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The Netherlands

Cognitive control and binding in context-based decision-making : normal and dopamine deviant populations

Wouwe, N.C. van

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Positive affect modulates flexibility and evaluative control

Abstract

The ability to interact with a constantly changing environment requires a balance between maintaining the currently relevant working-memory content and being sensitive to potentially relevant new information that should be given priority access to working memory. Mesocortical dopamine projections to frontal brain areas modulate working memory maintenance and flexibility. Recent neurocognitive and neurocomputational work suggests that dopamine release is transiently enhanced by induced positive affect. This ERP study investigated the role of positive affect in different aspects of information processing: in proactive control (context maintenance and updating), reactive control (flexible adaptation to incoming task-relevant information), and evaluative control in an AX-CPT task. Subjects responded to a target probe if it was preceded by a specific cue. Induced positive affect influenced the reactive and evaluative components of control (indexed by the N2 elicited by the target, and by the Error-Related Negativity elicited after incorrect responses) in an AX-CPT task, whereas cue-induced preparation and maintenance processes remained largely unaffected (as reflected in the P3b and the Contingent Negative Variation components of the ERP).

Introduction

Adequate interactions with a changing environment require a balance between *maintenance* of task-related information and *flexibility* to take new and potentially relevant information into account. First, maintenance and updating of relevant goals and intentions are important to successfully override distraction from the environment (proactive control). For example, on a busy working day you have to keep in mind to finish a presentation at the end of the day otherwise you will get distracted by currently incoming tasks. Second, instead of preparing proactively, a decision can also be based on currently available stimulus information by reactivating context information. Reactive control may be required for instance to resolve the conflict between overlearned action tendencies and actions indicated by the latest information. Finally, the outcome of the decision will be evaluated, which may give rise to increased control on future trials or a more optimal response choice.

Several studies have indicated that transiently induced positive affect modulates these cognitive functions, generally leading to enhanced flexibility. Some behavioural studies have pointed towards increased cognitive flexibility when affect is elevated; positive affect improved verbal fluency (Philips et al., 2002) and reduced interference between competing response alternatives (Kuhl & Kazén, 1999). On the other hand, positive affect has also been shown to increase response interference due to increased distractibility (Rowe, Hirsh, & Anderson, 2007). Studies of Dreisbach and Goschke (2004; Dreisbach, 2006; Dreisbach et al., 2005) pointed out that positive affect results in flexibility benefits, but also in maintenance costs (distractibility). The current experiment was designed to identify the modulating influence of positive affect (induced by a movie clip) on cognitive control, by means of event related potentials (ERPs).

Dopamine and positive affect

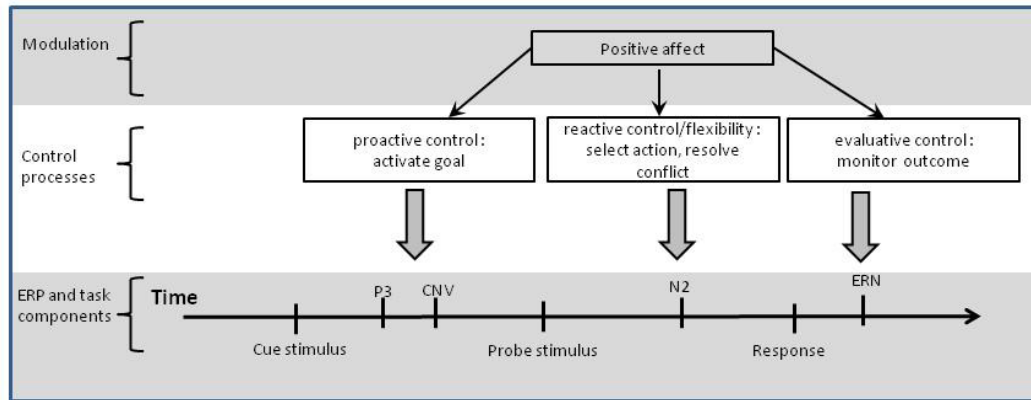
Ashby et al. (Ashby, Turken, & Isen, 1999; Ashby, Valentin, & Turken, 2002) suggested that the impact of positive affect on cognition is the result of a temporary increase of

dopamine (DA) release in midbrain DA-generation centres, which is propagated to dopaminergic projection sites in other brain areas, such as the prefrontal cortex (PFC). The resulting increase in DA levels serves to improve the ability to overcome dominant responses and results in more flexible behaviour. Dreisbach & Goschke (2004) compared the effect of individual differences in DA (identified on the basis of genetic polymorphisms and eye-blink rates) on flexibility and maintenance; participants with higher baseline levels of DA showed a similar performance pattern as did individuals with induced positive affect (Dreisbach & Goschke, 2004).

The link between mood elevation and increased level of DA was originally established by drug studies; DA agonists such as cocaine and amphetamine induce positive mood (Beatty, 1995), whereas DA antagonists flatten affect (Hyman & Nestler, 1993). Changes in DA levels affect dopaminergic activation in the brain areas that receive DA projections from the ventral tegmental area (mesocorticolimbic DA system), such as the anterior cingulate cortex (ACC), the dorsolateral PFC (DLPFC), hippocampus and amygdala (Ashby et al., 1999) and areas receiving DA projections from the substantia nigra to the striatum (nigrostriatal DA system). The mechanisms underlying the modulating influence of induced positive affect on different aspects of control, however, remain to be studied with more rigorous experimental scrutiny.

Figure 1

Schematic overview of control processes that may be modulated by affect during task performance and the related ERP components.



Aspects of control and neural correlates

Figure 1 provides a schematic overview of the control processes that may be modulated by affect during task performance, and the ERP components that are used to map this modulation.

Based on behavioral, neuroimaging, and computational modeling work, Braver and colleagues presented the Dual Mechanism of Control (DMC) theory, differentiating the importance of proactive and reactive mechanisms in flexible adaptive behavior (Braver, Barch & Cohen, 1999; Braver et al., 2001; DePisapia & Braver, 2004; Speer, Jacoby, & Braver, 2003). A core component of the DMC theory is the role of the PFC in proactive control and the involvement of the ACC in reactive control. In line with the DMC theory, the proactive use of abstract rules to control behaviour, for example by maintaining task-relevant information, is generally attributed to PFC functioning (Boettiger & D'Esposito, 2005).

Comparable to the flexible mode of processing mentioned by Dreisbach and colleagues (Dreisbach & Goschke, 2004; Dreisbach et al., 2005), reactive control involves information processing driven by the latest incoming information (i.e. the

probe) (Braver et al., 2007). The current study especially aims at measuring probe induced control processes that entail resolving possible interference between co-activated responses or correcting erroneous response tendencies (implemented by the ACC) by means of the ERP component that reflects response competition.

The ACC is either engaged in the reactive control process itself or in signaling the need for control, which is then applied by DLPFC. Output from the ACC triggers the DLPFC to reduce interference (Durstun et al., 2003; Kerns et al., 2004; MacDonald et al., 2000) and DLPFC subsequently biases posterior brain areas (i.e. parietal cortex) (Desimone & Duncan, 1995; Dove, Pollman, Schubert, Wiggins, & von Cramon, 2000; Gruber, Karch, Schlueter, Falkai, & Goschke, 2006).

Role of dopamine in control processes

Since several studies (Ashby et al., 1999; Dreisbach et al., 2004; Esch & Stefano, 2004; Wise, 1980; see also Berridge, 2007 for an orthogonal view) point to a relation between dopamine and positive affect, the role of dopamine in control processes will be discussed, i.e. proactive control, reactive and evaluative control.

With respect to the influence of dopamine on proactive and reactive control, modeling work, fMRI, drug and patient studies (Barch, 2004; Braver et al., 1999; Burgess & Braver, 2004; Speer et al., 2003; Cohen, Braver & Brown, 2002; Durstewitz, Seamans, & Sejnowski, 2000; Holroyd & Coles, 2002; Kimberg & D'Esposito, 2003) have provided evidence that dopaminergic modulations in the PFC especially affect proactive task preparation. For example, populations with a decline in DA functioning in PFC, such as older adults, are impaired in proactive control of behavior but show relatively intact reactive control over behavior (Braver et al., 2001). Additionally, systematic manipulation of DA in humans (c.f. for a review Barch, 2004) by using dopamine agonists pointed to enhancing effects of DA on maintenance of task-relevant information. Whereas the role of DA in PFC with proactive control is supported, it is less clear how and if DA might play a role in reactive control (Botvinick et al., 2001; Van Veen, Holroyd, Stenger, Cohen, & Carter, 2004).

Evaluative control on the other hand, which involves monitoring action outcomes, does seem to be sensitive to dopaminergic changes. Ridderinkhof, Ullsperger, Crone and

Nieuwenhuis (2004) emphasize the evaluative role of the ACC in decision making; the ACC monitors performance for unfavorable outcomes or errors. According to several authors (Cools, 2006; Frank, Woroch, & Curran, 2005), the evaluative function of the ACC is affected by DA fluctuations in the nigrostriatal DA system (i.e. the striatum is connected with the ACC in a basal-ganglia-thalamo-cortical circuit, Alexander, DeLong, & Strick, 1986). That is, DA modulates error sensitivity and the ability to adequately use feedback in reward based learning.

The effect of induced affect on ACC-mediated evaluative control can be investigated through the amplitude of the Error Related Negativity (ERN), a medial-frontal deflection peaking around 100 ms after an erroneous response (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The main generator of the ERN is most likely situated in the ACC (Holroyd & Coles, 2002). Evidence supporting that the ERN is sensitive to DA deviations comes from animal literature (Schultz, 2002), DA deviant populations, and drug studies (for a review see Overbeek, Nieuwenhuis, & Ridderinkhof, 2005). The ERN is relatively spared in patients with Parkinson's disease (PD) off medication, who have low striatal DA levels (Holroyd, Praamstra, Plat & Coles, 2002), but diminished in populations with high levels of DA, like PD patients on medication and patients with schizophrenia (Bates, Liddle, Kiehl, & Ngan, 2004; Ito & Kitagawa, 2006).

