



Positive affect increases cognitive control in the antisaccade task

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ABSTRACT

To delineate the modulatory effects of induced positive affect on cognitive control, the current study investigated whether positive affect increases the ability to suppress a reflexive saccade in the antisaccade task. Results of the antisaccade task showed that participants made fewer erroneous prosaccades in the condition in which a positive mood was induced compared to the neutral condition (i.e. in which no emotional mood was induced). This improvement of oculomotor inhibition was restricted to saccades with an express latency. These results are in line with the idea that enhanced performance in the positive affect condition could be caused by increased dopaminergic neurotransmission in the brain.

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1. Introduction

The question whether one's current emotional state influences one's cognitive abilities has been investigated in various domains. Positive mood has been shown to modulate cognitive functions, although the exact influence has been shown to vary between different functions: positive affect has been found to either impair or improve performance depending on the specific task. On the one hand, induced positive affect improves verbal fluency (Philips, Bull, Adams, & Fraser, 2002) and reduces interference between competing response alternatives in a Stroop-task (Kuhl & Kazén, 1999). On the other hand, positive affect has been shown to increase response interference due to increased distractibility (Rowe, Hirsh, & Andersen, 2007) and to impair performance on certain executive function tests (Oaksford, Morris, Grainger, & Williams, 1996). A series of studies by Dreisbach and colleagues revealed that positive affect results in flexibility benefits, but also in maintenance costs (distractibility) (Dreisbach, 2006; Dreisbach & Goschke, 2004; Dreisbach et al., 2005).

The exact effect of positive affect on cognitive control is therefore still unclear. To further delineate the modulatory effects of induced positive affect on cognitive control, we used a task that allowed us to study a specific aspect of cognitive control: the inhibition of reflexive eye movements ('oculomotor inhibition'). During the so-called antisaccade task, participants either make a saccadic eye movement towards the appearing stimulus after stimulus onset (i.e. prosaccade trials) or a saccade in the opposite direction as quickly as possible (i.e. antisaccade trials). Correct performance in

the antisaccade task requires the inhibition of the automatic response to the stimulus onset. Results typically show that antisaccade trials have longer saccade latencies than prosaccade trials and that participants frequently make an erroneous saccade to the stimulus onset in antisaccade trials (Everling & Fischer, 1998; Hutton & Ettinger, 2006).

Neuropsychological research has revealed that correct performance in the antisaccade task is subserved by brain areas that are also known to be involved in cognitive control. For instance, imaging studies have identified various frontal areas that are active during the antisaccade task such as the frontal eye fields and dorsolateral prefrontal cortex (Everling & Munoz, 2000; Funahashi, Bruce, & Goldman-Rakic, 1993). Lesion studies have revealed that successful inhibition in the antisaccade task relies heavily on frontal circuits (Guitton, Buchtel, & Douglas, 1985; Pierrot-Deseilligny, Rivaud, Gaymard, & Agid, 1991; Pierrot-Deseilligny et al., 2003). Furthermore, the amount of erroneous eye movements is known to be increased when a working memory task is performed simultaneously (Mitchell, Macrea, & Gilchrist, 2002) and successful performance in the antisaccade task is linked to working memory capacity (Eenshuistra, Ridderinkhof, & van der Molen, 2004; Roberts, Hager, & Heron, 1994). Therefore, oculomotor inhibition in the antisaccade task is generally linked to prefrontal cognitive control.

In the current study, it was investigated whether induced positive affect increases the ability to suppress a reflexive saccade in the antisaccade task. Participants performed the antisaccade task twice: once after seeing a neutral movie and once after seeing a movie which is expected to induce positive affect. The amount of erroneous eye movements was compared between the two sessions. In this analysis, a distinction was made between erroneous eye movements with express (80–130 ms) and regular (>130 ms) latencies, because

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these errors have been argued to reflect different and distinct phenomena (Klein & Fischer, 2005). Whereas express errors seem to reflect reflex-like prosaccades to the stimulus onset, erroneous eye movements with a regular latency reflect errors in the intentional processes associated with the execution of a correct antisaccade (Klein, Rauh, & Biscaldi, 2010). For instance, although erroneous eye movements with a regular latency are correlated with ('higher') cognitive measures, like executive function and working memory, similar correlations are absent for express errors (Klein et al., 2010).

