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Facing competition: Neural mechanisms underlying parallel programming of antisaccades and prosaccades

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ABSTRACT

The antisaccade task is a prominent tool to investigate the response inhibition component of cognitive control. Recent theoretical accounts explain performance in terms of parallel programming of exogenous and endogenous saccades, linked to the horse race metaphor. Previous studies have tested the hypothesis of competing saccade signals at the behavioral level by selectively slowing the programming of endogenous or exogenous processes e.g. by manipulating the probability of antisaccades in an experimental block. To gain a better understanding of inhibitory control processes in parallel saccade programming, we analyzed task-related eye movements and blood oxygenation level dependent (BOLD) responses obtained using functional magnetic resonance imaging (fMRI) at 3T from 16 healthy participants in a mixed antisaccade and prosaccade task. The frequency of antisaccade trials was manipulated across blocks of high (75%) and low (25%) antisaccade frequency. In blocks with high antisaccade frequency, antisaccade latencies were shorter and error rates lower whilst prosaccade latencies were longer and error rates were higher. At the level of BOLD, activations in the task-related saccade network (left inferior parietal lobe, right inferior parietal sulcus, left precentral gyrus reaching into left middle frontal gyrus and inferior frontal junction) and deactivations in components of the default mode network (bilateral temporal cortex, ventromedial prefrontal cortex) compensated increased cognitive control demands. These findings illustrate context dependent mechanisms underlying the coordination of competing decision signals in volitional gaze control.

1. Introduction

Executive control allows the flexible adaptation of behavior to the context of a situation, for example by suppressing an automatic response (Munoz & Everling, 2004). This inhibitory top-down control over reflex-like processes is termed *prepotent response inhibition* (Friedman & Miyake, 2004). It summons processes that regulate the selection of a weaker, but task-relevant response over a competing strong, but task-irrelevant response (Miller & Cohen, 2001).

Response inhibition can be modeled experimentally in the antisaccade task (Hallett, 1978), which requires participants to inhibit a reflex-like prosaccade to a sudden-onset peripheral target in favor of a volitional saccade to the mirrored target location (for a review see Hutton & Ettinger, 2006; Munoz & Everling, 2004; Pierrot-Deseilligny, Milea, & Mu, 2004).

Studies using functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) as well as single cell recordings in nonhuman primates have revealed a network of fronto-parietal and subcortical structures that is involved in the generation and control of saccadic eye movements (Jamadar, Fielding, & Egan, 2013; McDowell, Dyckman, Austin, & Clementz, 2008; Munoz & Everling, 2004). Cortical components of this network include the visual cortex, posterior parietal cortex (PPC) and frontal areas such

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as the frontal eye field (FEF) and supplementary eye field (SEF). Subcortical components comprise the basal ganglia, thalamus and superior colliculus (SC).

Within this network, increased activations in posterior parietal cortex, FEF and SEF are observed in antisaccades, thought to be mediating processes of vector transformation and response generation. Additionally recruited areas include the inferior frontal gyrus (IFG) and dorsolateral prefrontal cortex (DLPFC), areas thought to be involved in inhibition, goal maintenance and updating processes (Hutton & Ettinger, 2006; McDowell et al., 2008; Munoz & Everling, 2004). Conversely, a number of areas show reduced activations during saccades compared to rest or during antisaccades compared to prosaccades, incl. occipital gyrus, precentral and postcentral gyrus, middle (MTG) and superior temporal gyrus (STG), anterior cingulate, middle frontal gyrus (MFG) and ventromedial prefrontal gyrus (VMPFC) (Ettinger et al., 2008b; Pierce & McDowell, 2016; Sweeney et al., 1996). These areas likely mediate non-task processes and are reduced in activation in order to mediate successful task performance (Anticevic et al., 2012).

There are diverging opinions concerning the cognitive mechanisms underlying antisaccade performance. Classical approaches assume a consecutive processing sequence, in which the reflex-like prosaccade is initially cancelled by processes of goal redefinition (Hallett & Adams, 1980), a cancellation signal (Guitton, Buchtel, & Douglas, 1985) or an inhibitory signal (Roberts, Hager, & Heron, 1994), followed by the programming of the antisaccade. According to this view, antisaccade errors are a result of the insufficient inhibition or cancellation of the reflex-like prosaccade motor program.