A sizeable ERN is thought to reflect a phasic dip in DA level in the striatum that signals via the mesencephalic DA system to the ACC when outcomes are worse than expected. Individual and pharmacological differences in baseline striatal DA level limit the ability to produce an effective DA dip, and thereby limit the size of the ERN. For example, an increased DA level in the striatum due to D2-antagonist haloperidol is thought to prevent a phasic dip in DA level (Frank & O'Reilly, 2006). Indeed, the ERN in this condition is attenuated (Zirnheld et al., 2004).

Surprisingly, nonspecific DA enhancers amphetamine and caffeine have led to an increase in ERN and improvement of action monitoring in healthy volunteers (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; Tiegens, Ridderinkhof, Snel, & Kok, 2004). However, these substances do not only enhance the striatal DA level, but also that in the medial frontal cortex (MFC) (Acquas, Tanda & Di Chiara, 2002; Vollenweider, Maguire, Leenders, Mathys, & Angst, 1998). Similarly, an increase or decrease of the ERN can be used as an index of the locus of the DA enhancement by induced affect, i.e.

an increase in the ERN may reflect enhanced DA in MFC whereas a reduced ERN may reflect enhanced DA specifically in the striatum.

AX-CPT paradigm

The present study examines the effect of positive affect induction, compared to neutral affect induction, on behavioral and ERP components in a so-called AX-CPT task. This task enables us to measure different control processes, because performance in the task depends on the capability to update and maintain task-relevant cue information as well as on the flexibility to use unexpected probe information to select a response. The AX-CPT paradigm is a modified version of the classic Continuous Performance Test (CPT; Rosvold, Mirsky, & Sarason, 1956).

During each AX-CPT trial participants are presented with a sequence of letters on the computer screen. Unknown to the participants, these letter sequences are constructed as trials of cue-probe pairs. Subjects are instructed to respond to every letter with either a target or nontarget response by manually pressing a button. A target response, for example a left index finger press on a left button, is required when the target probe (*X*) is immediately preceded by a certain context cue (*A*). In every other case, for example in *AY*, *BX* or *BY* sequences, they have to respond to both cue and probe with a non-target response button, i.e., right index finger press on a right button.

Target trials (*AX*) occur on the majority of trials in the AX-CPT task; this frequency induces a strong bias to issue a target response, even on trials other than *AX* (*BX* and *AY*). In the AX-CPT task, increased use of proactive control (use of cue information to prepare for a response) will result in a stronger bias for a target response after an *A* cue, which impairs *AY* performance. When the *B* cue is used proactively, however, *BX* and *BY* performance will benefit from the preparation of a nontarget response specified by the *B* cue. Impaired proactive control would result in the opposite pattern: enhanced performance on *AY* trials, but performance costs on other trial types.

Predictions

Based on a proactive goal-driven account of control (Braver et al., 2001; Braver et al., 2007; Cohen et al., 2002) and the maintenance-flexibility balance (Dreisbach & Goschke, 2004; Dreisbach et al., 2005) the prediction can be derived that induced positive affect will modulate proactive control, although in opposite directions.

The maintenance-flexibility theory (Dreisbach, 2006; Dreisbach & Goschke, 2004; Dreisbach et al., 2005) predicts that positive affect increases flexibility but this occurs at the cost of maintenance. A reduced maintenance capability is reflected in improved *AY* performance and in impaired *BX* and *BY* performance (Note that if only flexibility is enhanced this will result in a selective *AY* improvement). According to a goal-driven account of control however, an increase in *DA* by the affect induction will result in a tendency towards enhanced proactive control, as expressed in improved *BX* and *BY* performance but impaired *AY* performance. Direct changes in reactive control with positive affect (i.e. not due to changes in proactive control) will be measured as the modulation of the probe related ERP component *N2*, although this is not predicted by a goal-driven account of control.

A goal-driven account of control and the maintenance-flexibility theory do not state predictions with respect to behavioral changes in evaluative control. Therefore, the modulating influence of positive affect on evaluative control will be measured by the ERPs exclusively and will be more exploratory because the ERP studies that investigated evaluative control (the *ERN*) and *DA* showed mixed results. Changes in the cue and probe related ERPs enable us to disentangle the modulating effect of positive affect on proactive, reactive and evaluative control mechanisms over time. For example, if positive affect exclusively modulates proactive control, this will be reflected in the cue related ERPs.

We examine the effect of the positive affect induction on proactive control by means of the Contingent Negative Variation (*CNV*) and *P3b*, measured in the cue-probe interval. The parietal *P3b* is a large positivity that peaks approximately 300 ms after stimulus presentation. It is thought to reflect context updating (Donchin & Coles, 1988; Polich, 2003) or motivational significance (Nieuwenhuis, Aston-Jones, & Cohen, 2005) because its amplitude increases when current context information in memory is refreshed by novel task-relevant information. The late wave of the *CNV* (Dias, Foxe, &

Javitt, 2003; Jonkman, Lansbergen, & Stauder, 2003) is a central negative deflection that precedes the signal to respond and correlates with expectation or general response preparation. It is also suggested to correlate with memory activity (Ruchkin, Canoune, Johnson, & Ritter, 1995).

With respect to the CNV and P3b we do not expect that emotion differentially affects the amplitude with *A* or *B* cues. If positive affect improves proactive control as predicted by a goal-driven account of control, we expect that this will be reflected in the increased amplitude of the P3b and CNV after *A* and *B* cue presentation. With an impairment of proactive control, the amplitudes will decrease. The P3b however is larger on relevant events that occur with lower probability (*B* cues in the AX-CPT task), which will increase the P3b after *B* cues compared to *A* cues (Ruchkin, Sutton, & Tueting, 1975) in both affect groups.

We examine the effect of the positive affect induction on probe- and response-induced control mechanisms (reactive and evaluative control) by means of the N2 and the ERN. The N2 is a negative potential occurring around 150-300 ms after high conflict trials (van Veen & Carter, 2002a; 2002b). The N2 has been proposed to reflect the need to inhibit an incorrect response tendency (Eimer, 1993; Jodo & Kayama, 1992), response conflict as triggered by the processing of irrelevant stimulus information (Nieuwenhuis, Yeung, Ridderinkhof, & van den Wildenberg, 2003; Yeung & Cohen, 2006), or action selection in the face of competing alternatives (Burle, van den Wildenberg, W.P.M., & Ridderinkhof, 2008). Each of these scenarios involves overcoming response interference (an aspect of reactive control that involves the ACC, as mentioned by DMC theory, Braver et al., 2007), and therefore we expect that if response competition is reduced with positive affect (either due to reduced proactive control or improved flexibility, which can only be concluded when integrating behavioral measures and the cue-based ERPs), the N2 will decrease in amplitude, but if response competition increases, the N2 will be enhanced as well.¹

¹ Studies on the N2 and cognitive control do not provide a unified theory with respect to the role of the N2. The N2 could either reflect response interference and the need for control or be involved in the actual control process (see Folstein & van Petten, 2008 for a review). We do not claim to support either one of these interpretations; especially not since the DMC theory emphasizes a dual role of the ACC (the source of N2; van Veen & Carter, 2002a; 2002b) in reactive control, either in resolving competition on the current trial or signaling the need for increased proactive control on future trials. In the discussion, both views of the N2 are used to interpret the data.

A goal-driven account predicts enhanced proactive control with positive affect which will be reflected in the cue-related ERPs and behavior, but also in the probe-related ERPs. As a consequence of increased proactive control, response competition will change in several trialtypes; reactive control will have to be engaged to a lesser extent after *X* probes in *BX* trials and to a larger extent after *Y* probes in *AY* trials, resulting in a decreased probe-related N2 on *BX* trials and increased N2 on *AY* trials. With reduced proactive control on the other hand, as predicted by the maintenance-flexibility theory, the opposite pattern will be reflected in the probe-related N2 on *AY* trials and again a smaller N2 on *BX* trials (when there is no maintenance of the *B* cue, the *X* probe with not conflict with this information).

Studies that investigated the effect of DA on the evaluative component of control and the ERN show mixed results; a DA increase in the striatum diminished the ERN, while a DA increase in MFC amplified the ERN. Therefore, the direction of the effect of positive affect has to be explored.

Method

Participants

Twenty five young adults (average age 24.8 years, 12 females) participated in this study. They were right handed and had normal or corrected-to-normal vision. The experiment lasted for two hours. This study was conducted in accordance with relevant laws and institutional guidelines and was approved by the local ethics committee from the Faculty of Social Sciences. Before starting the experiment each participant read and signed an informed consent. Participants either received course credits or €16 remuneration for their participation.

Task and apparatus

Questionnaires

Participants were randomly assigned to the positive or neutral affect group so they either viewed positive or neutral movie clips. At the end of the experimental session, participants chose on a 4-choice question how they experienced each movie clip (i.e. happy, neutral, compelling or their own description).

Movie clips

The effect of movie clips on positive and neutral affect was evaluated in a separate pilot-study (in subjects not participating in the present study). In that study, participants were assigned to a neutral or positive affect condition ($n=9$ and $n=8$, respectively) and performed two sessions of a cognitive task preceded by a movie clip. Each clip was assessed by participants who provided ratings of their emotional state on the Positive and Negative Affect Scale (PANAS) before and after viewing the clip. On the PANAS, participants have to rate whether each of 20 mood descriptions (10 positive and 10 negative) currently applies, resulting in scores for positive and negative affect. Positive movie clips significantly raised positive affect, $F(1, 8) = 8.03$, $p < 0.05$, whereas negative affect remained unchanged, $F(1, 8) = 3.21$, $p > 0.05$. The neutral movie clips did not change positive, $F(1, 7) = 0.15$, $p > 0.05$, or negative affect, $F(1,8) = 3.5$, $p > 0.05$.

