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The role of attention and motivation in proactive cognitive control processes

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Contents

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Chapter 1

Introduction

A prominent characteristic of human cognition is its versatility. We are capable of rapidly adapting to the changing internal and external environments that we perpetually encounter. We can swerve to avoid an unobservant driver, hit a baseball, make mental calculations to split a bill in a noisy restaurant, and maintain focus while taking an exam. This versatility is believed to depend on certain neural processes that dynamically tune sensory, perceptual, motor, and other internal mechanisms to assist in completion of internalized objectives. It involves the regulation, coordination, and sequencing of thoughts and actions (Braver, 2012) - and is known as cognitive control. Cognitive control is pervasive to adaptable human behavior, and as such is conceptually broad, encompassing multiple cognitive domains. These include, but are not limited to, updating and maintaining of contextually relevant information, attentional allocation, monitoring of performance, and activation or inhibition of appropriate or inappropriate behaviors (Miller and Cohen, 2001; Verbruggen et al., 2014).

In this introductory chapter, I expand upon this concept of cognitive control as arising from two different modes of control; reactive and proactive. Next we introduce response inhibition as a core mechanism in cognitive control research. We focus on response inhibition because it is an important facet of human cognition and is further implicated in a diverse range of cognitive dysfunctions, diseases, and disorders (e.g., Parkinson's disease, depression, attention deficit hyperactivity disorder (ADHD), substance abuse, and a variety of anxiety disorders - Chambers et al., 2009; Bari and Robbins,

2013). Further, I discuss the relationship between motivation and attention in response inhibition - a traditionally overlooked aspect. Lastly, I explain some of the the difficulties involved in researching proactive control as it relates to response inhibition and the methodologies that we used to overcome these difficulties to arrive at meaningful conclusions.

1.1 Reactive and proactive control

Cognitive control is typically investigated using response conflict paradigms such as the Stroop (Stroop, 1935), flanker (Eriksen and Eriksen, 1974), or Simon (Simon and Rudell, 1967) tasks. These tasks influence the necessity of control by artificially creating situations in which attaining a task-relevant goal is easily disrupted by task-irrelevant distractors. For example, in the Stroop task, color words such as "green" or "red" are presented to a participant in various ink colors, and subjects must name the ink color. The target stimulus (ink color) can correspond either to the same response as the target (congruent trials; e.g., red ink and the word "red") or to the alternative response (incongruent trials; e.g., red ink and the word "green"). Incongruent trials (but not congruent trials) elicit conflict likely due to the automaticity with which we read words, and enhanced control is required to override the tendency of responding with the printed color name in order to deliver a response of the ink color. If, for example, the authorities switched the red "don't walk" signs to green "don't walk" signs and vice versa overnight, it

would not be a good morning for pedestrians.

One dominant proposal in the field is that such cognitive control processes involve two broadly-defined components; the monitoring for conflict and response implementation (Botvinick et al., 2001; Botvinick and Cohen, 2014). Monitoring for conflict is a process in which higher-level goals ("respond to the ink color") are in juxtaposition with irrelevant ("respond to the word") response tendencies that need to be detected quickly and filtered accordingly. This process can, in one sense, be described as a "reactive" control process, such that control is engaged in reaction to the detection of conflict (Botvinick et al., 2001; Braver, 2012). Control may also be enacted "proactively" by a top-down goal directed strategy, and this is associated with stimulus expectancy and hence response expectancy, and might possibly operate (in some circumstances) independently of conflict monitoring. For example, a top-down attentional bias signal could focus attention on the ink color in a Stroop task, thus enabling subjects to name the ink color instead of (incorrectly) reading the word. Such biasing signals that direct performance based on a current task set or goal is an essential component of cognitive control. The proactive and reactive frameworks described above build upon models that distinguish between early expectation signals and late corrective processes (e.g., Braver, 2012), and are aimed at fully encompassing the rich complexity of cognitive control.

1.2 Basics of response inhibition

Another related hallmark of cognitive control research is response inhibition, and this is not without reason. Response inhibition can broadly be defined as the process by which a prepotent, routine, or dominant response is withheld. It is a key component of successful cognitive control due to its suppressive nature. Adaptive and contextually appropriate motor functioning requires complex coordination between motor activation and inhibition networks. Inhibitory mechanisms specifically play a fundamental role in typical cognitive functioning, development, and are implicated in a range of neurological and psychiatric conditions (Chambers et al., 2009). For example, obsessive-compulsive disorder (OCD) is a dysfunction in which sufferers are unable to defocus intrusive thoughts or inhibit compulsive behaviors, and they further show deficits in response inhibition tasks.

In a laboratory setting there are multiple tasks used to study response inhibition. The most influential paradigms are the go/no-go task (Drewe, 1975), the anti-saccade task (Hallett, 1978), and the stop-signal task (Logan and Cowan, 1984; Verbruggen and Logan, 2009a). To properly engage a response inhibition process, a task must require the cancellation of a movement that is prepotent and/or already initiated (Aron et al., 2014; Bari and Robbins, 2013). This is achieved in different ways in the different tasks. In the go/no-go task, frequent go trials and a regular fast pace of the task strongly emphasize the execution of a response, which is then prepotent also

during no-go trials, leading to a significant number of errors of commission. In the antisaccade task, the prepotency of an inappropriate relies on the automatic nature in which our eyes are drawn to a peripheral stimulus, which participants have to look away from. Yet, here we will focus on the stop-signal paradigm as it arguably can modulate motor prepotency in the most controlled fashion and has been highly successful in quantifying the latency and efficiency of response inhibition, and to investigate its underlying neural processes (e.g., Aron, 2011; Huster et al., 2013), which has made it a vastly popular tool both in basic as well as in clinical research (Kenemans, 2015; Verbruggen and Logan, 2008).

In the stop-signal task, a go-stimulus requiring a rapid choice-reaction (respond with one button to stimulus A and with another for stimulus B) is infrequently followed in rapid succession by a stop-stimulus, signaling the participant to halt the initiated response (see Chapters 2 and 3 for a prototypical example). There are (for now) two critical (and related) observations to be made in such a task; (i) subjects can inhibit their responses to a go-signal if the stop-signal arrives close in time relative to the go-signal, and (ii) subjects cannot inhibit their responses if the stop-signal arrives at a time further from the go-signal (and hence closer to response execution). These observations are critical as they put constraints on the types of mathematical models that can account for the data. A very influential model was proposed in Logan and Cowan (1984), and is known as the independent horse-race model. This model assumes that a race between a go process (triggered

by the go-signal) and a stop process (triggered by the stop-signal) occurs, and that the relative finishing times of the processes are determinants of inhibitory behavior in a given trial. The stop-process latency ("stop-signal response time"; SSRT) is covert, but can be recovered by assuming such a stochastic model (Verbruggen and Logan, 2009a). Specifically, the duration of the stop-process can be derived from the proportion of successful stop trials and the distribution of response times on go-trials (Verbruggen et al., 2013). However, as is noted in Elchlepp et al. (2016) and Verbruggen and Logan (2015), SSRT is not purely reflective of a unitary motor-related inhibitory process, but necessitates the involvement of other cognitive systems, which previews a central theme in this dissertation.

1.3 Neural components of reactive response inhibition

The neural underpinnings of response inhibition have been studied extensively, in particular in human neuroimaging, animal models, and in human brain dysfunction. To expand on the latter point, inhibitory deficits are well recognized in patients with frontal lobe trauma (e.g., Stuss and Alexander, 2000), and neuroimaging studies suggest that these response inhibition deficits in OCD are underpinned by physiological abnormalities within fronto-striatal circuits (e.g., Herrmann et al., 2003). Furthermore, patients with frontotemporal dementia show impairments associated with response inhibi-

tion (Rubia et al., 1999), and activation is sometimes attenuated in patients with attention deficit hyperactivity disorder (ADHD) and addictions (Seeley et al., 2009).

Most research has focused on reactive response inhibition, and so we will briefly discuss the major components thought to be involved in reactive response inhibition (Chambers et al., 2009). Ample evidence has been put forth using functional magnetic resonance imaging (fMRI) that the right inferior frontal cortex (rIFC) is critical for inhibitory behavior (e.g., Bari and Robbins, 2013; Chikazoe, 2010), and further with electrocorticography (ECoG) that such activity is present before the stop-process ends (Swann et al., 2013). See Swick et al. (2008) and Levy and Wagner (2011) for a discussion of the possible subregions of the rIFC that are thought to be activated. It has been proposed (Aron et al., 2004) that the rIFC signals inhibition directly via the subthalamic nucleus (STN), and possibly acts by increasing β synchronization between the two areas (based partially off evidence from deep brain stimulation in Parkinson's disease patients - Kühn et al., 2004; Schall and Godlove, 2012). It is further suggested that there exist different pathways for the go-signal and the stop-signal in relation to movement activation (striatum) and inhibition (STN). Some of the strongest evidence for this is seen in a rodent study (Schmidt et al., 2013) in which they demonstrate that two key basal ganglia pathways for action control resemble a race between a go-process and a stop-process. Downstream target neurons that project to systems important for control of orienting movements showed both

movement-related pauses (provoked by the striatum - direct pathway) and rapid increases in firing rate following stop-signals (provoked by the STN - hyperdirect pathway), which corresponded to outright stopping success. In this sense it is thought that the hyperdirect pathway cancels the movement initiated by the direct pathway. While there is a strong focus on the rIFC-STN axis, it is important to note that the presupplementary motor area (preSMA) is also implicated in stopping (Duann et al., 2009). The preSMA is functionally and structurally connected with the rIFC, and it is not yet clear whether the rIFC triggers the STN directly, or via the preSMA.

1.4 Specificity of response inhibition network

It has been suggested that inhibitory theories of rIFC are over-specified because they do not explain the contributions that the inhibitory module makes to broader cognition (e.g., Hampshire and Sharp, 2015b; Schall and Godlove, 2012). A possible limitation for the inhibitory perspective of rIFC function is that the tasks used do not control for potentially confounding cognitive demands. Thus, the possibility exists that activations seen in inhibitory paradigms do not equate to proof of an involvement in neural inhibitory processes, and could be explained by other mechanisms. Numerous studies identify rIFC with functions other than response inhibition (e.g., Erika-Florence et al., 2014; Corbetta et al., 2008; Hampshire and Sharp, 2015a). rIFC is consistently implicated in stimulus driven attentional capture (Asplund et

al., 2010) and this activation scales with the degree of stimulus unexpectedness (Shulman et al., 2009). In general, rIFC is often implicated as part of a network involved in orienting attention toward salient stimuli. Specific to the stop-signal task, activation of rIFC may be caused by cognitive functions such as attentional capture (Hampshire et al., 2010) or violations of event expectations (Zandbelt et al., 2013). However, it has also been argued (Aron et al., 2014) that any stimulus that is surprising, salient, or infrequent will recruit motoric inhibition, in that the tasks might have hidden inhibitory demands - and that this might be very fluid across different situations and tasks that invoke a wide range of unexpectedness, in turn causing a suppressive effect that is inherent to behavioral and even cognitive functioning (Wessel and Aron, 2017).

1.5 Proactive inhibition

To add to this complexity, a growing amount of interest in *proactive* response inhibition has recently been seen, which describes the tendency of slowing down responses when outright stopping *might* be required. Proactive response inhibition is often considered potentially more ecologically relevant given that it relates to a common phenomenon of response caution in uncertain environments rather than exclusively on imperative stimuli that might not always be available in everyday life (Aron, 2011). Proactive response inhibition likely relates to different modes of response caution in typical ev-

eryday situations and is probably related to expectation of event occurrence. For example, Go-trial response times are known to slow after a stop-trial (e.g. Ray Li et al., 2006) and furthermore recent computational work using hidden Markov models has shown that response time on go-trials scales with the trial-wise computation of a "subjective" stop-expectancy (Ide et al., 2013). Consistently, more proactive response inhibition is observed, the higher the proportion of stop-stimuli in a given task (e.g., Jahfari et al., 2010; Logan and Burkell, 1986). Further theoretical modeling has related this effect to an increased decision threshold of the go-process (e.g., Verbruggen and Logan, 2009b), and a range of fMRI studies have implicated the (reactive) response-inhibition network in this process as implementing "gradated" response inhibition (Aron et al., 2014). It has further been proposed that proactive inhibition involves an additional basal ganglia circuit (e.g. Frank et al., 2007).

1.6 Attention and motivation in proactive response inhibition

Given the role that attention plays in other cognitive control paradigms (Braver, 2012), it is not unlikely that it might play a part in response inhibition. Indeed, recent behavioral research has highlighted a possible role of earlier sensory processes in response inhibition (Bari and Robbins, 2013; Logan and Cowan, 2014; Verbruggen et al., 2014; Huster et al., 2014; Elch-

lepp et al., 2016). Consistent with such notions, differences in the attentional processing of stop-stimuli have been found to contribute to the behavioral outcome in the stop-signal task, with increased attention to the stop-stimulus being associated with successful (assumed to be reactive) response inhibition (e.g. Bekker et al., 2005; Boehler et al., 2009; Bengson et al., 2012), see Kenemans (2015) for a review.

Findings furthermore suggest that attentional processes may also be implicated in proactive response inhibition. Given that attention plays such an important role in other domains of proactive control, this seems quite natural. The same magnetoencephalograpic data that reported an increase in attentional processing (indicated by the event-related N1 component, believed to index the level of attentional discrimination of a visual stimulus (Hopf et al., 2002), of the stop-stimulus when successful, additionally suggested that the attentional processing of the go-stimulus in a stop-trial is enhanced when response inhibition is ultimately unsuccessful (Boehler et al., 2009). This differential processing of the go-signal took place before the processing of the stop-signal, suggesting that stopping success relies, at least partially, already on the perceptual processing of the go-stimulus. Due to the lack of an imperative stimulus about response inhibition, this effect likely reflects a proactive process that already plays out in go-stimulus processing, and suggests a role of controlled attentional processing also in proactive response inhibition.

The question of how motivational factors may influence proactive processes, as is noted in Schall and Godlove (2012), is particularly interesting,

due to possible motivational effects on attentional processes. Recently a series of studies was reported in which associating reward with one of two possible stop-stimulus colors also led to shorter SSRTs, despite the fact that any difference in global preparation was precluded since reward-related trials were presented in a random sequence together with all other trial types (Boehler et al., 2012; Boehler et al., 2014). In an EEG version of this experiment, it was demonstrated that enhanced attention to the reward-related color seems to play a role in bringing about the reward-related SSRT benefit (Schevernels et al., 2015). This experiment featured task blocks that were devoid of any reward associations. Using these trials as a comparison, it was demonstrated that the sensory/attentional N1 component to the go-stimuli of stop-trials was enhanced throughout the reward-related block. Given that reward was exclusively related to stop-stimuli, this effect on the go-stimuli (of stop-trials) was considered a context effect in the sense that attention is increased globally (Jimura et al., 2010). This set of findings highlights that reactive response inhibition is amenable to motivational manipulations, with the EEG data suggesting that this might be achieved through attentional mechanisms. Given the effect also on go-stimulus processing, these effects seem to again be partly proactive in nature.

1.7 Outline

The literature review above paints a multifaceted picture of response inhibition, with clues that both reactive and proactive response inhibition also depend on afferent processes pertaining to the level of attentional deployment to the task stimuli, and to motivational factors. Given the central role that response inhibition (deficits) play in basic research and in common brain-related disorders, a better understanding of the full process structure seems important. The research presented in this thesis aims to extend our knowledge about the relationship between attention, motivation, and proactive response inhibition. The inferential approaches we applied can be split into two general strategies. The first strategy was to examine the go-locked N1 component and its relationship with response time in stop-signal tasks with differing task contexts. In Boehler et al. (2009) the attentional processing of the go-stimulus in a stop-trial was enhanced when response inhibition was unsuccessful. This implies that varying attentional processing of the go-stimulus is behaviorally relevant, yet this work was limited with respect to the study of proactive response inhibition. It involved only a small subset of trials (i.e., stop-trials) that could have been affected by the overlapping processing of stop-stimuli in trials where those were presented particularly rapidly after the onset of the go-stimulus. Further, it was not clear whether participants actively engaged differential attentional processing strategically. This earlier work might have identified randomly oscillating variations in

attentional focus towards the go-stimuli, and not strategically deployed levels of attention. In **Chapter 2** we use EEG and address these issues by examining go-trials and their relationship with response time in a multilevel single-trial approach, using the relationship between response time and stopping-success to strengthen the argument. This was done in a standard relevant-stop context and contrasted with an irrelevant-stop context, i.e. ignore the stop-signals. In this sense, this created a baseline, and differences between relevant and irrelevant are bound to be caused by proactive control processes in go-trials.

In **Chapter 3** we adopt this same basic strategy to both replicate the study in Chapter 2 and to further explore proactive control under a different motivational context. We investigated contextual effects of rewarding successful reactive response inhibition on proactive inhibition by posting a monetary reward on some stop-trials. We anticipated to replicate our earlier results for the standard relevant-stop context. For the reward-stop context, however, we hypothesized two different possible patterns of results: (i) The relationship between response times and N1 amplitudes could be similar or even more pronounced, or (ii) the global attentional increases driven by reward context (as seen in Jimura et al., 2010) might interfere with the relationship between visual attention and response speed.

One thing to note about the first strategy is that it at least partly sets up a reverse inference (Poldrack, 2006), in that within-context fluctuations in brain activity measures are used to infer a cognitive process (varying levels

of attention in different trials). The second general strategy was aimed at counter this limitation by explicitly manipulating the proportion with which response-inhibition trials were presented, and by lateralizing stimuli and examining pretarget alpha oscillations in a stop-signal task and a go/no-go task. The manipulation of expectation by a cue is similar in spirit to Zandbelt et al. (2013) and Verbruggen and Logan (2009a) but with the extension of looking at pre-target attentional processes. In spatial attention tasks, cued attention to a location has been repeatedly associated with increased alpha band activity over ipsilateral regions, and decreased alpha activity over contralateral regions prior to stimulus presentation (e.g., Vollebregt et al., 2015; Bengson et al., 2012). That these modulations happen prior to stimulus presentation, means that they are by definition target-independent, and suggests that they are driven by top-down processes (see, Romei et al., 2008; Capotosto et al., 2009; Capotosto et al., 2012, for further evidence suggesting it is top-down). In **Chapter 4** we record EEG data while participants complete a lateralized-go and centralized-stop stop-signal task with explicit cues about the probability of the centralized-stop occurring. We analyze posterior alpha oscillations as in between the cue and the go-target to look at possible spatial attention modulations across the cue levels. In **Chapter 5** we extend this same logic to a go/no-go task (similar to Bengson et al., 2012). Additionally, in this task the uncued hemifield always had a distractor, and the cues were high or low pitch tones. In this manner, the task was more in line with the previous spatial attention tasks examining pretarget alpha oscilla-

tions, in that it had distractors (see, Slagter et al., 2016, for a discussion), and was not as temporally complex (i.e., there was no second signal) as the experiment in Chapter 4. In Chapter 5 we follow this up by looking at midfrontal theta power. Theta has been shown (Cohen and Donner, 2013) to be a robust power modulation that is non-phase-locked and more closely linked to conflict and behavior than it's phase-locked counterpart (i.e., the ERP waveform). Theta-band oscillations are modulated during action monitoring tasks, specifically in tasks that elicit conflict between competing responses (e.g., Cohen, 2016; Cohen and Donner, 2013; Huster et al., 2013; Oehrn et al., 2014). Recent research has highlighted that midfrontal theta is a signature of the engagement of a system that responds flexibly to errors and the possibility of errors (Cavanagh et al., 2014; Cohen, 2014; Cohen, 2016). In Chapter 5 we apply this logic to a go/no-go task and examine midfrontal theta as a function of both expectation and behavior.

Both inferential strategies employed in this thesis are aimed at modeling the complexity involved in proactive control and response inhibition, and are unique contributions to the field. That is to say, we are either looking at single-trial relationships to understand proactive processes at a finer level (a method that is not normally used in the literature), or we are investigating (what we believe to be) a clear marker of proactive processes using data that occurs before any relevant inhibitory stimuli (an index that has not received much attention). We further, in Chapters 2-4, model the behavioral response time and accuracy data in a sequential sampling framework to detail

the complexity involved in decision-making processes in such tasks.

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Chapter 2

Strategic down-regulation of attentional resources as a mechanism of proactive response inhibition[1]

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Abstract

Efficiently avoiding inappropriate actions in a changing environment is central to cognitive control. One mechanism contributing to this ability is the deliberate slowing down of responses in contexts where full response cancellation might occasionally be required, referred to as proactive response inhibition. The present electroencephalographic (EEG) study investigated the role of attentional processes in proactive response inhibition in humans. To this end, we compared data from a standard stop-signal task, in which stop signals required response cancellation (stop-relevant), to data where possible stop signals were task-irrelevant (stop-irrelevant). Behavioral data clearly indicated the presence of proactive slowing in the standard stop-signal task. A novel single-trial analysis was used to directly model the relationship between response time and the EEG data of the go-trials in both contexts within a multilevel linear-models framework. We found a relationship between response time and amplitude of the attention-related N1 component in stop-relevant blocks, a characteristic that was fully absent in stop-irrelevant blocks. Specifically, N1 amplitudes were lower the slower the response time, suggesting that attentional resources were being strategically down-regulated to control response speed. Drift diffusion modeling of the behavioral data indicated that multiple parameters differed across the two contexts, likely suggesting the contribution from independent brain mechanisms to proactive slowing. Hence, the attentional mechanism of proactive response control we report here might coexist with known mechanisms that are more directly

tied to motoric response inhibition. As such, our study opens up new research avenues also concerning clinical conditions that feature deficits in proactive response inhibition.

2.1 Introduction

Adaptive motor behavior requires a complex coordination of motor activation and inhibition. Inhibitory mechanisms play a fundamental role in everyday behavior, in cognitive development, and in a range of neurological and psychiatric conditions, including attention-deficit hyperactivity disorder (ADHD), Parkinson's disease, and substance abuse (Chambers et al., 2009). In a laboratory setting the stop-signal paradigm has often been used to quantify the latency and efficiency of response inhibition (Logan and Cowan, 1984), and to investigate its underlying neural processes (Aron, 2011; Huster et al., 2013).

In the stop-signal task, a go-stimulus requiring a rapid choice-reaction is infrequently followed by a stop-stimulus, signaling the participant to halt the initiated response. Task behavior can be characterized as a race between a process that triggers (go-process) and cancels (stop-process) a motor action. The stop-process latency (stop-signal response time; SSRT) is covert, but can be recovered by assuming a stochastic model, such as the Independent Race Model (Verbruggen and Logan, 2009b).

Traditionally, the research focus was on processes related to this reactive form of inhibition (triggered by the stop-stimulus), which has been found to be related to a response-inhibition network involving the right inferior frontal gyrus, the pre-supplementary motor area, and the subthalamic nucleus (Aron et al., 2014; see also Cai et al., 2014). Recently, however, proac-

tive response inhibition has received increasing attention. Proactive response inhibition is considered potentially more ecologically relevant, in that it describes the tendency of slowing down responses when outright stopping might be required, which likely relates to response caution in everyday situations (Aron, 2011). Mathematical modeling has mostly related this effect to an increased decision threshold of the go-process (e.g., Verbruggen and Logan, 2009a), and a range of experimental studies have implicated the (reactive) response-inhibition network in this process as implementing gradated instead of complete response inhibition (Aron et al., 2014).

Although the core neural processes of reactive and probably also proactive inhibition likely reside within the response-inhibition and extended motor network, recent behavioral research and theorizing has highlighted a possible role of earlier sensory/attentional processes in response inhibition (Bari and Robbins, 2013; Logan et al., 2014; Verbruggen et al., 2014b; Huster et al, 2014). Consistent with such notions, differences in the attentional processing of stop-stimuli have been found to contribute to the behavioral outcome in the stop-signal task, with increased attention to the stop-stimulus being associated with successful response inhibition (Bekker et al., 2005; Boehler et al., 2009; Kenemans, 2015).

Yet, some recent findings suggest that attentional processes may also play a role in proactive response inhibition; specifically, we have reported magnetoencephalograpic data showing that the attentional processing of the go-stimulus in a stop-trial is enhanced when response inhibition is ultimately

unsuccessful (Boehler et al., 2009; see also Knyazev et al., 2008). While this implies that varying attentional processing of the go-stimulus is behaviorally relevant, this earlier work was limited in important ways with respect to the study of proactive response inhibition. Specifically, it involved only a small subset of trials (i.e., stop-trials), which furthermore could theoretically have been affected by the overlapping processing of Stop-stimuli in trials where those were presented particularly rapidly after the onset of the Go-stimulus. Additionally it was not clear whether participants actively engaged differential attentional processing as a means of strategic proactive inhibition. Rather, this earlier work might have identified the influence of randomly oscillating variations in attentional focus towards the go-stimuli, and not strategically deployed levels of attention driven by task-relevant stimuli. Here, we address these questions by (i) using EEG measures for which we adopt a single-trial framework to model electroencephalographic activity during go-trials as a function of response slowing (Pernet et al., 2011), and (ii) by including additional task blocks in which stop-stimuli were task-irrelevant to provide a baseline condition which should be devoid of proactive response inhibition.

2.2 Methods

2.2.1 Participants

Sixteen healthy right-handed subjects (mean age 24.7 years, SD 5.0, 8 males) took part in the study. All subjects were neurologically intact and had normal or corrected-to-normal visual acuity.