If induced positive affect increases cognitive control, as observed in the Stroop-task (Kuhl & Kazén, 1999), this should result in stronger oculomotor inhibition, reflected by a decreased number of erroneous eye movements on antisaccade trials. The analysis of express and regular latencies will provide insight in whether this possible improvement is related to an increased inhibition of reflex-like prosaccades or related to reduced errors in intentional processes, as measured by erroneous eye movement with a regular latency.

2. Method

2.1. Participants

Twelve students of the Utrecht University, aged between 18 and 25 years, served as paid volunteers. Six participants were male. All reported having normal or correct-to-normal vision. They were naive as to the purpose of the experiment. All participants gave their informed consent prior to their inclusion in the study.

2.2. Apparatus

An Intel Core2 computer controlled the timing of the events. The displays were presented on a LaCie 22" monitor with a resolution of 1024×768 pixels. Eye movements were registered with the Desktop Mount EyeLink1000. The EyeLink1000 has a temporal resolution of 1000 Hz and a spatial resolution that is smaller than 0.5° . Although the system can compensate minimal head movements, the participant's head was stabilized using a chin rest. The distance between the monitor and the chin rest was 65 cm. Participants performed the experiment in a sound-attenuated and dimly lit room.

2.3. Stimuli, procedure and design

Participants performed two sessions: the positive affect condition and the neutral condition. The time between these two sessions was at least 24 h. The order of the sessions was counter-balanced between participants. The order of each session was the following: first questionnaire, calibration procedure, practice trials of eye movement task, movie fragment, second questionnaire, experimental trials eye of movement task. These elements will now be discussed in detail.

2.3.1. First questionnaire

In the questionnaire participants indicated on a five-point scale whether they were refreshed vs. tired, calm vs. anxious, alert vs. unaware, amused vs. sober and positive vs. negative (Isen, Daubman, & Nowicki, 1987). Zero on this scale indicates the first extreme (i.e. 0 is positive, 5 is negative).

2.3.2. Calibration procedure

Each session started with a nine-point grid calibration procedure. Participants were required to saccade towards nine fixation points sequentially appearing at random in a 3×3 grid. In addition, simultaneously fixating the central fixation point and

pressing the space bar recalibrated the system by zeroing the offset of the measuring device at the start of each trial.

2.3.3. Practice trials of eye movement task

See Fig. 1 for an example of the display sequence. Participants viewed a display containing a plus sign (0.70°) on a black background in the centre of the display, which was used as fixation point. The color of the plus sign indicated the type of trial: red indicated an antisaccade trials and green indicated a prosaccade trial. Half the trials were prosaccade trials and the other half were antisaccade trials. After 1000 ms the fixation point disappeared and 250 ms after the fixation point offset one circle (1.30° in diameter) appeared at a distance of 10° either to the right or left side. The circle appeared at the same Y coordinate as the fixation point. The target was presented for 1500 ms. Afterwards all objects were removed from the display. The practice of the eye movement task consisted of 40 trials.

Participants were instructed to fixate the central fixation point until target onset and to then move their eyes towards or away from the target location (depending on the task). It was stressed that one had to make a single accurate saccade toward the correct location. Participants heard a short tone when the saccade latency was higher than 600 ms or shorter than 80 ms. The sequence of trials was counterbalanced and randomized for each participant.

2.3.4. Movie fragment

The movie fragment in the neutral condition contained two crossroads in Amsterdam showing normal traffic. In the positive affect condition, participants could choose between different movie fragments: a fragment from a Disney movie ("The Little Mermaid" or "Lion King") or a sketch from a Dutch comedy program ("De Lama's"). In contrast to the movie fragment in the neutral condition, the fragment in the positive affect condition was hypothesized to induce positive affect.

2.3.5. Second questionnaire

Participants filled out the same questionnaire as discussed above.

2.3.6. Experimental trials of eye movement task

The eye movement task as outlined above consisted of 200 trials.