Experimental paradigm

The AX-CPT paradigm was used to measure context processing and cognitive control. During each AX-CPT trial participants were presented with a sequence of letters on the computer screen. Unknown to the participants, these letter sequences were constructed as trials of cue-probe pairs (types *AX*, *AY*, *BX*, *BY*). Subjects were instructed to respond to every letter with either a target or non-target response. Depending on their timing, some of these letters were considered cues and others probe in the final data-analysis.

A target response was required only when the target *X* probe was immediately preceded by an *A* cue. In every other case participants had to respond with a non-target response. Incidental No-go probes, which required participants to refrain from responding, were included to ascertain that attention would be sustained following the *B* cue. A no-go probe consisted of a red octagon with the word “stop” printed on it.

Each trial started with the presentation of a fixation cross for 1200 ms, followed by a letter (cue), appearing for 200 ms. Subjects were allowed to respond within 1200 ms (on both cue and probe stimuli). After presentation of the first letter a fixation cross was shown again (1700 ms), succeeded by the second letter (probe, 200 ms). Letters were presented in black against a white background. Target trials consisted of the sequence of the category *A* (cue) and *X* (target). For *AX* trials, the actual letters 'A' and 'X' were used. Nontarget trials were mixed with the *AX* target trials. In nontarget trials the *B* cue and *Y* probe category could be filled in by any letter of the alphabet with exception of 'A', 'K', 'X', and 'Y'.

Design

AX trials occurred prevalently (63,5%) throughout the experiment to induce a strong tendency to make a target response to the *X* probe. Other trialtypes (*AY*, *BY*, *BX*) occurred with a frequency of 9.4% throughout the experiment. Thus *A* cues were followed by an *X*-probe on 470 of the trials (87% of *A* cues) and by a *Y*-probe on 70 of the trials (13% of *A* cues). No-go trials (*B* cue-no-go probe) occurred with a frequency of 8.1% throughout the experiment. The total number of trials was 740. Response button assignments were counterbalanced. Participants were randomly assigned to one of the affect conditions: a positive affect or a neutral affect condition.

Procedure

The intention of the experiment and standard EEG procedures were explained to the participants after which they completed an informed consent form. This was followed by an *AX*-CPT task instruction and two short practice blocks. The first experimental run was preceded by a positive or neutral affect induction. Positive affect was induced by showing participants fragments of a movie clip from the *Lion King* and the *little Mermaid*. In the neutral condition, a fragment of streets scenes was presented. For both conditions, fragments lasted five minutes and were presented before each experimental

run. The experimental runs lasted for about thirty minutes and consisted of five mini-blocks.

Psychophysiological recordings

The electroencephalogram (EEG) data was recorded from 24 electrode sites (Fz, FCz, Cz, Pz, CPz, P3, C3, F3, F4, C4, P4, F5, F1, F2, F6, AF3, FC3, CP3, O1, AF4, FC4, CP4, O2, Oz) using an elastic cap with Ag/AgCl electrodes (Biosemi). Eye movements were recorded with electrode pairs placed above and below the eye (vertical electro-oculogram) and from the outer canthi of each eye (horizontal electro-oculogram). EEG signals were referenced to CMS and DRL during DC recording (<http://www.biosemi.com/faq/cms&drl.htm>). Offline, the signals were re-referenced to the mastoids. Signals were amplified with a Biosemi ActiveTwo system and the data were digitized at 256 Hz.

Data reduction and statistical analysis ERPs

Most ERPs were calculated for correct response trials only but we calculated the ERN for correct and incorrect responses to *AY* trials separately. Trials with artifacts were rejected from further analysis. EEG was corrected for ocular artifacts (Gratton, Coles, & Donchin, 1983). The corrected data were used to compute average ERPs for each stimulus and channel of interest. Five participants were excluded from analysis; three due to technical problems with the EEG equipment and two due to their ERP signals that deviated more than 2 SD from the average group ERP amplitudes.

P3b

For the P3b, EEG over electrode site Pz was filtered at 0.01 Hz (high pass) and 30 Hz (low pass), slope 24dB/octave. The cue-related P3b was aligned to a baseline of -200 to 0 ms before the average cue presentation point. *B* cues showed later peak amplitude than *A* cues, likely because they were less prevalent than *A* cues and took more time to process, which is confirmed by longer reaction times. Therefore the mean P3b

amplitude over Pz was calculated in a window of 240 to 610 ms after cue presentation for *A* cues and 350 ms to 640 ms for *B* cues, corresponding to the latencies between which the grand averages exceeded half of the peak amplitude.

CNV

For the CNV, EEG over Cz was filtered at 0.01 Hz (high pass) and 30 Hz (low pass), slope 24dB/octave. The CNV was time-locked to the probe stimulus and baseline corrected -500 to -300 before the response to the cue was given (cue-based RTs were less than 330 ms, so this correction is based on a segment before cue presentation). A segmentation window of -200 ms to 0 ms prior to the probe was used to calculate the mean amplitude.

Both CNV and P3b following the cue were analyzed by comparing the amplitudes in an analysis of variance (ANOVA) with the within-subjects factor Cue (*A* cue, *B* cue) and the between-subjects factor Affect (neutral, positive).

N2

The N2 was obtained by filtering the EEG with a 12 Hz low-pass filter. A 2 Hz high-pass filter (slope 24dB/octave) was used filter out the P3b, as the N2 tended to be absorbed by the rising flank of the P3b, which may obscure the measurement of N2 amplitude differentially across conditions (Donkers & van Boxtel, 2004; Donkers, Nieuwenhuis & van Boxtel, 2005). The signal was referenced to a baseline of -200 to 0 ms before probe presentation. Probe-locked N2 mean amplitudes over FCz, Fz, FC3 and FC4 were calculated over a window of 230 ms to 300 ms following the probe. To verify whether the N2 (and the ERN) indeed reach their maximum at FCz (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Van Veen & Carter, 2002a, 2002b) and to exclude distributional effects of affect, a repeated measures ANOVA was performed, within-subject factor Location (FCz, Fz, FC3, FC4; averaged over CPT conditions) and between-subjects factor Affect (neutral, positive). More specific comparisons were performed by means of simple contrasts at FCz in two additional ANOVAs, with within-subject factors Trialtype (*AY*, *AX*, *BX*, *BY*) and Trialtype (*BX*, *AX*, *BY*) and for both ANOVAs the between-subjects factor Affect (neutral, positive). We expect that modulations of proactive control due to the affect induction will be visible when comparing *AY* with *BY* and with *BX* and when comparing *BX* with *BY*.

ERN

For the ERN, EEG over FCz, Fz, FC3 and FC4 were filtered off-line with a 12 Hz low-pass filter and a 2 Hz high-pass filter, slope 24dB/octave (Donkers & van Boxtel, 2004; Donkers et al., 2005). A window of 0 ms to 100 ms after the probe-related response was used to calculate the mean amplitude of the ERN on *AY* trials for correct and incorrect responses. It was aligned to -100 to 0 ms before a response was given. Similar to the N2 analysis, we first investigated distributional effects of affect and verified whether the ERN reaches its maximum at FCz. Thus, we performed an ANOVA, on the ERPs of pooled correct and error *AY* trials, with within-subject factor Location (FCz, Fz, FC3, FC4) and between-subjects factor Affect (neutral, positive).

To test the effect of affect on the ERN in errors versus correct performance at FCz, we performed a repeated measures ANOVA with the within-subject factor *AY* (correct, error) and a between-subjects factor Affect (neutral, positive).

Behavioral analysis

Trials with reaction times shorter than 150 ms and longer than 1100 ms were removed from the analyses. Two pairs of repeated measures ANOVAs were performed, each pair consisting of an accuracy and reaction times analysis. One pair of analyses was performed with the within-subject factor Trialtype (*AY*, *AX*, *BX*, *BY*) and the other pair with the within-subject factor Trialtype (*BX*, *AX*, *BY*) and both with the between-subjects factor Affect (positive, neutral). Again we used simple contrasts to test changes in *AY* compared to *BY*, *BX* and *AX* and changes in *BX* compared to *BY*.

Results

Subjective measurements

The question presented at the end of the experiment indicated that the positive movie clips were rated positively in most of the cases (7 of 9 participants, one rating was missing and one participant rated the positive movies as neutral). The neutral movie clips were judged as neutral by most of the participants (8 out of 10, one missing and one participant rated the neutral movie as boring).

Figure 2

Reaction times by Trialtype and Affect induction. Error bars represent standard errors.

