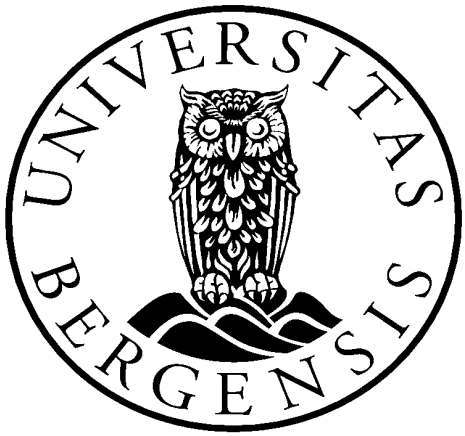
The effect of Recent Trial History on Response Times and Beta-Burst rates in the Stop-Signal task

Jørgen Kransberg



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Supervisor: Marco Hirnstein, Department of Biological and Medical Psychology (UiB)

Co-Supervisor: Carsten Bundt, Department of Cognitive and Clinical Neuroscience (UiO)

Co-Supervisor: András Puszta, Department of Cognitive and Clinical Neuroscience (UiO)

Abstract

There is no consensus on a direct neural marker of inhibition in the literature on response inhibition. Previous studies have investigated the relationship between inhibition and EEG-derived measures, such as the P3 and N2 ERP-components as well as activity in the beta (15-30 Hz) frequency-band. Recent studies have observed sudden peaks of activity in the beta frequency-band, only apparent at the single-trial level. Beta-Burst rate, that is, the number of such peaks occurring on each trial, have been proposed as reflecting response inhibition. The aim of the current study was to investigate the relationship between sensorimotor post-go Beta-Burst rates and behavioral measures of response inhibition. Thirty-four participants performed the Stop-Signal task while their brain activity was recorded with EEG. The length of trial-sequences within each block were predetermined with the purpose of investigating how the number of preceding trials influenced task performance and Beta-Burst rates. We expected shorter response times and lower Beta-Burst rates prior to unsuccessful compared to successful inhibition. We also expected both response times and Beta-Burst rates to be negatively correlated with Sequence-Length. The results showed shorter response times prior to unsuccessful compared to successful inhibition. There was however no difference in Beta-Burst rates prior to unsuccessful compared to successful inhibition. The results also showed that Sequence-Length had no effect on neither response times nor Beta-Burst rates. In sum, the current study did not find evidence for a relationship between Beta-Burst rates and behavioral measures of response inhibition, questioning the role of Beta-Burst rates as a neural marker of inhibition.

Keywords: Response inhibition, beta-bursts, EEG, stop-signal, cognitive control

Sammendrag

Det er ingen konsensus rundt en direkte nevral markør for inhibisjon i litteraturen om responsinhibering. Tidligere studier har undersøkt forholder mellom inhibering og EEG-baserte mål, som P3 og N2 ERP-komponentene og aktivitet i beta (15-30 Hz) frekvensbåndet. Nylige studier har observert plutselige økninger i aktivitet i beta frekvensbåndet som kun er synlige på single-trial nivå. Beta-Burst rate, som er antallet slike plutselige økninger i aktivitet i hver enkelt trial, har blitt foreslått å reflektere inhibisjon. Formålet med denne studien er å undersøke forholdet mellom sensorimotor post-go Beta-Burst rater og atferdsmål på responsinhibering. Trettifire deltakere utførte Stop-Signal task mens hjerneaktiviteten deres ble målt med EEG. Lengden på trial-sekvenser var predeterminert med det formål å undersøke hvordan antallet foregående trials påvirket deltakernes atferd. Vi forventer kortere responstider og lavere Beta-Burst rater før mislykket sammenlignet med vellykket inhibering. Vi forventet også at både responstider og Beta-Burst rater var negativt korrelert med Sekvenslengde. Resultatene viste kortere responstider før mislykket sammenlignet med vellykket inhibering. Det var imidlertid ingen forskjell i Beta-Burst rater før mislykket sammenlignet med vellykket inhibering. Resultatene viste også at Sekvenslengde hadde ingen effekt på hverken responstider eller Beta-Burst rater. Totalt sett støttet ikke resultatene en sammenheng mellom Beta-Burst rater og responsinhibering.