2.2.2 Stimuli

On each trial a traffic light symbol was presented above a central fixation dot on a gray background. The traffic symbols were green go-signs, directed to either the LEFT or the RIGHT, and red stop-signs. The LEFT-pointing go-sign required a button press with the right index finger, and the RIGHT-pointing one required a button press with the right middle finger. In contrast to our earlier related work (e.g., Boehler et al., 2009) the target stimuli were presented in isolation without additional distractor items. This choice-reaction stimulus either lasted for the full stimulus duration (go-trial) or was rapidly followed by a stop stimulus (stop-trial). Two block types were used: stop-relevant and stop-irrelevant (see Schmajuk et al., 2006 and Boehler et al., 2010, for similar task designs that used stop-irrelevant stop-trials as a sensory baseline condition to investigate reactive response inhibition). In stop-relevant blocks subjects were instructed to withhold their response when a stop-stimulus was encountered, whereas in stop-irrelevant blocks subjects were instructed to ignore the stop-stimulus completely and to always respond

LEFT or RIGHT to the go-stimulus, see Figure 1. Participants were told not to slow down their response strategically. Still, proactive slowing is typically observed in such settings (Verbruggen et al., 2005).

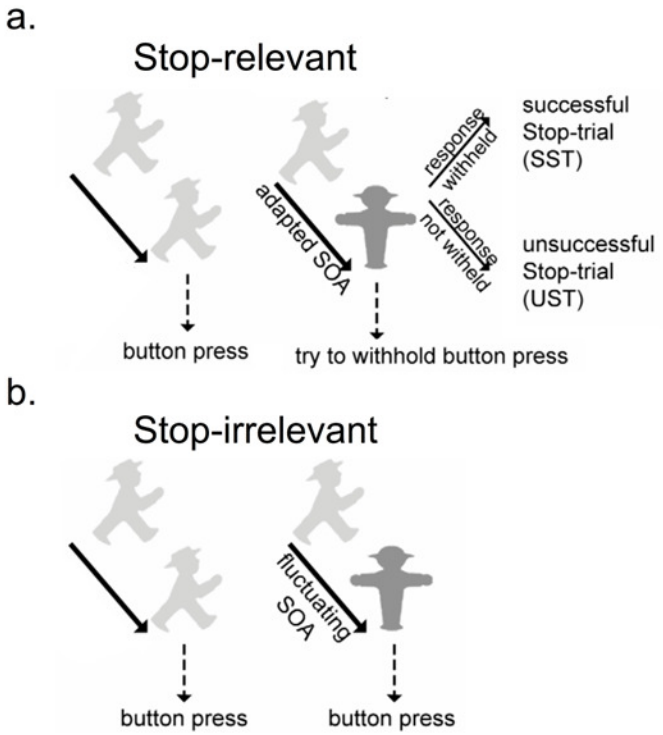

Figure 2.1: **Paradigm.** Participants performed a standard (stop-relevant) stop-signal task (a) and a stop-irrelevant version (b) in separate blocks. Response inhibition was required upon presentation of a stop-stimulus in the stop-relevant but not the stop-irrelevant blocks.

2.2.3 Procedure

Go-trials accounted for 75 percent of all trials, and stop-trials for the remaining 25 percent. There were twelve experimental blocks each consisting

of approximately 90 trials. At the midpoint there was a small break and instructions were changed (e.g., stop-relevant to stop-irrelevant), with the next run having the opposite sequence. In total, there were 285 stop-trials and 846 go-trials, equally distributed across the stop-relevant and stop-irrelevant task blocks. Given the fast ABBA sequence of different block types, the actual block order was not further counterbalanced across participants. The overall duration of stimulus presentation was 700 ms for each trial, and trials were interleaved by intertrial intervals that varied randomly between 1000 and 1400 ms. For go-trials in both stop-relevant and stop-irrelevant blocks the visual display was constant for the 700 ms duration, whereas for stop-trials the go-stimulus was replaced by a stop-sign after a certain stop-signal delay (SSD), which would then stay on screen until the end of the 700 ms duration. For relevant stop trials an adaptive staircase procedure was used to control stopping performance by incrementing (after a successful stop trial) or decrementing (after an unsuccessful stop trial) the stop-signal delay by 17 ms. This procedure enabled the reliable calculation of the stop-signal response time (SSRT), which reflects the time required to inhibit a motor response. As a matched routine, we took the end value of the adapted stop-signal delay from stop-relevant blocks as the initial value in subsequent stop-irrelevant blocks and then randomly alternated it by 17 ms on each subsequent irrelevant trial.

2.2.4 Recording and Analysis

Basic Behavioral Analyses. All response time analyses were performed using repeated-measures analysis of variance (rANOVA). Differences in accuracy for go-trials were tested between blocks using a non-parametric $\chi 2$ test of differences in proportions. To estimate the SSRT the integration approach was used. This approach defines the SSRT = (nth rank-ordered RT) - (mean stop-signal delay), with n equal to the number of RTs in the go-trial RT distribution multiplied by the overall probability of responding given a stop signal. Simulations showed that under most circumstances the integration approach yields consistent and unbiased estimates of the SSRT (Verbruggen et al., 2013). Note, however, that stop-trial data were only of peripheral interest here, as the main analyses focus on the go-trials from the two different task blocks[2].

Drift Diffusion Models. Drift diffusion models are a description of a binary choice process defined by three main parameters (Ratcliff, 1978), and have been used frequently in the study of proactive inhibition (e.g., Verbruggen and Logan, 2009a). These parameters are the response threshold (a), the mean rate of approach to a threshold, known as drift rate (v), and processes that precede and succeed the actual decision process and give rise

[2]Note that the assumptions of the independent race model were nevertheless tested in order to evaluate whether the SSRT could reliably be estimated. Specifically, (1) the SSD was longer during unsuccessful than successful Stop-trials, $t(15) = 7.65$, $p < 0.001$; (2) that go-trial RT was slower than RTs on unsuccessful Stop-trials, $t(15) = 8.94$, $p < .001$; and (3) to show that RT on unsuccessful stop trials increase as a function of SSD we tested a correlations difference from zero ($r = 0.34$, $t(779) < 0.001$)).

to a nondecision time (t0). Hierarchical Bayesian estimation was used to model the parameters using the Hierarchical Drift Diffusion Model (HDDM) software (Wiecki et al., 2013). Model fit was assessed using the deviance information criterion (Spiegelhalter et al., 2002; DIC, with smaller DIC indicating better fit). Five nested candidate models were fit to the data; a null model (model 0), a full model (model 1, including a, v, and t0), and 3 reduced models; a model without t0 (model 2), a model without v and t0 (model 3), and a model without a and t0 (model 4). These models were chosen to test for differences in the parameters in a principled sequential manner, and for alignment with previous modeling efforts (Verbruggen and Logan, 2009a). 20,000 posterior samples were drawn for each model using Markov-Chain Monte Carlo methods. We used a burn-in of 5,000 and a thinning factor of 3. Each model was checked for convergence using the Gelman-Rubin diagnostic (Gelman and Rubin, 1996). Furthermore, posterior-predictive checks were made as an added assurance of proper fit. After model selection, posterior distributions were probed to determine differences directly in the parameters between the stop-relevant and stop-irrelevant task contexts. This is accomplished by examining the proportion of posterior samples falling above or below the two estimated posterior distributions of any specific parameter, resulting in a probability that one posterior distribution is greater or less than the other (see Kruschke, 2010 for an overview of Bayesian methodology).

EEG Recording EEG was recorded from 64 electrodes mounted in a custom-designed electrocap (Electro-Cap International, Eaton, Ohio), referenced to the right mastoid during recording (SynAmps amplifiers from Neuroscan; El Paso, TX). Additionally, horizontal and vertical EOG electrodes recorded blinks and eye movements, for which participants were additionally monitored online via a video camera in the EEG chamber. Electrode impedances were kept below 2Ω for the mastoids, below 10Ω for the electrooculogram (EOG) electrodes, and below 5Ω for all the remaining electrodes. All EEG channels were continuously recorded with a band-pass filter of 0.01-100 Hz at a sampling rate of 500 Hz per channel.

EEG Preprocessing EEG data were algebraically re-referenced to the average-mastoid offline. A coarse (visual) inspection was performed on the continuous data of each subject to exclude stretches of data with common EEG artifacts. This was followed by an Ocular-correction ICA analysis using the vertical EOG as the blink marker channel in Brain Vision Analyzer 2 (Brain Vision analyzer software, Brain Products GmbH, Munich, Germany). The correct responses of the go-trials from the two task blocks were then epoched from -200 to 1200 ms and corrected using the pre-stimulus baseline prior to further analysis. In the end, 97.8% of the data epochs were preserved. Go-locked event-related potential (ERP) analyses. In order to focus on the inferoposterior visual N1 component, an averaged topography was plotted across both block types and used to define 10 posterior electrodes, 5 on the

right and 5 on the left, as well as the time-range of interest, determined as 130 ms to 190 ms, to represent the visual N1 elicited by the Go-signal. This time-range and set of electrode averages was then also used for the statistical analysis (see e.g., Vogel and Luck, 2000, for similar a similar choice of channels and time-range). It is important to note, however, that the ERP analysis here is of peripheral interest, given that the between-block comparison is rather unspecific.

Single-trial ERP data The main analysis of interest investigated the relationship between single-trial ERP data and response speed on go-trials in the two different task blocks. To this end, single-trial ERP analysis was carried out using the software package LIMO EEG (Pernet et al., 2011; also see Gaspar et al., 2011). Single-trial analysis fits a general linear model of the form

$$y_{e,s} = X\beta_{e,s} + noise \tag{2.1}$$

to trials of EEG data (y), for all analyzed electrodes (e) and sampling points (s) in the N1 time window. The five predictors in the design matrix X were the categorical stop-relevant and stop-irrelevant go-trial types, the single-trial normalized (per subject, per condition) response times, and a noise variable. Below, we describe some more details of the statistical analysis, as implemented in LIMO EEG (see Pernet et al., 2011 for more details). A generalized Moore-Penrose pseudo-inverse algorithm was used to estimate the beta parameters for each subject. Model fit was assessed per individual by

examining R^2, the amount of variance explained in the EEG by the design matrix. These coefficients were tested using a restricted intercept-only model to develop an F-test that determines the amount of variance being explained over this restricted model with the full model. This results in F-values for each sampling point and electrode considered in the model, with degrees of freedom dependent on total number of predictors in the restricted model (i.e. number of predictors in the full model - 1) and trial number. At the second level of the analysis each of the subjects' five estimated beta coefficients were synthesized to probe for statistical significance using nonparametric (bootstrapping) methods. The general linear model allows directly testing for the covariation of single-trial ERPs with response time using a bootstrap-t approach. This determines the significance and direction of beta parameters per sample point. We used a robust one sample t-test that tests if the average effect significantly differs from zero. The observed t-values were first computed. The data were then centered and five-thousand bootstraps were made. Subjects were drawn randomly with replacement. For every bootstrap, a one-sample t-test was performed on the bootstrap sample, subsequently storing the t-value. These bootstrapped t-values provide an approximation of the t-distribution under H0. The p-values are then computed by comparing the observed t-values to the bootstrapped t-distribution. Since tests are performed on multiple electrodes and sampling points, as is typical for this approach (e.g., Pernet et al., 2011; Gasper et al., 2011), testing will give rise to false positives. To account for multiple comparisons, we used temporal

clustering by which only clusters with a mass (sum of t values) bigger than the 95% percentile of the null distribution are considered significant. In this case, the null distribution corresponds to the maximum cluster value across electrodes measured at each bootstrap computed on nullified data (Pernet et al. 2015). In a similar vein, for a repeated-measures ANOVA, the observed F-values were first calculated. Following this, an F-table under H0 was made. First, the data was centered for each condition so that each cell of the ANOVA had a mean of zero. Second, the centered data was used to estimate F-distributions under H0. Subjects were sampled with replacement and the associations between observations were kept. Five-thousand bootstraps were made. P-values were obtained by comparing the observed and bootstrapped F-values, and multiple comparison corrections were handled in the same manner as the 1-sample t-tests.

2.3 Results

2.3.1 Behavioral Performance

The average response times to go-stimuli in the stop-relevant blocks were 466.7 ms (SD 116.6), which were slower than ($F(1,15) = 50.37$; $p < .0001$) those in the stop-irrelevant blocks 402.3 ms (SD 85.2). This result indicates that participants were employing proactive response slowing in the stop-relevant blocks, as expected. Overall, accuracy in go-trials was high: in the stop-relevant blocks it was 98.9 percent, but slightly lower ($\chi^2 = 8.12$, p =

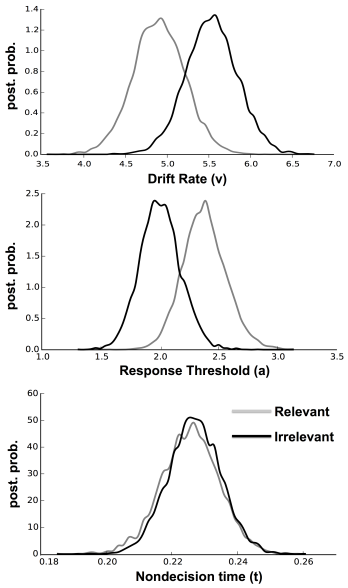

Figure 2.2: **Hierarchical drift diffusion model results.** Drift rate (top), response threshold (middle), and non-decision time (bottom) posterior probability densities for both the irrelevant and relevant conditions of Model 1.

0.004) in the stop-irrelevant blocks at 98.3 percent. The SSRT was calculated using the integration method yielding an estimate of 242.8 ms (SD 37.5), a value in line with previous research.

The DIC-based model selection procedure evidenced that the full model (model 1, DIC = -25009.1) best accounted for the data. The next closest candidate model was the reduced model 2 (DIC = -24915.6), followed by model 3 (DIC = -24782.1), model 4 (DIC = -24658.7), and the model 0 (DIC = -22925.4). Based on this selection criteria and posterior predictive checks, model 1 was chosen for further analysis. Two of the three estimated main parameters showed significant differences between block types. In par-

ticular, robust effects were observed for a raised response threshold (a) and lower drift rates (v) in the stop-relevant blocks. Posterior distributions of the three main parameters are shown in figure 2. With respect to the response threshold, the $p(a_{relevant} > a_{irrelevant}) = 0.94$, showing that indeed the response threshold is raised in the relevant blocks. The drift rate is lower in the relevant blocks, $p(V_{irrelevant} > V_{relevant}) = 0.92$. As seen in Figure 2, non-decision time did not show evidence of being different between the blocks, $p(t0_{irrelevant} < t0_{relevant}) = 0.46$. Overall, the results evidence a more conservative response process in the stop-relevant blocks, and the fact that the effects were seen on two parameters suggests that this was brought about by multiple processes. Indeed, it is possible that model 2 (full model sans t0) is the better fitting model, given that DIC is known to be somewhat biased towards a model with greater complexity (Plummer, 2008). However, the parameter estimates of model 1 and model 2 are similar, so interpretation of the other two parameters remains exactly the same.

2.3.2 Go-locked N1 ERP Analysis

The average topography between 130 and 190 ms post Go-stimulus is shown in Figure 3a for correct Go-trials collapsed across the stop-relevant and stop-irrelevant blocks. Channel locations and time range for further analysis were selected based on this average across both block types. The N1 electrodes were separately averaged in the left and right hemisphere (see black dots in Fig. 3a) and a rANOVA was used to test for differences between block type

(stop-relevant vs. stop-irrelevant) and laterality (left vs. right hemisphere) for the data averaged between 130 and 190 ms post go-stimulus. Both block type ($F(1, 15) = 5.60$, $p = 0.032$, $\eta^2 = 0.004$) and laterality ($F(1, 15) = 6.12$, $p = 0.026$, $\eta^2 = 0.049$) showed significant differences, but there was no significant interaction between the two ($F(1, 15) = 1.19$, $p = 0.29$, $\eta^2 = 0.0002$). Note, that laterality was only of peripheral interest, that we had no clear expectations, and that the main effect only indicates that N1 amplitudes were generally larger over one hemisphere. The mean amplitudes of the stop-relevant N1 were slightly more negative than in the stop-irrelevant condition. Thus, a simplistic mapping between response speed and mean N1 amplitudes did not hold[3]. Yet, these effects are, as indicated by the effect size, quite small. Indeed, this between-block comparison is necessarily quite unspecific, and our a-priori analysis plan was to investigate the relationship between response time and N1 amplitudes within the two different block types, for which we applied the single-trial-based analysis presented below.

2.3.3 Systematic Variation in Sensory Processing of Go-signals

Single Subject Analysis and Model Fit. For each participant, all ten inferoposterior N1 electrodes were individually taken into an analysis with the EEG signal modeled as a linear function of the response time to inves-

[3]In fact, in our further linear models N1 analyses there were no categorical differences between block type in a similar (the models also included RT, an error term, and were bootstrap tests) repeated measures ANOVA

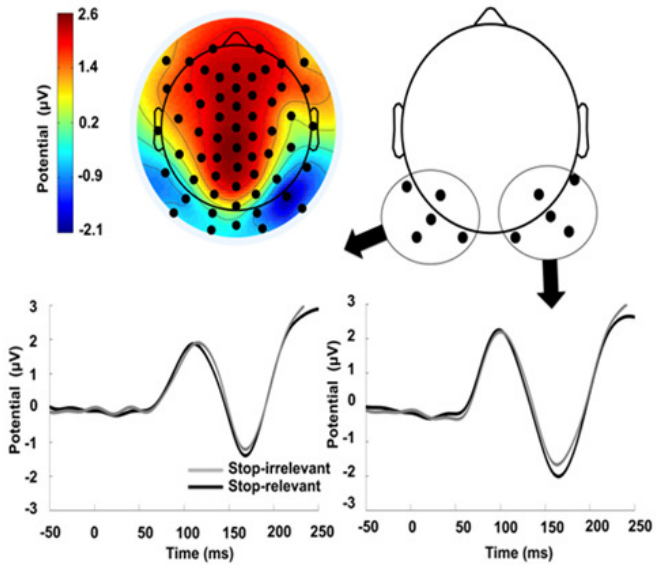

Figure 2.3: **ERP results.** Topography for go-trials averaged between 130 and 190 ms collapsed across the two task blocks on the top left, and sensors chosen to represent visual N1 on the right. Sensor plots from the average of the five electrodes averaged on the left and the right are displayed at the bottom (plotted using a 30-Hz low-pass filter for displaying purposes)

tigate relationships to go-stimulus processing across sampling points. Only correct go-trials were taken into consideration. For the first-level statistical analysis, single-trial ERPs were estimated for each individual at each of the 10 electrodes selected above between 130 and 190 ms. This resulted in beta coefficients for the categorical block parameters, response time parameters, and noise, for each of these 10 electrodes and for each sample point.

As expected, there was variation between individuals, electrodes, and sampling points modeled in terms of the estimated R^2. The F-values were queried for a maximum value F-statistic across individuals and sampling

points. The maximum F-values had a range from 4.14 to 16.18 over all individuals, with a mean of 8.35 (SD = 3.8). For each participant the max F-values were found to be significantly different from a restricted model, using number of linear predictors in the restricted model and participant trial numbers to calculate the appropriate degrees of freedom. Given that each individuals model had sample points within the N1 range that were significantly explained by the design matrix, it was concluded that the model fit was adequate to continue testing at the second level.

Second Level Analyses. Group-level differences in the stop-relevant blocks of the RT beta parameters were tested using a bootstrapping 1-sample t-test procedure to synthesize individuals. Within the N1-related electrodes chosen for analysis, a generalized pattern of N1 attenuation emerged as RT increased. Estimated t-values peaked with significant effects that were centered roughly on 160 ms. Seven of the 10 electrodes showed significant effects using uncorrected t-tests and an $\alpha \pm$ level of 0.05. Electrodes started to show RT effects in the relevant beta parameters capturing the relationship between RT and EEG amplitude starting at 138 ms and lasting until 190 ms. A 1-dimensional temporal cluster analysis was further run on the model to correct for multiple comparisons. This analysis evidenced 3 electrodes, 2 confined to the right posterior, and 1 to the left at a corrected p = 0.05 alpha level. The bootstrapped mean Betas and 95% confidence interval for each of the three electrodes that survived temporal cluster correction are plotted in Figure 4.

This shows the mean change in mV per standard deviation unit of RT for the stop-relevant blocks to further illustrate this relationship.

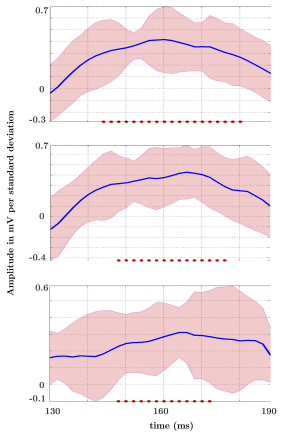

Figure 2.4: **Bootstrap mean Beta and 95% Confidence Intervals.** Evolution of mean Beta parameter for the three electrodes that survived multiple comparison correction in the 1-sample t-test of the stop-relevant block. Sampling points that survived correction are marked as red dots.

Differences in Slowing Between Blocks A rANOVA was used to directly test the differences between the stop-relevant and stop-irrelevant trials and their relationship to response slowing. The purpose of testing the difference between the blocks was to ensure that the differences seen in the relevant blocks was not simply due to response time fluctuations that would be seen in an arbitrary forced choice task. Five thousand bootstrapped F-statistics were used in the analysis. Significant differences were found to begin at 155

ms, and continue until 175 ms post stimulus dependent on electrode, peaking at 164 ms, which is clearly within the time course seen for the 1-sample t-test. As in the 1-sample test, 7 of the 10 electrodes were found to be different between the two beta parameters in the uncorrected tests, and these differences overlapped between the tests. Three electrodes were found to be significant at $\alpha = 0.05$ using the same clustering correction technique used in the 1-sample test. Two of these cluster-corrected electrodes overlapped with the previous 1-sample cluster-corrected analysis, both in the right hemisphere. Thus, to summarize, Go-trials in stop-relevant blocks displayed a systematic positive relationship between N1 amplitudes and RTs, and this relationship was significantly stronger than in the stop-irrelevant blocks, for which no clear relationship was found.

2.4 Discussion

The present EEG study investigated the neural processes underlying proactive response inhibition during the stop-signal task in human subjects, focusing on early attentional mechanisms. Based on a comparison of go-trials from different trial blocks in which stop-stimuli were either task-relevant or not, we found that participants indeed employed proactive response slowing in the relevant blocks, and a hierarchical drift diffusion model indicated that this mostly relied on a combination of differences in decision thresholds as well as in drift rates. This effect was accompanied by a significant rela-

tionship between the single-trial amplitudes in the visual N1 component in the stop-relevant but not the stop-irrelevant task blocks. Given that the N1 component is believed to index the level of attention paid to the go-stimulus, these results seem to reflect a down-regulating strategic process that proactively slows go-stimulus processing when the response to this stimulus might have to be canceled.

The role of visual attention in response inhibition. Go-stimuli elicited a classic inferoposterior N1 component. This component has been found to be larger the more attention that is paid to a stimulus, which is thought to index the selective attentional processing of the visual stimulus in mid- and high-level visual areas (Vogel and Luck, 2000), and which has been found to ramify into differences in response speed in attentional tasks (e.g., Talsma et al., 2007). The traditional ERP analysis did not find evidence for an inverse link between overall N1 amplitude and response speed, given that the condition with slower responses had slightly larger N1 amplitudes (and a very small effect size). Yet, given the difference in task requirements this between-block comparison is necessarily quite unspecific (see also below) and our main interest was to look at the relationship between fluctuations of response times and EEG activity within the respective blocks. In line with our expectation, a multilevel single-trial EEG framework indicated that such a link does exist when looking at fluctuations within the stop-relevant condition. Specifically, this analysis demonstrated that as response time increased,

the N1 component attenuated, but only in the stop-relevant blocks.

In general, the role of attention and other perceptual mechanisms has mostly been neglected in the response inhibition literature (although see e.g. Sharp et al., 2010 for a discussion concerning whether response-inhibition-related fMRI activity might not in fact reflect activity in the ventral attentional system). Yet, in order to cancel a pre-potent response it is clear that first all relevant external stimuli need to be detected. The overarching view is to attribute differences in stopping latencies solely to differences seen in the efficacy of a single centralized response-related inhibitory control process (Verbruggen et al., 2014a). The current research suggests a clear role for early perceptual/attentional modulations in the stop-signal task. In this vein, the attenuation of N1 amplitudes as responses slow can be considered as an index of the discrimination dedicated to the go-stimulus. A down-modulated go-stimulus processing therefore appears to be advantageous for later inhibition via the positive relationship between successful inhibitory behavior and longer response times in the independent race model. While our inference is in principle a reverse one (inferring that attention was affected by looking at a neurophysiological marker without explicitly modulating it through our task design), we point to the tight and rather specific link between the N1 component and attention.

Crucially, the present data indicate that the relationship between response slowing and attentional processing of the go-stimulus is indeed under proactive control. An alternative notion would have been that attentional go-

stimulus processing randomly fluctuates (e.g., as a function of general attentiveness). Yet, under a random-fluctuation account one would expect similar modulations also in a task context when Stop-stimuli are not task-relevant, which is counter to what we found here. Although this between-block comparison might by itself not rule out non-strategic contributions, our notion dovetails with recent work on Bayesian dynamic belief models, which have found a very strong relationship between Go-trial response time and the inferred subjective probability of a stop-stimulus, which was also interpreted as being strategic in nature (Ide et al., 2013). Although perceptual mechanisms are usually neglected in response-inhibition studies, there is still some supporting evidence that attention to go-stimuli plays a role in adjusting response tendencies. Previous MEG work has shown that the go-stimulus N1 component was less pronounced in successful stop-trials as opposed to unsuccessful stop trials, suggesting that paying less attention to the Go-stimulus slows down responding, which in turn makes successful inhibition more likely (Boehler et al., 2009; see also Knyazev et al., 2008). Furthermore, when perceptual distractors in a stop-signal task were presented over whole trials, inhibitory behavior was impaired, and this impairment scaled with the degree of discrimination difficulty (Verbruggen et al., 2014b). To add to this, using pre-stimulus oscillatory EEG it was shown that a failure to lateralize occipital alpha activity in response to an attentional cue was predictive of false alarms (Bengson et al., 2011). Taken together, these studies suggest that the way in which sensory systems are adjusted to detect relevant stimuli

is an important aspect of response inhibitory behavior.