More recent theoretical accounts explain antisaccade performance in terms of parallel programming, often linked to a competition model or the horse race metaphor (Hutton & Ettinger, 2006; Kristjánsson, 2007; Massen, 2004; Munoz & Everling, 2004; Reuter & Kathmann, 2004). The competition model assumes that co-ordinates for the exogenously triggered prosaccade and the endogenously initiated antisaccade are programmed immediately after the stimulus is presented. These signals compete until one of them reaches a threshold for execution (Hutton, 2008; Massen, 2004). These theoretical accounts thus postulate that an antisaccade error is a result of differences in the relative strength of the prosaccade and antisaccade programs. In other words, an antisaccade error occurs when the correct antisaccade is programmed too slowly (Massen, 2004) or activated inefficiently (Hutton, 2008). Inverse correlations between prosaccade latencies and antisaccades errors (Ettinger et al., 2005; Taylor & Hutton, 2009) underscore this point: The faster the opposing saccade signal reaches threshold (shorter latencies) the more likely it wins the competition (higher directional error rates).

In a test of the parallel processing account, Massen (2004) manipulated the frequency, i.e. the prior probability or expectancy, to perform an antisaccade or prosaccade. Antisaccade latencies and direction errors increased with decreasing antisaccade probability, suggesting that antisaccade programming was slowed and the prosaccade decision signal more often won the competition for execution. A recent fMRI study employed a similar frequency manipulation in a mixed antisaccade/prosaccade design (Pierce & McDowell, 2016). It was observed that performance of prosaccades in blocks where they were infrequent was accompanied by increased activation in frontal, temporal and parietal cortex, similar to levels seen during antisaccades, suggesting that this manipulation induced significant cognitive control demands during infrequent prosaccades (Pierce & McDowell, 2016).

The present study built on the horse race model and induced competition between saccade signals via a frequency manipulation (Massen, 2004). We investigated neural correlates, using BOLD fMRI in an event-related design, to gain a better understanding of inhibitory processes in cognitive control. Specifically we raised the question if and where executive control functions triggered by the competition between antisaccade signals and prosaccade signals can be observed on a neural level.

To explore the robustness of the effect of frequency manipulation on saccade performance, we additionally performed a behavioral laboratory experiment in an independent sample using the same task as in the fMRI experiment (see supplementary material).

2. Materials and methods

Participants were recruited from amongst the University of Bonn student population. They had normal or corrected to normal vision and reported no history of neurological or psychiatric disorders and no current use of any psychoactive medications. The study was approved by the ethics committee of the Department of Psychology at the University of Bonn and participants provided written informed consent.

2.1. Participants

The sample consisted of 16 participants (8 female, 8 male; age: $M = 22.94$, $SD = 1.92$, range 19–26; handedness: 13 right-handed; 2 left-handed; 1 unknown).

2.2. Experimental design

Participants lay supine on the scanner bed and viewed the stimuli via a first surface reflection mirror on an MRI head-side projector screen (NordicNeuroLab 32″ LCD monitor, height: 39.2 cm width: 52.35 cm, resolution: 1024 × 768 pixels, 120 Hz) positioned at the rear end of the scanner bore. The distance from the screen via the mirror to the participants' eyes was a total of 172 cm.

The experiment consisted of a mixed antisaccade and prosaccade task. The factors were *Condition* (antisaccade, prosaccade) and *Frequency* (frequent antisaccades, infrequent antisaccades). The task was written in ExperimentBuilder Version 1.10 (SR Research Ltd.).

All stimuli were white and were presented on a black background. Each trial consisted of a central fixation cross, a central cue and a peripheral target (Fig. 1). The central fixation cross (0.73° × 1.3°) was presented for a jittered duration ranging from 1.8 to 7.8 s ($M = 4.8$ s). The fixation cross was followed immediately by a cue, consisting of a central vertical (0.13° × 1.3°) (antisaccade condition) or horizontal (1.3° × 0.13°) (prosaccade condition) bar that indicated the type of eye movement to perform upon target onset. The target stimulus, a circle of 0.2° diameter, appeared 200 ms after cue onset pseudo-randomly 4.3° to left or right of the cue. The central cue remained visible whilst the target was present for 800 ms.

In the antisaccade condition participants were instructed to perform a saccade to the mirror image location of the peripheral target and back to the center as quickly and accurately as possible. In the prosaccade condition participants were instructed to perform a saccade to the position of the peripheral target and back to the center as quickly and accurately as possible.

The frequency of antisaccade trials was manipulated across blocks. Thus, blocks contained either 75% antisaccade trials (18 out of 24 trials) and 25% prosaccade trials in the frequent antisaccade (AS75) condition or 25% antisaccade trials (18 out of 72 trials) and 75% prosaccade trials in the infrequent antisaccade (AS25) condition (Table 1). The number of antisaccade trials was similar in both types of experimental blocks to eliminate possible confounds due to differences in trial frequencies.