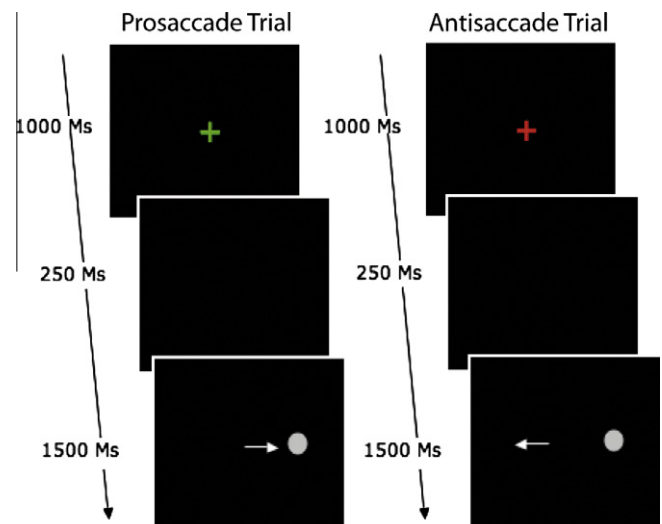


Fig. 1. Example of the display sequence for the antisaccade task. The arrow in the third panel of the antisaccade task depicts the direction of the required eye movement and was not present in the experiment.

3. Data analysis

3.1. Questionnaire

For the analysis of the questionnaire we used paired *t*-tests with the within-subjects variable time (before vs after movie).

3.2. Eye movement task

Saccades were automatically detected using software developed by SR Research. Thresholds for detecting the onset of saccadic movements were accelerations of 8000 (deg/s²), velocities of 30.0 (deg/s), and distances of 0.5 (deg) of visual angle. Movement offset was detected when velocity fell below 30.0 (deg/s) and remained at the level for 10 consecutive samples.

Saccade latency was defined as the interval between target onset and the initiation of a saccadic eye movement. Trials were excluded when the latency of the saccade was lower than 80 ms or higher than 600 ms (see e.g., Nijboer, Vree, Dijkerman, & Van der Stigchel, 2010), or further than two and a half standard deviations away from the subject's mean latency. Moreover, trials were excluded from analysis in which no saccade, too early or a too small first saccade ($<3^\circ$) was made. The endpoint of the first saccade had to have an angular deviation of less than 22.5° from the center of the target or the mirrored target location. In the first case, the saccade was classified as a prosaccade; in the second case the saccade was classified as an antisaccade. In other situations, the saccade was classified as an error and not analyzed.

An Analysis of Variance (ANOVA) with Condition (positive affect vs neutral) and Task (prosaccade vs antisaccade) as within-subjects factors was used to analyze effects on saccade latency. Only trials in which the first eye movement was initiated correctly (either a prosaccade or antisaccade, depending on the task) were included in the saccade latency analysis.

To investigate the effect of induced positive affect on errors, a paired *t*-test was run on antisaccade trials with Condition (positive affect vs neutral) as the factor. Additional comparisons were made between the positive affect and neutral conditions for the percentage erroneous eye movements with express (80–130 ms) and regular (>130 ms) latencies.

4. Results

4.1. Questionnaire

In the neutral condition, none of the questions was responded to differently before or after participants saw the movie (p 's > 0.05). In the positive affect condition, participants were more amused ($t(11) = 5.00$; $p < 0.001$) and more positive ($t(11) = 2.35$; $p < 0.05$) after the movie fragment when compared to before they saw the movie.

4.2. Antisaccade task

The exclusion criteria led to a total loss of 7.1% of trials.

4.2.1. Saccade latency

A main effect of Task was observed ($F(1, 11) = 101.4$; $p < 0.0001$). The mean latency of correct prosaccades was lower (mean = 141 ms; st. dev. = 22 ms) than correct antisaccades (mean = 207 ms; st. dev. = 33 ms). The main effect of Condition was not significant ($F < 1$). No significant interaction between Task and Condition was observed ($F(1, 11) = 1.35$; $p = 0.28$).