Turning from go- to stop-stimulus processing, related studies have shown that the attentional processing of the stop-stimulus plays an important role in determining behavioral outcome, with enhanced attention for successful stop-trials (e.g. Dimoska and Johnstone, 2008; Bekker et al., 2005; see also Salinas and Stanford, 2013 for a related finding in a countermanding saccade task, and Kramer et al., 2013), or alternatively with the N1 as a marker of visual attention already reflecting an inhibitory mechanism (Kenemans, 2015). One interesting question here relates to the relationship between these modulations of the attentional processing of the go-stimulus vs. stop-stimulus in a given stop-trial, with one suggestion being that attentional resources need to be shared across these different components (Boehler et al., 2009; Pessoa, 2009). Given that at the moment of go-stimulus presentation participants cannot know yet that a given trial will be a stop-trial, this implies that such "anticipatory" resource sharing with a potentially upcoming stop-stimulus should also happen on regular go-trials. Yet, on the basis of the present data we cannot decide whether the observed effects relate to the anticipation of possible (relevant) Stop-stimuli or whether the attentional processing of go- and stop-stimuli proceed largely independently.

Although go-stimulus processing naturally precedes stop-stimulus processing, this does not necessarily imply that such effects are the earliest in time that possible mechanisms contributing to proactive slowing could be occurring. For example, a number of studies have related proactive response

slowing to neural activity that precedes a given stop-trial altogether (Cai et al., 2011; Majid et al., 2013; Zandbelt et al., 2013). Similarly, it is likely that attentional control settings are implemented before the presentation of a given trial. Such preparatory effects may in fact be particularly likely in the present case in a blocked strategic way because of the non-selective nature of our manipulation. In contrast, other work investigating proactive inhibition has employed selective stopping paradigms in which, for example, one of two possible go-responses might have to be inhibited (Aron, 2011), which might require a more refined and selective mechanism than in our case where a global mechanism of slowing down all responses is likely applied.

Relationship to motor-level inhibition and drift diffusion models. Given the wealth of existing research linking proactive slowing to parts of the response-inhibition network (see, e.g., Zandbelt and Vink, 2010; Van Belle et al., 2014; Boehler et al., 2011; Jahfari et al., 2010; Chikazoe et al., 2009; Lavallee et al., 2014), we do not consider the present effect as the only mechanism underlying proactive response slowing. Rather, we assume that different mechanisms co-exist, and that neurophysiological measures might be more sensitive to the transient effect described here (but see an fMRI study by Li et al., 2009 for possible involvement of sensory areas in response slowing, as well as van Belle et al., 2014, for the involvement of dorsal attentional control areas in proactive response inhibition; and Jahfari et al., 2015, for the interplay between the prefrontal cortex and basal ganglia system with perceptual

systems in response inhibition), but which may be less sensitive than fMRI to mechanisms that act more directly on the motoric level. Consistent with this notion of multiple mechanisms, our diffusion drift model of the behavioral data indicated that more than one parameter was affected. Specifically, we replicated an effect on decision thresholds that has been described previously (Verbruggen and Logan, 2009a), but we also found a pronounced effect on drift rates. The latter has also been reported before, but was found to be difficult to interpret (Logan et al., 2014; but see, White et al., 2014). One possible explanation is that decision-threshold adjustments are implemented within the stopping network, whereas drift rate reflects the attentional mechanism we describe here. The latter seems to intuitively fit well, given that the attentional processing of a task stimulus clearly relates to the speed with which it is being discriminated. It seems possible that the balance between these different mechanisms is adjusted based on strategy differences, as well as possibly being related to specific features of a given task. In the present study, we have focused a-priori on attentional processes. Additionally, possible subsequent mechanisms in frontal or even subcortical areas that might be more directly related to adjusted decision thresholds might be difficult to pick up with EEG due to anatomical reasons (but see, O'Connell et al., 2012; Twomey et al., 2015). Another aspect in which the drift diffusion data seems relevant concerns the fact that the comparison between the stop-relevant and the stop-irrelevant blocks is necessarily somewhat unspecific. Specifically, it is likely that more than just proactive inhibition differed between the blocks.

The fact that the stop-relevant blocks featured the possibility of having to cancel a response creates a dual-task situation (and as far as representing this task rule, this is also true for Go-trials), which has been suggested as an additional contributing factor to response time differences (Verbruggen and Logan, 2009a; see Zandbelt and Vink, 2010 for an attempt to circumvent this problem by parametrically varying the expectation of Stop-trials). Yet, the fact that non-decision time appears to not exert its effect between blocks in the response process indicates that a dual-task hypothesis is not very likely to account significantly for the observed data (see Verbruggen and Logan, 2009a for an extended discussion related specifically to the stop-signal task). Consistent with this, the faster response times in stop-irrelevant blocks were accompanied by lower accuracy, in line with a generally faster response mode that comes at some cost for response accuracy. Still, the comparison probably suffers from some global differences between the blocks, which in our opinion might in part have given rise to the N1 differences in the ERP between blocks, which featured larger N1s in the stop-relevant blocks and might reflect the generally increased task requirements of the stop-relevant blocks. In contrast to that, we consider it a major strength of the single-trial-based approach, which was the main analysis of interest here, that such global differences should play less of a role as far as differences in behavior and EEG activity across trials within the different blocks is concerned. Given that the task requirements remain stable across those trials, we believe that our main finding of a single-trial-based covariation between response time and the N1

component should be mostly unaffected by global block differences.

Conclusion. In the current report we present evidence that strategic modulations of the attentional processing of go-stimuli in a stop-signal task relate to the degree of proactive response slowing on a single-trial level. Specifically, an inverse relationship between single-trial amplitudes of the visual N1 component and response speed during go-trials was found in a context that might require response inhibition, while no such relationship existed when response inhibition was never required. This is in accordance with recent results suggesting a strong dependency between go-trial behaviors and stopping (White et al., 2014). The present attention-related effect likely coexists with additional proactive inhibition mechanisms. Our findings specifically emphasize the role of proactive attentional modulations in inhibitory control, thus contributing to a more multifaceted view of proactive control. Yet, integration of these disparate parts will be important to better understand inhibitory deficiencies in the future.

2.5 Acknowledgements

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Chapter 3

Motivational context for response inhibition influences proactive involvement of attention[1]

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[1]Langford, Z.D., Schevernels, H., & Boehler, C.N. (2016). "Motivational context for response inhibition influences proactive involvement of attention." *Scientific Reports, 6*

Abstract

Motoric inhibition is ingrained in human cognition and implicated in pervasive neurological diseases and disorders. The present electroencephalographic (EEG) study investigated proactive motivational adjustments in attention during response inhibition. We compared go-trial data from a stop-signal task, in which infrequently presented stop-signals required response cancellation without extrinsic incentives ("standard-stop"), to data where a monetary reward was posted on some stop-signals ("rewarded-stop"). A novel EEG analysis was used to directly model the covariation between response time and the attention-related N1 component. A positive relationship between response time and N1 amplitudes was found in the standard-stop context, but not in the rewarded-stop context. Simultaneously, average go-trial N1 amplitudes were larger in the rewarded-stop context. This suggests that down-regulation of go-signal-directed attention is dynamically adjusted in the standard-stop trials, but is overridden by a more generalized increase in attention in reward-motivated trials. Further, a diffusion process model indicated that behavior between contexts was the result of partially opposing evidence accumulation processes. Together these analyses suggest that response inhibition relies on dynamic and flexible proactive adjustments of low-level processes and that contextual changes can alter their interplay. This could prove to have ramifications for clinical disorders involving deficient response inhibition and impulsivity.

3.1 Introduction

Successful motor control is dependent on the interacting dynamics of activation and inhibition mechanisms. The latter mechanisms play a fundamental role in typical and in atypical cognitive functioning, e.g. in attention-deficit hyperactivity disorder (ADHD), schizophrenia, and Parkinson's disease (Chambers et al., 2009). The stop-signal task is a highly influential response-inhibition paradigm, which has been developed to characterize the behavioral components of motor inhibition, in particular the stop-signal response time (SSRT, Logan and Cowan (1984)), and to investigate the neural processes involved (Aron et al., 2007).

Research has focused mainly on mechanisms triggered by the stop-stimulus, dubbed reactive stopping, for which a network of brain areas has been identified as being relevant. The most influential instantiations of this network recruit the right inferior frontal gyrus, the pre-supplementary motor area, and the subthalamic nucleus (Aron et al., 2014; Chambers et al., 2009), and are stop stimulus activated. A parallel line of research has shifted focus towards preparatory inhibitory mechanisms, in part because of their ecological relevance (Aron et al., 2014; Verbruggen and Logan, 2009b; Aron, 2011), and possible derailment in disorders such as ADHD (Bhaijiwala et al., 2014). The hallmark of this proactive form of inhibition is in delayed response times in situations where outright stopping *might* be required. A delayed response to a go-stimulus increases the probability of successful inhibition for any given

trial (Verbruggen and Logan, 2009a) and preparatory processes are generally believed to benefit reactive inhibition (Chikazoe et al., 2009). Moreover, it has been shown that response speed can be adjusted on a very short time scale (Verbruggen and Logan, 2009b) and that it is possibly related to the computation of a trial-wise (subjective) expectation of encountering a stop-signal in the upcoming trial (Ide et al., 2013). Experimental studies have suggested that proactive response inhibition recruits the same reactive response-inhibition network as described above, which then implements partial instead of complete inhibition (Aron et al., 2014; Jahfari et al., 2015; Wessel and Aron, 2013). Given the wealth of converging evidence, this is one likely explanatory mechanism in the variation seen in proactive inhibition, but not necessarily the only explanation, or the sole mechanism involved (Belle et al., 2014; Jahfari et al., 2015; Li et al., 2009; Huster et al., 2014).

It is typically assumed that in both reactive and proactive inhibition behavior ultimately depends on the action of a central response-inhibition module (Stuphorn, 2015). Yet, computational work suggests that a large portion of the time needed to implement response inhibition is taken up by non-inhibitory processes related to the processing of the stop-stimulus (Boucher et al., 2007; Salinas and Stanford, 2013; Verbruggen et al., 2014), and some recent experimental work has varied inhibition demands while controlling for such processes (Erika-Florence et al., 2014; Hampshire, 2015) (suggesting a less modular system, e.g. Hampshire and Sharp (2015)). Parallel to reactive control (Bekker et al., 2005; Kenemans, 2015), in the domain

of proactive inhibition the involvement of attentional processes has recently been emphasized (Elchlepp et al., 2016; Verbruggen et al., 2014; Dimoska and Johnstone, 2008; Jahfari et al., 2015). In a first relevant study, magnetoencephalographic data showed that the attentional processing even at the level of the go-stimulus of a stop-trial varies in a way that affects behavior, in that it is enhanced when response inhibition is ultimately unsuccessful (Boehler et al., 2009). In this same vein, a recent electroencephalographic (EEG) study of ours showed evidence that for go-trials the inferoposterior N1 component (an index of selective attentional processing (Luck et al., 2000)) was being systematically down-regulated as response times were slowed, but only when outright stopping was contextually relevant (Langford et al., 2016). Since this work focused on go-trials, it clearly relates to proactive response inhibition, meaning that the respective fluctuations in RT and attention are cautionary and preparatory in nature, rather than being related to outright inhibition.

In addition to the involvement of attention in response inhibition, there is a growing body of evidence implicating early attentional processes in reward-related processes (Kiss et al., 2009; Hickey et al., 2010; Hopf et al., 2015; Donohue et al., 2016), as well as in their interaction (Greenhouse and Wessel, 2013). Consistent with this convergence on early attentional processes, it has recently been shown that reward can also play a modulatory role, in particular in measures of reactive response inhibition (Scheres et al., 2001; Rosell-Negre et al., 2014). We have recently reported a series of studies in

which associating reward with one of two possible stop-stimulus colors also led to shorter SSRTs, despite the fact that any difference in global preparation was precluded since reward-related trials were presented in a random sequence together with all other trial types (Boehler et al., 2012; Boehler et al., 2014). In an EEG version of this experiment, we have demonstrated that enhanced attention to the reward-related color seems to play a role in bringing about the reward-related SSRT benefit (Schevernels et al., 2015). Furthermore, this experiment featured task blocks that were devoid of any reward associations. Using these trials as a comparison, we demonstrated that the sensory/attentional N1 component to the go-stimuli of stop-trials was enhanced throughout the reward-related block. Given that reward was exclusively related to stop-stimuli, we considered this effect on the go-stimuli (of stop-trials) a context effect in the sense that attention is increased globally (Jimura et al., 2010). Currently we investigated contextual effects of rewarding successful reactive response inhibition on proactive inhibition; although this is a indirect as far as motivational effects on proactive inhibition goes, it circumvents the problem that simply rewarding a cautious response mode will lead to rather trivial response slowing.

The present study used single-trial EEG analyses to model the covariation of the visual N1 with response time framed within the context of proactive response inhibition. We focused on go-trials and the relationship between the sensory/attentional N1 component and response time in rewarded-stop (RS) and standard-stop (SS) task blocks (see Figure 3.1c). For the SS blocks,

we anticipated to replicate our earlier results of a systematic relationship between single-trial response times and N1 amplitude (Langford et al., 2016). For the RS context, however, we hypothesized two different possible patterns of results: **(1)** The relationship between response times and N1 amplitudes could be similar or even more pronounced, given that we consider it a marker of proactive response inhibition, which is generally a useful process when trying to successfully inhibit a response (Chikazoe et al., 2009), and could hence be more strongly engaged in a task context in which successful inhibition can be rewarded. **(2)** The global attentional increases driven by reward context(Schevernels et al., 2015; Jimura et al., 2010) might interfere with or abolish the relationship between visual attention and response speed. This might be particularly relevant here because of the rapid temporal succession of go- and stop-stimuli, which might preclude a fully specific reward effect on stop-stimuli without simultaneously also enhancing the processing of go-stimuli presented at the same spatial location. We further modeled the behavioral data as an evidence accumulation process to pick apart the perceptual decision making dimensions that are relevant for differentiating between the SS and RS task blocks (see, Logan and Cowan (2014), Verbruggen and Logan (2009b), and Jahfari et al. (2015) for further motivation related to response inhibition, and see (White et al., 2014) specifically for the relationship between RT and stopping behavior in a drift-diffusion framework). Specifically, we were interested to see whether evidence accumulation would proceed faster in a reward context, which would be consistent with an

attentional increase to go-stimuli.

3.2 Methods

The analysis in the current manuscript is based on previously reported data (Schevernels et al., 2015). In the present manuscript, we focus on procedures and methods pertinent only to the current analyses, which largely focus on go-trials from a task context in which successful inhibition in stop-trials would yield a reward vs. one where this was not the case, which were not analyzed for the previous report.

3.2.1 Participants

Twenty healthy right-handed students participated in the experiment (6 males, $\overline{age} = 22$). All participants had normal or corrected-to-normal vision and no history of psychiatric or neurological disorders. Written informed consent was obtained from all participants. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethical Committee of the Faculty of Psychology and Educational Sciences at Ghent University. Participants received a base compensation of 20 euros and an additional performance-dependent bonus described in the next section. Because of a considerable number of missed and incorrect go-trials (16.5%, with a range of 1% and 9% for the remaining participants), the data of one participant were excluded from all analyses as already done in the earlier report

using this dataset (Schevernels et al., 2015).

3.2.2 Stimuli and Procedure

Throughout the experiment a black rectangular box and a white fixation dot were presented on a grey background at the center of the screen. Go stimuli were green traffic light symbols pointing to the left or to the right. The target go stimulus was presented centrally above the fixation dot, and was additionally surrounded by two green traffic symbols on both sides that were balanced in congruency (i.e., directly next to the target was one stimulus with the same orientation and one with the opposite, again flanked by their respective mirror image) and had to be ignored. This exclusively served to globally increase the attentional load of the task, without varying the congruency level. Participants were asked to respond rapidly with the index finger (left mouse button) or middle finger (right mouse button) of their right hand according to the orientation of the central go traffic sign. A don't-walk traffic sign was used as a stop stimulus. The color of this signal could either be blue or pink with equal proportions. In both go and stop trials the total stimulus presentation duration was 600 ms, followed by a randomly-distributed inter-stimulus interval of 1000 to 1400 ms. Participants completed two blocks, a reward block and a no-reward block, and block order was counterbalanced across participants.

Participants started with a short practice run, including 34 go trials and 20 stop trials with 10 blue stop signals and 10 pink stop signals. In stop trials

the interval between a go and stop stimulus (go-stop delay) was constantly adapted to create and maintain a 50 percent rate of correct stopping. A staircase procedure was implemented that increased the go-stop delay by 34 ms after a successful stop trial (SST) and decreased it by 34 ms after an unsuccessful stop trial (UST), with a minimum of 34 ms and a maximum of 567 ms delay (starting value: 200 ms). Pink and blue stop trials shared the same staircase, which hence controlled the stopping-success rate over all stop trials within a block. Reward was only assigned to one of the stop signal colors at the start of the reward block, i.e. immediately after training for half of the participants (for whom the reward contingency was then explicitly removed at the end of the block) and after the no-reward block for the other half. Block order and the color of reward-predictive stop signals (pink or blue) were counterbalanced across subjects. Two long blocks were used to minimize possible carry-over effects related to reward-related colors(Hickey et al., 2010; Libera and Chelazzi, 2009).

Both experimental blocks consisted of 5 runs of 100 trials each, yielding a total of 320 go trials and 180 stop trials (90 trials for each color) per block. In the reward block participants could win points for successful response inhibition in reward-related stop trials, but not in reward-unrelated stop trials. At the end of every run the amount of points gathered in that run was shown. Participants were also informed that these points would be added up at the end of the block, yielding an extra bonus of between 0 and 6 euro based on a specified transformation from points to money. Subjects were asked to

respond as fast as possible and not to slow down their responses during the experiment, which is important when evaluating stopping performance (Verbruggen et al., 2013). Additionally, to further prevent such slowing, participants were told that the collected points in a run would be set to zero in case they significantly slowed down their responses. Since this procedure turned out to be quite effective, this correction was never actually used.

3.2.3 Recording and Analysis

EEG recording

EEG data was collected with a 64 channel Biosemi ActiveTwo system (Biosemi, Amsterdam, Netherlands) using a standard 10-20 system, sampling data at 256 Hz. External electrodes were attached to the left and right mastoid and at the outer canti of both eyes and directly above and below the left eye. Data were re-referenced offline to the average of the left and right mastoid and a low-pass FIR filter was applied at 30 Hz (-6 dB attenuation at 33.7 Hz). Blinks were removed using independent component analysis. Go-locked epochs were made with a time window from -200 to 1000 ms, using the pre-stimulus period for baseline correction. Automatic artifact rejection was performed on these epochs with a subsequent visual inspection to reject missed artifacts. Automatic rejection removed trials with values that fell outside of -/+150 mV. Furthermore, epochs including horizontal eye movements were detected by a step function in the bipolar eye channel (with a

threshold of 60 mV, window size of 400 ms and window step of 10 ms). After all rejection techniques were applied an average of 94.6 percent of epochs remained.

Single-trial EEG analysis of the inferoposterior N1 and exploratory extension to later time-ranges

The inferoposterior N1 locations were chosen to be closely aligned with previous work (Vogel and Luck, 2000). These included 6 posterior electrodes, 3 on the right and 3 on the left of a standard 10/20 EEG system (specifically, O1, O2, PO7, PO8, P7, P8). The 6 electrodes were used in further analyses to examine the visual N1 with the time range defined between 130 ms and 190 ms after the onset of a go-signal. Such inferences in principle are reverse inferences (inferring that attention was affected by looking at a neural marker without explicit modulation), yet there is a highly specific link between the N1 component and attention.

The analysis of interest investigated the relationship between single-trial EEG data and response speed on go-trials in the SS and RS blocks. To this end, single-trial EEG analyses were carried out using the package LIMO EEG (Pernet et al., 2011). At the first-level single-trial analysis fits a general linear model of the form $y_{e,s} = X\beta_{e,s} + noise$ to trials of EEG data (y), separately for all analyzed electrodes (e) and sampling points (s) in the N1 time window (130 ms to 190 ms post go-stimulus). The five predictors in the design matrix X were the categorical SS and RS go-trial types, the single-trial normalized

(per subject, per condition) reaction times, and a noise variable.

Two types of tests were run on the group level data as implemented in LIMO EEG: **(1)** We used robust one sample t-test that tests if the average effect (mean of β values) significantly differs from zero. This determines the significance and direction of β parameters per sample point in each context, individually. In this approach, subject beta weights are drawn randomly with replacement to provide an approximation of the t-distribution under the null hypothesis (i.e. bootstraps on centered data). **(2)** In a similar manner we computed paired samples t-tests to generate a null distribution of t-values to test β differences between contexts. To account for multiple comparisons, we used temporal clustering by which only clusters with a mass (sum of t values) bigger than the 95th percentile of the null distribution are considered significant. In this case, the null distribution corresponds to the maximum cluster value across electrodes measured at each bootstrap computed on the nullified data (**pernet2015**).

Following these analyses, we determined the electrode for each subject individually that maximized model fit based on R^2. This vector of maximized R^2 electrodes was then used to run another 1-sample t-test on the β parameter of the RT predictor in the SS condition. To visualize this effect we plotted the model predictions by normalizing the max R^2 electrodes for each individual, aligning by response time, and averaging over all individuals.

While we had strong hypotheses about the visual N1 based on past research, a more exploratory analysis was undertaken to probe other later

effects known to be attention related. In this analysis we used the same procedure as with the N1 but with a broader time window (200-600 ms post-go presentation) and the full electrode montage. The same 1-sample t-tests and paired samples t-tests from the N1 analysis were used to determine differences in the population.

Behavioral analysis

Behavioral data was analyzed concerning standard response-time and accuracy parameters before (Schevernels et al., 2015), which we reproduce here when needed. Additionally, we ran drift diffusion models. Drift diffusion models encapsulate a mathematical description of a binary choice process and are defined by three central parameters (Ratcliff, 1978), that have also have been used to quantify the decision processes of proactive inhibition (Verbruggen and Logan, 2009b; Jahfari et al., 2010; Logan and Cowan, 2014). These parameters are the response threshold (a), the rate of approach to the threshold, known as drift rate (v), and processes that precede and succeed the actual decision process giving rise to nondecision time (t_0).

Bayesian estimation was used to model the parameters using the Hierarchical Drift Diffusion Model (HDDM) software (Wiecki et al., 2013). Seven candidate models were fit to the data; a null model, a full model (including a, v, and t_0), and 5 reduced models; a model without t_0, a model without a, a model with only a, a model with only v, and a model with only t0. 40,000 posterior samples were taken for each model, with a burn-in of 10,000

samples, and a thinning factor of 3. Five percent of the behavioral data was assumed to come from a uniform distribution that is not adequately explained by drift diffusion processes (e.g., physiological interruption, task unrelated distractions (Ratcliff and Tuerlinckx, 2002)). Each model was checked for convergence using the Gelman-Rubin diagnostic(Gelman and Rubin, 1996).

Model fit was assessed using the deviance information criterion, DIC (Spiegelhalter et al., 2002). DIC penalizes how well the model fits the data, as well as the number of parameters used to explain the data. Furthermore, posterior-predictive checks were made on each model to assess the performance and reasonableness of the model estimates and to check the models ability to reproduce the observed data. After model selection, posterior distributions were probed to determine differences directly in the parameters between the SS and RS task contexts. This is accomplished by examining the proportion of posterior samples falling above or below a two specified posterior distributions, resulting in a probability that one posterior distribution is greater or less than the other (Kruschke, 2010).

3.3 Results

3.3.1 Single-trial EEG analyses

Attentional covariation with response time

As a first exploration the average of all N1-related electrodes were plotted as a function of individually normalized response time in Figure 3.1a. These data were time-locked to the onset of the go-signal of *correct* go-trials. A clear N1 effect is seen within the analysis window (130 to 190 ms). This N1 effect visibly dampens as RT increases in the SS context, but is sustained across RT in the RS context. To explore this quantitatively a robust multilevel single-trial EEG methodology was applied in two separate steps. This is an optimal method as it can account for both categorical (context) effects and parametric (RT) effects within a unitary model. In the first-level statistical models regression parameters were estimated for each individual at each of the inferoposterior N1 electrodes for each time-sample between the defined time window. These included linear predictors for the categorical RS and SS factor, continuous response time per context, and noise, for each of the 6 electrodes and for each sample point.