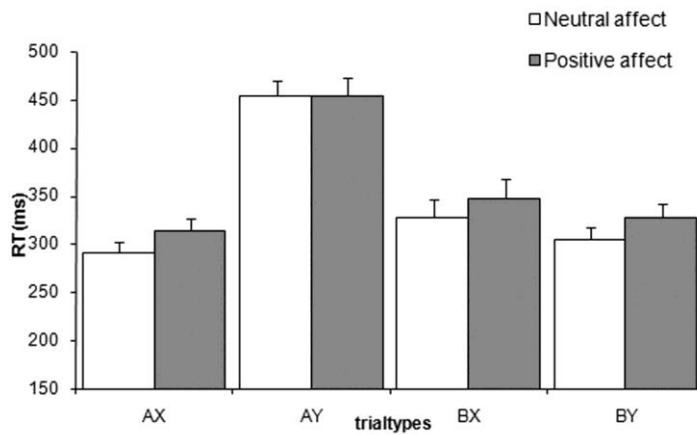
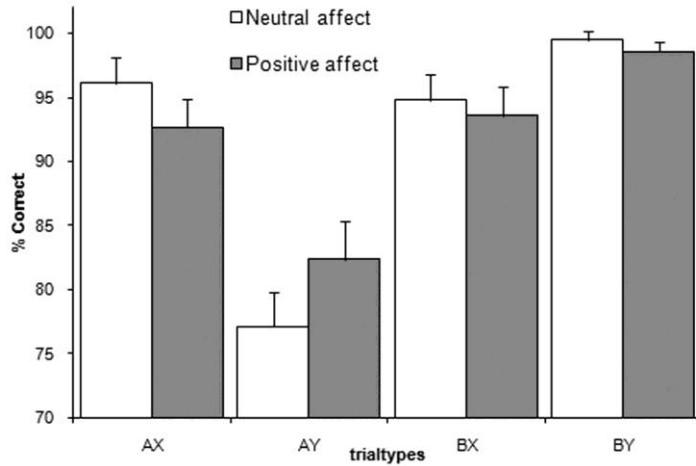


Figure 3

Accuracy by Trialtype and Affect induction. Error bars represent standard errors.



Behavioral results

Reaction time

Figures 2 and 3 show the mean reaction time and mean accuracy for responses on AX, AY, BX and BY trials. The reaction times yielded a significant main effect of Trialtype (AY, AX, BX, BY), $F(3, 54) = 155.35, p < 0.001$. Simple contrasts revealed that responses on AY trials ($M_{AY} = 455$ ms) were slower than responses on AX ($M_{AX} = 303$ ms), $F(1, 18) = 309.20, p < 0.001$, BX ($M_{BX} = 338$), $F(1, 18) = 144.00, p < 0.001$ and BY trials ($M_{BY} = 317$ ms), $F(1, 18) = 194.72, p < 0.001$. We found no interaction with Affect, $F(3, 54) = 0.87, p = 0.42$, nor a main effect of Affect, $F(1, 18) = 0.73, p = 0.40$.

The second analysis with Trialtype (BX, AX, BY) revealed a main effect of Trialtype as well, $F(2, 36) = 18.21, p < 0.001$, but no interaction with Affect, $F(2, 36) = 0.04, p = 0.91$, nor a main effect of Affect, $F(1, 18) = 1.21, p = 0.29$. Simple contrasts showed

that performance on *BX* trials was slower than performance on *AX*, $F(1, 18) = 22.54, p < 0.001$, and *BY* trials, $F(1, 18) = 13, p < 0.01$.

Accuracy

The accuracy analysis indicated a main effect of Trialtype (*AY*, *AX*, *BX*, *BY*), $F(3, 54) = 60.99, p < 0.001$. Simple contrasts revealed that performance on *AY* trials ($M_{AY} = 80\%$) was less accurate than performance on *AX* ($M_{AX} = 94\%$), $F(1, 18) = 66.78, p < 0.001$, *BX* ($M_{BX} = 94\%$), $F(1, 18) = 51.82, p < 0.001$ and *BY* trials ($M_{BY} = 99\%$), $F(1, 18) = 115.35, p < 0.001$. There was a nearly significant interaction between Trialtype and Affect, $F(3, 54) = 3.00, p = 0.07$.

Simple contrasts indicated that performance on *AY* trials ($M_{AY-pos} = 82\%$) versus *AX* trials was less impaired in the positive affect condition ($M_{AX-pos} = 93\%$), as opposed to the neutral condition ($M_{AY-neutr} = 77\%$, $M_{AX-neutr} = 96\%$), $F(1, 18) = 5.83, p < 0.05$. No main effect of Affect was found, $F(1, 18) = 0.001, p = 0.98$.

The second analysis yielded a main effect of Trialtype (*BX*, *AX*, *BY*) as well, $F(2, 36) = 13.59, p < 0.001$, but no interaction with Affect, $F(2, 36) = 0.87, p = 0.43$, nor a main effect of Affect, $F(1, 18) = 0.71, p = 0.41$. Simple contrasts showed that performance on *BX* trials was less accurate than performance on *BY* trials, $F(1, 18) = 17.48, p < 0.01$.

Figure 4

Grand average ERPs (P3) elicited by the A cue (solid lines) and the B cue (dashed lines) at Pz, separately for positive and neutral affect. Dark and light gray horizontal bars indicate windows of analysis for respectively A and B cues.

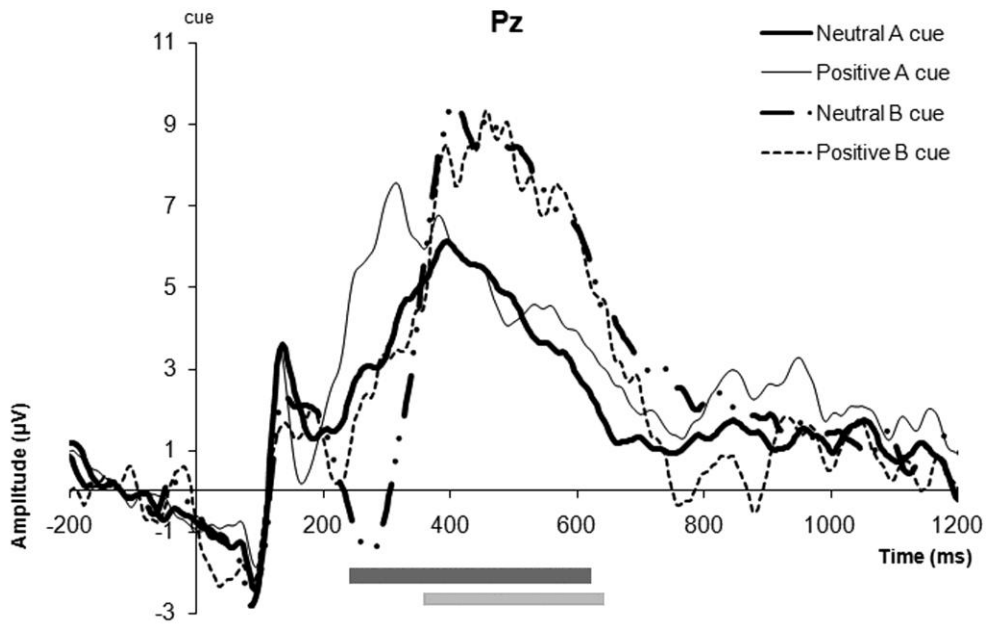
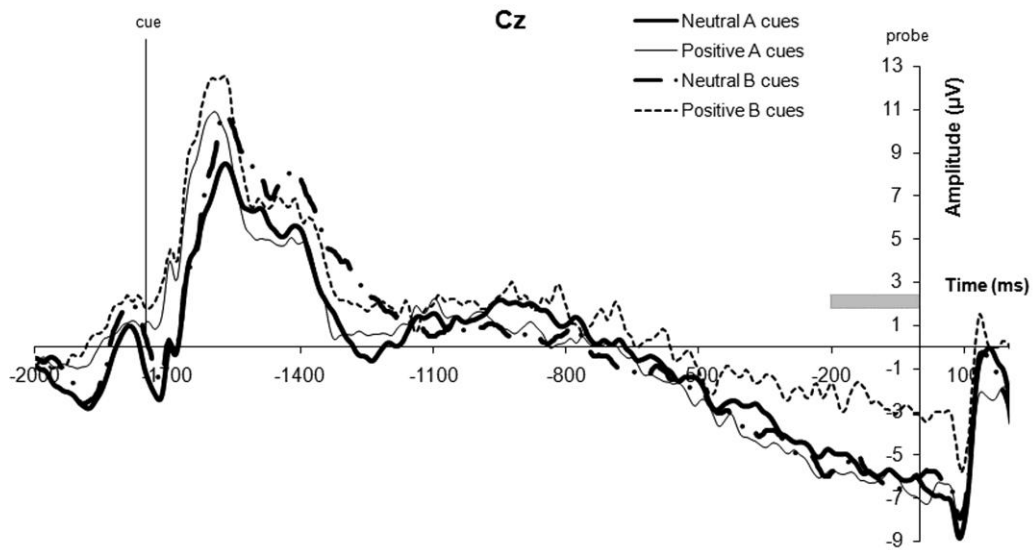


Figure 5

Grand average ERPs (CNV) evoked by the A cue (solid lines) and the B cue (dashed lines) at Cz, separately for positive and neutral affect. The gray horizontal bar indicates window of analysis.



ERP Results

P3

Figure 4 shows the P3b amplitude elicited by A and B cues. At Pz, the B cue elicited a larger positive deflection ($M_{B-cue} = 7.48 \mu\text{V}$) than the A cue ($M_{A-cue} = 4.74 \mu\text{V}$), $F(1, 18) = 14.61$, $p < 0.01$. No main effect of Affect was present, $F(1, 18) = 0.03$, $p = 0.86$, nor an interaction of Affect and Cue, $F(1, 18) = 0.62$, $p = 0.44$.