4.2.2. Percentage erroneous eye movements

Participants made fewer erroneous prosaccades in the positive affect condition (mean = 0.167; st. dev. = 0.115) than in the neutral condition (mean = 0.222; st. dev. = 0.119; $t(11) = 3.03$; $p < 0.02$; see Fig. 2).

Furthermore, participants made fewer erroneous prosaccades with express latencies in the positive affect condition (mean = 0.099; st. dev. = 0.091) than in the neutral condition (mean = 0.146; st. dev. = 0.125; $t(11) = 2.81$; $p < 0.02$). There was no difference between the positive affect (mean = 0.067; st. dev. = 0.056) and neutral condition for regular latencies (mean = 0.074; st. dev. = 0.052; $t(11) = 0.72$; $p = 0.48$).

5. Discussion

The current study investigated whether positive affect increases the ability to suppress a reflexive saccade in the antisaccade task. Evidence that positive affect was indeed induced in the positive affect condition was provided by pre- and post-test questionnaires in which participants confirmed that they were more positive and amused after seeing the movie compared to before the movie. Results of the antisaccade task showed that participants made fewer erroneous prosaccades in the condition in which a positive mood was induced compared to the neutral condition (i.e. in which no emotional mood was induced). There were no effects on saccade latency, indicating that positive affect did not influence the speed of responding.

Correct performance in the antisaccade task requires the inhibition of the automatic response to the target. Because a failure of oculomotor inhibition will result in the execution of an erroneous eye movement toward the stimulus, the lower amount of erroneous eye movements in the positive affect condition points to an increased cognitive control. This is in line with the idea that positive affect results in better cognitive performance when competing response alternatives are present (Ashby, Isen, & Turken, 1999; Ashby, Valentin, & Turken, 2002; Kuhl & Kazén, 1999).

To account for the influence of positive mood on cognitive abilities, Ashby and colleagues proposed a neurobiological theory of the influence of positive affect (Ashby et al., 1999, 2002). According to their theory, induced positive affect leads to temporary increase of dopamine release in mid-brain DA-generation centres. This dopamine release is subsequently propagated to dopaminergic projection sites in other brain areas, most prominently the prefrontal cortex and the striatum (Williams & Goldman-Rakic, 1993). Increased dopamine levels in the prefrontal cortex enhance the ability to overcome dominant responses and increase cognitive

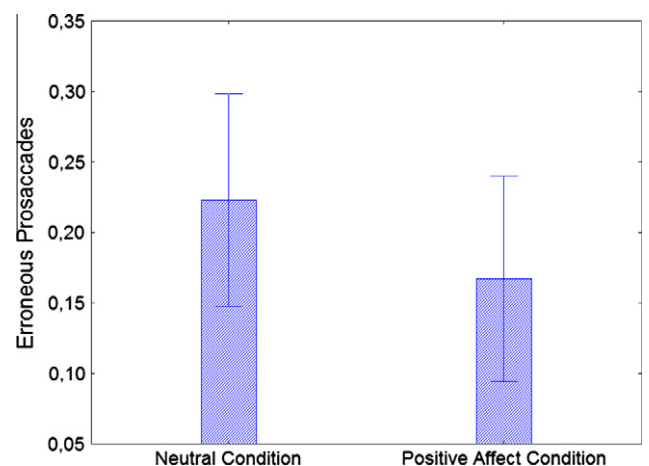


Fig. 2. The fraction erroneous prosaccades in antisaccade trials for both the neutral and positive affect condition. Error bars indicate 95% confidence intervals.

flexibility, resulting in altered cognitive performance on tasks involving cognitive control. Evidence for this theory originates from studies which have shown that DA agonists that enhance dopaminergic activity strengthen positive affect (Beatty, 1995). Furthermore, there is ample evidence that DA selectively modulates cognitive control processes (Braver, Barch, & Cohen, 1999; Reynolds, Braver, Brown, & Van der Stigchel, 2006).