Standard-stop go-trials and response time

Covariational RT and EEG differences within the SS context were tested using a bootstrapping 1-sample t-test procedure to 'synthesize' individuals' β

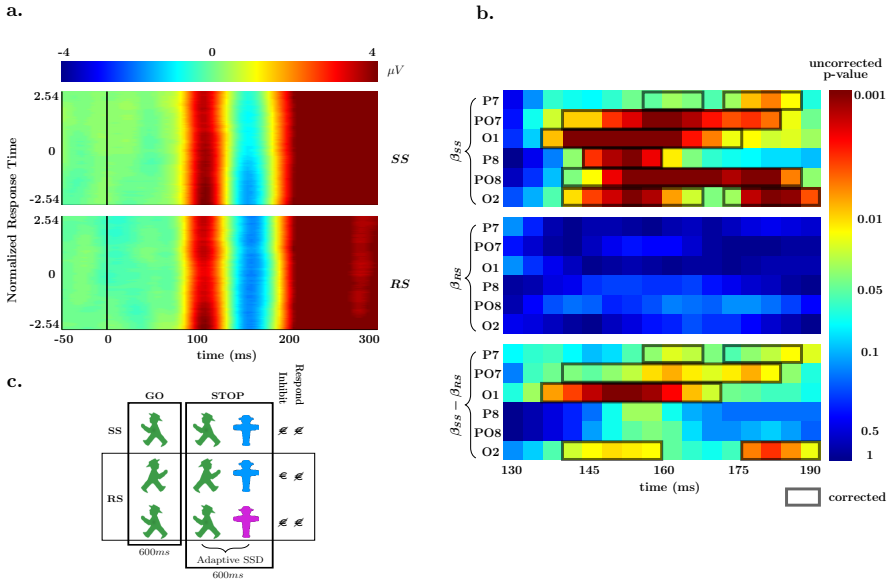

Figure 3.1: **Response Time Effects. a.** Averaged N1 EEG electrodes (O1, O2, PO7, PO8, P7, P8) data plotted by normalized (per individual) response times in the standard-stop context (top) and rewarded-stop (bottom). **b.** Uncorrected p-values for the standard-stop (SS, top) 1-sample t-test, reward-stop (RS, middle) 1-sample t-test, and paired-sample t-test (bottom) for six electrodes. Significant temporally corrected values at $\alpha = 0.05$ are overlaid. **c.** Stimuli and paradigm (flanking stimuli are not shown) showing go-trials, stop-trials, and potential payouts for stop-trials in both the SS and RS blocks. The color coupled to reward (blue in the example) for a successful inhibition was instructed before the block began.

parameters for statistical testing at the second level of analysis. This is a test that probes the relationship between RT and N1 amplitudes. Within the N1 electrodes chosen for analysis, a clear pattern emerged in the SS trials. As RT slowed in the SS context the inferoposterior N1 voltages systematically became less negative across all individuals. Note that while the analysis is performed separately for multiple time-points, correcting for multiple com-

parisons using a 1-dimensional temporal cluster correction showed evidence that such an effect was indeed present in all analyzed electrodes and peaked around 160 ms (see β_{SS} of Figure 3.1b).

Further testing was done by constructing an optimized electrode vector from each individual's electrodes using the maximal r^2 as the decision criteria for inclusion (see(Rousselet and Pernet, 2011) for the motivation behind optimized averaging in EEG research). The same 1-sample t-test was run on this optimized vector of electrodes to support the previous finding. Cluster-corrected significant differences were found from ∼148 ms until ∼167 ms. The model was then inverted and the predicted voltages given by this optimized electrode vector were normalized for each individual and subsequently collapsed across individuals to develop a predicted-voltage plot of RT * post-go time * voltage to visualize the effect (see Figure 3.2). Included in Figure 3.2 is an overlay that depicts the relationship between RT and the probabilities of either successful or unsuccessful inhibition (as predicted using the horse race model(Logan and Cowan, 1984)).

Rewarded-stop go-trials and response time

Go trials from the RS blocks were submitted to the same second level covariational approach used to test the β parameters in the SS blocks. The N1 attenuation effect as a function of RT seen in the SS task was completely absent from the RS blocks. The p-values for the 1-sample t-test are shown in the middle plot (β_{SS}) of Figure 3.1b. This indicated that there was a lack of

evidence for a covariational relationship between RT and the N1 at a single trial level (i.e., accept null $\beta = 0$, even in the absence of multiple comparison corrections).

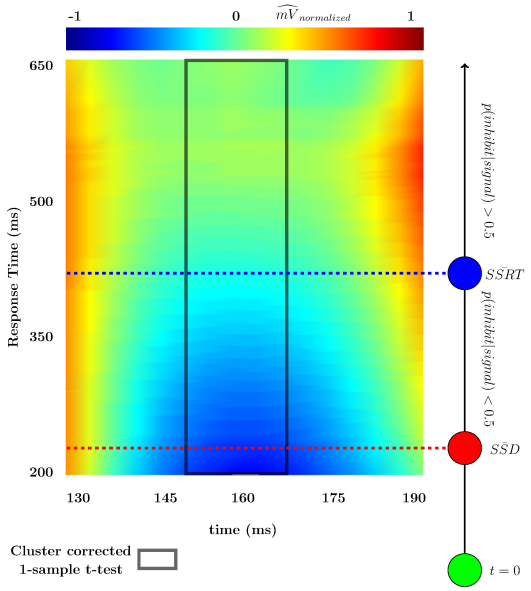

Figure 3.2: **Model Prediction.** Predicted voltages individually normalized and then averaged across individuals plotted by response time and time since go presentation. Electrode plotted for each individual was the electrode from the optimized r^2 vector. Significant cluster from 1-sample t-test overlaid on top. To the right the relationship between SSRT, SSD, and $p(inhibit|signal)$ are shown in relation to go-RT and the single trial N1.

RT differences between contexts

To test that the relationship between RT and N1 amplitude seen in the 1-sample t-tests are more than qualitatively different between SS and RS contexts, a paired-samples bootstrapping t-test was applied to the RT beta coefficients. Four of the six electrodes were shown to be different between

the RS and SS contexts using the same 1-dimensional temporal cluster correction method, as shown in the bottom panel (β_{SS} - β_{RS}) of Figure 3.1b. To summarize the covariational RT effects we observed a relationship between single-trial N1 amplitude and response speed, with longer response times being related to slower behavioral responses, similar to what we have observed in an earlier study in a standard stopping context (Langford et al., 2016). Importantly, this relationship was exclusive to the SS and absent in the RS context.

Categorical differences between contexts

Group-level differences in the categorical factors RS and SS were tested using the same paired samples bootstrapping t-test. This is generally related to a standard ERP, but controlling for RT, and additionally modeling noise. For reference to a common (ERP) analytic scheme, the standard ERP is shown averaged over the 6 N1 electrodes in Figure 3.3a. For the paired samples t-test all 6 of the electrodes tested showed more negative amplitudes in the RS N1 at an uncorrected threshold of $\alpha = 0.05$, as is seen in Figure 3.3b. After controlling for multiple comparisons using a 1-dimensional cluster correction, 3 of the 6 were found to be different (specifically PO7, P8, and PO8). These effects started at $\sim$140 ms post-stimulus and continued (for some electrodes) until the end of the tested interval of 190 ms. Go-trials in the RS blocks had more negative amplitude than in the SS context. This result extends our earlier observation in this dataset of an enhanced N1 ERP amplitude for the

go-stimuli presented during stop-stimuli (Schevernels et al., 2015).

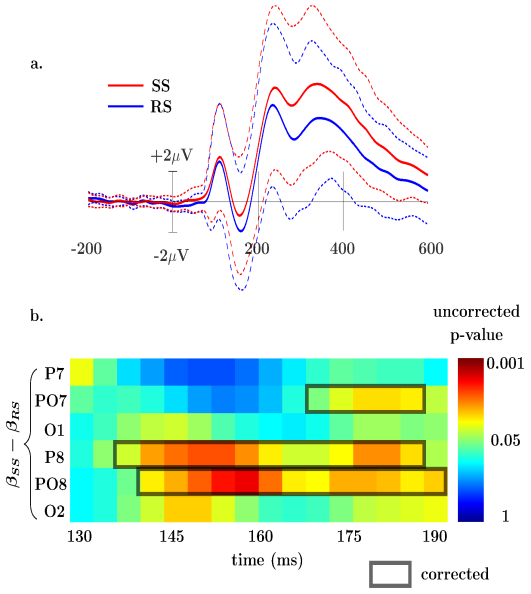

Figure 3.3: **Categorical Effects. a.** Standard ERP of all 6 N1 electrodes averaged with 95% confidence interval. **b.** Time course of p-values from the paired samples t-tests with the tested range of 130 to 190 ms post-go presentation for the categorical β parameters of the SS and RS blocks.

N2-P3 complex

While the N1 component was our explicit a-priori focus of interest, we temporally extended our analysis of possible EEG-response-time covariation in the same fashion to identify the possible involvement of later neurocognitive mechanisms. The same model was applied to the full scalp in the window of 200-600 ms after the go-signal in correct go-trials separately for the SS and RS task context. Cluster-corrected 1-sample t-tests of the RT parameters showed evidence of a covariational effect peaking around ∼320 ms in both

the SS and RS blocks, and a later effect peaking around $\sim$ 400 ms in the RS blocks. The earlier component in both blocks was a negative wave (N2) and had a fronto-central scalp distribution (Figure 3.4b), and became more negative with increased RT. The later RS component was a positive-going wave (P3) with a central-posterior scalp distribution (Figure 3.4b), and became more positive with increasing RT. A paired-samples t-test was done on both the categorical and RT β parameters to test for differences between the SS and RS contexts. No differences were found after cluster correction for either paired-samples test. Therefore, as opposed to the N1 component, neither the go-locked N2 nor P3 were sensitive to reward availability for stopping, while also showing some relationship to RT that was largely independent of the two different task contexts.

3.3.2 Behavioral Analysis

Standard analysis

Correct go-trial mean RTs were similar in the RS block (426.5 ± 9.5 ms) compared to the SS block (420.3 ± 8.6 ms). Go-trial accuracy in the RS block ($96.6 \pm 1.1\%$) was also similar to the SS block ($96.9 \pm 0.6\%$). The average stop-signal delay in the SS blocks was 229.3 ± 9.4 ms, and the average SSRT in the SS blocks was 177.3 ± 4.2 ms (reproduced here for Figure 3.2, see (Schevernels et al., 2015) for further details related to SSRT, stop-trial behavior, and other calculations not pertinent to the current analyses).

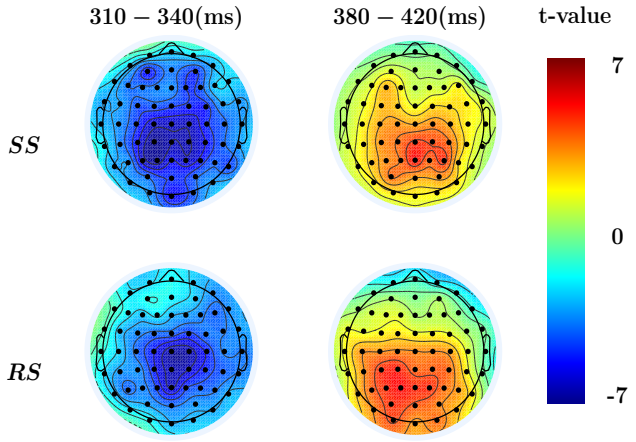

Figure 3.4: **1-sample t-test on EEG-response-time relationship between 200 and 600 ms.** t-values from 1-sample RT β t-tests averaged within the significant cluster corrected time ranges for both the early and late components. The data indicate a negative relationship between RT and EEG data in the N2 range, and a positive one in the P3 range, albeit only for the RS context after multiple-comparison correction

Hierarchical Drift Diffusion Model

Drift-diffusion models incorporate a framework for forced choice decisions that can be used to account for accuracy and response times in a given trial. Models were fit using a hierarchical Bayesian estimation scheme to sample from parametric distributions corresponding to the rate of sensory/perceptual accumulation of go-stimulus information (drift, v), level of response caution (response threshold, a), and the combined time needed for nondecision processes (nondecision time, t_0). Model selection for the HDDM started by fitting a null model, i.e. ignoring differences between go-trials from the SS and RS contexts. SS and RS differences in response threshold, drift, and nondecision time were then successively added to the model, and in combi-

nation (by taking away the initial constraints of equality across SS and RS). Table 4.1 reports the improvement in model fit of Deviance Information Criterion (DIC) for the three best hypothetical models considered, as well as the null model. Based upon DIC, the model with all parameters included was selected for further analyses.

As seen in the bottom of Table 4.1, the differences between the parameter distributions in the RS and SS are different (as seen in the high probabilities that the sampled distributions are dissimilar) for the model that includes all parameters. The RS blocks, relative to the SS blocks, have a raised response threshold, a steeper drift, and also a smaller amount of time devoted to nondecision processes (t_0). Notably, the standard mean and accuracy analyses above had indicated very similar go-trial behavior in the two task blocks. The DDM analysis indicates that this is brought about in different ways, in which in RS blocks short nondecision times and faster drift-rates are compensated for by a raised response threshold in order to maintain a similar response speed, as participants were instructed to do.

3.4 Discussion

The current EEG study investigated neural processes underlying proactive response inhibition during the stop-signal task in human subjects and focused on early attentional mechanisms. The analysis was based on a comparison of go-trials from different trial blocks in which successful response inhibition

	model	$DIC_{difference}$	
	Null	–	
	a, v	-145	
	v, t_0	-101	
	a, v, t_0	-229	
	a	v	t_0
SS	2.57(0.29)	5.05(0.39)	0.18(0.01)
RS	3.06(0.32)	5.52(0.40)	0.15(0.02)
Group Variation	1.36(0.21)	1.58(0.20)	0.06(0.01)
p(high > low)	0.90	0.81	0.92

Table 3.1: **Top** Differences in DIC of tested model from the null model (lower DIC is better). **Bottom** Parameter estimates from (a,v,t) model, group variability, and the probability that the greater value between SS and RS is indeed greater for each of the parameters (i.e. $p(a_{RS} > a_{SS})$; $p(v_{RS} > v_{SS})$; $p(t_{0_{SS}} > t_{0_{RS}})$).

was either explicitly motivated by reward prospect, or not, thereby probing for differences due to motivational context. We found a significant relationship between the single-trial amplitudes in the attention-related visual N1 component and response speed in the standard stop-signal task, but not in the rewarded-stop task blocks. This was accompanied by an overall more pronounced N1 in the reward context. Additionally, we found evidence for relationships between RT and EEG in the N2 and P3 time-ranges, which however did not differentiate clearly between the two motivational task contexts. Finally, despite overall mean behavior being highly similar across blocks, we observed differential results of a drift diffusion analysis, in which

the mere difference in context led to differences in all key parameters. Specifically, a stop-trial-related reward context modulated go-trials to have higher drift rates and lower nondecision times, together with a elevated decision threshold.

Visual attention in a standard stop signal task The classic inferoposterior N1 component is thought to index the selective attentional processing and discrimination of the visual stimulus in mid- and high-level visual areas, and to generally indicate how much attention is paid to a visual stimulus (Vogel and Luck, 2000; Luck et al., 2000). The single-trial ERP analysis demonstrated that as response times increased, the amplitudes of this component decreased, albeit only in the SS block. The damping of N1 amplitudes as responses slow is likely an indication of a down-regulated discrimination of the go-stimulus. Mathematical modeling of the stop-signal task predicts that the slower the response the more probable successful stopping is to occur (Verbruggen and Logan, 2009a). Therefore, a down-modulated go-stimulus processing, as measured by the N1 component, is arguably advantageous for later inhibition. Consistent with this notion, an analysis of stop-trials in this same dataset had indicated that the N1 to go-stimuli was larger for trials that ultimately were not successfully inhibited (Schevernels et al., 2015).

These results in the SS block of the present study replicates our recent report, in which we found the same relationship between N1 amplitudes and response times for go-trials in a standard stop-signal task (Langford et al.,

2016). Additionally, this earlier work indicated that this relationship is likely under proactive top-down control because it was absent in control blocks in which stop-stimuli were task-irrelevant, which abolishes any need not just for outright inhibition but also for strategic response slowing. Notably, in this previous report this absent relationship coincided with a slightly attenuated average N1 ERP component for this stop-irrelevant task context. The present findings are important not least as a replication because our previous report was the first description of this relationship between single-trial N1 amplitudes and go-trial response times. Additionally, some factors were different in the present work, which therefore do not seem to be critical for this relationship to arise. In particular, in the present work, the target stimulus was always flanked by four distractor items, thereby increasing the need for selective attention, whereas our earlier work (Langford et al., 2016) did not feature such distractors. Simultaneously, the present comparison between task blocks is arguably more specific than in our earlier work, in that the two blocks were very similar in many regards, and still yielded different results. Most notably, go-trial performance was extremely similar across the two blocks, so that the present differential results arise in an absence of any clear overt behavioral difference. Additionally, the two blocks did not differ strongly in task requirements. Specifically, our earlier comparison between stop-relevant and stop-irrelevant blocks compared tasks that differed quite fundamentally in requirements, in that a regular stop-signal task sets up a dual-task situation (with opposing requirements to go or stop) that the stop-

irrelevant blocks did not have(Verbruggen and Logan, 2009b). Based on drift-diffusion modeling we argued that it was not the dual-task nature that drove behavior in the stop-relevant blocks, in particular not on a single-trial level. The present data corroborates that the presence of the relationship between N1 amplitudes and response time is not majorly dependent on such task requirements, because those were highly comparable across blocks in the present study.

Another important aspect relates to the relationship between the categorical/ERP differences across conditions (SS vs. RS) and the within-condition variation. A very recent related report investigated the role of visual attention in proactive (inhibitory) control (Elchlepp et al., 2016). These authors found that attention to the go-stimulus is enhanced if this stimulus might change into a stop-stimulus (or an additional control condition requiring a double response). This was interpreted as reflecting the involvement of attentional processes in proactive control, albeit not necessarily in an inhibition-specific fashion. In addition, those authors found that attentional modulations of go-stimulus processing disappeared when stop-stimuli were auditory rather than visual. These results could be interpreted as representing an effect of monitoring a stimulus for a relevant change, much like in our paradigm. We see our categorical reward-context modulation of the N1 as a related process, in which attentional monitoring is generally ramped up in order to optimally detect a reward-relevant stimulus. In our mind, such categorical effects are highly relevant, but suggest that the within-condition

variation is the aspect that is more closely linked to behavior.

Extending the analysis window beyond the planned N1 time- and electrode-range, we found evidence for a relationship between RT and EEG activity in the N2 and P3 time-range, both of which were enhanced (N2 more negative, P3 more positive) as RT increased. While the P3 effect only survived multiple-comparison correction for the RS context, there was no evidence for a real difference between the contexts for either time-range. The results therefore seem to suggest that also later processes scale with RT, without clearly differentiating between the two motivational contexts. A specific interpretation of these relationships seems difficult, given that they concern simply go-trials. Yet, given the context of the stop-signal task, one could speculate about a link to components that are typically found in response inhibition, where N2 and P3 components play a prominent role both in the Go/Nogo task and the stop-signal task (Enriquez-Geppert et al., 2010). Having a related signature in slow-vs-fast go-trials might speak towards additional neurocognitive processes that deliberately slow down responses. Yet, this interpretation is naturally speculative at this point.

Prospect of reward The main contribution of the present study is delineating the effect of a reward context (for successful stop-trials) on the above relationship between attention and response time in go-trials. This contextual modulation had two clear effects. **(1)** The average N1 amplitude was enhanced for go-trials from the reward-relevant task context, probably indi-

cating a generally increased level of attention. **(2)** There was no covarying relationship between the N1 and response time on the single-trial level. This pattern is consistent with the second hypothesis raised in the introduction, namely that a context effect of RS stop-trials generally increased the amount of attention paid also to the go-stimuli, and that this process simultaneously overrides any fine-grained relationship between attention and response speed.

Concerning the behavioral data, one thing to note in particular is that the mean response times were nearly identical in the two task contexts. Overall go-trial response time is typically a bit artificial in the stop-signal task since it strongly relies on the instruction given to the participants (and their compliance to it), i.e. if successful stopping would be the only priority, simply refraining from a button press would be the most successful strategy (typically discouraged by instruction). Along similar lines we instructed participants not to slow down their responses during RS blocks compared to the SS blocks. Yet, despite this high degree of similarity in behavior, the reward context had an effect on go-trial ERPs as well as on the single-trial relationship between RT and the N1 amplitude. This is interesting not least because reward effects often take highly specific forms of benefitting precisely and exclusively the rewarded task aspect, and since stopping performance in this task was specifically enhanced for reward-related stop-trials and not for randomly intermixed no-reward stop-trials that differ only in the color of the stop-stimulus (Schevernels et al., 2015). As indicated above, these effects likely represent context effects in the sense that a reward-anticipation-related

increase in attention to possible stop-stimuli automatically entails enhanced attention to the go-stimuli that always rapidly precede them (in stop-trials) or are presented in isolation (in go-trials) (Elchlepp et al., 2016). While this seems plausible, given the tight temporal succession of events in stop-trials, it should be noted that we and others have also speculated in the past that there is a sharing of attentional resources between go- and stop-stimuli in a given stop-trial (Boehler et al., 2009; Pessoa, 2009). Moreover, it is often found that attention away from a stimulus that might interfere with obtaining a reward is generally considered a feasible mechanism e.g. in delayed gratification context (O'Connor et al., 2012). It seems possible that a reward context also changes these relationships.

Multiplicity of influences on drift diffusion processes As highlighted in the previous section, standard go-trial performance was highly similar across the two task contexts. Yet, looking at drift diffusion modeling, we found that there were in fact subtle changes in the sub-processes of evidence accumulation that jointly determine behavioral outcome. Specifically, both drift rate increased and decision threshold increased, while nondecision time decreased in the RS blocks. A decrease in nondecision time and increase in drift rate leads to quicker response processes, whereas the raised decision threshold led to slower, more conservative responding. Therefore, it seems that the decision threshold compensates for the changes in the other two parameters. While the reason for this might be a bit artificial, in that it likely

relates to the instruction not to slow down responses across the two task contexts, it still illustrates nicely a fine-grained process structure, in which even near-identical mean behavior can arise from different constellations of different drift diffusion parameters (i.e. modeling RT distributions and accuracy is more powerful than summary measures). This finding is generally reminiscent of results from a stop-signal task that used stimuli with different image quality, where it was found that reduced drift rate and increased nondecision time for low-quality images was compensated for by an increased decision threshold (Jahfari et al., 2015). It seems likely that our presented results are strongly related, just the source of variation in stimulus processing is internal rather than external, and likely strategically employed. When speculating about the relationship of these parameters to our electrophysiological results, it seems reasonable to link the N1 modulations to differences in drift rate and possibly nondecision time, in that early attentional processes plausibly map onto both the rate of and starting time of evidence accumulation, which might furthermore relate to anticipatory attentional processes that precede the actual trial (e.g., (Bengson et al., 2012)). The increased decision threshold, in turn, might relate to a process more linked to the motor output level, which we did not capture in our analysis of the EEG data (see, O'Connell et al. (2012), Elchlepp et al. (2016), and Dippel and Beste (2015) for a broad discussion of candidate neural signatures). Given the fact that for obtaining reward going slow would be beneficial (but was explicitly discouraged), the changes in drift rate and nondecision time might also reflect a

non-instrumental context effect, which is then counteracted by an increased decision threshold.

Conclusion The current manuscript provides new evidence of a strategic and dynamic modulation in the attentional processing of go-stimuli in a standard stop-signal task using single-trial ERP analysis. Go-trial amplitudes were dampened the more delayed a response. This confirms our previous work using the same general methodology (Langford et al., 2016), and is in line with work using both a different analysis strategy (comparing go-stimulus N1 amplitudes for stop-trials in which inhibition was ultimately successful vs. not) and neuroimaging modality (MEG) (Boehler et al., 2009). Yet, under a motivational context in the form of reward for successful response inhibition, this modulation was not evident, though attention was in fact generally increased by the mere presence of the probability of a reward. This went along with subtle changes in the relationship between different drift diffusion parameters that overall still resulted in near-identical mean behavior. Together, it appears that a reward context, even if not directly relevant for the processes studied, can introduce changes in a global attentional state, perhaps towards a sustained strategic proactive control mode (Locke and Braver, 2008; Jimura et al., 2010), thereby having an impact on all stimuli and trials that are comprised in this environment. More generally, it furthermore explicitly links differences in proactive slowing to (contextual) motivational factors, hence suggesting additional possible pathological mech-

anisms for patient populations with deficits in proactive response inhibition like ADHD (Bhaijiwala et al., 2014).

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Author contributions statement

Z.L.,H.S. and N.B. conceived the experiment, Z.L. and H.S. conducted the experiment(s), Z.L. and N.B. analyzed the results. All authors reviewed the manuscript.

Additional information

The authors declare no competing financial interests.

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Chapter 4

Anticipatory α oscillations indicate an active role of attention in a cued expectancy stop-signal task

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C. Nico Boehler

Abstract

Response inhibition is required for contextually appropriate behavior, and is implicated in numerous cognitive dysfunctions. The current electroencephalographic (EEG) study investigated proactive adjustments of attention in a lateralized cued stop-expectancy task in order to extend previous work suggesting a role of attentional modulations in proactive response inhibition. In this task participants are given a cue indicating the probability of the current trial containing a delayed centralized stop-signal, as well as a directional cue indicating the hemifield of a delayed go-signal. Prestimulus alpha power was modulated by the stop-expectancy in the delay period between the cue and the lateralized go-signal, with greater lateralization of alpha power as stop-expectancy increased. This was accompanied by response time slowing and a larger attention-related N1 component as stop-expectancy increased. Further a drift-diffusion process model indicated that as stop-expectancy increased participants employed different evidence accumulation processes and a more cautious response model. Together this pattern of results speaks towards a highly flexible proactive response inhibition process that recruits different systems to achieve the goal of inhibiting responses. Yet, the direction of this effect seems to depend substantially on exact task requirements and expectation, and speaks against a simple notion directly binding down-modulated attention to proactive inhibition in a way that generalize across cued expectations of the probability of response inhibition being necessary.

4.1 Introduction

Motor control requires interacting cortical networks that issue both activation and inhibition signals to produce contextually appropriate output. Dysfunction of inhibitory networks is implicated in multiple neurological pathologies and disorders, such as Alzheimer's disease and attention-deficit hyperactivity disorder (Chambers et al., 2009). While these inhibitory networks have been extensively researched there remains debate about their specificity (Hampshire and Sharp, 2015; Aron et al., 2015), as well as how sensory and perceptual processes come to bear on behavioral outcomes.

