccades minus prosaccades) for the CNV and the N2 activation of the prosaccade trials (prosaccade map only) and the antisaccade trials (antisaccade map only). However, we did not find evidence for a direct tradeoff between these two processes in our study (prosaccades: r = -0.15, p = .35; antisaccades: r = 0.15, p = .35). This observation is in line with the idea that successful control usually results from a mixture of proactive and reactive mechanisms, that might be at least partly independent, depending on the specific task context and (trait or state) characteristics of

the individual (Braver, 2012). In a previous ERP study using a continuous performance task, positive mood influenced reactive control selectively (indexed by the N2 following targets), but not proactive control (reflected by the P3 and the CNV following cues; van Wouwe, Band, & Ridderinkhof, 2011). Our new ERP results add to these earlier findings by showing that positive mood can trigger a shift from pro- to reactive control relative to neutral mood when inhibition is required, instead of just dampening one of these mechanisms.

Noteworthy, such hypothesized changed balance between pro- and reactive control in positive compared to neutral mood might also explain why we did not find a significant group difference at the behavioral level (i.e., lower performance for positive mood). However, caution is needed with this interpretation, as the lack of mood effect on behavior might be attributed to specific methodological factors. For example, accuracy was relatively high, and therefore we cannot exclude that tasks requiring more inhibitory control (e.g., antisaccade tasks with a waiting period between the offset of the cue and the onset of the target, i.e. a "gap", see Fischer & Weber, 1997) might reveal mood-related effects at the behavioral level as well, in addition to changes in the strength of specific ERP (topographical) components. On the other hand, the absence of a change in behavioral performance by positive mood in our study cannot be attributed to an ineffective MIP. Both the VAS and PANAS scores clearly showed higher levels of positive affect after the MIP in the positive relative to the neutral mood group, either due to an increase after the positive MIP (in the VAS), or a decrease after the neutral one (in the PA of the PANAS).

In earlier studies, changes in dopamine (DA) activation after the induction of positive mood, modulating phasic DA increases either in the prefrontal cortex (Dreisbach & Goschke,

2004) or the striatum (van Wouwe, et al., 2011), were proposed to explain positive mood effects on cognitive control. Similarly, the ERP results of our study, showing a modulation of the CNV and the N2 concurrently, might be explained by changes in DA activation in these brain structures, in line with the tenets of an older neuro-anatomical model accounting for effects of positive affect on prefrontal-based cognition (Ashby, et al., 1999) and the assumption that pro- and reactive control might arise from non-overlapping networks (see Braver, 2012). However, additional pharmacological and neurophysiological studies are necessary to corroborate the dependence of amplitude variations at the level of the CNV and N2 components on phasic vs. tonic dopaminergic activation, and their association with different types of cognitive control, as well as their sensitivity to transient changes in positive affect (see also Dreisbach & Goschke, 2004).

Conclusions

Altogether, these new findings show that positive mood does not simply modify inhibition, as measured using a standard antisaccade task. Instead, it is associated with subtle changes in the way pro- and reactive control mechanisms dynamically come into play during the preparation and execution of saccadic eye movements. To investigate this, we first delineated the actual sequence of electrophysiological effects associated with the performance of pro- and antisaccades. We found differences in activation between pro- and antisaccades at the level of the CNV in anticipation of target-onset, as well as at the level of the N2 and P3a following target-onset. Interestingly, positive mood influenced the CNV and N2 components in opposite directions, while leaving the P3 components unaffected. Relative to neutral mood, positive

mood blurred the differentiation between pro- and antisaccades during the preparation phase, at the level of the CNV. Following target onset, positive mood enhanced inhibitory control at the level of the N2. These new findings contradict the assumption that this mood state alters motor inhibition *per se*, but instead they suggest that positive mood alters the way proactive and reactive control components are engaged to solve the challenge posed by antisaccadic eye movements. We reckon that these effects could arise due to changes in DA-dependent neural pathways, connecting midbrain structures to the prefrontal cortex. Because these neurophysiological effects depending on positive mood were evidenced in the absence of clear change in the (inhibition of) behavior, future studies are needed to assess whether some of these effects might reflect an active compensatory process or not.