Interestingly, the antisaccade task has been shown to be modulated by dopamine levels in the brain. For instance, patients with schizophrenia have higher error rates and longer latencies than controls on antisaccade tasks (Fukushima, Fukushima, Morita, & Yamashita, 1990; Sereno & Holzman, 1995), similarly to advanced Parkinson patients (Kitagawa, Fukushima, & Tashiro, 1994). Because these disorders have been linked to an imbalance in dopaminergic states in the brain, these abnormalities in the antisaccade task may be due to disturbances in dopaminergic neurotransmission. Although we did not measure dopamine levels directly in the current experiment¹, we speculate that the observed modulations of positive affect on the antisaccade task might therefore be due to changes in dopaminergic levels in the brain. Higher levels of dopamine result in the enhanced ability to overcome dominant responses. Such fluctuations in DA levels might be expected to modulate activity particularly in those oculomotor circuits that are densely innervated by dopaminergic projections.

Results further showed that the effect of induced positive affect on oculomotor inhibition was restricted to the eye movements with short latencies (80–130 ms). It is known that these erroneous 'express' saccades reflect a different and distinct phenomenon than erroneous saccades with a longer latency (>130 ms) (Klein & Fischer, 2005; Klein et al., 2010). Therefore, it seems that the induced positive affect exclusively improves the oculomotor inhibition of reflex-like prosaccades. This finding might seem inconsistent with the idea that induced positive affect increases cognitive control, because it has been suggested that only errors with a regular latency are correlated with ('higher') cognitive measures, like executive function and working memory (Klein et al., 2010). Although speculative, it is interesting to consider the possible neural mechanisms underlying the effect of induced positive affect on the oculomotor inhibition of reflex-like prosaccades. When a saccade is required in the direction opposite to the visual hemifield in which a stimulus onset occurs, several distinct but interrelated oculomotor processes come into play: (1) active fixation of the oculomotor system, (2) intentional saccade initiation, and (3) selective suppression of saccades until the program of the appropriate eye movement has been fully developed. To maintain fixation rather than respond to any trivial change of information in the visual field, the superior colliculus (SC) contains fixation neurons that fire during fixations but cease firing just before and during saccades, whereas movement neurons display the opposite pattern (Munoz & Wurtz, 1993). Because express saccades occur when activity in the fixation cells is reduced (Munoz & Wurtz, 1992), an increased activity of fixation neurons in the SC result in increased control over reflexive saccades. One of the areas that might modulate the fixation neurons in the SC, is the dorsolateral prefrontal cortex (DLPFC). Indeed, in a recent computational model of the oculomotor system, express saccades were found in trials in which there was a relatively small input from the DLPFC to fixation

neurons in the SC (Meeter, Van der Stigchel, & Theeuwes, 2010). The DLPFC projects densely to the intermediate and deep layers of the SC (Goldman & Nauta, 1976; Johnson & Everling, 2006; Yeterian & Pandya, 1991). Johnson and Everling (2006) concluded that DLPFC neurons projecting to the SC are mostly involved in inhibiting prosaccades. They speculated that such neurons might project to fixation neurons in the rostral SC. There are also indirect connections from DLPFC to fixation neurons in the SC, via the basal ganglia and the Substantia Nigra pars reticulara (SNr) (Hikosaka, Nakamura, & Nakahara, 2006; Hikosaka, Sakamoto, & Miyashita, 1993). Since SNr neurons are tonically active and are GABAergic, it is generally thought that SNr delivers a constant inhibition to saccade neurons to help maintain fixation. Because the richest projections from the dopamine generators in the mid-brain are found in the prefrontal cortex (including the DLPFC) and the striatum (including the caudate nucleus) (Williams and Goldman-Rakic, 1993), fluctuations in DA, such as those elicited by positive affect, most likely modulate fixation neurons in the SC, be it through the direct or indirect route.

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¹ Dopaminergic modulations have been linked to blinking rate (e.g. Colzato, van den Wildenburg, & Hommel, 2008). When analyzing the amount of blinks recorded during the experimental trials in the current study, there was indeed a trend for more blinks in the positive affect condition than in the neutral condition ($t(11)=1.78$; $p=0.10$). It must be noted, however, that this experiment was not designed to measure blink rate. To perform a speeded eye movement in response to a visual stimulus, a low amount of blinks is beneficial. Blinks were therefore likely to occur in between experimental trials. Eye blinks were not recorded in this time period, however.

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