The stop-signal task has been used to determine both the timing and efficiency of inhibitory processes, and to clarify the neural signatures involved. In the stop-signal task, a go-stimulus requiring a rapid perceptual judgment is infrequently followed by a stop-stimulus, signaling a need to inhibit the response. Behavior in the stop-signal task is usually modeled as a race between two competing processes - a go- and a stop-process. The stop-process latency (the presumed time the stop-process wins) can be estimated (stop-signal response time - SSRT) by assuming a stochastic model. The research focus has been on processes initiated by the stop-stimulus. This line of research - reactive inhibition - has implicated a specific "response-inhibition network" involving the right inferior frontal cortex (Aron et al., 2014; Cai et al., 2014). Yet, computational work suggests that a large portion of the time needed to implement response inhibition is taken up by non-inhibitory processes related

to the processing of the stop-stimulus that would likely precede activity in this network (Boucher et al., 2007; Salinas and Stanford, 2013; Verbruggen et al., 2014; Erika-Florence et al., 2014; Hampshire, 2015; Hampshire and Sharp, 2015).

Proactive inhibition, a concept related to preparatory processes and response caution, has emphasized the involvement of attentional processes in response inhibition (Elchlepp et al., 2016; Verbruggen et al., 2014; Dimoska and Johnstone, 2008; Boehler et al., 2009; Kenemans, 2015). Two recent electroencephalographic (EEG) studies of ours provided evidence that for go-trials the inferoposterior N1 component (Luck et al., 2000) was being strategically down-regulated as response times (RT) were slowed when outright stopping was contextually relevant (Langford et al., 2016a), and was further modulated by motivational context (Langford et al., 2016b). Given that these analyses exclusively looked at go-trials (i.e., no stop-stimuli were presented in these trials, hence not triggering outright inhibition), differences were arguably impelled by proactive processes, with fluctuations in RT and attention being preparatory in nature, and not outright inhibition.

Importantly, this earlier research technically entailed at its core a reverse inference, since we used variations in the N1 component across trials related to RT in order to interpret this finding as reflecting attention. In the current study we aimed to directly modulate preparatory processes in a fashion similar to Zandbelt et al. (2013) and Verbruggen and Logan (2009) in order to provide more direct evidence for an attentional involvement in proactive

inhibition. We used a lateralized stop-signal task with a probabilistic stop-expectation cue to manipulate preparatory attentional processes. Further, we focused on a clearly preparatory time-frequency signal that has been used extensively in cueing tasks to examine spatial attention. Cued attention to a location has been associated with increased alpha band activity over ipsilateral regions, and decreased alpha activity over contralateral regions prior to stimulus presentation (e.g., Vollebregt et al., 2015; Bengson et al., 2012). We analyzed alpha-band activity prior to the onset of a cued lateralized go-signal that could be expected to be followed by a centralized stop-signal at different occurrence rates.

4.2 Materials and Methods

4.2.1 Participants

19 participants (aged 18-26, 6 male) behavioral and EEG data were collected from the Ghent University subject pool. 3 participants were removed from all analyses because of data quality over the whole scalp, leading to substantial data loss.

4.2.2 Stimuli and Procedure

Participants were seated 80 cm from a computer screen. Experimental control and visual presentation were done using Neurobehavioral Systems Pre-

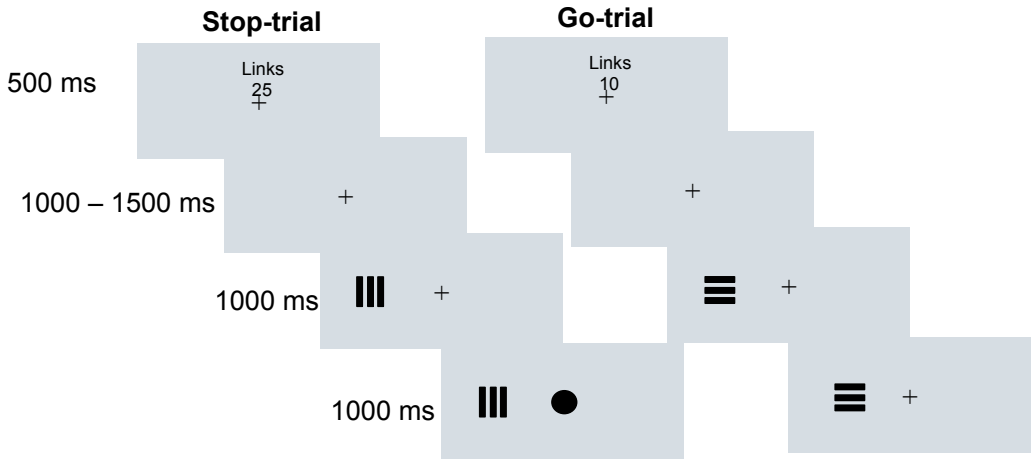

Figure 4.1: Paradigm. In this task participants were to respond as fast as possible to the orientation of the grating in a go-trial, while also stopping their response when a delayed stop-signal (black circle) appeared. The stop-signal was controlled using an adaptive procedure to ensure stopping rates of 50%. The cue indicated the side (Links vs. Rechts) of the upcoming go-signal, and also explicitly states the percentage of trials that are stops in the specific condition (10, 25, or 50%).

sentation software. The task reported in this manuscript had blocks that were interleaved with another similar task that presented go-stimuli centrally. Trial sequence proceeded with a visual cue consisting of a directional cue, either "Links" (left) or "Rechts" (right), below a level cue consisting of integers "10" (Lo), "25" (Mid), or "50" (Hi) above a central fixation cross. Visual cues were left on the screen for 400 ms. Directional cues and level cues both appeared randomly and in equal proportions. After the cue there was a randomly selected 1000-1500 ms interval between cue offset and the first target onset. The first target was a go-signal, these consisted of either vertical or horizontal gratings presented in the cued hemifield. Participants were to respond with their right and left index fingers using the 'z' key or the 'm' key depending on the orientation of the go-signal. Participants completed half of the blocks using one response mapping, and the second half using the opposite (which was counterbalanced across subjects, and practice trials were given after the switch). The level cue was used to represent the percentage of trials, in the upcoming trial, that consisted of not only the go-signal, but also a second stop-signal. The stop-signal was a gray circle presented at the location of the central fixation cross for 100 ms. The instructions given to the participants were to respond as fast as possible to the go-signal, but to not respond in trials which had a stop-signal. A subject-specific staircase procedure was adapted online to control the timing of the second signal to produce roughly equal stop-trial failures and successes. For the initial estimates participants first completed a short trial block and were told to ignore

the gray dots (before task instructions) and only to respond to the vertical and horizontal gratings. These response times were then averaged and the initial estimates of the staircase procedure were set to one standard deviation below the mean. There were separate staircases for all combinations of hemifield and response hand and all percentage levels of the level cue. If participants responded slower than 3 standard deviations above the initial estimates for any go-trial, they were given a 200 ms feedback after the trial indicating that they went too slow. There were 8 blocks of 45 trials, preceded by 1 practice block and task instructions. After each block participants took a self-timed break and received feedback regarding the response time and the percentage of correct stop-trials out of the total in the preceding block. Eye movements were monitored online using a video camera placed above the computer monitor and participants were discouraged from making horizontal eye movements.

4.2.3 Behavioral Analyses

Repeated measures ANOVA (rANOVA) were used to test for differences in response time using factors cue level, response side, and hemified of presentation. This same procedure was done on the proportion of correct stop-trial data to check if the adaptive-SSD procedure was successful at keeping all levels at roughly 50% stopping rates. To estimate the SSRT the integration approach was used at each of the cue levels. This approach defines the SSRT = (nth rank-ordered RT) - (mean stop-signal delay), with n equal to

the number of RTs in the go-trial RT distribution multiplied by the overall probability of responding given a stop signal. Simulations showed that under most circumstances the integration approach yields consistent and unbiased estimates of the SSRT (Verbruggen et al., 2013). The SSRT was then tested using a rANOVA with cue level as a factor. For multiple comparison correction given a significant omnibus test in any of the rANOVAs we used Bonferroni-Holmes corrected p-values in pairwise paired samples t-tests (denoted as p_{bh}).

Drift-diffusion models are a description of a stochastic binary choice process defined by three main parameters (Ratcliff, 1978). These parameters are the response threshold (a), the mean rate of approach to a threshold, known as drift rate (v), and processes that precede and succeed the actual decision process and give rise to a nondecision time (t0). Hierarchical Bayesian estimation was used to model the parameters using the Hierarchical Drift Diffusion Model (HDDM) software (Wiecki et al., 2013). Model fit was assessed using the deviance information criterion (Spiegelhalter et al., 2002; DIC, with smaller DIC indicating better fit). 5000 posterior samples were drawn for each model using Markov-Chain Monte Carlo methods. We used a burn-in of 1,000 and a thinning factor of 3. To account for individual differences in overall performance in this set of models we separately estimated v, a, and t0 as within-subject factors. These models take into account that a subject whose performance in, for example, the Lo condition is better than another subject, that also this might transfer to the Hi condition. Given that

only level played a role in the standard analysis of response times, we simplified the analyses and did not consider hemifield, nor response hand in the DDM. In these models we chose the Lo condition as the baseline and modeled the three parameters relative to the estimate of Lo. Each model was checked for convergence using the Gelman-Rubin diagnostic (Gelman and Rubin, 1996) and by looking at the autocorrelation of the sampled chains. Since we modeled the baseline condition as Lo, differences were probed by taking into account how many sampling points in the other conditions fell above or below zero. This resulted in a probability estimate that one posterior estimate is greater or less than the baseline Lo condition (see Kruschke, 2010 for an overview of Bayesian methodology). When needed, to further test between Hi and Mid, we simply looked at how many sampled points in one estimate were greater or less than the other.

4.2.4 EEG Acquisition and Analyses

EEG data were acquired at 512 Hz using a 64 channel Actichamp system with locations placed according to a standard international 10-20 system. The montage included two horizontal EOG (HEOG) channels and two mastoid channels. Data was imported into EEGLAB (Delorme and Makeig, 2004) using Cpz as the reference, which was then recovered algebraically. Bad electrodes were removed after importing and continuous data was prepared for Independent Component Analysis (ICA). Data were high-pass filtered at 1 Hz and split into ICA-appropriate go-locked epochs from -2000 ms to 1000

ms. First, epochs with data outside of $\pm$ 500 μV were rejected, and then followed by a further visual inspection for nonstationary epochs. Extended infomax ICA was then run on these epochs and subsequently the IC weight and sphere matrices were transferred back to the unfiltered continuous raw data. Artifactual components containing blink artifacts or other artifacts clearly distinguishable from brain-derived EEG signals were removed from the continuous data. Artifact-free continuous data were high-pass filtered at 0.5 Hz. Previously removed bad electrodes were interpolated, and segmented into 360 time-frequency appropriate epochs from -2500 ms to 2000 ms surrounding the target. Trial rejection was then done in four steps. (1) Trials with more than one response were removed. (2) Trials in which the participant responded faster than 100 ms, or received post-trial feedback indicating that they responded too slow were removed. (3) Trials containing bipolar HEOG (low-pass filtered at 20 Hz) $\pm$ 50 μV from the start of the cue to the start of the target, were removed. (4) EEG channels with data values exceeding $\pm$ 100 μV from the start of the cue to 500 ms after target onset were removed. Average percentage of trials after all rejection steps 93% of the total collected trials. The trial-averaged EOG channel was then visually inspected for fluctuations outside of $\pm$ 2 μV to detect individuals with consistent horizontal eye movements between cue and target over the whole experiment.

EEG time-frequency decomposition

The stimulus-locked epoched EEG times series were decomposed into their time-frequency representation by convolving them with a set of complex Morlet wavelets.

$$e^{i2\pi tf}e^{-t^2/2\sigma^2} \tag{4.1}$$

Where i is the complex operator, t is time, f is frequency from 2 to 50 Hz in 30 logarithmically spaced steps, and σ defines the width of each wavelet (x\2πf), where x increased logarithmically from 4 to 10. Following convolution data were reshaped back into individual trials. From the complex signal an estimate of condition specific power at each time point was calculated.

Pre-target α

To determine location and timing of pre-target alpha lateralization the selection procedure was independent of possible conditional differences. Scalp topographies collapsed over condition of raw power at 10 Hz were first plotted over the period preceding the go-signal for both left and right cues. Based upon the alpha activity in these topographies two clusters of five electrodes each were chosen for subsequent analysis. In the left posterior cluster electrodes O1, PO3, P5, P7, and PO7 were chosen, and in the right posterior cluster electrodes O2, PO4, P6, P8, and PO8 were chosen. These clusters were then averaged and time frequency power for each possible lateralization combination (left cue-left cluster, left cue-right cluster, right cue-right clus-

ter, and right cue-left cluster) were computed and subsequently collapsed to compute ipsilateral and contralateral time-frequency maps for each individual. These maps were used to examine pre-target power activity. The time range from -600 ms to -100 ms and frequency range from 8-13 Hz were chosen as the testing window because of the high pre-target alpha activity. For each subject and cue level (Hi, Mid, and Lo) separately a lateralization index was then computed.

$$\alpha LI = \frac{\alpha_{contralateral} - \alpha_{ipsilateral}}{\alpha_{ipsilateral} + \alpha_{contralateral}} \tag{4.2}$$

in which raw alpha power in each hemisphere is expressed relative to the total alpha power at both sites. To determine if alpha power lateralization was modulated by cue level a rANOVA was performed within the predefined time window using the αLI. We used the Bonferroni-Holmes p-value correction when necessary to test pairwise differences.

Early sensory evoked components

To examine early attention-related ERPs the data were first low-pass filtered at 30 Hz and then a -200 ms to 1000 ms period around the target stimuli was defined. Given that the SSD was rather long (i.e., possible stop-stimuli were presented relatively late during go-stimulus processing; see Results) we used all trials to calculate the ERP waveforms to characterize go-stimulus processing across the different experimental conditions.. Data were baseline

corrected from -200 ms to 0 ms before the target. Topographical maps were made in the typical time ranges that the P1 and N1 visual evoked components are seen for left and right presented targets, independent of condition. Based on these maps, it was decided that the post target ERPs included all of the same electrodes as in the alpha analyses. The time domain waveforms were then plotted averaged over individuals and conditions for ipsilateral and contralateral electrode sites. Based on these waveforms the P1 was defined in the range of 100 ms to 140 ms, and from 180 ms to 220 ms for the N1. The mean voltage of each participant was included in a rANOVA ran with condition (Lo, Mid, Hi), hemifield of presentation (left and right), as well as hemisphere (contralateral and ipsilateral) as within-subject factors. As with the behavioral analyses we used the Bonferroni-Holmes p-value correction to control for multiple comparisons.

4.3 Results

4.3.1 Behavioral Analyses

rANOVA with factors cue level, response side, and hemifield was used to test for differences between correct go-trial response times (see Table 4.1 for a summary of behavioral data). Only cue level was significant, $F(2,30) = 9.62$, $p < 0.001$ (all other F's < 1). Post-hoc pairwise paired t-tests were done over the three different cue levels. Hi was significantly slower than Mid ($t(15) = 2.62$, $p_{bh} = 0.029$ and slower than Lo ($t(15) = 3.55$, $p_{bh} =$

0.009), and furthermore Mid was slower than Lo (t(15) = 2.70, $p_{bh} = 0.037$), after correcting for multiple comparisons. The same analyses was run on the proportion of correct go-trials out of all go-trials for each individual, and there was no evidence that go-accuracy was different over and of the factors (all F's < 1).

The proportion of successful stops over cue level was 0.48 (0.04) for Hi, 0.45 (0.07) for Mid, and 0.48 (0.10) for Lo. Stopping accuracy over cue level, response side, and hemifield was assessed using a repeated measures ANOVA on the proportion data. This analyses suggested that the adaptive procedure held the proportion of stops roughly equal over the cue levels, F(2,30) = 2.48, p =0.10. There were no further interactions between the factors in the analysis. A rANOVA was used to test the differences between failed stop-trials and correct go-trials, as expected failed stop-trials (559.8 (94.4)) were faster than go-trials. Furthermore, a rANOVA was used to test if there were differences in the SSRT over cue level, and there were not F(2,30) = 0.18, p = 0.83. The mean SSRT was 196.8 (80.72).

The mean drift rate of the Lo condition was 2.51 (0.21). The group level estimate of Hi drift was much lower with no samples being greater than zero (p(Hi > Lo) = 0), with a mean -0.30 (0.08) below the Lo condition. The estimates of the Mid condition were marginally lower than the Lo group (p(Mid > Lo) = 0.12), with a mean estimate of -0.08(0.07) lower than the Lo condition. That is to say as the probability of a stop signal became greater the drift rate became lower. This same pattern held for the response threshold,

	go-RT±sem	SSRT±sem	SSD±sem	go-Accuracy±sem
Lo	563±23	200±33	332±21	0.92±0.06
Mid	573±21	204±16	353±30	0.92±0.07
Hi	596±22	187±12	406±29	0.91±0.07

	a±sd	v±sd	t_0±sd
Lo	1.50±0.09	2.51±0.21	0.32±0.04
Mid_{diff}	0.07±0.03	-0.08±0.07	0.005±0.002
Hi_{diff}	0.12±0.03	-0.30±0.08	0.005±0.003

Table 4.1: Top. Means and standard error of the means for behavioral results. Bottom. Drift-diffusion model parameters. Mid and Hi are reported as a difference from the baseline Lo condition.

except in the opposite direction. As stop-trials became more probable the response threshold was raised. The Lo condition had a response threshold of 1.50 (0.09). For the Hi threshold the estimate again had no samples containing zero (p(Hi < Lo) = 0), with a mean estimate of 0.12(0.03) above the Lo condition. The Mid condition was also greater than the Lo condition (p(Mid < Lo) = 0.003), with a mean of 0.07 (0.03) greater than the Lo condition. Nondecision time was estimated to be 0.32(0.04) in the Lo condition. Both the Mid (p(Mid < Lo) = 0.05, mean = 0.005(0.002)) and Hi (p(Hi < Lo) = 0.06, mean = 0.005(0.003)) were slightly higher than the Lo nondecision time, but the Hi and Mid factors were similar (p(Mid > Hi) = 0.52).

4.3.2 Pretarget alpha lateralization

To test for differences in pre go-stimuli alpha power we first plotted the condition-averaged and subject-averaged contralateral and ipsilateral power from -1500 ms to 500 ms surrounding the target, see Figure 5.2. Following this we computed the alpha lateralization index for each condition and individual separately and submitted this to a rANOVA. The rANOVA rejected the hypothesis that there were no differences in the alpha lateralization index, F(2,30) = 5.67, p = 0.008. Pairwise t-tests were then computed on all combinations of cue level using Bonferroni-Holms correction of p-values. This showed that Lo was greater than both Mid (t(15) = 2.74, $p_{bh} = 0.023$), and Lo was greater than Hi (t(15) = 2.95 $p_{bh} = 0.022$). However, there was no significant difference between Hi and Mid ($p_{bh} = 0.56$). We further tested if the Lo condition was different than zero using a one-sample t-test, which indicated that it was not, t(15) = 0.62, p = 0.55.

Early sensory evoked components

The P1 component showed no main effect of level in the tested interval, F(2,30)=1.66, p=0.21. However, there was a significant main effect of hemisphere and level, F(2,30) = 5.04, p = 0.013. Splitting this data to test for simple effects did not result in any significant effects (all t's < 1.43). The trend however was that for Lo and Mid the ipsilateral P1 was slightly higher than contralateral, and for Hi it was exactly the same. For the N1, there was a main effect of level, F(2,30) = 5.09, p = 0.012, but there were no other

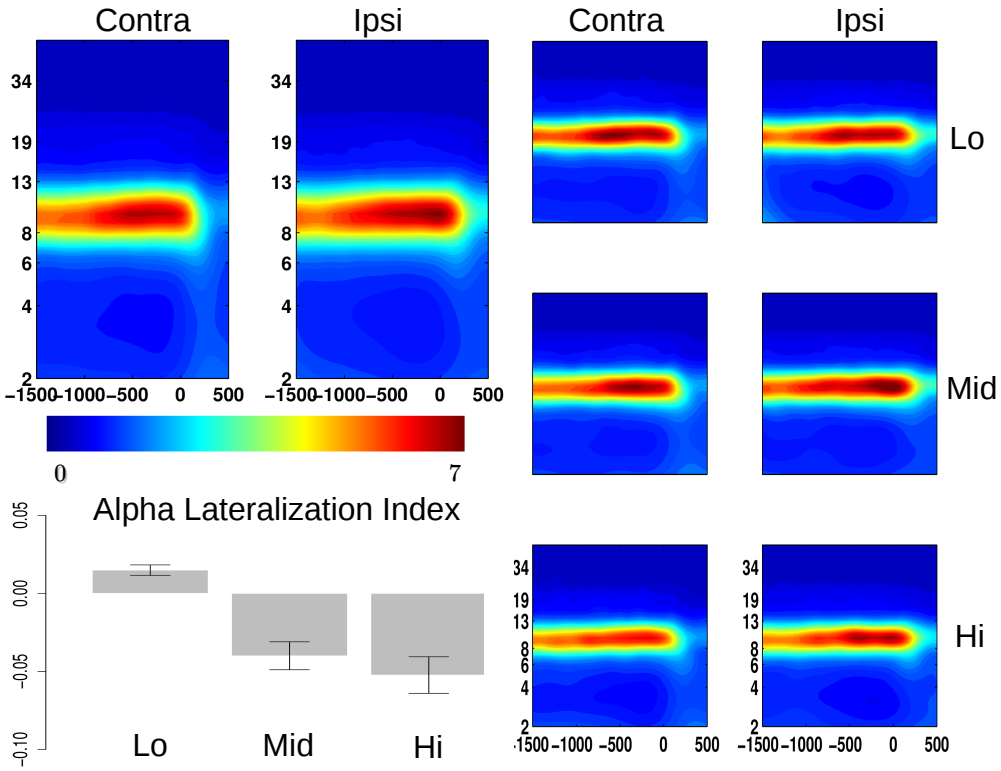

Figure 4.2: **Alpha lateralization.** Top left. Contralateral alpha power and ipsilateral alpha power plotted around the go-signal from -1500 ms to 500 ms for all conditions combined. Right. Contralateral and ipsilateral alpha power split by level. Bottom left. Alpha lateralization index split by level.

significant main effects. Pairwise t-tests were then used to compare across levels using Bonferroni-Holmes correction. This showed that Lo had higher amplitudes than Hi (t(15) = 4.02), $p_{bh} < 0.001$, and Mid also had higher amplitudes than Hi (t(15) = 2.48, $p_{bh} = 0.023$, however mid was not lower than Lo (t(15) = -1.94, $p_{bh} = 0.056$. There was also a significant interaction between hemisphere and level, F(2,30) = 3.77, p=0.34. Examining simple effects it was seen than in the Hi level ipsilateral N1 amplitudes were more negative than contralateral, t(15) = -2.42, p = 0.028, however this was not the case for the Lo or Mid level. The trend for the N1 is that the higher the probability that a stop signal occurs the more pronounced the amplitudes, overall.

4.4 Discussion

In this study we looked at pre-stimulus alpha lateralization as a function of a cued expectation for the current trial to have a second stop-stimulus. We found that as cued expectation for a stop-trial went up response times slowed. Drift-diffusion modeling of the go-trial data suggested that speed and accuracy came to fruition in different manners for the different expectation cues. Specifically, as the probability of a stop trial rises, drift rate falls, response threshold rises, and non-decision time becomes longer. Furthermore, there was a clear trend in the lateralization of alpha power over the different levels of expectation; as expectation for a stop-trial became higher the lat-

eralization index became lower. This was accompanied by a N1 effect over the different levels of expectation. As the probability of a stop-trial became higher, the N1 became more pronounced.

Over all conditions ipsilateral power is seen to be higher. However, this is clearly a mixture that is modulated by expectancy of a stop-signal. Specifically, when looking at the α lateralization index, which normalizes for total α power, we report that in the Lo condition contralateral power is greater than ipsilateral power, whereas in the Mid and Hi condition ipsilateral power is greater than contralateral power. This is interesting because in the Lo condition we see an almost "flipped" lateralization than what would be expected based on research on pre-stimulus alpha power (thought it is actually an absent lateralization). This was a pattern seen in Slagter et al. (2016), in which the side of target appearance was always fixed, instead of randomly appearing in different hemifields (e.g., Thut et al., 2006). Of course, in the current study, target hemifield was not fixed over the experiment, and varied randomly, yet we still observed this flipped lateralization when the probability of a temporally delayed central stop-stimulus (a "distractor" relative to the go-signal, but not to the task) was low (presented on only 10% of trials). However, when the probability of the central stop-stimulus was raised we saw a clear lateralization in the other direction; ipsilateral power was higher than contralateral. This pattern of results is not easily reconciled with the current literature on prestimulus α lateralization. However, a stop-signal task is temporally more complex (unless the SSD=0, similar to a go/no-go task)

than a simple Posner-like cueing task used in most studies, yet we do report a clear modulation by expectation.

The visual N1 component was also modulated by level of expectation. In both Langford et al. (2016a) and Langford et al. (2016b) we report on two completely centralized stop-signal tasks and the relationship between the same N1 component and response time. Both studies showed that as response time slowed the N1 component dampened. What we see here is quite different. While manipulating the expectancy in this experiment we see a bilateral N1 component that is more pronounced as expectancy goes up, and this is accompanied by systematically slowed responses. So, slower response times in this case are (at a categorical level) related to a more negative N1 component. The real difference in this case is that participants were possibly able to adapt a different strategy when the go-stimuli were lateralized, as opposed to centralized. This is quite possibly also the reason for the "flipped" alpha asymmetry described above.