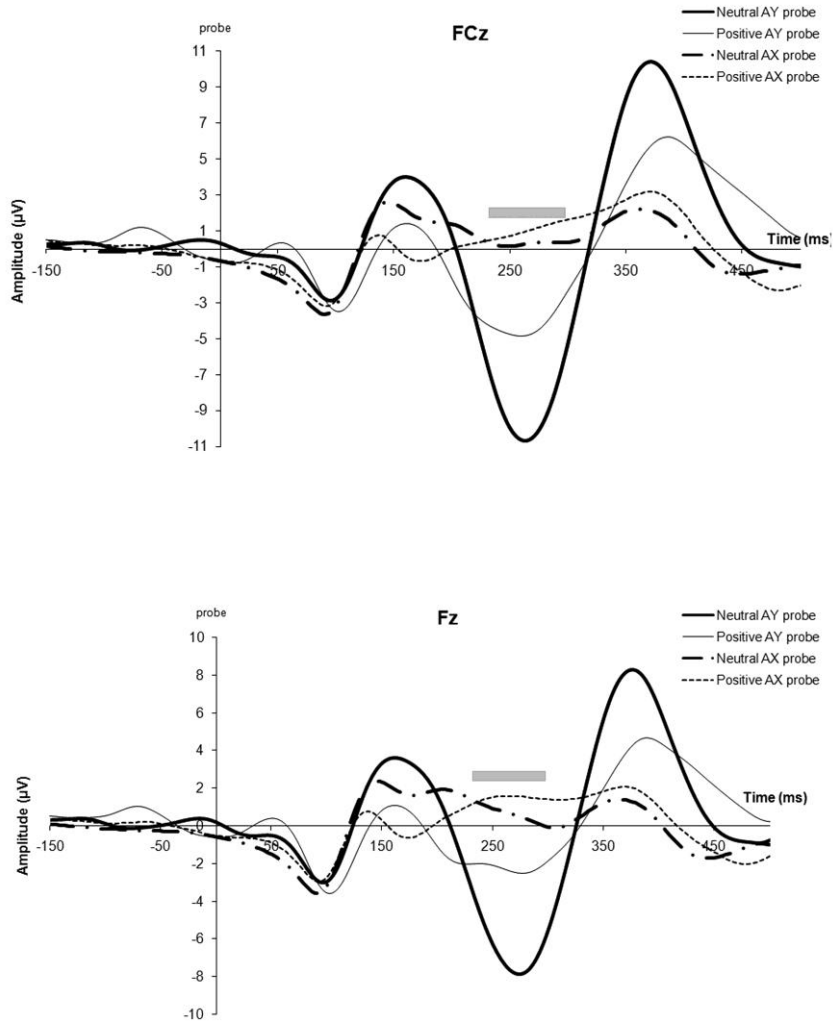
CNV

Figure 5 shows the CNV amplitude elicited by A and B cues. Note that the CNV was measured time-locked to the probe; thus, the $t = 0$ represents probe presentation. At Cz,

the main effect of Cue for CNV amplitudes showed a nearly significant effect, $F(1, 18) = 3.34$, $p = 0.08$. Activity in response to the A cue ($M_{A-cue} = -6.09 \mu\text{V}$) was more negative than to the B cue ($M_{B-cue} = -4.49 \mu\text{V}$). No main effect of Affect was present, $F(1, 18) = 0.39$, $p = 0.54$. However, the interaction of Affect with Cue did almost reach significance, $F(1, 18) = 4.24$, $p = 0.05$. This effect is mainly caused by a reduced CNV to the B-cue and a slightly increased CNV to the A-cue in the positive affect group ($M_{posBcue} = -3.05$, $M_{neutrBcue} = -5.93$, $M_{posAcue} = -6.45$, $M_{neutrAcue} = -5.73$).

Figure 6

a. Grand average ERPs (N2) evoked at FCz, Fz, FC3 and FC4 by the nontarget Y probes following an A cue (solid lines) and the target X probes following an A cue (dashed lines) at FCz, separately for positive and neutral affect. The gray horizontal bar indicates window of analysis.



POSITIVE AFFECT MODULATES COGNITIVE CONTROL

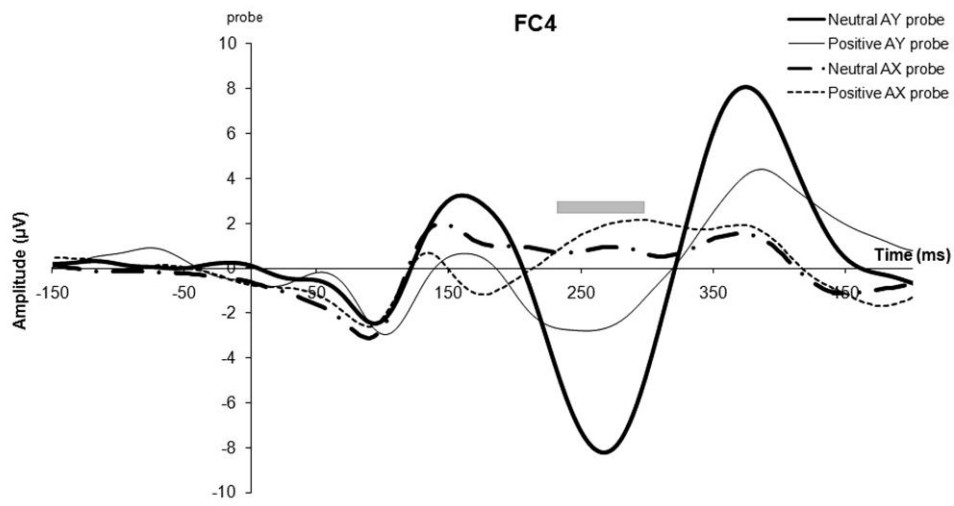
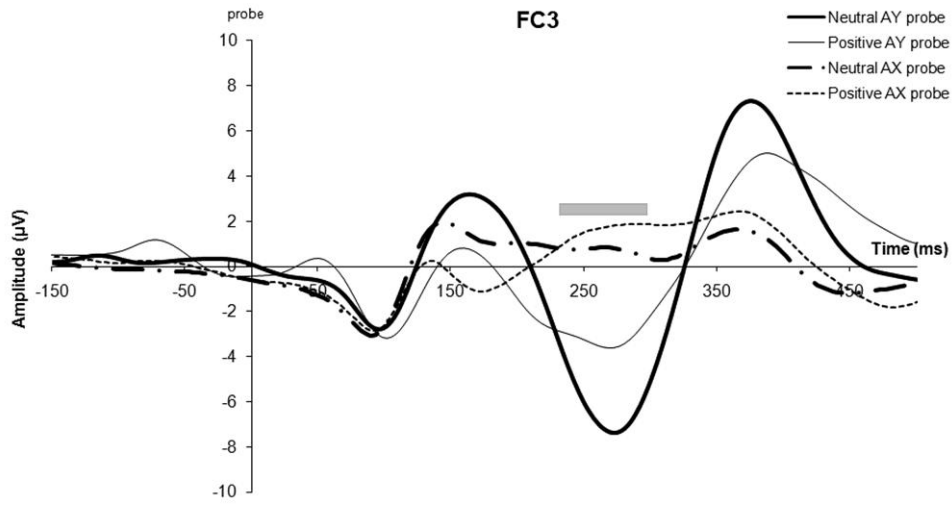
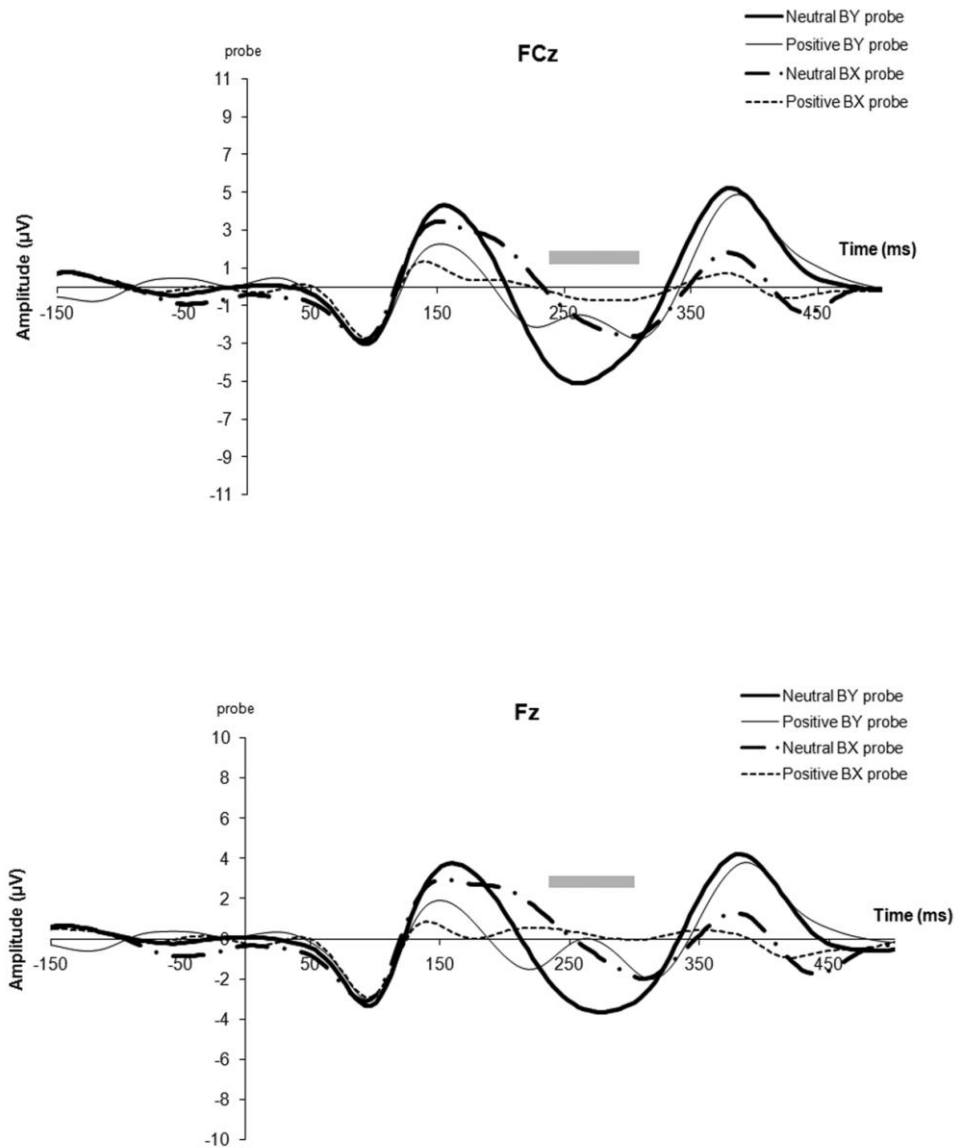
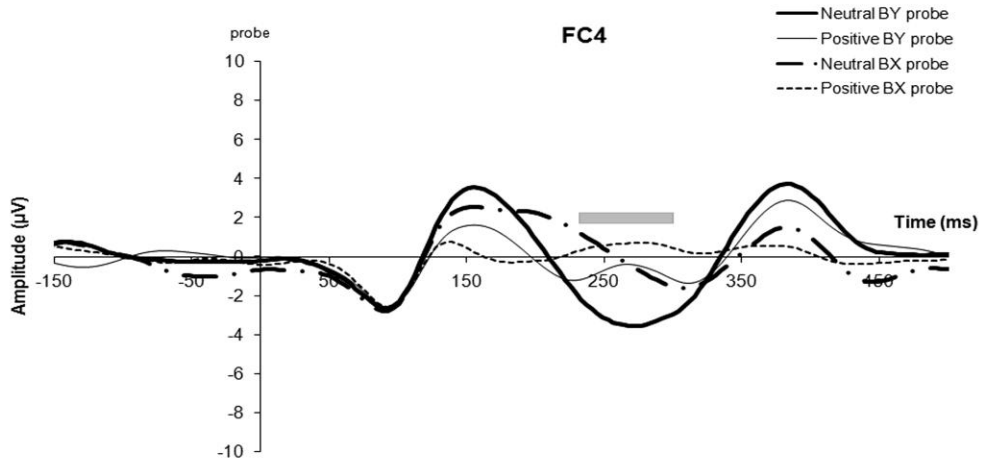
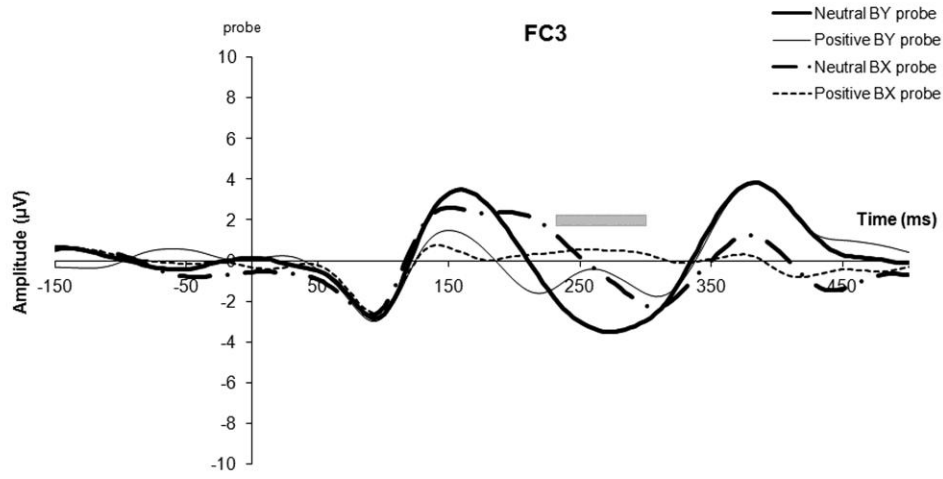