Nøkkelord: Response inhibition, beta-burst, EEG, stop-signal, kognitiv kontroll

Preface

First, I would like to thank my supervisors Marco Hirnstein, Carsten Bundt and András Puszta who have provided valuable feedback and guided me during the development of my thesis. I also want to thank René Huster who gave me the chance to work with the MICC-lab. I want to thank everyone at the MICC-lab. You have taken good care of me during this last year of my studies.

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1. Introduction

The ability to adapt behavior in response to changing contextual demands are crucial in successfully navigating the environment. One aspect of such cognitive control processes is response inhibition (RI), which is the ability to cancel an already initiated movement (Band et al., 2003). Imagine seeing a friend in public. You raise your hand thinking you should wave to get their attention. A moment later, you realize that the person you saw was not your friend after all. If you realized the mistake early enough, you might be able to lower your hand before you are fully invested in waving to a stranger. The ability to stop an already initiated movement can save someone from embarrassing situations, but more importantly, it has been related to developmental and psychiatric disorders such as ADHD (Wodka et al., 2007) and schizophrenia (Enticott et al., 2008). Further development of our understanding of the neural underpinnings of RI can help shed light on aspects of these disorders and how to treat them.

When inhibition is applied pre-emptively, it is called proactive inhibition and when it is applied in response to a stimulus it is called reactive inhibition. Proactive inhibition can be observed behaviorally by measuring how participants adjust their performance throughout the task. This is typically observed in conditions requiring a response to be canceled (Rawji et al., 2022). The latency of reactive inhibition is difficult to measure directly due to successful inhibition being apparent by the absence of behavior. It can however be computed based on the performance of participants on the Stop-signal task (SST).

The high temporal resolution of electroencephalography (EEG) makes it well suited to study the short-latency processes related to inhibition. Different neural markers of brain activity measured with EEG have been proposed as reflecting inhibition, such as the P3 and N2 ERP-components (Eichele et al., 2010; Steinhauser et al., 2012) and desynchronization (i.e., weakening of the signal power) of EEG-activity in various frequency bands (Beyer et al., 2012; Cavanagh & Shackman, 2015). However, there is currently no consensus in the

literature regarding a direct neural marker of inhibition (Huster et al., 2020). Wessel (2020) proposed that single-trial bursts of activity in the beta frequency band (15-29Hz), called Beta-Bursts, could reflect response inhibition. Beta-burst rates have been shown to predict successful inhibition of a prepotent response (Soh et al., 2021; Wessel, 2020), and to correlate with partial electromyography (prEMG) measures of motor inhibition (Hannah et al., 2020; Jana et al., 2020). However, some researchers have failed to replicate these findings (Enz et al., 2021; Errington et al., 2020). In addition, when using scalp EEG, only 15% of all trials contain Beta-Bursts (Jana et al., 2020). This has been suggested as being a consequence of the low signal-to-noise ratio in scalp EEG, making it difficult to detect all the Beta-Bursts occurring.

Behavioral measures of response inhibition have been known to be influenced by the global task context (i.e., across the task) such as how likely it is that a response must be canceled (Verbruggen et al., 2019). A few studies have also investigated the effect that the preceding trial-sequences have on subsequent performance (Chang et al., 2017; Eichele et al., 2010; Hu et al., 2015). However, most of these studies have investigated this effect using other inhibition tasks than the SST. Exploring how stopping performance in the SST is influenced by the sequence of preceding trials, can shed light on how humans track and learn from their recent experiences.

In this study we investigated how behavioral and neural measures of proactive inhibition were influenced by the recent trial history. We observed how reaction times and brain activity varied based on the sequence of preceding trials in the SST. We also wanted to explore the relationship between behavioral measures of proactive inhibition and the novel measure of Beta-Burst rate. By investigating how they both vary under the same conditions we hope to understand how they are related.

1.1. Response Inhibition

The ability to inhibit a response has been investigated in different paradigms. However, the findings from different paradigms must be generalized with caution. What the participants are required to do, i.e., their performance, depends on the paradigm. This means that the performance on one task might not involve the same processes as the performance on another task. The SST is one of the paradigms most widely used to study response inhibition (Logan & Verbruggen, 2014). In this paradigm, researchers can measure the timing of the prepotent response, as well as compute an indirect measure of the reactive inhibition. The SST also allows researchers to examine how participants adjust their performance by observing how the timing of their prepotent response varies throughout the experiment.