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TABLES

Table 1. Results of the MIP. Means (Standard Deviation) and results of the group comparison (based on independent-samples t-tests) between the happy and neutral mood groups (df=38). ** indicates $p \leq .01$

FIGURES

Figure 1. A) Illustration of the trial sequence for prosaccades (left panel) and antisaccades (right panel). The color of the fixation cross (cue) indicated the trial type (green for prosaccades; red for antisaccades). The arrow indicates the correct direction of the saccade, but was not shown in the actual experiment. B) Procedure. C) EOG for correct prosaccades (full lines) and antisaccades (dotted lines) to the right (positive) and left (negative) hemifield.

Figure 2. VAS scores for A) feelings of happiness and B) pleasantness showed a selective increase in levels of positive mood in the positive but not the neutral mood group. Behavioral performance showed C) decreased accuracy and D) increased latencies in anti- compared to prosaccades, equally so in both mood groups (** indicates $p \leq .01$).

Figure 3. A) Grand average ERPs for the preparatory activity preceding target, recorded at a representative fronto-central midline electrode position (A2) for prosaccades (full lines) and antisaccades (dotted lines) with B) the corresponding voltage maps (top view) for the positive (red frame) and neutral (blue frame) mood group, separately. Negative values are plotted upwards; the gray frame indicates the window used for ERP data analyses ("late" CNV component). C) Grand average ERPs following target for pro- and antisaccades separately (collapsed across groups) at a representative fronto-central midline electrode position (C22/FFCz) to highlight the N2 (left panel) and at a representative central electrode position (A1/Cz) for the subsequent P3 (right panel). Note that the zero on the x-axis indicates target onset.

Figure 4. Results of the spatiotemporal cluster analysis (from stimulus onset until 600 ms after stimulus onset) for pro- and antisaccades, and for the positive and the neutral mood group separately. Three main ERP activities were found, namely the N2, P3a and P3b. At the level of the N2, different maps were found for pro- compared to antisaccades. During the P3a window, one dominant map was present, while two maps were found during the P3b. Occipital views of the five dominant maps are displayed. Note that the amplitude differences for these maps were normalized (i.e. the amplitude value at each electrode was divided by the GFP).

Figure 5. Results of the switch/repeat analysis for the A) neutral mood group and the B) positive mood group. CNV amplitudes are shown at a representative electrode (A2) for prosaccades following prosaccades ("Pro repeat"; full black line) and following antisaccades ("Pro switch"; dotted black line), and for antisaccades following antisaccades ("Anti repeat"; full gray line) and following prosaccades ("Anti switch"; dotted gray line).