Behavioral performance was in line with previous research and race-model predictions. The SSRT was not modulated by level of expectation and was furthermore similar to SSRT estimates in similar tasks (e.g., Verbruggen and Logan, 2009; Zandbelt et al., 2013). The probability of a successful stop-trial was held roughly equal across levels using an adaptive staircase procedure, and failed stop-trials were faster than go-trials. As expected RT on correct go-trials slowed as expectation of a stop-trial went up. There were no accuracy differences seen in a simple rANOVA analysis of go-trial proportions

between cue level. However, in a possibly more interesting modeling approach of both RT and accuracy we see that there are clear differences. Specifically, as stop-expectancy increases, we report an increase in response threshold, a decrease in drift rate, and a slight increase in nondecision time. In Langford et al. (2016a) we report the same types of drift-diffusion models only using an uncued stop-relevant (respond to the stop-signal) and stop-irrelevant (ignore the stop-signal) blocks. When participants were in the stop-relevant block they had an increase response threshold and a decrease in drift rate, as we also report here. In a very broad sense, this means that as participants expect a stop-signal they take on a more cautious response mode.

Proactive response inhibition has recently implicated the role of attentional processes as an explanatory factor (e.g., Elchlepp et al., 2016), though it is argued that differences seen in response inhibition are related solely to response inhibition. This contribution strengthens the former argument in multiple ways; (i) we have used a clear proactive component of allocation of spatial attention that occurs before any go- or stop-signal is presented. (ii) We show that this component is modulated directly by a cue that represents the probability of a stop-trial, arguably mimicking sequence effects seen in normal stop-signal tasks. (iii) We have replicated our previous findings using sequential sampling models, and have shown that while keeping the (arguably) same behavioral response mode patterns, participants adapt a qualitatively different strategy of information gathering based solely on the difference in stimuli presented (centralized vs. lateralized). This can be

seen in both the modulation of alpha lateralization effect and the N1 component. This pattern of results speaks towards a highly flexible proactive response inhibition process that recruits different systems to achieve the goal of inhibiting responses; and is clearly not purely inhibitory in nature.

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Chapter 5

Cued expectation modulates prestimulus alpha lateralization in a go/no-go task

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C. Nico Boehler

Abstract

The current electroencephalographic (EEG) study investigated adjustments of attention in a lateralized cued-expectancy go/no-go task. In this task participants were presented with an audiovisual cue that represents both the hemifield of the relevant stimuli (visual) and the odds of the stimuli requiring inhibitory behavior (auditory). Cued attention to a location has been associated with increased alpha-band activity over ipsilateral (irrelevant) regions, and decreased alpha activity over (relevant) contralateral regions prior to stimulus presentation. Prestimulus alpha power was modulated by expectancy in the delay period between the cue and the target stimuli. At high odds of no-go trials less alpha power was observed in the contralateral than in the ipsilateral hemisphere, whereas at low odds contralateral and ipsilateral power were similar. In addition, a larger bilateral attention-related N1 component in the high odds condition further indicated that sensory processing of the stimuli was heightened. This was accompanied by response time slowing indicating a more cautious response mode when the expectancy of an inhibition trial was high. Shifting to oscillatory activity that represents the recruitment of a dynamic action monitoring system, we observed that midfrontal theta activity in successful inhibition trials was increased when the odds of an inhibition trial was low. These results indicate a dynamic system of preparatory attention, action monitoring, and ultimately response inhibition, that is modulated by expectancy and jointly contributes to the ultimate behavioral output.

5.1 Introduction

Recent electroencephalography (EEG) and magnetoencephalography (MEG) research have shown that behavioral states that require top-down control (e.g. top-down attention, information retention) are accompanied by modulations in alpha-band oscillatory power. Cortical regions that are task-irrelevant typically see increases in alpha power, and regions that are task-relevant see decreases in alpha power. Observations supporting this have been made over multi-modality studies (e.g., Frey et al., 2014), in working memory tasks engaging either dorsal or ventral processing streams (Jokisch and Jensen, 2007), and in spatial attention tasks (e.g., Rihs et al., 2007). One theoretical account for these modulations posits that alpha fluctuations reflect the cortical flow of information, engaging in targeted inhibition of task-irrelevant (competing) networks and simultaneous disinhibition of task-relevant networks(Jensen and Mazaheri, 2010; Klimesch et al., 2007). This, in turn, would encourage maintenance and/or amplification of relevant sensory stimuli, while suppressing distracting stimuli.

Specific to visuospatial attention tasks, cued attention to a location has been repeatedly associated with increased alpha-band activity over ipsilateral regions, and decreased alpha activity over contralateral regions prior to stimulus presentation (e.g., Vollebregt et al., 2015; Frey et al., 2015). That these oscillations are specifically involved in the orienting of spatial attention is supported in part by studies showing a modulation in alpha-band power

by cue reliability (Gould et al., 2011; Dombrowe and Hilgetag, 2014), its sensitivity over a broad range of experimental manipulations (e.g., Haegens et al., 2011), and work showing that TMS pulses at alpha frequencies impaired visual detection contralateral to the occipital and parietal stimulation sites (Romei et al., 2008), consistent with a spatially specific suppressive role of alpha-band activity. However, there are disparate findings regarding the alpha-band modulation in relation to the (clear) presence or absence of competing distractors in the uncued hemifield (e.g., Rihs et al., 2007), which conflicts with the claim of targeted inhibition when a network is task-irrelevant (see Slagter et al., 2016, for a discussion).

Another related line of research has further shown that prior perceptual expectations about forthcoming content also affects sensory processing (Summerfield and Koechlin, 2008; Fischer et al., 2011; Fründ et al., 2014). For example, prior knowledge about the direction and velocity of moving targets enhances their detectability (Alink et al., 2010) and aids in object recognition (Kleinschmidt et al., 2002). Alpha oscillations have been also linked to expectation - they are suggested to entrain to predictable sequences of stimuli, amplifying stimulus evoked responses (Rohenkohl et al., 2012), and content-based predictions of letters lead to modulations in prestimulus alpha-band power(Mayer et al., 2016). Furthermore, alpha-band power has recently been suggested to be involved in balancing speed-accuracy tradeoffs in a time-pressure task (Limbach and Corballis, 2016), which might further be involved with stimulus prediction / expectation (as discussed below). The possibility

of reconciling these effects with accounts of alpha oscillations is an appealing notion because of the (pre-stimulus) temporality involved and suggested links with attentional biasing.

In the response inhibition literature multiple studies have implicated attentional processing of the stimuli involved in different tasks as an explanatory factor (Elchlepp et al., 2016; Verbruggen et al., 2014; Dimoska and Johnstone, 2008; Jahfari et al., 2015; Boehler et al., 2009; Zandbelt et al., 2013; Vink et al., 2015). Modeling suggests that a large portion of the time needed to implement response inhibition is taken up by processes related to the accumulation of appropriate sensory evidence for a given behavior (Boucher et al., 2007; Salinas and Stanford, 2013; Verbruggen et al., 2014). Relevant recent work has demonstrated a direct link between the selective attention / visual discrimination N1 ERP component and go-trial response time in a stop-signal task on a single-trial level, suggesting that attentional processing is strategically regulated (Langford et al., 2016a), and further modulated by contextual factors (Langford et al., 2016b).

In such tasks the variation seen in response time (and hence the single-trial N1 ERP) seems to partially be driven by a continual tracking of trial history (Ide et al., 2013) informing subjective expectations about likely trial contents, thereby predicting upcoming stimuli. This underlying hidden Markov model of prior expectations were furthermore found to be highly predictive of response times in inhibition tasks. In this sense, a prior-trial judgment about forthcoming stimulus-identities (and hence the appropriate behavior)

is taken into consideration, affecting response time, which in turn correlated with attentional sensory processing in our earlier work (Langford et al., 2016a). Conceiving of perceptual decisions as processes that stochastically accumulate sensory information towards a decision bound, this work found context-dependent, arguably expectation-based, biasing effects on the accumulation processes that determine decision times and outcomes (i.e. involved in negotiating speed-accuracy tradeoffs).

Tying these lines of attentional alpha-power modulations and response inhibition together, past research has investigated pre-stimulus alpha lateralization in a go/no-go task by comparing successes and failures in signal-inhibit trials (Bengson et al., 2012). This work evidenced a greater lateralization of alpha power in successful inhibition trials. Here, we extend this work to different probability levels of cued no-go expectation. This also ties in with our earlier work suggesting that attention is actively modulated in the service of response inhibition, at least in a stop-signal task (Langford et al., 2016a; Langford et al., 2016b). In addition to alpha-power asymmetry, we were also interested in target processing, focusing on early attentional components (P1 and N1), as well as midfrontal oscillatory activity in the theta range thatis a sensitive marker for the recruitment of an action monitoring system. (e.g. Cohen, 2016; Yamanaka and Yamamoto, 2010).

5.2 Materials and Methods

5.2.1 Participants

22 participants (aged 18-34, 5 male). Two of them were removed from all analyses, one because of data quality over the whole scalp, and another because of data quality in the main analysis electrodes and because of a consistent HEOG movements.

5.2.2 Stimuli and Procedure

Participants were seated 80 cm from a computer screen. Experimental control and visual presentation were done using PsychoPy (Peirce, 2007). The trial sequence proceeded with an audiovisual cue consisting of either a low-frequency (LoF) 500 Hz or high-frequency (HiF) 1000 Hz tone generated from a bilateral speaker arrangement. These 100 ms auditory cues were presented simultaneously with a directional (left of right) visual arrow cue that lasted 250 ms. LoF and HiF tones and left and right arrow cues were all randomized across trials but in equal proportions. After the audiovisual cue there was a randomly selected 1700-2000 ms interval between cue offset and target onset. Targets were the integers 1 through 5 and subtended 2° of visual angle. They were presented 11° horizontal to, and 3° vertically below screen center for 100 ms. Subjects were instructed to respond to the numbers 1, 2, 4, and 5 (go), but not respond to the number 3 (no-go). The right index finger was used on right cue trials, and the right middle finger for left cued trials. This

procedure basically cued which button to use for go trials, and hence still represents a basic detection task (i.e., the target stimulus does not need to be discriminated beyond whether it is a go or a no-go stimulus); this was implemented in order to ensure that participants were attending the cued side (see (Bengson et al., 2012), where the lack of such information was considered a possible problem). In the LoF cued trials the no-go target would appear randomly with a frequency of 1 out of 10, and in the HiF cued trials the no-go target would appear randomly with a frequency of 1 out of 2. The target was followed by a 1000 ms response period followed by a randomly chosen inter-trial interval between 450 and 750 ms. There were 9 blocks of 80 trials, preceded by 1 practice block and task instructions. After each block participants took a self-timed break and received feedback regarding the response time and the percentage of correct rejections in the preceding block.

5.2.3 EEG Acquisition and Analyses

EEG data were acquired at 512 Hz using a 64 channel Biosemi ActiveTwo system (Biosemi, Amsterdam, Netherlands) with standard electrode locations according to the international 10-20 system. External electrodes were attached both to the left and right mastoids, at the outer canthi of both eyes (HEOG), and directly above and below the right eye (VEOG). Bad electrodes were removed after recording and continuous data was prepared for Independent Component Analysis. Data were high-pass filtered at 1 Hz

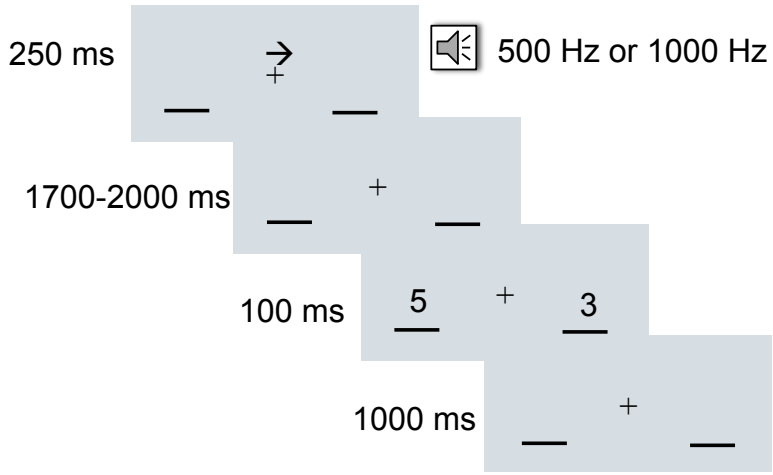

Figure 5.1: Trials stared with an audiovisual cue. Participants were to respond when the digits on the cued side (arrow) were 1,2,4, or 5, but to not respond when the digit was a 3. The auditory tone either cued a low odds of a 3, or a high odds of a 3.

(see Winkler et al. (2015) for a discussion) and split into ICA-appropriate target-locked epochs from -2500 ms to 1000 ms. First, epochs with data outside of ± 500 muV were rejected, and then a probability rejection was used to reject improbable data (kurtosis rejection function in EEGLAB (Delorme and Makeig, 2004), with single-channel limits set at 6 standard deviations and all-channel limits set at 3 standard deviations), followed by a further visual inspection for nonstationary epochs. Extended infomax ICA was then run on these epochs and subsequently the IC weight and sphere matrices were transferred back to the unfiltered continuous raw data. Artifactual components containing blink artifacts or other artifacts clearly distinguishable from brain-derived EEG signals were then removed from the continuous data. Artifact-free continuous data were then high-pass filtered at 0.5 Hz

and average referenced. Previously removed bad electrodes were interpolated, and segmented into 720 time-frequency appropriate epochs from -3000 ms to 2000 ms surrounding the target. Trial rejection was then done in four steps. (1) Trials where the incorrect response finger was used and no-response go-trials were removed. (2) Trials with response times faster than 100 ms and slower than 800 ms were removed. (3) Trials containing bipolar HEOG (low-pass filtered at 20 Hz) ± 50 muV from the start of the cue to the start of the target, were removed. (4) EEG channels with data values exceeding ± 100 muV from the start of the cue to 500 ms after target onset were removed. Average trial count after all rejection steps was 654 ± 71, or 91% of the total collected trials. The trial-averaged EOG channel was then visually inspected for fluctuations outside of ± 2 muV to detect individuals with consistent horizontal eye movements between cue and target over the whole experiment; based on this criteria one participant was excluded from all further analyses, as already noted in the Participants section. In a final step a surface Laplacian transform was then applied to the scalp channels (see, Perrin et al., 1989; Cohen, 2014b, for details and motivation).

EEG time-frequency decomposition

The stimulus-locked epoched EEG times series were decomposed into their time-frequency representation by convolving them with a set of complex Morlet wavelets.

$$e^{i2\pi tf}e^{-t^2/2\sigma^2} \tag{5.1}$$

Where i is the complex operator, t is time, f is frequency from 2 to 50 Hz in 40 logarithmically spaced steps, and σ defines the width of each wavelet (x\2πf), where x increased logarithmically from 4 to 10. Following convolution data were reshaped back into individual trials. From the complex signal an estimate of condition specific power at each time point was calculated.

Pre-target α

To determine location and timing of pre-target alpha lateralization, we used a selection procedure that was orthogonal to possible conditional differences. Scalp topographies collapsed over condition of raw power in the alpha band (8-13 Hz) were first plotted over the period preceding the target in 100 ms steps for both left and right cues. Based upon the alpha activity in these topographies two clusters of three electrodes each were chosen for subsequent analysis. In the left posterior cluster electrodes O1, PO3, and PO7 were chosen, and in the right posterior cluster electrodes O2, PO4, and PO8 were chosen. These clusters were then averaged and time-frequency power for each possible lateralization combination (left cue-left cluster, left cue-right cluster, right cue-right cluster, and right cue-left cluster) were computed and subsequently collapsed to compute ipsilateral and contralateral time-frequency maps for each individual. The ipsilateral maps were then subtracted from the contralateral maps to examine pre-target alpha-power lateralization. The time range from -1100 ms to -100 ms was chosen as the testing window because of the high pre-target alpha activity throughout this period. For each

subject and cue level (HiF and LoF) separately a lateralization index was then computed.

$$\alpha LI = \frac{\alpha_{contralateral} - \alpha_{ipsilateral}}{\alpha_{ipsilateral} + \alpha_{contralateral}} \tag{5.2}$$

in which raw alpha power in each hemisphere is expressed relative to the total alpha power at both sites. To determine if alpha power lateralization was modulated by cue level, a paired samples t-test was performed within the predefined time window using the αLI. Subsequently, in the HiF condition the no-go data were split by behavioral outcome (failed and successful no-go trials) and tested using the αLI in the same time and frequency window.

For midfrontal theta power we first decibel normalized the data using the period from -200 ms to 0 ms before the cue. Next, we plotted theta power collapsed across condition from -500 to 1000 ms surrounding the go-signal and further plotted a scalp topography of theta power averaged around the time of the peak alpha power (see Results). From these plots it was determined that the electrode FCz would be appropriate for our analyses, as well as the time range from 350 to 500 ms following the go-signal. Data was then split by cue level and behavioral type. Given that in the Lo condition there were, for some people, very few no-go-trials that were also failures, we did not consider to test failures. We then tested go-trials against successful no-go-trials, and further go and no-go trials over cue level. In all cases we used simple paired samples t-tests after equalizing trial numbers per individual by the lowest

observed number between the two variables tested.

Early stimulus-evoked components

To examine early attention-related ERPs the data were first low-pass filtered at 30 Hz and then a -200 ms to 1000 ms period around the target stimuli was defined. Data were then baseline corrected from -200 ms to 0 ms before the target. Topographical maps were made in the normal time ranges that the P1 and N1 visual evoked components are typically seen for left and right presented targets, independent of condition. Based on these maps, it was decided that the post target ERPs were centered around the same electrodes used in the post-target alpha analyses, but were not as spatially confined (see results). To account for this four more electrodes were considered in the ERP analyses, in addition to the ones also included in the alpha analysis. Specifically, the left cluster included O1, PO3, PO7, P5, and P7, while the right cluster included O2, PO4, PO8, P6, and P8. The time domain waveforms were then plotted averaged over individuals and conditions for ipsilateral and contralateral electrode sites. Based on these waveforms the P1 was defined in the range of 100 ms to 140 ms, and from 180 ms to 220 ms for the N1. The mean voltage of each participant was included in a repeated-measures ANOVA ran with condition (HiF and LoF), hemifield of presentation (left and right), as well as hemisphere (contralateral and ipsilateral) as within-subject factors.

5.3 Results

5.3.1 Behavior

Repeated-measures ANOVA (rANOVA) was used to test for differences in response time in the go-trials using presentation side and cue level as factors. As expected, there was a main effect of cue level with LoF cues producing significantly faster responses, $F(1,19) = 13.24$, $p=0.002$. There were no significant differences for hemifield $F(1,19) = 2.04$, $p=0.17$; and no significant interaction between cue level and hemifield, $F(1,19) = 0.49$, $p = 0.49$. Accuracy in the no-go-trials was assessed using cue level and hemifield as factors in a rANOVA. Similar to the response time data, there was a significant main effect of cue level; no-go-trials following HiF cues were inhibited in greater proportions, $F(1,19) = 12.77$, $p = 0.002$ than following LoF cues. Neither hemifield ($F(1,19) = 0.56$, $p = 0.46$) nor the interaction between hemifield and cue level ($F(1,19 = 0.51$, $p = 0.48$) were significantly different. A rANOVA showed that the proportion of correct go-responses (i.e. left-cue responses made with index finger or right-cue responses made with middle finger) did not vary over cue level ($F(1,19) = 0.001$, $p = 0.97$), and further there were absolutely no incorrect responses made on no-go trials that elicited a response (no-go-fail). A 1-sample t-test was used to test if the proportion of correct go-responses was different from 1, which did not reach statistical significance ($t(19) = -1.72$, $p = 0.1$). The analyses of behavioral data show evidence that in the HiF trials participants gave slower responses in go-trials,

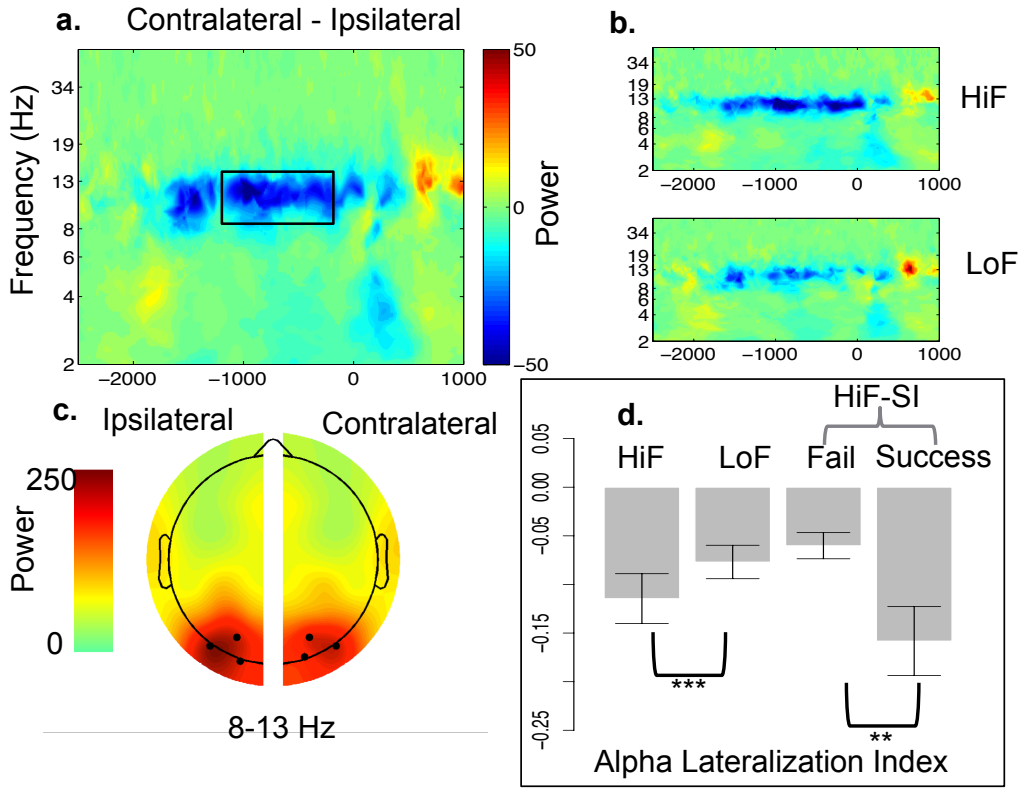

Figure 5.2: Top left: median (over subjects) difference between contralateral and ipsilateral power. Top right: Median HiF difference. Middle right: Median LoF difference. Bottom left: topography of raw alpha power split between contralateral and ipsilateral averaged over -1100 ms and -100 ms before target. Bottom right: Mean αLI with standard error of the mean.

and were more accurate in not responding in no-go-trials. Given the pattern of correct responses in go and no-go-fail trials, missed cues (resulting in attentional orienting to the wrong hemifield) are highly unlikely to account for any differences seen in either the behavioral or EEG data.

5.3.2 Pre-target α lateralization

To inspect alpha lateralization preceding the targets, raw power in the ipsilateral hemisphere (e.g., channel PO8 when the cue pointed right) and in the contralateral hemisphere (e.g., channel PO8 when the cue pointed left) were first plotted as scalp topographies (seen averaged over laterality in Figure 5.2b, with analysis electrodes marked) averaged over conditions. Subsequently, raw power data for each participant were averaged over the marked electrodes and ipsilateral power spectra were then subtracted from the contralateral power and the median (over participants) was plotted, see Figure 5.2a. For each participant and cue level the alpha lateralization index from 8-13 Hz and -1100 ms to -100 ms before target presentation for the mean of the three channels marked in Figure 5.2b was then calculated. A paired samples t-test was then used to test for differences between the HiF and LoF cue levels. This test showed a strong effect of cue level on the αLI, t(19) = -4.29, p = 0.0004 (d = -0.96, 95% CI -1.66 -0.26). As can be seen in Figure 5.2c in the HiF condition there is a sustained negative contralateral-minus-ipsilateral power difference preceding the target that is nearly absent in the LoF condition.

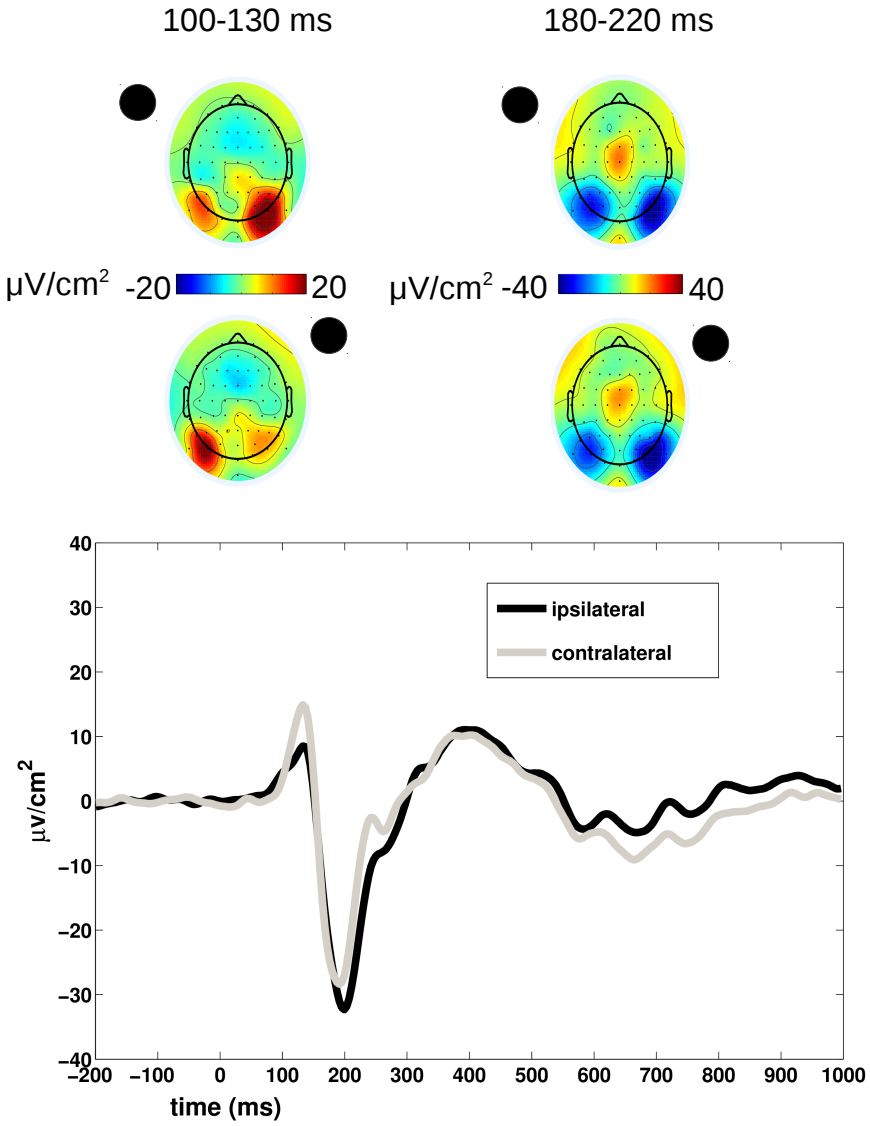

Figure 5.3: Top: mean topographies in the tested areas for the P1 and the N1. Black dots indicate the hemifield that the stimuli were presented in. Bottom: mean ERP waveform for both contralateral and ipsilateral hemispheres for the electrodes included in the analyses.