1.1.1. The Stop-signal Task

The SST tests the ability of the participant to cancel an already initiated motor response (Logan & Verbruggen, 2014). The task consists of two different trial types; go-trials and stop-trials. On all trials, an arrow pointing leftwards or rightwards, is presented to the participants in each part of the trial. This arrow represents the go-signal, informing the participant that they have to quickly press a button with the hand corresponding to the direction of the arrow. In 25% of all trials, a second arrow is presented shortly after the go-signal. This second arrow is the stop-signal, and when presented it informs the participant that they must cancel their original response to the go-signal. The 75% of trials where only the go-signal is presented are go-trials. The 25% of trials where the stop-signal is presented after the go-signal are stop-trials.

The SST is based on the horse-race model (Logan & Verbruggen, 2014). The model describes a race between the go-process and the stop-process, with the winning process deciding whether the go-response will be executed or inhibited. In the context of the SST, the go-process is initiated at the presentation of the go-signal, and the stop-process at the

presentation of the stop-signal. The outcome of the race as well as of the trial depends on the speed of each process, and the duration of the time interval between the initiation of the two processes.

The time interval between the go-signal and the stop-signal is called the stop-signal delay (SSD) (Verbruggen et al., 2019). This delay between the two stimuli varies throughout the task so that the participant's ability to stop a response can be assessed under conditions with different degrees of difficulty. A long SSD means that the go-response is closer to completion before the stop-signal is presented – making it more difficult to cancel the go-response. A short SSD means that the go-response is further away from completion – making it easier to stop.

There are two main approaches to determine the SSD and how it will vary throughout the task (Verbruggen et al., 2019). If using the fixed approach, the SSD on a specific stop-trial will be randomly chosen from a predetermined pool of SSDs. The fixed approach was originally the most used approach but has since been mostly replaced by the tracked approach (Verbruggen et al., 2019).

The tracked approach involves a staircase algorithm where the SSD on a specific stop-trial depends on the outcome of the previous stop-trial. If the previous stop-trial was a successful stop (SS), then the SSD on the next stop-trial will increase with a step-size (usually between 25-50 msec, Verbruggen et al., 2019), making it harder to stop. Similarly, if the previous stop-trial was an unsuccessful stop (US), then the SSD on the subsequent stop-trial will decrease by a step-size, making it easier to stop. By adjusting the SSD based on the participant's performance, the staircase algorithm makes it so that the overall probability of stopping is approximately 50% for all participants.

One difference between the two approaches is that due to the staircase procedure, the task difficulty when using the tracked approach is equal across participants. In the fixed

approach, all participants are exposed to the same pool of SSDs independently of how they perform throughout the task. That means that the task will be easier for participants who are inherently better at stopping, compared to participants who are relatively worse at stopping. Such differences in task difficulty might influence the results. For instance, when exploring effects on response times, task difficulty could influence the results by differentially influencing the degree of fatigue each participant experiences.

One of the factors leading to the widespread use of the SST paradigm, is that it allows for calculation of the latency of the inhibition process (Verbruggen et al., 2019). This measure is called the stop-signal reaction time (SSRT) and is one of the performance measures in the stop-signal task.

1.1.2. Outcome Measures in SST

The participants try to balance the need to respond quickly on go-trials with the need to correctly stop in stop-trials. How participants balance the need for speed versus the need for accuracy can be investigated in the SST by measuring how quickly they respond to the go-signal, and how often they successfully inhibit a response.

Go-signal reaction time (GoRT) is the time it takes for a participant to respond to the go-signal, averaged across all go-trials (Eagle et al., 2008). It is assumed to represent the time of the go-process described in the horse-race model (Band et al., 2003). In addition, go-accuracy is a measure of the percentage of go-trials that the participant responded to correctly.

The latency of the inhibition process is called Stop-signal reaction time (SSRT) and is the presumed time it takes for the stop-process to complete. SSRT is the most used behavioral marker of RI and has been regarded as the gold standard for measuring the latency in reactive inhibition (Huster et al., 2020). However, there is still controversy around the use of this measure in the literature. SSRT can, for instance, be calculated in different ways, leading to less standardization of the measure, potentially reducing reliability.