Figure 1

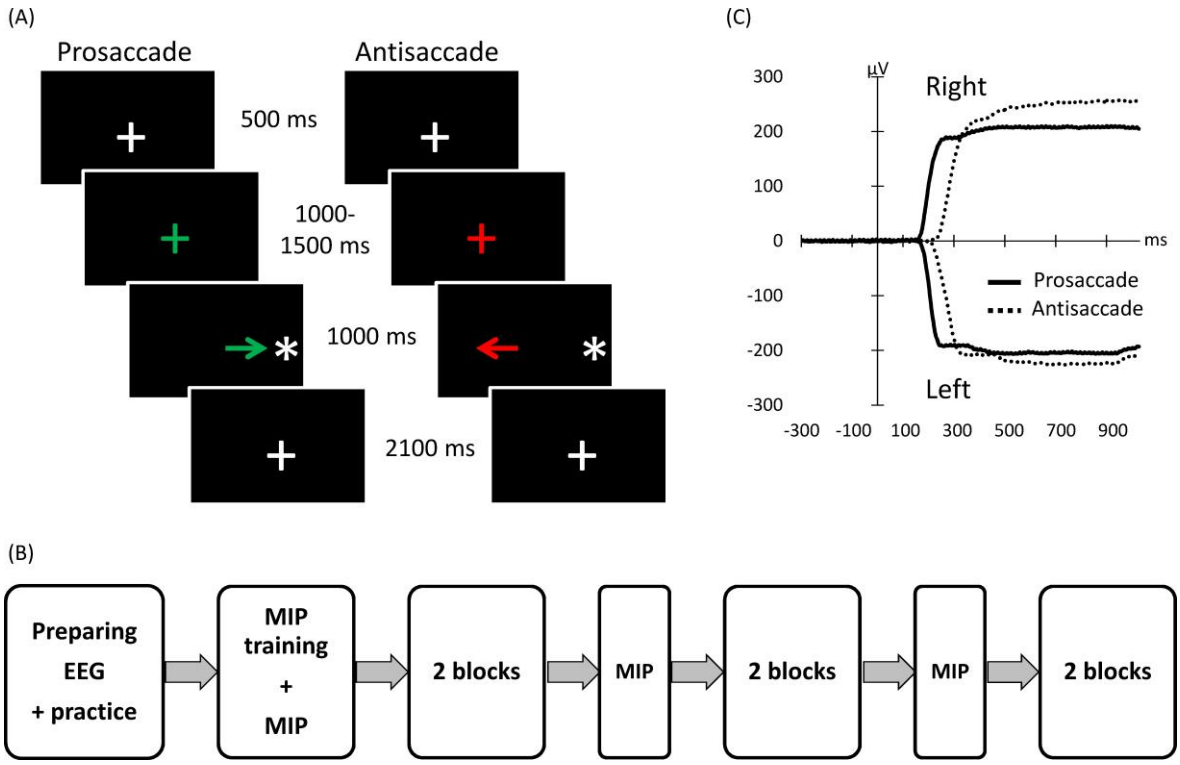


Figure 2

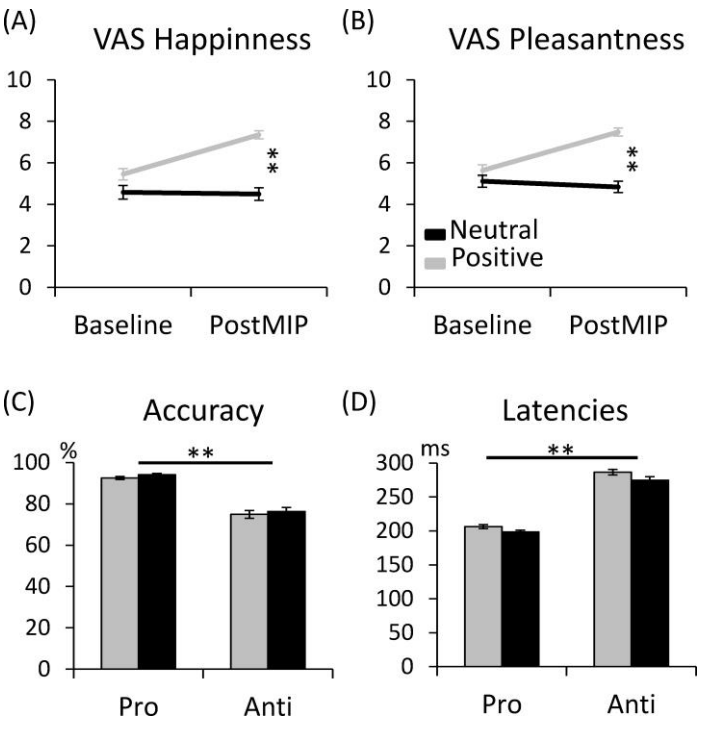


Figure 3

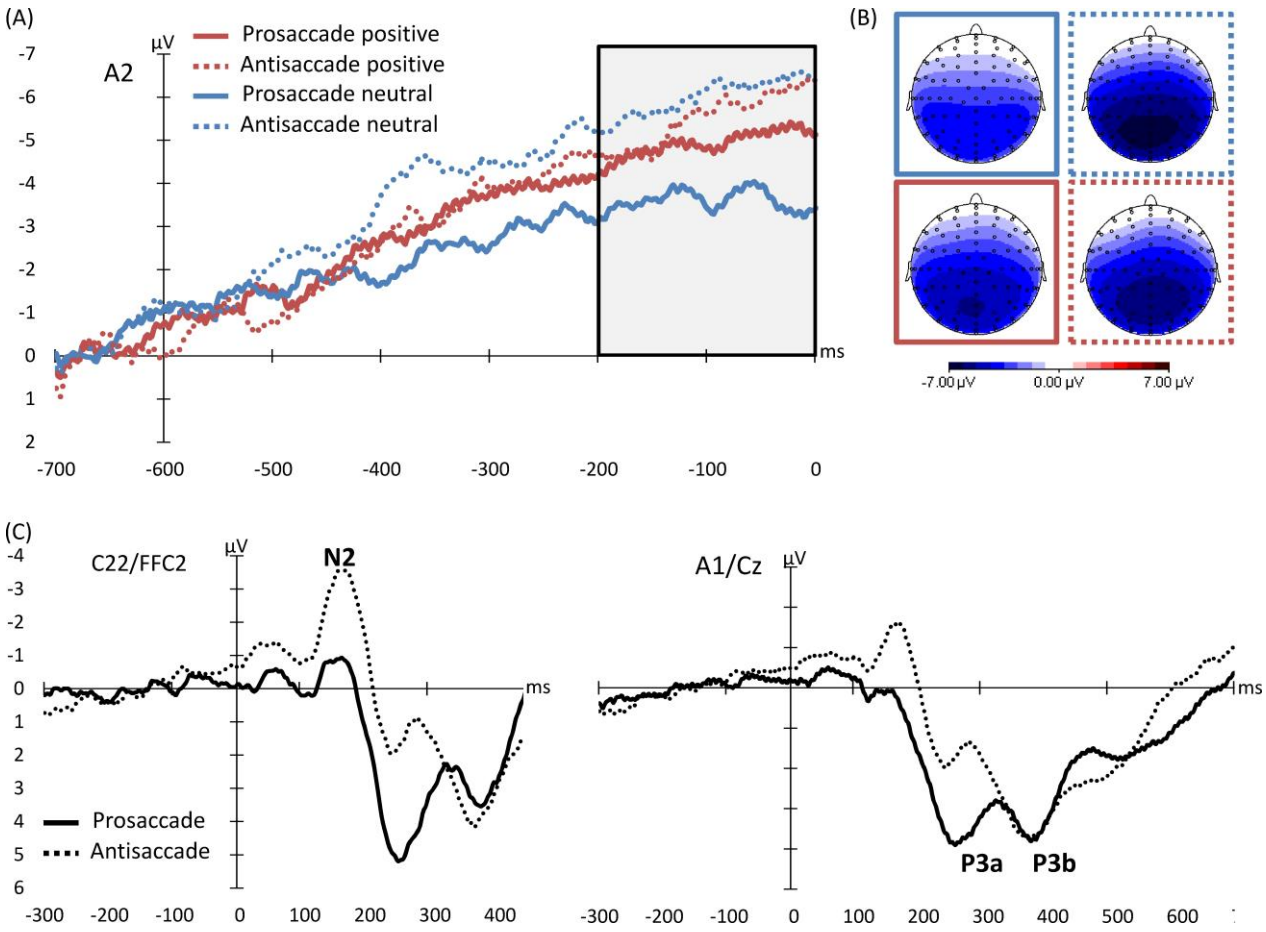

(A)
A2
µV
Prosaccade positive
Antisaccade positive
Prosaccade neutral
Antisaccade neutral
ms
(B)
-7.00 µV
0.00 µV
7.00 µV
(C)
C22/FFC2
N2
Prosaccade
Antisaccade
A1/Cz
P3a
P3b