Figure 6

b. Grand average ERPs (N2) evoked at FCz, Fz, FC3 and FC4 by the nontarget Y probes succeeding an B cue (solid lines) and the target X probes following an B cue (dashed lines) at FCz, separately for positive and neutral affect.



POSITIVE AFFECT MODULATES COGNITIVE CONTROL



N2

Figure 6 shows the N2 elicited by the different trial types (*AX*, *AY*, *BX*, *BY*), time-locked to the probe and at different locations, FCz, Fz, FC3, FC4. The first ANOVA revealed a more negative N2 on FCz ($M_{FCz} = -2.54 \mu\text{V}$) compared to the other locations, $F(3, 54) = 38.21$, $p < 0.001$, $M_{Fz} = -1.29 \mu\text{V}$, $M_{FC3} = -1.35 \mu\text{V}$, $M_{FC4} = -1.27 \mu\text{V}$. However, no significant interaction between Location and Affect, $F(1, 18) = 1.27$, $p = 0.30$ was present.

The second ANOVA, specific for location FCz, revealed a main effect of Affect, $F(1, 18) = 6.39$, $p < 0.05$. The N2 amplitudes evoked in the positive affect condition were smaller than in the neutral affect condition ($M_{pos} = -1.41 \mu\text{V}$, $M_{neutr} = -3.68 \mu\text{V}$). Furthermore, a main effect of Trialtype (*AY*, *AX*, *BX*, *BY*), $F(3, 54) = 72.30$, $p < 0.001$, on N2 amplitude was observed.

Simple contrasts showed that the N2 amplitude observed after *AY* trials ($M_{AY} = -6.66 \mu\text{V}$) was significantly larger than after *AX* trials ($M_{AX} = 0.63 \mu\text{V}$), $F(1, 18) = 81.43$, $p < 0.001$, *BX* trials ($M_{BX} = -1 \mu\text{V}$), $F(1, 18) = 98.55$, $p < 0.001$ and *BY* trials ($M_{BY} = -3.15 \mu\text{V}$), $F(1, 18) = 44.96$, $p < 0.001$. These effects were modulated by affect; the increase in N2 amplitude on *AY* versus *AX* trials was significantly smaller in the positive affect condition ($M_{AY-pos} = -4.29 \mu\text{V}$, $M_{AX-pos} = 0.99 \mu\text{V}$) than in the neutral affect condition ($M_{AY-neutr} = -9.02 \mu\text{V}$, $M_{AX-neutr} = 0.27 \mu\text{V}$), $F(1, 18) = 6.15$, $p < 0.05$.

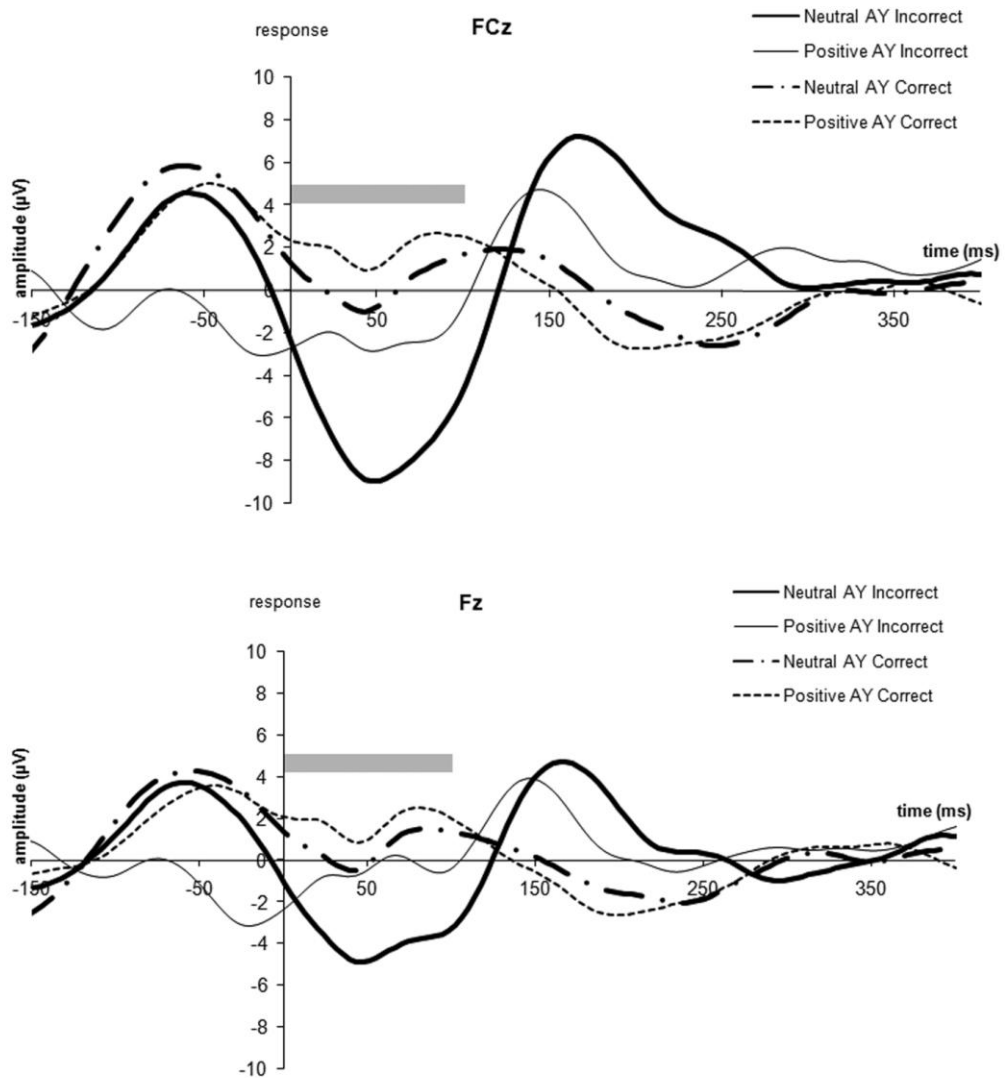
Similar results were observed for the interaction of Affect and trial types *AY* versus *BX*, $F(1, 18) = 10.91$, $p < 0.01$. The difference in N2 amplitudes between *AY* and *BX* trials was larger in the neutral affect condition ($M_{BX-neutr} = -1.49 \mu\text{V}$) as opposed to the positive affect condition ($M_{BX-pos} = -0.52 \mu\text{V}$). Likewise, the results of the contrast comparing *AY-BY* in interaction with Affect revealed a nearly significant effect, $F(1, 18) = 3.9$, $p = 0.06$. The N2 amplitude was more negative on *AY* trials as opposed to *BY* trials ($M_{BY} = -3.14 \mu\text{V}$). Additionally, the increase in negative activity of the N2 on *AY* versus *BY* trials was significantly larger in the neutral affect condition ($M_{BY-neutr} = -4.78 \mu\text{V}$), as compared to the positive affect condition ($M_{BY-pos} = -1.81 \mu\text{V}$).