The two most common ways to calculate the SSRT are the mean method and the integration method. The mean method computes SSRT by subtracting the mean SSD from the mean GoRT (Verbruggen et al., 2019). The mean SSD is determined by finding the mean of the inhibition function. This function describes the relationship between SSD and the probability of responding on a stop-trial. The SSD at which the probability of stopping is 50% is the mean of the inhibition function, as well as the mean SSD.

The integration method is based on the rationale that the upper limit of GoRT on unsuccessful stop-trials is equal to the sum of SSD and SSRT (Band et al., 2003). To find the trials with the upper limit of GoRT, all individual go-trials are ordered from short to long GoRT. Then the number of go-trials are multiplied with the stop-accuracy to find the *nth* GoRT. For example, if we order 120 go-trials based on their GoRT, and we have a stop-accuracy of 50%, the *nth* GoRT would be the GoRT on the 60th go-trial (nGo-trial x stop-accuracy = 120 x 0.5 = 60). The SSRT is then estimated by subtracting the mean SSD from the *nth* GoRT. When using the integration method, go-trials where the participant made the wrong response or failed to respond, are counted as a go-trial in relation to the ordering of go-trials, but the GoRT is then set to 1000 msec (Verbruggen et al., 2019).

Verbruggen and colleagues (2019) ran simulations with the two different methods to investigate which one leads to the most reliable estimation of SSRT. They showed that the mean method was more influenced by the right tail of the GoRT distribution. This means that the mean method might lead to unreliable SSRT estimations if used on samples with a non-gaussian distribution of GoRT. Verbruggen and colleagues also showed that the mean method is heavily influenced by the rate of go-trials with no response (go-omissions). For these reasons, they recommended using the integration method with replacement of go-omissions to calculate SSRT.

SSRT is a measure of reactive inhibition, that is, inhibition of a response after a signal to do so has been presented (Eagle et al., 2008). Proactive inhibition, that is, inhibition happening in anticipation of a signal to stop (Rawji et al., 2022), and can be observed as adjustments in the latency of the participants´ responses.

1.1.3. Performance Adjustments

Participants typically adjust their performance throughout the task (Rawji et al., 2022). One of the observed performance adjustments in the literature is pre-Error-Speeding (pre-ES) (Nelson et al., 2010; Thakkar et al., 2014), which is that participants typically have shorter GoRT prior to US-trials compared to SS-trials.

In a study conducted by Rawji and colleagues (2022), they investigated the effect of proactive inhibition on the neural pattern of activation in the motor cortex. Rawji and colleagues used a variation of the SST where participants had to perform two different types of blocks. Half of the blocks were standard SST blocks with both go- and stop-trials, while the other half were pure-go blocks with only go-trials. The key difference between the blocks were the presence of stop-trials in the standard blocks which were thought to promote the use of proactive inhibition. Having participants perform both types of blocks allowed the researchers to investigate how potentially having to inhibit a response affects the performance. They stimulated the motor cortex using transcranial magnetic stimulation (TMS) resulting in measurable muscular contractions called motor-evoked potentials (MEPs). The amplitude of these MEPs reflects the corticospinal excitability (CSE) (Rawji et al., 2022). By stimulating the motor cortex at different times during the task, one can measure variations in the amplitudes of the MEPs to estimate the CSE over time.

They found significantly shorter GoRT in the pure-go block compared to standard SST blocks. The presence of stop-trials thus seemed to recruit proactive inhibition, behaviorally evident as a slowing down of the go-response.

They also found that the patterns of variations in CSE differed when comparing the two types of blocks. The results showed that the rate of increase in CSE was the same in both conditions, but that onset of the CSE rise was later in the standard blocks. Rawji and colleagues (2022) interpreted this as instead of altering the threshold for initiating a movement, rather the rise of CSE seems to be delayed in conditions promoting the use of proactive inhibition.

In sum, Rawji and colleagues (2022) observed shorter GoRTs, as well as a later onset of the CSE rise in the conditions that promoted the use of proactive inhibition. They demonstrated a relationship between proactive inhibition and adjustments in GoRT, while shedding some light on how proactive inhibition is implemented at a neural level. These findings demonstrate how proactive inhibition can be behaviorally operationalized as adjustments in GoRT.