Figure 4

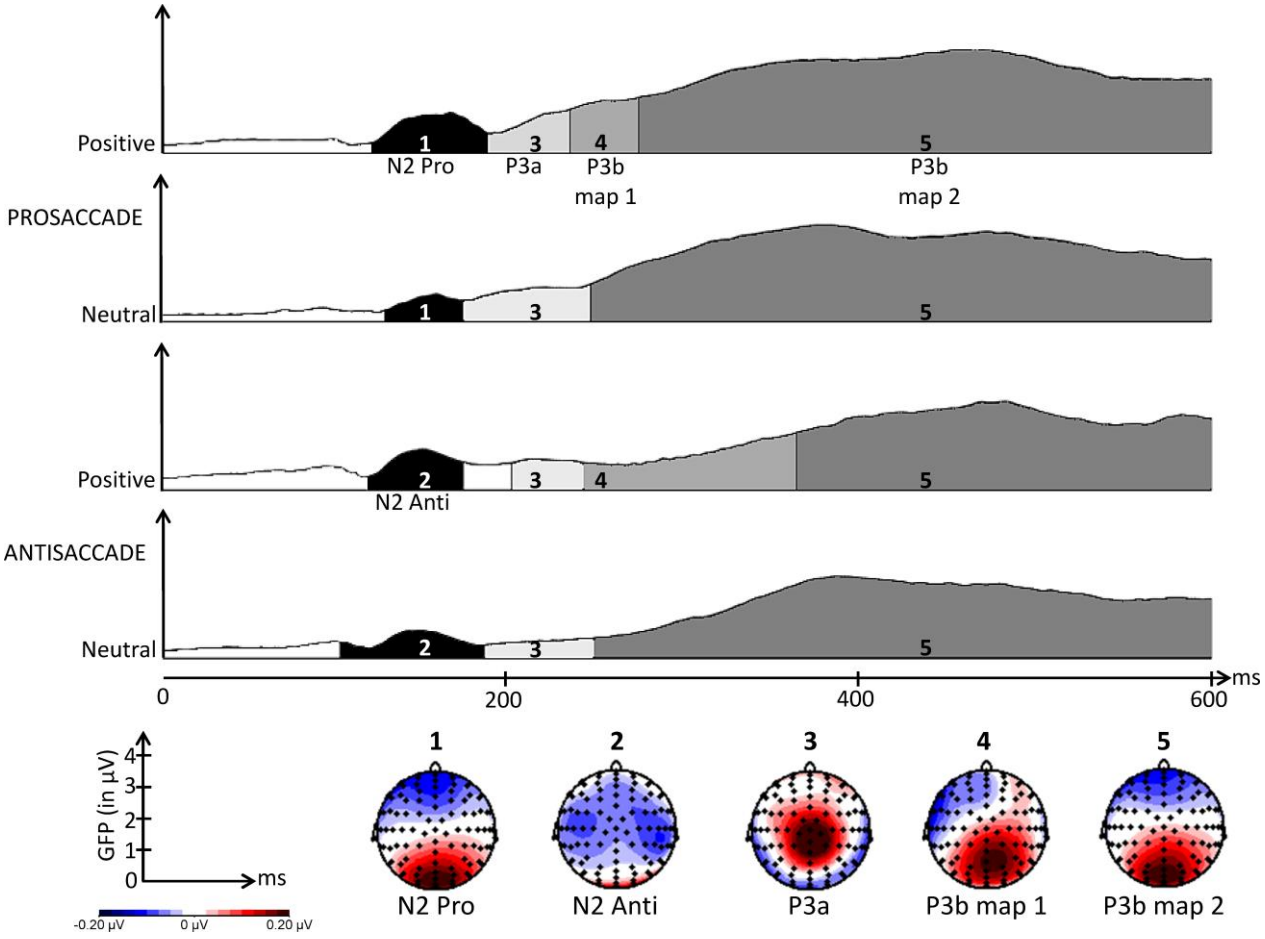
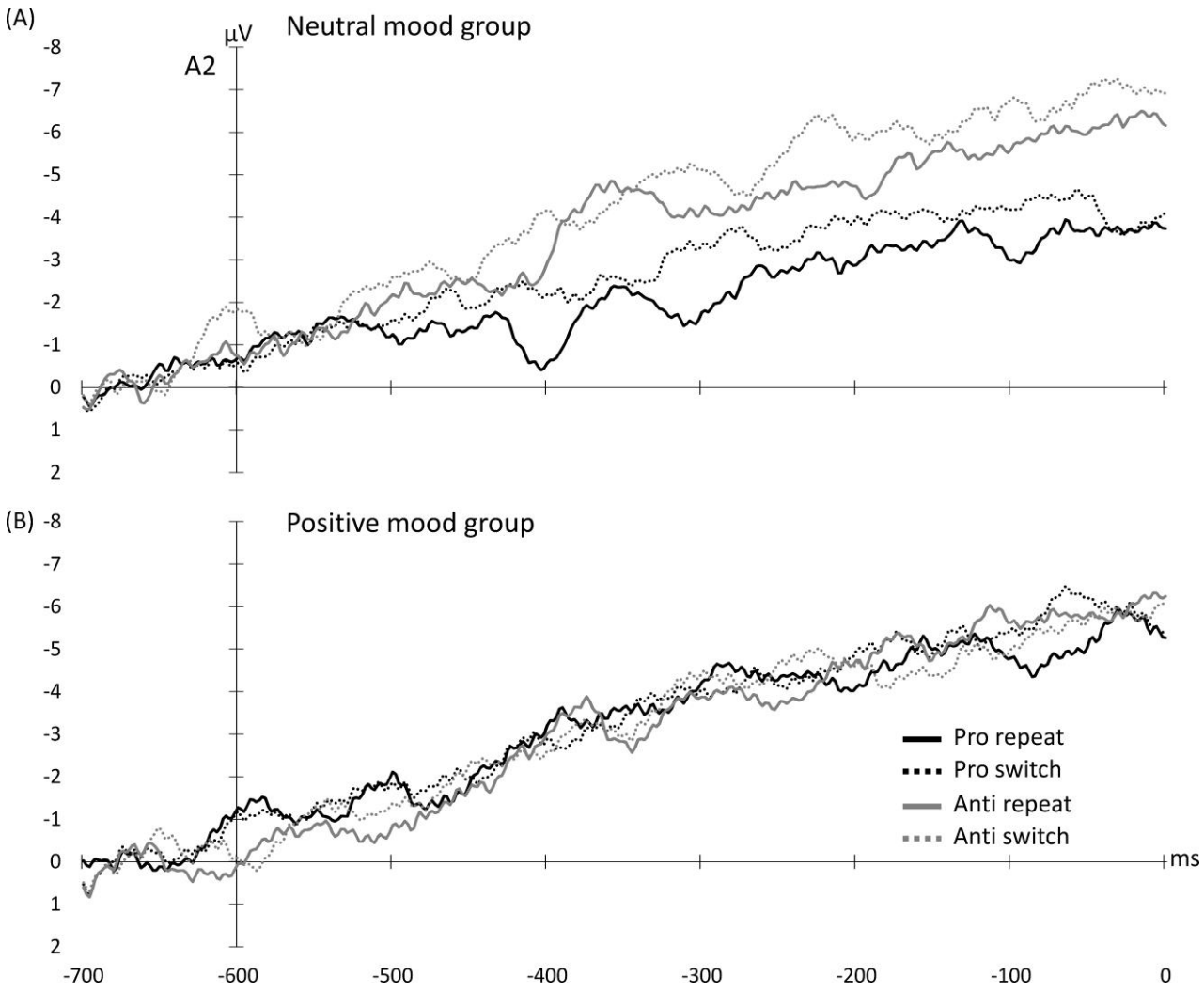

Positive
1
N2 Pro
3
P3a
4
P3b
map 1
5
P3b
map 2
PROSACCADE
Neutral
1
3
5
Positive
2
N2 Anti
3
4
5
ANTISACCADE
Neutral
2
3
5
ms
0
200
400
600
GFP (in µV)
4
3
2
1
0
ms
-0.20 µV
0 µV
0.20 µV
1
2
3
4
5
N2 Pro
N2 Anti
P3a
P3b map 1
P3b map 2

Figure 5


(A)
μV
A2
Neutral mood group
-8
-7
-6
-5
-4
-3
-2
-1
0
1
2
(B)
Positive mood group
Pro repeat
Pro switch
Anti repeat
Anti switch
ms
-700
-600
-500
-400
-300
-200
-100
0