The third ANOVA yielded a main effect of Trialtype (*BX*, *AX*, *BY*), $F(2, 36) = 54.61$, $p < 0.001$, on N2 amplitudes, but no main effect of Affect, $F(1, 18) = 2.67$, $p < 0.12$. Furthermore, simple contrasts showed that the N2 amplitude observed after *BX* trials was significantly larger than after *AX* trials, $F(1, 18) = 14.89$, $p < 0.01$, but significantly smaller compared to *BY* trials, $F(1, 18) = 65.81$, $p < 0.001$. This *BX-BY* effect was

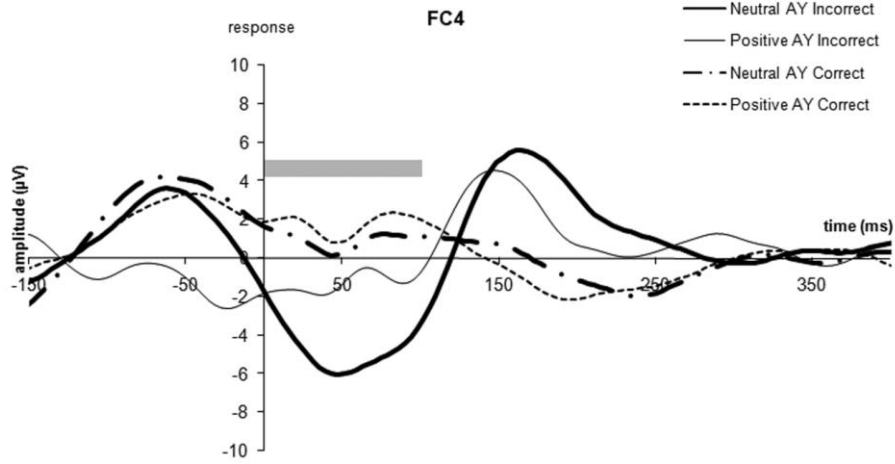
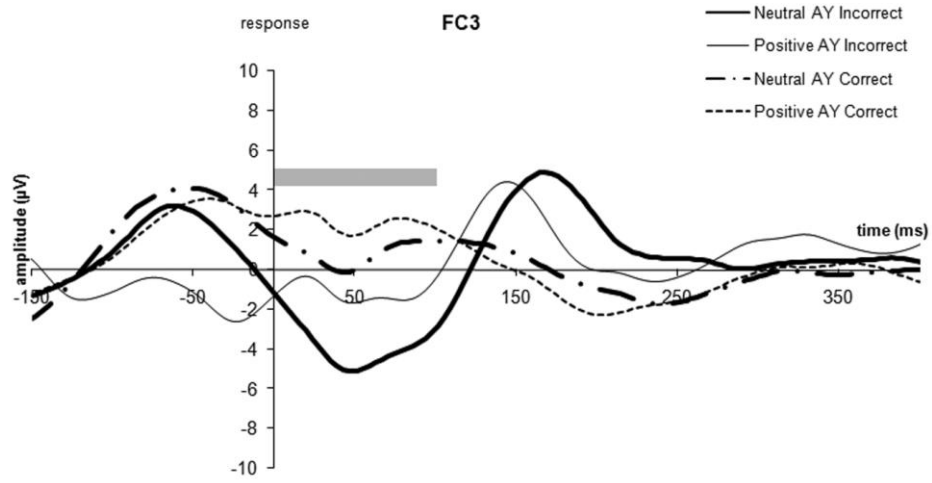
modulated by affect, $F(1, 18) = 10.28, p < 0.01$, although not in an expected direction; this effect was mainly caused by an increased difference in N2 amplitudes between positive and neutral affect on *BY* trials, ($M_{BY-pos} = -1.81 \mu V, M_{BY-neutr} = -4.48 \mu V, t(18) = -2.80, p \leq 0.05$), while there was no significant effect of affect on *BX* trials ($M_{BX-pos} = -0.52 \mu V, M_{BX-neutr} = -1.49 \mu V, t(18) = -1.05, p = 0.31$).

Figure 7

Grand average ERPs (ERN) evoked at FCz, Fz, FC3 and FC4 by correct and incorrect responses on AY trials at FCz, separately for positive and neutral affect. The gray horizontal bar indicates window of analysis.



POSITIVE AFFECT MODULATES COGNITIVE CONTROL



ERN

As anticipated, only the *AY* condition elicited sufficient numbers of errors to reliably calculate an ERN. The mean number of *AY* error trials used to create the ERN for each subject was 13 and for correct trials 46. Figure 7 shows the ERN elicited by *AY* correct and error trials at FCz, Fz, FC3 and FC4. The ERN was significantly more negative on FCz ($M_{FCz} = -1.68 \mu\text{V}$) compared to the other locations, $F(3, 54) = 17.04, p < 0.001, M_{Fz} = -0.44 \mu\text{V}, M_{FC3} = -0.40 \mu\text{V}, M_{FC4} = -0.79 \mu\text{V}$. Additionally, the difference between positive ($M_{pos} = -0.07 \mu\text{V}$) and neutral ERN ($M_{neutr} = -3.3 \mu\text{V}$) amplitude was larger at FCz compared to the other locations ($M_{Fz_pos} = 0.61 \mu\text{V}, M_{Fz_neutr} = -1.49 \mu\text{V}, M_{FC3_pos} = 0.66 \mu\text{V}, M_{FC3_neutr} = -1.47 \mu\text{V}, M_{FC4_pos} = 0.32 \mu\text{V}, M_{FC4_neutr} = -1.89 \mu\text{V}$), $F(1, 18) = 3.59, p < 0.05$.

The second ANOVA, specific for location FCz, revealed a significant difference between correct ($M_{AY_correct} = 1.15 \mu\text{V}$) and incorrect responses ($M_{AY_incorrect} = -4.51 \mu\text{V}$) on the ERN amplitude, $F(1, 18) = 65.32, p < 0.001$. The analysis yielded a significant main effect of Affect as well, $F(1, 18) = 10.33, p < 0.01$. The ERN was (on *AY* correct and error trials) more negative in the neutral condition ($M_{AY_neutr} = -3.3 \mu\text{V}$) compared to the positive affect condition ($M_{AY_pos} = -0.07 \mu\text{V}$). Most important, a significant interaction effect of response correctness on *AY* and Affect was found, $F(1, 18) = 4.54, p < 0.05$. This effect is mainly the result of *AY* error trials that showed a significantly reduced ERN amplitude in the positive affect condition ($M_{AY_pos} = -2.15 \mu\text{V}$) compared to the neutral affect condition ($M_{AY_neutr} = -6.87 \mu\text{V}$), $t(18) = -4.04, p \leq 0.001$.

Discussion

The main objective of this study was to investigate the modulating influence of positive affect on cognitive control processes. The current study shows that positive affect enhances flexibility and modulates the ability to evaluate performance, but it does not change goal maintenance or preparation.

Relation to other studies

Both goal-driven accounts (Braver et al., 2001; Braver et al., 2007; Cohen et al., 2002) and the flexibility-maintenance theory (Dreisbach & Goschke, 2004; Dreisbach et al., 2005) predicted that positive affect would modulate proactive control, although in a different direction. Our results provide moderate support for part of these theories, although not entirely as predicted.

Our study revealed improved flexibility with positive affect; an improvement of *AY* performance, where a cue-induced bias has to be overcome by using probe information. This improved flexibility is similar to the findings of Dreisbach (2006), but unlike Dreisbach we found no additional maintenance deficiency (impairment on *BX* and *BY*). As we used a relatively long interval between cue and probe, a maintenance deficiency would have resulted in poor *BX* and *BY* performance. The behavioral data are largely confirmed by the ERP data: the cue related P3b was unaffected by positive affect, while the ERN and N2 amplitude decreased in *AY* trials and the N2 remained equal for both affect conditions in *BX* trials.

The CNV data indicate that the cue-information was adequately prepared in the positive affect group, but not significantly different from the neutral affect group. However, the differential effect of positive affect on *A* and *B* cues in the CNV (although this effect failed to reach significance, $p=0.05$) suggest some support for a goal-driven account, when explaining this effect in terms of a Stimulus Preceding Negativity. The CNV consists of a response preparation component and stimulus anticipation component (Van Boxtel & Böcker, 2004). In order to perform the task correctly, *A* and *B* cues both ask for motor preparation whereas a *B* cue is more predictive because it asks for a nontarget response regardless of the probe presented. Therefore, anticipation to *B* cues for the upcoming probe is less necessary compared to *A* cues, which is reflected in a reduced SPN subsequent to a *B* cue. This indicates that the positive affect group more selectively prepare (in a proactive way) for task relevant information.