Researchers have investigated time-frequency EEG data collected from participants doing the SST to further understand the mechanisms underlying proactive inhibition. By studying changes in the brain happening before the stop-signal it is possible to see how changes in brain activity can be related to stopping performance and behavioral measures of proactive inhibition.

1.2. Relationship between RI and Time-Frequency EEG

One avenue of research related to proactive inhibition and the stop-signal task has investigated time-frequency analyses of EEG-data. By doing so, it is possible to investigate changes in the frequency of the recorded EEG-data over time.

1.2.1. Averaged Beta

Raud and colleagues (2020) found differences in the beta (15-25) and mu (9-14 Hz) frequency bands prior to a successful stop-trial (SS) compared to an unsuccessful stop-trial

(US). Specifically, they observed weaker desynchronization (i.e., weaker decrease of the power of the EEG-signal) in the beta-frequency band before successful stop trials compared to unsuccessful stop trials. This means that successful stop trials were associated with a higher beta power compared to unsuccessful stop trials. They used electromyography (EMG) to probe corticospinal excitation in the pre- and post-go period and observed that motor cortical activity differed prior to SS vs US. In addition, they observed smaller motor-evoked-potentials (MEPs) in the hand that was cued to potentially stop, indicating a relationship between proactive inhibition and MEPs.

Some researchers have raised concerns with the growing amount of literature failing to indicate a relationship between changes in trial-averaged beta-band EEG-data and proactive inhibition (Wessel, 2020). This has led researchers to explore single-trial oscillations in the beta frequency band to further investigate how time-frequency analyses of EEG data are related to inhibitory processes.

1.2.2. From Averaged to Beta-bursts

Desynchronization of trial-averaged beta power has been related to initiation of a movement (Raud et al., 2020). However, Feingold and colleagues (2015) compared trial-averaged beta to single-trial beta and found that power variation in trial-averaged beta could be explained by beta oscillations occurring as brief bursts at the single-trial level. They proposed that the observation of beta desynchronization and synchronization is the result of different probabilities of beta-bursts occurring. Investigating single-trial beta-bursts can tell us more about the temporal dynamics of beta oscillations. Trial-averaged beta oscillations typically last for several seconds. Beta-Bursts, however, last between 90-115 msec (Feingold et al., 2015). Feingold and colleagues (2015) also noted a high trial-to-trial variability in the occurrence of beta-bursts, where only a subset of the observed Beta-Bursts were synchronous

across trials. This can indicate that trial-averaged beta is less sensitive to the distinction between synchronous and non-synchronous oscillations in the beta frequency band.

The discovery of Beta-Bursts has led researchers to investigate how variations in single-trial oscillations are related to different aspects of response inhibition. Wessel (2020) found a relationship between GoRT and sensorimotor Beta-Burst rates in the time-window following the go-signal. He analyzed a large SST dataset (N = 234) to see if there was a relationship between Beta-Burst rate and movement initiation and cancellation.

1.2.3. Beta-Bursts & Proactive Inhibition

Wessel (2020) proposed that Beta-Bursts extracted from sensorimotor (C3 and C4) or frontal (FCz) electrode sites can operationalize inhibition related to movement initiation (proactive inhibition) or movement cancellation (reactive inhibition), respectively. The results showed that sensorimotor Beta-Burst rates increased after the presentation of the go-signal and then decreased up until the go-response. There was also observed a lateralization of the Beta-Burst rates meaning that Beta-Burst rates recorded at electrode sites contralateral to the responding hand were more strongly sustained. This can be interpreted as less inhibition of the responding hand compared to the non-responding hand.

Based on his findings, Wessel (2020) proposed that sensorimotor Beta-Burst rates in the post-go time window reflects a tonic proactive inhibition of the motor system. To investigate this proposition, he explored the within-subject relationship between Beta-Burst rates and response times. Wessel tested different time bins ranging from a 0-25 msec to 525-575 msec after the go-signal. Sensorimotor Beta-Burst rates were positively correlated with GoRT in all post-go time bins, with the strongest correlation found in the 75-125 msec and 125-175 msec time bins. This was interpreted as meaning that subjects showing high post-go sensorimotor Beta-Burst rates also showed systematically longer GoRT. Wessel also observed that Beta-

Burst rates correlated negatively with SSRT, meaning that subjects showing a higher rate of Beta-Bursts also showed shorter SSRT.