To test for differences in the αLI in relation to inhibitory behavior, we compared no-go-success and no-go-fail trials. Due to the low trial count for LoF no-go-fail trials for some participants, this analysis was confined to the HiF condition, in which we furthermore matched the number of the conditions by randomly selecting trials to match the trial counts to the condition with fewer trials. Following a similar logic as before, a paired samples t-test showed that the αLI was significantly more negative for no-go-success trials compared to no-go-fail trials, t(19) = -2.72, p = 0.013 (d = -0.61, 95% CI -1.28 0.06), see Figure 5.2d.

Early evoked components

To examine early evoked stimulus processing we submitted the subject-specific P1 and N1 components to a rANOVA using hemisphere (contralateral and ipsilateral), hemifield (left and right), and condition level (HiF and LoF) as within-subject factors. We observed an N1 effect that showed that the N1 was more pronounced in the HiF condition, F(1,19) = 4.72, p = 0.042. There was also a main effect of hemisphere, with the N1 being more negative in the ipsilateral hemisphere, F(1,19) = 8.49, p = 0.009. However, there was no interaction between any the factors. There was no main effect of condition for the P1, F(1,19) = 2.76, p = 0.11. The P1 amplitudes were higher in the contralateral hemisphere, F(1,19) = 18.90, $p < 0.001$. There were no interaction effects seen in the analyses. See Figure 5.3 for plots of both the P1 and the N1.

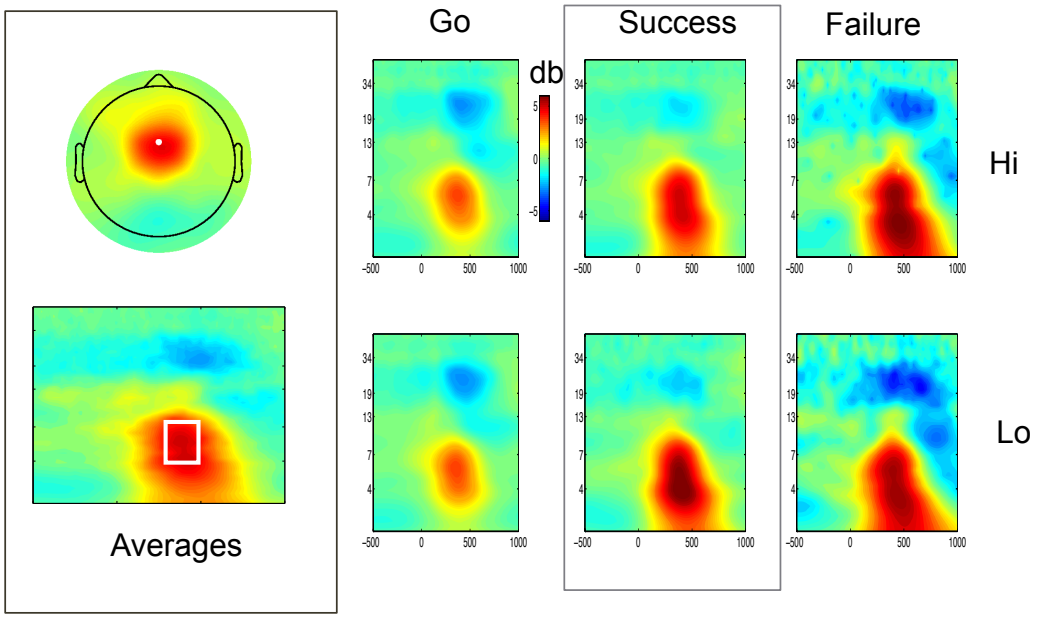

Figure 5.4: Top left: Total decibel normalized theta power averaged from 350-500 ms over all conditions. Bottom left: Theta plotted averaged over all conditions. Tested area is marked with a white square. Right: Theta power split by condition and trial behavior.

5.3.3 Post-target θ

Theta power was examined after the target from 350-500 ms between 4-8 Hz. All tests equalized trial numbers across conditions by random selection. No differences were found between go-trials over cue level, $t(19) = -1.12, p = 0.27$. Collapsing over cue level a large effect was found between no-go-success and go-trials, $t(19) = -8.1, p < 0.001$(d = -1.79, 95% CI -2.57 -1.012), with higher theta band activity for no-go-success trials. Further, a difference was found between no-go-successes over cue level, $t(19) = -3.19, p = 0.005$ (d = -0.71, 95% CI -1.39, -0.04) meaning theta power was higher during a no-go-success trial in the low-expectation condition relative to the high expectation. No differences were evident between no-go-success and no-go-fail trials $t(19) = 0.71, p = 0.32$. no-go-fail trials were not tested across expectation level because of low trials numbers in the LoF cell for some participants.

5.4 Discussion

The main focus of the current EEG study was the lateralization of posterior alpha-band oscillations preceding target stimuli in a go/no-go task, whose identities were stochastically predicted by an auditory expectation cue. This manipulation both slowed response times and raised the proportion of correct inhibition trials in high expectation trials. We found a significant increase in the lateralization of alpha power when the cue predicted that the forthcoming

stimulus has a high likelihood to require response inhibition (the number '3'). In regards to early sensory processing, we observed a more pronounced N1 component in the high expectation trials and no differences in the P1. Additionally, we found increases in midfrontal theta power in signal-inhibittrials vs. signalrespond trials, and further observed an increase in successful signal-inhibit trials in the low expectation trials vs. hi expectation trials.

Alpha lateralization effects are often reported based on post-hoc analyses of behaviorally identical trials (e.g. hits and misses). This implies that trial-by-trial performance may be determined by a (possibly) random oscillatory state (i.e. not really a top-down driven state, but a necessary one for a given behavioral outcome). In the current study the spatial cue was always predictive of the relevant hemifield, and the opposite hemifield was always irrelevant to the response. The auditory tones, however, where only stochastically predictive at different levels of the stimuli. In this manner, we manipulated expectancy and were able to use all trials over the different levels, as well as look at fail and success trials. We observed that expectancy clearly modulates posterior alpha oscillations in a manner that is consistent with a spatially specific suppressive role of alpha-band activity, and further lends support to the idea that these alpha modulations are driven by content-based expectations. Such oscillations for example, entrain to predictable sequences of stimuli, amplifying stimulus-evoked responses (Rohenkohl et al., 2012), and content-based predictions of stimuli have been seen to modulate prestimulus alpha-band power (Mayer et al., 2016). In the current manuscript we

show that this can be driven by a more cognitive-based task that is thought to depend on the relationship between attentional components and the negotiation of speed-accuracy tradeoffs (see, Limbach and Corballis, 2016, for a similar discussion in a response pressure task). This argument is supported by recent research in inhibitory tasks, which we will now detail.

Shifting to the relationship between attention and response inhibition, recent research in such inhibitory tasks has shown that response time scales on a single-trial level with the visual evoked N1 component in tasks where inhibiting a response might be required, but not when a simple decision is made, or when attention was generally triggered strongly by a reward context (Langford et al., 2016a; Langford et al., 2016b). Given the positive correlative relationship between response time and the probability of inhibiting a response, this N1 effect is arguably driven by predictions about stimuli in the upcoming trial (see, Ide et al., 2013, for a further discussion) - which creates a speed-accuracy tradeoff. In the present work, there was a more prominent bilateral N1 ERP in trials with a high expectation cue, however no evidence for a difference in the visual P1 was found. While this N1 effect might seem to be in conflict with our past research - in the sense that the high expectation condition had slower responses - the categorical difference across probability levels might reflect more cognitive operations, more generally linked to expectation and attention, in a way that is possibly not directly mapped onto response speed in an instrumental way for response inhibition. Yet, at the very least, attentional orienting and processing is responsive to

our task manipulations, signifying a role of attention at least in the global sense.

A possibility suggested in a similar study was that these alpha effects could possibly be due to the misperception of a cue (Bengson et al., 2012) Given that the response mapping in the present study was consistent with the cued side, and the amount of incorrect (left respond right-right respond left) responses in inhibition-fail trials was zero, it is highly unlikely that any behavioral or electrophysiological result could be attributed to a misperception of the cue. While there was a significant increase in lateralization in the high predictive condition, the cue that was predictive of a low signal-inhibit probability showed nearly a complete absence of lateralized alpha activity. However, it should be noted that in Bengson et al. (2012) the directional cue was 150 ms shorter than the currently reported cue, so we can only rule out that the misperception of the cue did not play a role in the current study. We further observed a higher lateralization index in successful versus unsuccessful inhibition trials, which is in direct alignment with this earlier work (Bengson et al., 2012). Given that this past research only analyzed alpha power as a function of behavioral outcome, related to our previous point, it was an open question if the observed differences were specifically top-down control of attention, or possibly random fluctuations. Given the differences seen between the expectation levels this is not likely the case for the current study.

Midfrontal theta has been shown (Cohen and Donner, 2013) to be a

robust power modulation that is non-phase-locked and more closely linked to conflict and behavior than it's phase-locked counterpart (i.e., the ERP waveform).Theta-band oscillations in medial prefrontal cortex are modulated during action monitoring tasks, specifically in tasks that elicit conflict between competing responses (e.g., Cohen, 2016; Cohen and Donner, 2013; Huster et al., 2013; Oehrn et al., 2014). Recent research has highlighted that midfrontal theta is a signature of the engagement of a dynamic system that responds flexibly to errors and the possibility of errors (Cavanagh et al., 2014; Cohen, 2014a; Cohen, 2016). Here, we look at the modulation of midfrontal theta as a function of expectation and behavior, reporting increases in power in correct signal-inhibit trials when the trial was unexpected to be an inhibition trial. We found a direct link between expectation of an action before stimuli appear and midfrontal theta. In this manner the expectation cue creates situations that are more consistent with proactive control (high-expectancy) and reactive control (low-expectancy). Previous studies have shown that frontal EEG responses are stronger during trials that require reactive control (Bartholow et al., 2005; Folstein and Van Petten, 2008), and the current results are in line with this finding. In both expectation levels a signal-respond trial is expected as it never drops below a fifty-percent frequency, and this is probably the reason that we only see the theta modulations over expectation in signal-inhibit trials, and not signal-respond (something which could be explored in the future).

In the current manuscript we have ruled out the idea that differences seen

in earlier work (Bengson et al., 2012) in the lateralization of pre-target alpha power linked to subsequent behavior were due to random fluctuations and/or misallocation of attention to the uncued side. Instead it was shown that they are driven by a clearly top-down process that was related to expectation of trial content. This increased lateralized alpha effect when expectation was high was also followed by an increase in sensory processing of the stimuli as evidenced by N1 amplitudes. Future work should be aimed at linking these two components together in similar tasks. Furthermore, we have demonstrated a link between a dynamic action monitoring system and expectation as shown in increases in midfrontal theta power

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Chapter 6

Discussion

As presented in this thesis, response inhibition is a key component of successful cognitive control due to the fact that a changing environment often requires inhibiting an automatic or otherwise prepotent response on short notice. While the research interest has traditionally been focused on *reactive* processes, which refers to response inhibition that is triggered by an explicit sensory stop-stimulus, interest in *proactive* response inhibition has recently seen a steep rise. Proactive inhibition describes the tendency of slowing down responses when outright inhibition *might* be required (Aron, 2011) usually resulting in a slower response as opposed to reactive processes yielding outright inhibition. Yet, research thus far has mostly focused on a central motoric inhibition mechanism without acknowledging the possible role of afferent processes like the attentive processing of task-relevant stimuli. Attention plays a dominant role in other cognitive control paradigms (Braver, 2012) and it is not unlikely that it also plays a part in response inhibition. Indeed, recent research has highlighted a possible role of earlier attentional processes in response inhibition (e.g. Bari and Robbins, 2013; Logan and Cowan, 2014; Verbruggen et al., 2014; Huster et al., 2014; Elchlepp et al., 2016; Bekker et al., 2005; Boehler et al., 2009; Kenemans, 2015). Further detailing how these attentional processes relate to proactive inhibitory processes is the focal point of this thesis.

In this Discussion I will first give a brief tour of the more important methodological background, emphasizing some innovative aspects over previous work. Following this I will focus on the specific findings of the re-

search presented in thesis, and their relationship to past research in the field. Though I will already talk about the results of the sequential sampling models in this overview, I will expand upon the concept in a separate section, in which I detail the commonalities across the studies. These models play an important role in the first three studies; most notably as they don't just describe response time and accuracy as summary measures (and random noise), but as a specific process that unfolds over a trial (and experiment). Following this, I will further discuss the relationship between the presented research and cognitive dysfunctions. Inhibitory mechanisms are heavily implicated in cognitive and neurological dysfunction (Chambers et al., 2009), and while the research presented in the thesis is very basic and fundamental, it likely speaks towards these disorders. Lastly, I will discuss the future research outlook in proactive control and response inhibition.

6.1 Methodological background

6.1.1 Multilevel single-trial EEG analyses

In both **Chapter 2** and **Chapter 3** we used a novel multilevel single-trial electroencephalography (EEG) approach to investigate attentional components involved in response inhibition. Given it plays a central role in the thesis I will briefly touch upon the general logic and purpose behind the method. Single-trial analyses refer to methods that model some type of variance within subjects, and they are not traditionally used in neuroimaging

research. Traditional techniques have focused on demonstrating differences between means calculated over experimental conditions. By studying variability across trials, single-trial analyses can allow us a different view of cortical processes in EEG research. This modeling approach, for example, can be used to make a systematic mapping between a stimulus quality and brain activity (e.g, Rousselet and Pernet, 2011). In this thesis we specifically focus on modeling EEG activity and behavioral (response time) variability. As discussed in Chapter 1 and throughout the thesis response time and response inhibition have a a relationship that is grounded in both theoretical and empirical work. These single-trial models enabled us to look at covariation between EEG measurements and response times, which allows us to further strengthen arguments related to attention and response inhibition.

6.1.2 Visual N1 ERP

Though I will discuss the findings of the single trial approach further below, here I will present the basics of the inferoposterior N1 component that was the focus of both chapters (and indeed also presented in **Chapter 4** and **Chapter 5**). The visual N1 is a negative-going component that is generally seen in the time range of 150-200 ms (though this time range does vary) after any visual stimulus, and is seen most clearly in posterior occipito-temporal areas. A corpus of research has focused on factors that modulate the amplitude of the visual N1. These studies provide evidence suggesting that, while the visual N1 is a sensory component evoked by any visual stimulus,

it also reflects a benefit of correctly allocating attentional resources. Amplitude effects on the N1 are absent during simple reaction time tasks, which only require subjects to make a rapid response to stimuli (Luck et al., 2000; Mangun and Hillyard, 1991) - which is a finding that suggests that the N1 is linked to visual discrimination processes (see also, Hopf et al., 2002).

6.1.3 Preparatory α-band power

As will be discussed below, in **Chapter 4** and **Chapter 5** we switch focus from the visual N1 to a time-frequency signal that is thought to be involved in preparatory attention. Past EEG research has provided evidence of a preparatory signal in alpha-band oscillatory power in states that require top-down control. Regions that are task-irrelevant see increases in alpha power, and regions that are task-relevant see decreases in alpha power. Evidence supporting this has been made over multi-modality studies (e.g., Frey et al., 2014), in working memory tasks engaging either dorsal or ventral processing streams (Jokisch and Jensen, 2007), and in spatial attention tasks (e.g., Rihs et al., 2007). Specific to visuospatial attention tasks, cued attention to a location has been repeatedly associated with increased alpha-band activity over ipsilateral regions, and decreased alpha activity over contralateral regions prior to stimulus presentation (e.g., Vollebregt et al., 2015; Frey et al., 2015). That these oscillations are specifically involved in the orienting of spatial attention is supported in part by studies showing a modulation in alpha-band power by cue reliability (Gould et al., 2011; Dombrowe and Hilge-

tag, 2014), its sensitivity over a broad range of experimental manipulations (e.g., Haegens et al., 2011), and work showing that transcranial magnetic stimulation (TMS) pulses at alpha frequencies impair visual detection contralateral to the occipital and parietal stimulation sites (Romei et al., 2008), consistent with a spatially specific suppressive role of alpha-band activity.

There has also been further work aimed at detailing the links between the prefrontal cortex (PFC) and the α oscillations that we are interested in examining. A series of studies, starting with Capotosto et al. (2009), recorded simultaneously EEG and TMS in simple spatial attention tasks and found that stimulation of core regions of the dorsal attention network (DAN, specifically the frontal eye fields, intraparietal sulci) modify prestimulus alpha power in posterior cortices, suggesting a role for prefrontal and parietal regions of the DAN in alpha modulations. In another line of inquiry Marshall et al. (2015) diffusion imaging data that quantifies the white matter tracts (medial superior longitudinal fasciculus, SLF) that connect frontal cortices to parietal areas further supports the notion of frontal control over posterior alpha oscillations. To note, the medial SLF projects to areas overlapping with the dorsal attention network and other control-related prefrontal regions (e.g. dorsolateral PFC-dlPFCm, which is central to the next section). They demonstrated a relationship between hemispheric asymmetry of the SLF in individuals and their ability to exert top-down control of alpha oscillations. Specifically, subjects with a stronger SLF volume in the right compared to the left hemisphere had a better ability to modulate right compared to left

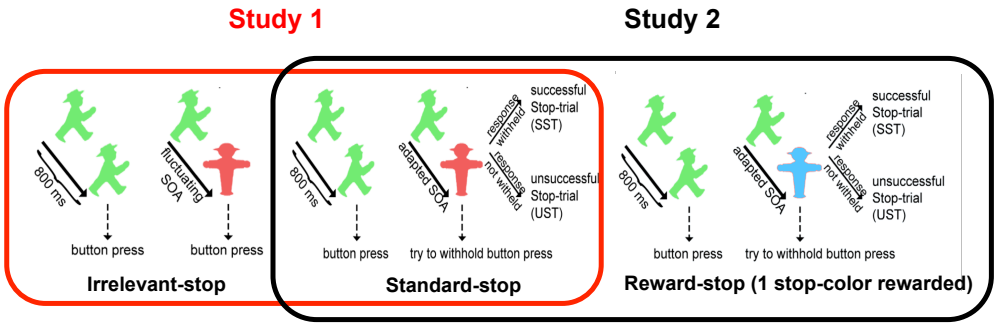

Figure 6.1: Commonalities between the experimental paradigm in the first two chapters. In these two studies a standard visual stop-signal task was used, along with two different contextual changes. In Study 1 an irrelevant-stop task was used, in which participants were given the same stimuli in the standard-stop task, but they were to ignore the stop-stimuli and to respond only to go-stimuli. In Study 2 there was a defined color on the stop-stimuli (blue in the figure) in some blocks that rewarded successful inhibition of a response.

hemisphere oscillations. All of this taken together suggests that such alpha-band oscillations are driven by prefrontal areas that are generally thought to be involved in cognitive control, and are arguably a marker of proactive allocation of spatial attention.

6.2 Overview of empirical findings

In **Chapter 2** we specifically investigated single-trial ERPs and the relationship with response time in one standard form of a stop-signal task (relevant-stop context), and a sensory baseline in which participants were to ignore the stop-signals (irrelevant-stop context) with a focus on the visual N1 component, see Figure 6.1 for an overview. We made a comparison be-

tween go-trials from these different trial blocks. Given the focus on go-trials we argue that our analyses were clearly picking up a proactive process (as far as any link to response inhibition is concerned), since stop-stimuli were not available for processing. Further, the contrasts between relevant- and irrelevant-stopping contexts supports this argument. We found that participants employed proactive response slowing in the relevant blocks, relative to the irrelevant blocks. Hierarchical drift diffusion models indicated that this slowing of responses mostly relied on a combination of decision thresholds as well as in drift rates (more on this in the next section). This slowing effect coincided with a positive relationship between the single-trial amplitudes in the visual N1 component in the stop-relevant context. Put simply, this means as response times slowed, the visual N1 went closer to zero μV, but was still present as a negative wave. This was not the case in stop-irrelevant task blocks, in which the visual N1 stayed constant over response time. As discussed above, the N1 component is believed to index the level of attention paid to a stimulus discrimination process. As the relationship between the N1 and response time holds only in the relevant-stop context, we argued that this pattern of results reflects a strategic damping process that proactively slows go-stimulus processing when the response to this stimulus might have to be canceled (see Figure 6.2 for an illustration of the results of this study reflecting the modeled results of the analysis). It is interesting to note as well that there appears to be no *strong* differences in the visual N1 between the two contexts in a categorical sense, at least when we took into account re-

sponse time. What happens in this case is that the relationship between the fluctuations in response time and the N1 in the irrelevant-stop context are centered in the middle of the positive relationship seen in the relevant-stop context. In a simple case of a comparison between the visual N1 between contexts by taking the traditional method of averaging all trials, you would find only a very weak relationship, which however disappears when taking response-time variation across trials into consideration. This fact highlights a real strength of the single-trial methodology that we applied.

The study in **Chapter 3** was similar in spirit to the previous chapter, and investigated the same visual N1 component underlying proactive response inhibition during the stop-signal task. The analysis was based on a comparison of go-trials from different trial blocks in which successful response inhibition in stop-trials was either explicitly motivated by reward prospect, or not (see Figure 6.1 for an overview, and the overlap with Chapter 3). In this sense the context was changed to probe for differences due to an explicit motivation of reactive response inhibition; yet, what we probed was the influence of such a context on proactive inhibition in go-trials. We found a significant positive relationship ($\beta > 0$) between the single-trial amplitudes in the visual N1 component and response speed in the standard-stop context, but not in the rewarded-stop context. However, there was an overall more pronounced N1 in the reward context suggesting a global increase in attentional processing when reward was a possibility. This is still at its core related to proactive response inhibition given we only looked at go-trials. The standard-stop con-

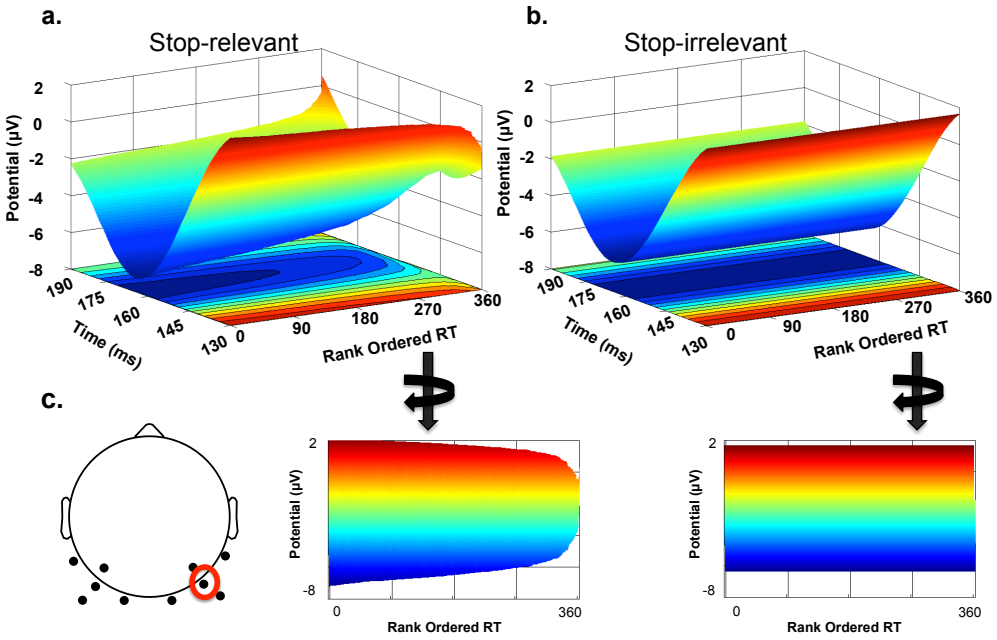

Figure 6.2: A different view of the results in Chapter 2. In this figure we see the predicted values plotted over response time, potential, and time from the go-stimulus in the (a) stop-relevant and (b) stop-irrelevant contexts from one posterior electrode (c). These are then rotated to show the differences only over response time. This is very similar to Figure 1 seen in Chapter 3, and the general pattern is the stop-relevant condition shows the same pattern as the standard-stop condition in Chapter 3.

text results were a replication of the findings reported in Chapter 2 in the relevant-stop context, whereas the reward context likely created a context in which attention is generally increased for all task-relevant stimuli (likely related to the fast succession of go- and stop-stimuli in a stop-trial) that might not have left room for subtle relationships between response speed and N1 amplitude (see Figure 6.2). In contrast to the clear difference on the N1, we also saw non-zero relationships between response time and EEG in the N2 and P3 time-ranges that did not differentiate clearly between the two contexts. While it did not differentiate between motivational context, it very well might be interesting to analyze this finding in tasks similar to the study in Chapter 2, which we had originally refrained from given a very clear a-priori hypothesis concerning the N1 based on earlier work (Bekker et al., 2005; Boehler et al., 2009). Finally, in Chapter 3, despite overall mean response time and go-accuracy being similar across blocks, we observed differential results of a drift diffusion analysis. Specifically, a stop-trial-related reward context drove go-trials to have increased drift rates and decreased nondecision times, together with an elevated decision threshold. These results also suggest an overall context effect of reward that extends to go-trial processing (despite go-trial performance not being directly relevant for obtaining reward), where enhanced attention to the go-stimulus likely results in the faster drift rate and (at least partly) the shorter non-decision time, whereas the elevated threshold likely reflects the ambition of keeping the responses in a similar RT range as in the standard-stop context (not least to

comply with instructions).