There are three possible explanations for the differential effect of positive affect on proactive control in the current study relative to that of Dreisbach (2006). First, the difference in results may be the result of the type of affect induction. The current study induced positive affect before each experimental run (to avoid disturbing effects of, for example, affective pictures on the ERPs); Dreisbach (2006) continuously presented

affective pictures throughout the experiment. The pre-experimental affect induction may have had a milder, more tonic effect compared to the repetitive presentation of affective stimuli. However, several studies (Forgas & Moylan, 1987; Gross & Levenson, 1995; Hagemann et al., 1999; Harle & Sanfey, 2007) indicate that movieclips are quite effective in eliciting a positive mood that can last for approximately 30 minutes (Sinclair, Mark, Enzle, Borkovec, & Cumbleton., 1994). Brief electrical stimulation of basolateral nuclei in the amygdala (in rats) resulted in a similar time course of DA release from the VTA into the striatum (up to 30 minutes) (Floresco, Yang, Philips, & Blaha, 1998).

Second, one of the neural mechanisms mentioned by Dreisbach and colleagues explaining the maintenance-flexibility theory (Dreisbach & Goschke, 2004; Dreisbach et al., 2005) is that positive affect increases phasic DA in PFC which improves updating of working memory and switching of cognitive set (flexibility), but reduces stability of representations in working memory, which is the same mechanism as mentioned by Braver et al. to explain updating and maintenance in WM (Braver et al., 1999; Braver & Cohen, 2000). This mechanism may nicely explain the results found by Dreisbach et al., however, this explanation is based on the assumption that positive affect mainly results in a phasic dopamine changes. If positive affect mainly produces a tonic dopamine change, which cannot be excluded and may depend on the type of affect induction, this will probably lead to predictions as derived from goal-driven accounts (increased maintenance). Future studies relating parametrical changes in affect (from unexpected reward to continuously presented positive stimuli) to cognitive changes in working memory maintenance vis-à-vis updating or flexibility could provide more clarity with respect to tonic and phasic changes in positive affect and in what way they modulate cognitive performance.

Third, it could be argued that maintenance impairments of affect would only show up under excessive working-memory maintenance demands, for instance with longer inter-stimulus intervals (stretching over many seconds) than were used in the current study. Some drug studies have failed to find effects of DA on working memory (Turner et al., 2003), although the effect may also depend largely on individual differences in baseline DA (Kimberg & D'Esposito, 2003).

With the exception of the modulatory effect of positive mood on the *B* cue related CNV, suggesting more selective anticipation; positive affect did not change proactive

updating or maintenance. The increased flexibility with positive affect, as indicated by relatively few *AY* errors, may be explained by the modulatory effect of DA in the striatum. DA increases in the striatum are involved in some forms of cognitive flexibility (i.e. switching between relevant stimulus information), as substantiated by pharmacological fMRI studies with healthy controls and Parkinson's patients on and off medication (Cools, Barker, Sahakian, & Robbins, 2001; Cools, Sheridan, Jacobs, & D'Esposito, 2007). These studies indicated that a DA enhancement in the striatum improves the efficacy of using incoming response-relevant stimulus information to control behavior. Moreover, recent modeling work (O'Reilly, 2006; Seamans & Yang, 2004) poses that the modulatory effect of DA on projections from the basal ganglia to the PFC may enable more rapid and selective updating compared to the slower and more diffuse effects of direct projections of DA to the PFC. Enhanced flexibility results in diminished conflict and/or a decreased need for inhibition on *AY* trials in the positive affect condition compared to the neutral affect condition. This effect on action selection in reactive control is reflected in the smaller N2 amplitude.

In the current study we assumed that the N2 reflects response competition or conflict and indicates the need for reactive control. In contrast to this view, a recent review points out that the N2 reflects the actual control process (see Folstein & van Petten, 2008). Based on this interpretation, we would have to conclude that control is decreased in the positive compared to the neutral affect condition on *AY* trials, which may indicate a reactive control deficiency or the result of reduced proactive control. However, the N2 amplitude on *AY* trials is still significantly larger than on *AX* trials in the positive affect condition ($M_{AY-pos} = -4.29 \mu V$, $M_{AX-pos} = 0.99 \mu V$, $t(8) = -5.77$, $p < 0.001$) and performance improved on *AY* trials, which indicates that there does not seem to be a reactive control deficiency. Likewise, the cue-based ERPs were not significantly reduced and *BX* and *BY* performance costs did not increase with positive affect, indicating that proactive control was not impaired.

Our findings also seem to be in line with findings from Harmon-Jones and colleagues (Gable & Harmon-Jones, 2008; Harmon-Jones, Lueck, Fearn, & Harmon-Jones, 2006). They posed that positive affect states vary in intensity of approach motivation which differentially modulates attention; i.e. high approach motivation narrows attention whereas low approach motivation broadens attention. According to Gable and Harmon-Jones' (2008) definition of low approach motivation, the current

study employed a positive affect induction with low approach motivation, which may thus have broadened attention. This seems to resemble our findings: a broader attentional set may reduce a task-induced *A*-cue bias and enable updating the latest (probe) information.

Orthogonal to theories that emphasize the role of dopamine in positive affect, Berridge (2007) posed that an increase in dopamine enhances the motivation to gain reward. Comparable to drug induced dopaminergic changes that increase ‘wanting’, the positive film clips may have enhanced the motivation to gain reward. However, this may be difficult to distinguish from ‘liking’ or positive affect based on behavioral measures and it would not have necessarily led to different predictions.

Positive affect and evaluative control

The reduced ERN as a result of positive affect is in line with other studies that investigated the modulating effect of affect on the ERN: the amplitude of the ERN was found to be larger for individuals high in negative emotionality than for individuals low in negative emotionality (Hajcak, McDonald, & Simons, 2004; Luu, Collins, & Tucker, 2000). Additionally, negative affect, as induced by IAPS pictures and negative feedback, results in a larger ERN (Wiswede, Münte, Goschke, & Rüsseler, 2009; Wiswede, Münte, & Rüsseler, 2009) compared to embodied positive affect, established by mimicking the features of a smile, which lead to a smaller ERN (Wiswede, Münte, Krämer, & Rüsseler, 2009).²

The reduced ERN with positive affect also seems to correspond to results of studies on dopaminergic modulations of the striatum (Frank & O’Reilly, 2006; Zirnheld et al., 2004; Ito & Kitagawa, 2006): positive affect may have increased DA in the striatum, explaining why participants in the positive affect condition might not have been able to show the DA dips during error processing, as indicated by the reduced ERN in the positive compared to neutral affect conditions.

At first glance, our interpretation that increased DA levels in the striatum due to positive affect were responsible for the reduced ERN appears to be contradicted by

² The studies by Wiswede et al. were published after we had already submitted and revised a previous version of this study and were therefore only referred to in the discussion.

studies in which DA agonists caffeine and amphetamine caused a larger ERN and improved action monitoring (de Bruijn et al., 2004; Tieges et al., 2004). The crucial difference, however, is that these drugs release DA not only in the striatum, but also in MFC (Vollenweider et al., 1998; Acquas et al., 2002). We argue that the increased ERN in those studies was a reflection of a DA increase mostly in the MFC, where DA-induced processing benefits should result in enhanced effects on evaluative control, as expressed in the ERN. Converging evidence for the interpretation that the locus of increased DA release critically determines whether the ERN increases or decreases comes from studies that investigate the ability to learn from negative feedback. Simulations of a variety of DA-related effects by Frank et al. (2004; 2005) have indicated that a smaller ERN is elicited in the same conditions where learning from negative feedback is diminished. According to Frank et al., striatal increase of DA limits the phasic reductions (dips) of DA due to errors and negative feedback. As a result, a less pronounced dopaminergic error signal is projected to the MFC, hence giving rise to a smaller ERN. In line with this reasoning, we predict that positive affect will reduce the ability to learn from negative feedback.

Finally, the interpretation of our cue-based ERPs relies on a null finding and therefore has to be interpreted with caution. It is conceivable that this either results from overly conservative measurements or the mood induction may not have been strong enough to affect cue-based processes in the current experimental paradigm as previously discussed.

Limitations

A few limitations of the current study should be mentioned. To begin with, we did not find an effect of affect on the reaction times of the AX-CPT but only on accuracy. Note, however, that the CPT was originally designed to measure accuracy (hits, misses, and false alarms) and is not specifically sensitive to differences in reaction times. Most studies using the CPT only report effects of their experimental manipulation in terms of accuracy scores (for a review see Riccio, Waldrop, Reynolds, & Lowe, 2001).

Note that in the current study, no negative affect induction was included for comparison. It could be argued that the effect of positive affect is merely due to

increased arousal and a similar finding could have been found after inducing negative affect because emotional states in general are thought to increase arousal (LeDoux, 1996). Or, the effects found in the current study could be mediated by a moderate negative affect induced by the neutral affect condition, although this seems unlikely, since the affective rating of the movie clips used in the current study does not suggest that the neutral movie clips induce negative affect.

A negative affect induction was not included in this study because a number of studies comparing negative and positive affect have already demonstrated that the effects of positive affect on flexibility cannot be attributed to a result of increased arousal (Isen, 1997; Dreisbach & Goschke, 2004, Dreisbach, 2006). Higher arousal states alone do not improve flexibility but are thought to merely increase the likelihood of the dominant response (Berlyne, 1967; Easterbrook, 1959). Moreover, Wiswede, Münte, Krämer and Rüsseler (2009) recently showed that negative affect, induced by IAPS pictures, increases the ERN compared to positive or neutral IAPS pictures. Negative affect is not as clearly related to DA as positive affect, i.e., a decrease in DA produces flattened affect rather than negative affect (Hyman & Nestler, 1993).

Conclusion

The ability to adapt to a constantly changing environment asks for maintenance of currently task-relevant information against distracting irrelevant information, and requires flexibility to update new incoming relevant information. Positive affect modulates flexibility and the ability to evaluate performance (N2 and ERN) in an AX-CPT task, whereas preparation after the cue and maintenance of cue information remains equal to the neutral affect condition (P3b and CNV). The effect of positive affect on the ERN and improved flexibility is attributed to a dopaminergic increase in the striatum.