Although we consider the use of single-trial RT information to analyze the EEG data both novel and highly informative, the research in **Chapter 2** and **Chapter 3** technically entailed a reverse inference (Poldrack, 2006). Specifically, we used variations in the N1 component across trials related to response time in order to interpret this finding as reflecting attention, but did not try to manipulate attention directly. The mathematical models used in the stop-signal literature (Independent Horse Race models) have specific empirically-validated model predictions for go-trial response time distributions in such tasks (which our data all met), and with the further use of a well-validated EEG component known to be reflective of discriminatory processing, we feel that the conclusions regarding attention and response time in the previous chapters are firmly grounded and correctly interpreted. However, partially driven by the fact we were modeling a reverse inference, in **Chapter 4** and **Chapter 5** we aimed to directly modulate preparatory processes in a fashion similar to Zandbelt et al. (2013) and Verbruggen and Logan (2009) to provide more direct evidence for attentional involvement in proactive inhibition. To further this, we switched focus from the visual N1 component to a neurophysiological signal that is present even prior to the presentation of any target stimuli, specifically alpha-power lateralization, using a cue-target sequence with lateralized go-stimuli (e.g., Rihs et al., 2007). This research was directly driven by both our past findings, the past findings of others, as well as mathematical modeling work on expectations in the

stop-signal task. We will first briefly discuss the modeling work, and finally move on to the specific findings in each of the chapters.

Part of the motivation for the research in **Chapter 4** and **Chapter 5** is the role that expectation plays in recent mathematical models of the stop-signal task (e.g. Shenoy and Yu, 2011; Ide et al., 2013; Yu and Cohen, 2009). These models are hidden Markov models (Baum and Petrie, 1966) that predict the probability of a stop-trial given past observations, and accumulation of evidence (in some sense similar to a drift-diffusion process). In Shenoy and Yu (2011) it was shown that this class of models could account for classic stopping behavior in a stop-signal task, specifically that the stop failure rate grew with an increasing stop-signal delay, and that there are faster stop failures than correct go-trials. Further, these models could account for sequence effects, i.e., slowing on go-trials after runs of stop trials and speeding of go-trials after runs of go-trials with the model predicting actual RT on Go-trials with very high precision. While these models are clearly not the full story when it comes to response inhibition behavior (e.g., they do not speak directly to the implementation of response inhibition, except if it is framed exclusively as a decision), they do explain a lot of variance seen in the stop-signal task, and are interesting in that they are, in many senses, a model of expectation and hence proactive processes.

In **Chapter 4** we examined the pre-stimulus alpha lateralization as a function of a cued expectation for the current trial to have a second stop-stimulus. The task was one in which the cue carried two sources of infor-

mation. It was explicit in the frequency/probability at which the current trial would include a centralized stop-signal (10%, 25%, 50%), and was always predictive of the hemifield in which the go-signal would be presented. We found that as cued expectation for a stop-trial went up response times slowed, as expected. Drift-diffusion modeling of the go-trial data suggested that response time distributions came about in different manners for the different expectation cues. Specifically, as the probability of a stop trial rises, the drift rate lowered, response thresholds raised, and non-decision times became longer. This was accompanied by a clear trend in the lateralization of alpha power over the different levels of expectation; as expectation for a stop-trial became higher, the lateralization index became lower. This was accompanied by a larger bilateral visual N1 effect over the as expectation became higher.

The prediction going into Chapter 4 was that as response time slowed (stop-signal expectation became higher) we would see a smaller go-locked N1, and a lower lateralization in alpha-band power before the go-stimulus across the different probability conditions. This prediction was based off of the results in Chapter 2 and Chapter 3. However, as described above, this is not what the results indicated at all. There is, in this case, a very clear modulation of both alpha-band power and the visual N1 going in the opposite direction. As touched upon in the Introductory chapter the dual-mechanisms of control account (Braver, 2012) posits the idea that subtle differences between otherwise similar tasks might lead to significant changes

in preferred cognitive control strategy. These differences could be expected to result in shifts in both behavioral performance and also in neurophysiology. In our task the behavioral performance appears to be in line with a standard stop-signal task, however a possible reason for the unexpected differences in the EEG signals is simply that the go-stimuli in Chapter 4 were presented in a lateralized fashion but stop-stimuli centrally (hence also requiring monitoring this central position), and this fact afforded participants a different strategy to arrive at the same behavior. Furthermore, the stop-signal task is both spatially and temporally more complex than the usual cued Posner tasks used to examine spatial attention and alpha-band lateralization, adding another layer to the interpretation.

In **Chapter 5** the main focus was again the lateralization of posterior alpha-band oscillations, and this was partially due to the unexpected outcome seen in Chapter 4. In this study we used go/no-go task and similar to Chapter 4 the cue carried two types of information. An auditory expectation cue stochastically predicted the identity of the upcoming stimuli (either a go, or a stop signal), and a spatial cue was fully predictive of the relevant side. The main differences to Chapter 4 is that go/no-go task is less complex temporally (no second-signal) and we further included distractors in the irrelevant hemifield - which could make a difference in alpha lateralization (Slagter et al., 2016). This expectation manipulation both slowed response times and raised the proportion of correct inhibition trials in high expectation trials. We found a significant increase in the lateralization of al-

pha power when the cue predicted that the forthcoming stimulus had a high probability to require response inhibition. We furthermore observed a more pronounced N1 component in the high expectation trials. Additionally, we found increases in midfrontal theta power in no-go trials vs. go trials, and further observed an increase in successful no-go trials in the low expectation trials vs. high expectation trials.

6.3 Sequential sampling models

A common theme in Chapter 1, Chapter 2, and Chapter 3 is a formulation of a sequential sampling model known as a drift-diffusion model (DDM). DDMs are decision making models of two-choice (it can be extended, however) tasks (see Figure 6.3 for a simple graphical depiction of a DDM). Each possible choice is represented as an upper and lower boundary, specifically to our case these boundaries are defined as a correct-go (upper) and an incorrect-go (lower) response. Each decision is modeled as an accumulation (a Wiener process) of noisy information indicative of one choice or the other, with sequential evaluation of the accumulated evidence at each time step (Ratcliff and Rouder, 1998; Smith and Ratcliff, 2004). The rate at which the accumulation process approaches the upper or lower boundaries is called the drift-rate (v) and represents the relative strength of accumulating evidence for or against a particular boundary. The distance between the two boundaries is the threshold (a) and determines how much evidence must be accumulated

until a boundary is passed and hence a response is made. The smaller the distance between the upper and lower boundaries the faster a response is in general (holding other parameters constant), however this will also lead to a greater influence of noise and hence could make crossing a boundary easier based purely on noise, leading to errors. Basic sensory perception, movement initiation, and response execution are all aggregated in the models by a single non-decision time parameter (t). The finishing times of this process gives rise to the distributions of response times for both correct and incorrect choices. Given the involvement of multiple reactive and proactive processes in forming behavior in the stop-signal task we believe that these models (and similar formulations) are integral to understanding the full complexity of response inhibition. They are particularly interesting when contextual changes are made, because participants might dynamically change response modes.

In **Chapter 2** we replicated an effect on response thresholds that was previously described (Verbruggen and Logan, 2009), with thresholds being higher in the relevant-stop context. We also found an effect on drift rates, with drift rate being higher in the irrelevant-stop context. The drift-rate effect has also been reported before, but was found to be difficult to interpret (Logan and Cowan, 2014). The differences seen in these parameters suggest that in the relevant-stop context participants employed a more conservative response mode (i.e., go responding was less susceptible to a noisy response), and we suggested that the dynamically modulated level of attention towards the go-stimulus is related to drift rate, whereas other, likely more motor-level

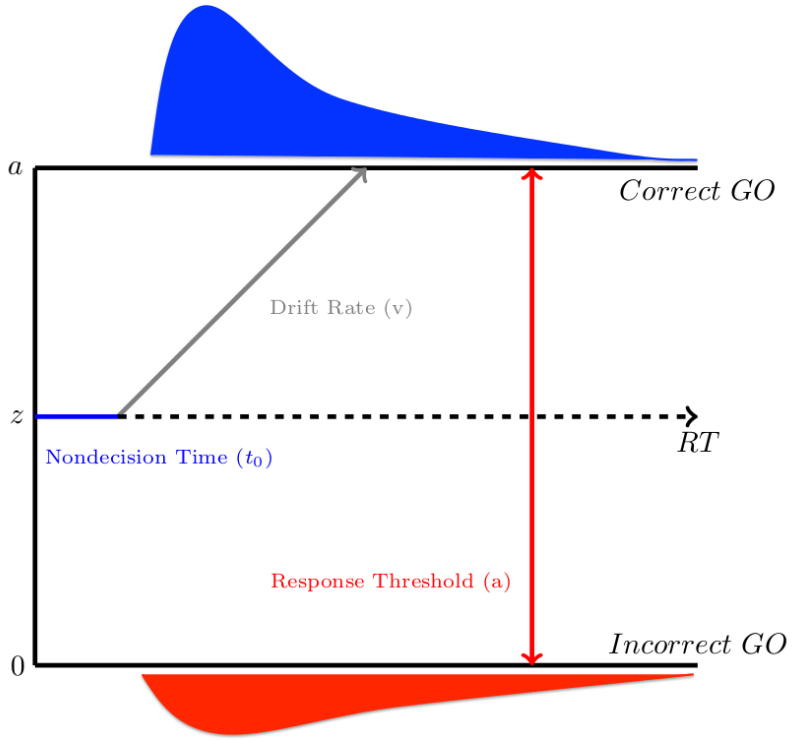

Figure 6.3: Graphical depiction of a drift-diffusion model. In a drift-diffusion process evidence is accumulated stochastically (v) towards one of two bounds, which define the response threshold (a). In the cases seen in this thesis the boundaries are either a correct-go or an incorrect-go response. As evidence accumulation crosses a boundary a response is executed. The nondecision time (t_0) defines all processes that are not part of the decision process. z is the starting point in the process, and can be biased based upon the paradigm (in our modeling we assume that there is no bias).

processes probably account for differences in threshold.

In **Chapter 3** under motivational changes in context both the drift rate increased and the decision threshold increased, while nondecision time became smaller in the rewarded-stop blocks. A decrease in nondecision time and increase in drift rate leads to quicker response processes, whereas the raised decision threshold leads to slower, more conservative responding. Therefore, it seems that the decision threshold compensates for the changes in the other two parameters. A decrease in nondecision time and increase in drift rate leads to quicker response processes, whereas the raised decision threshold led to slower, more conservative responding. Therefore, it seems that the decision threshold compensates for the changes in the other two parameters. While the reason for this might be a bit artificial, in that it likely relates to the instruction not to slow down responses across the two task contexts, it still illustrates nicely a fine-grained process structure, in which even near-identical mean behavior can arise from different constellations of distinguishable drift diffusion parameters (i.e. modeling RT distributions and accuracy is more powerful than summary measures). This finding is generally reminiscent of results from a stop-signal task that used stimuli with different image quality, where it was found that reduced drift rate and increased nondecision time for low-quality images was compensated for by an increased decision threshold (Jahfari et al., 2015). It seems likely that our presented results are strongly related, just the source of variation in stimulus processing is internal rather than external.

It seems likely that our presented results are strongly related. When speculating about the relationship of these parameters to our electrophysiological results, it seems reasonable to link the N1 modulations to differences in drift rate and possibly nondecision time, in that early attentional processes plausibly map onto both the rate of and starting time of evidence accumulation, which might furthermore relate to anticipatory attentional processes that precede the actual trial (e.g., Bengson et al., 2012). The increased decision threshold, in turn, might relate to a process more linked to the motor output level, which we did not capture in our analysis of the EEG data (see, O'Connell et al. (2012), Elchlepp et al. (2016), and Dippel and Beste (2015) for a broad discussion of candidate neural signatures).

Chapter 4 gave evidence that as stop-expectancy increases there is an increase in response threshold, a decrease in drift rate, and a slight increase in nondecision time. In a very broad sense, this means that as participants expect a stop-signal they take on a more cautious response mode, which is in line most clearly with Chapter 2. Indeed, this was actually the prediction of the diffusion process when the experiment was designed, and further motivates the role of expectation in response inhibition research. However, it does indicate that we should possibly be a bit more cautious in the interpretation of electrophysiological signals and their relation to diffusion processes, because it is clearly not the whole story when comparing results from Chapter 2 and Chapter 4 - given that we arrive at seemingly different EEG interpretations. A more powerful method would be to relate the EEG components

directly to the DDM parameters on a single trial level (see Cavanagh et al. (2011) for an example).

6.4 Possible clinical relevance

A host of neurological and cognitive disorders and diseases are known to be associated with general cognitive control dysfunction as well as response inhibition dysfunction. Cognitive control functions appear to be diminished in clinical populations with Huntington's and Parkinson's diseases (e.g Brown and Marsden, 1990; Sawamoto et al., 2008; Lawrence et al., 1996), depression (e.g. McIntyre et al., 2013), as well as patients with frontal lobe damage (e.g., Roca et al., 2010), to name a few. Specific to reactive response inhibition, it has been demonstrated to be implicated multiple times in patients with ADHD (e.g., Overtoom et al., 2002; Chambers et al., 2009; Senderecka et al., 2012; Alderson et al., 2007), obsessive compulsive disorder (e.g., Chamberlain et al., 2006; Menzies et al., 2007), Parkinson's Disease (e.g., Vriend et al., 2015; Alegre et al., 2013). Furthermore, motivational differences are seen between healthy participants and clinical populations, and the overlap is substantial; e.g., Parkinson's disease (e.g., Pagonabarraga et al., 2015), ADHD (e.g., Cubillo et al., 2012), depression (e.g. Yang et al., 2014),and Alzheimer's disease (e.g. Landes et al., 2001). In the current thesis we have demonstrated that proactive control processes are heavily influential in response inhibition and that gaining a richer understanding will surely depend on understand-

ing the proactive involvement of sensory and attentional processes. We have further demonstrated how motivational modulations come to bear on such attentional processes, altogether sketching a multifaceted picture of different and possibly interacting determinants of response inhibition behavior. As such, it will be critical in the future to further distinguish deficits in a core motoric inhibitory function from related deficits in more general proactive control and attention, as well as motivational impediments, when considering related deficits as a diagnostic tool, and when devising clinical interventions.

6.5 Outlook

Taken together, we believe that the research presented in this thesis points towards a highly flexible control system that is involved in response inhibition. One of the strengths of this thesis is in its novel use of nontraditional methodology. We have shown that response time and visual attention covary in response inhibition on a single-trial level, and that this covariation can be modulated by contextual changes. This is a finding that can only be seen by using methods that go beyond simple condition-wise averages across all trials. Further, using drift-diffusion models we have shown that these response time distributions are driven by different processes under different contexts. These two methodologies allowed us to find slight differences that an orthodox approach would not allow, and we would argue should be applied more often in the literature. In another line of research, turning to

pre-target alpha-power lateralization, we specifically used a marker of what we would argue is quite related to the idea of proactive control given the pre-target temporality involved, finding further corroboration of a general involvement and relevance of attention in proactive response inhibition. As is clear in both the present EEG analyses and in the drift-diffusion models response inhibition is a dynamic and flexible process that unfolds on a very fast scale and differs across trials in a meaningful way (and generally in line with recent conceptual work seen in Elchlepp et al. (2016)) This dynamic and varying nature is, in our view, an important feature that needs to be integrated more systematically also in future work, both on the topic of the present thesis, as well as likely also in a wider context of cognitive functions.

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Chapter 7

Nederlandse samenvatting

Een opvallend kenmerk van de menselijke cognitie is zijn veelzijdigheid. We kunnen ons snel aanpassen aan een continu veranderende interne en externe omgeving. We zijn bijvoorbeeld in staat om snel uit te wijken om een botsing te voorkomen met een niet-oplettende bestuurder, een honkbal te raken met een knuppel, mentale berekeningen te maken wanneer we een rekening moeten splitsen in een druk restaurant, en ons te concentreren terwijl we een moeilijk examen maken. Deze veelzijdigheid is afhankelijk van bepaalde neurale processen die zich dynamisch afstemmen op sensorische, perceptuele, motorische en andere interne mechanismen en ons zo helpen bij het bereiken van onze interne doelstellingen. Het gaat om de regelutatie, cordinatie, en het ordenen van gedachten en handelingen (Braver, 2012), en wordt cognitieve controle genoemd. Cognitieve controle is belangrijk voor menselijk adaptief gedrag, en is een breed concept dat meerdere cognitieve domeinen omvat zoals: het bijwerken en beheren van contextueel relevante informatie, het sturen van aandacht, het bijhouden van prestaties, en de activering of inhibitie van gepaste of ongepaste gedragingen (Miller and Cohen, 2001; Verbruggen et al., 2014).

Responsinhibitie is een belangrijke component van succesvolle cognitieve controle. Een veranderende omgeving vereist vaak dat een automatische of anderszins overheersende reactie op korte termijn onderdrukt of geinhibeerd kan worden. Alhoewel traditioneel onderzoek vooral heeft gekeken naar reactieve processen, waarbij inhibitie wordt getriggerd door een expliciete sensorische stop-stimulus, is er recentelijk meer belangstelling voor proactieve

responsinhibitie. Proactieve inhibitie is de neiging om reacties te vertragen wanneer een regelrechte stop nodig is Aron, 2011 wat meestal resulteert in een langzamere reactie in tegenstelling tot algehele inhibitie zoals bij reactieve processen. Onderzoek heeft zich tot nu toe vooral gericht op een centraal motorisch inhibitie mechanisme zonder rekening te houden met de mogelijke rol van andere processen, zoals aandachts verwerking bij taakrelevante stimuli. Aandacht speelt een belangrijke rol in andere cognitieve controle taken (Braver, 2012) en het is dus niet onwaarschijnlijk dat dit ook een rol speelt bij responsinhibitie. Recent onderzoek heeft inderdaad de mogelijke rol van vroegere aandachtsprocessen bij responsinhibitie gevonden (Bari and Robbins, 2013; Logan and Cowan, 2014; Verbruggen et al., 2014; Huster et al., 2014; Elchlepp et al., 2016; Bekker et al., 2005; Boehler et al., 2009; Kenemans, 2015). Hoe aandachtsprocessen en proactieve inhibitie processen precies aan elkaar gerelateerd zijn is de focus van dit proefschrift.

In hoofdstuk 2 onderzochten we single-trial ERP's en de relatie met reactietijden in een standaard vorm van de stop-signaal taak (stop-relevante context), en een zintuiglijke basislijn waarbij deelnemers geinstrueerd werden om de stop-signalen te negeren (stop-irrelevante context). Specifiek keken we naar de visuele N1 component. De vertraagde reactietijden vielen samen met een positieve relatie tussen de single-trial ERP amplitudes in de visuele N1 component in de stop-relevante context. In hoofdstuk 3 onderzochten we dezelfde visuele N1 component onder gelegen aan pro-actieve responsinhibitie tijdens de stop-signaal taak. De resultaten van de standaard-stop

taak waren een replicatie van de bevindingen in de stop-relevante context gerapporteerd in hoofdstuk 2, terwijl een belonings conditie een algemene verhoging van aandacht voor alle taak-relevante stimuli creerde. In hoofdstuk 4 onderzochten we pre-stimulus alpha lateralisatie in functie van een gecuede verwachting dat de huidige taak een tweede stop-stimulus zou bevatten. Er was een duidelijke trend in de lateralisatie van alpha power over de verschillende niveaus van verwachting; als verwachting voor een stop-signaal hoger werd, werd de lateralisatie index lager. In hoofdstuk 5 onderzochten we opnieuw de lateralisatie van posterieure alpha-band oscillaties, en gebruikten we een go/no-go taak. Vergelijkbaar met hoofdstuk 4 werd er een cue gepresenteerd die twee soorten informatie kon bevatten, spatiale en verwachtings informatie. We vonden een aanzienlijke toename van de lateralisatie van alpha power wanneer de cue voorspelde dat het zeer waarschijnlijk was dat de volgende stimulus een responsinhibitie zou vereisen.

Bij elkaar genomen, zijn wij van mening dat het onderzoek beschreven in dit proefschrift laat zien dat het systeem dat betrokken is bij responsinhibitie zeer flexibel is. We hebben aangetoond dat reactietijd en visuele aandacht covariren in responsinhibitie op een single-trial niveau en dat deze covariatie kan worden gemoduleerd door contextuele veranderingen. Deze bevinding kan enkel worden gevonden door gebruik te maken van werkwijzen die verder gaan dan het eenvoudig condition-wise middelen over alle trials. Met betrekking op pre-stimulus alpha-power lateralisatie, hebben we een specifieke marker gebruikt waarvan we beweren dat deze is gerelateerd

aan proactieve controle gezien de pre-target timing, waardoor we verdere bevestiging vinden van een algemene betrokkenheid en relevantie van aandacht in proactieve responsinhibitie. Zoals blijkt in zowel de huidige EEG analyses en de drift-diffusion modellen is responsinhibitie een dynamisch en flexibel proces dat zich in een zeer snelle tijd ontvouwt en verschilt per trial op een zinvolle manier. Dit is een belangrijk kenmerk dat systematisch moet worden gentegreerd in toekomstig onderzoek, zowel in het onderwerp van dit proefschrift, evenals in een bredere context van cognitieve functies.

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% Name/identifier study:Strategic down-regulation of attention
% Author: Zachary Langford
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* On which platform are the raw data stored?
  - [X] researcher PC
  - [X] research group file server: in the near future the data will also be stored on our research group file server
  - [ ] other (specify):

* Who has direct access to the raw data (i.e., without intervention of another person)?
  - [X] main researcher
  - [X] responsible ZAP (when the data will be stored on the research group file server, the data will be available to both the main researcher and the (co-)promotors
  - [ ] all members of the research group
  - [ ] all members of UGent
  - [X] other (specify): Marty Waldorff at Duke University also has full access to the raw data.

3b. Other files

* Which other files have been stored?
  - [X] file(s) describing the transition from raw data to reported results. Specify: see methodology section in the article
  - [X] file(s) containing processed data. Specify: Preprocessing steps.
  - [X] file(s) containing analyses. Specify: see findings section in the article.
  - [X] files(s) containing information about informed consent. Specify: filed at Duke University.
  - [ ] a file specifying legal and ethical provisions. Specify:
  - [X] file(s) that describe the content of the stored files and how this content should be interpreted. Specify: Analysis was all done with a script.
  - [ ] other files. Specify: ...

* On which platform are these other files stored?
  - [X] individual PC
  - [ ] research group file server
  - [ ] other: ...

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  - [X] main researcher
  - [ ] responsible ZAP
  - [ ] all members of the research group
  - [ ] all members of UGent
  - [ ] other (specify): ...

4. Reproduction

* Have the results been reproduced independently?:
[ ] YES / [X] NO

* If yes, by whom (add if multiple):
  - name:
  - address:
  - affiliation:
  - e-mail:

% Data Storage Fact Sheet (versie 7 maart 2014)

% Name/identifier study:Motivational context and attention
% Author: Zachary Langford
% Date: 17 maart 2017

1. Contact details

1a. Main researcher

- name: Zachary Langford
- address: Henri Dunantlaan 2, 9000 Gent
- e-mail: zachary.langford@UGent.be

1b. Responsible Staff Member (ZAP)

- name: prof. Nico Boehler
- address: Henri Dunantlaan 2, 9000 Gent
- e-mail: nico.boehler@UGent.be

If a response is not received when using the above contact details, please send an email to data.pp@ugent.be or contact Data Management, Faculty of Psychology and Educational Sciences, Henri Dunantlaan 2, 9000 Ghent, Belgium.

2. Information about the datasets to which this sheet applies

* Reference of the publication in which the datasets are reported: Langford, Z. D., Schevernels, H., and Boehler, C. N. (2016b). Motivational context for response inhibition influences proactive involvement of attention. Scientific Reports, 6(October

):35122.

* Which datasets in that publication does this she
et apply to?: the sheet applies to all the data us
ed in the publication

3. Information about the files that have been store
d

3a. Raw data

* Have the raw data been stored by the main researc
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If NO, please justify:

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  - [ ] all members of the research group
  - [ ] all members of UGent
  - [X] other (specify): Hanne Schevernels

3b. Other files

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  - affiliation:
  - e-mail:

% Data Storage Fact Sheet (versie 7 maart 2014)

% Name/identifier study:Anticipatory Î± oscillatio
ns indicate an active role of attention in a cued
expectancy stop-signal task
% Author: Zachary Langford
% Date: 17 maart 2017

1. Contact details
==========
========

1a. Main researcher

- name: Zachary Langford
- address: Henri Dunantlaan 2, 9000 Gent
- e-mail: zachary.langford@UGent.be

1b. Responsible Staff Member (ZAP)

- name: prof. Nico Boehler
- address: Henri Dunantlaan 2, 9000 Gent
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% Data Storage Fact Sheet (versie 7 maart 2014)

% Name/identifier study:Cued expectation modulates prestimulus alpha lateralization in a go/no-go task
% Author: Zachary Langford
% Date: 17 maart 2017

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1a. Main researcher

- name: Zachary Langford
- address: Henri Dunantlaan 2, 9000 Gent
- e-mail: zachary.langford@UGent.be

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