The correlations between Beta-Burst rates and GoRT and SSRT, can be interpreted as evidence supporting Wessel's proposal that Beta-Bursts reflect a tonic inhibition of the motor system (i.e., proactive inhibition). More proactive inhibition could lead to a delayed initiation of the go-response, and thus a longer GoRT. Such a delay of the initiation of the go-response could be beneficial in making the participant more likely to cancel that go-response when a stop-signal is presented. Together these findings indicate that sensorimotor post-go Beta-Burst rates reflect proactive inhibition of the motor system. Such an effect of proactive inhibition on GoRT can be expected to be observed behaviorally as pre-ES (Rawji et al., 2022).

Another study investigating the relationship between sensorimotor post-go Beta-Burst rate and proactive inhibition was conducted by Soh and colleagues (2021). They found evidence for a relationship between Beta-Bursts centered around the go-signal and proactive inhibition. In their experiment, participants had to perform a variation of the SST which included standard blocks with both go- and stop-trials, as well as pure-go blocks with only go-trials. The idea was that the degree of proactive inhibition recruited in the two types of blocks would differ. The pure-go blocks are thought to not promote the use of proactive inhibition, the participant can simply respond to the go-signal without the possibility of having to cancel their response. The standard blocks are thought to recruit proactive inhibition in the sense that participants must be prepared to cancel their response in the case of a stop-signal being presented. By having experimental conditions that differ regarding the recruitment of proactive inhibition, they could compare the performance and neural activity between the two conditions and interpret any differences as being due to the degree of proactive inhibition recruited.

Soh and colleagues found longer GoRT in the standard blocks compared to the pure-go blocks. Indicating that the possibility of having to cancel a response causes participants to respond more cautiously. The results also showed that sensorimotor post-go Beta-Bursts rates were higher in standard blocks compared to pure-go blocks. If post-go Beta-Burst rates are an index of proactive inhibition, then there should be higher beta-burst rates in conditions recruiting a higher degree of proactive inhibition (Huster et al., 2020). The results also showed that Beta-Burst rates were positively correlated with GoRT across participants. This correlation was observed in both conditions, indicating a relationship between sensorimotor post-go Beta-Burst rates and GoRT, independently of proactive inhibition. In addition, there was a within-subject correlation between the degree of GoRT-slowing and the degree of change in beta-bursts rates when comparing the two conditions. This could mean that higher degrees of proactive inhibition (evidenced by more slowing) are related to higher Beta-Burst rates.

In sum, Soh and colleagues (2022) found an increased rate of sensorimotor post-go Beta-Bursts in the standard blocks compared to pure-go blocks. The standard blocks involve a possibility of having to cancel a response and thus promote the use of proactive inhibition. These findings support the idea of sensorimotor post-go Beta-Burst rates reflecting proactive inhibition.

Together, these studies demonstrate a relationship between sensorimotor post-go Beta-Burst rates and proactive inhibition. This relationship is further strengthened by the observation of differences in Beta-Burst rates both when comparing different types of trials, and when comparing conditions promoting different levels of proactive inhibition. The two types of blocks used by Soh and colleagues (2022) differed in the global (task-wide) probability of a stop-signal, differentially promoting the use of proactive inhibition. However,

the local performance context, that is the preceding trial sequences, have also been shown to influence performance on inhibition tasks.

1.3. Recent Trial History

Varying global parameters of the SST such as the overall probability of a stop-signal, have been known to influence performance (Ramautar et al., 2006). Less research has investigated the effects of local performance context such as Sequence-Length, which is the number of consecutive go-trials prior to a stop.

In a study by Eichele and colleagues (2010) they observed that response times as well as the amplitude of the N2 ERP-component progressively changed across trials preceding an error. Based on an existing connectionist model of conflict adaptation, they hypothesized that response conflict induced by the stimulus, and indexed by the N2 ERP-component, could account for error-preceding brain activity. To test this, they analyzed single-trial scalp EEG data recorded while participants (N = 70) performed a variation of the Erikson Flanker Task.

They found that the N2-amplitude progressively decreased (i.e., became more negative) leading up to a trial where the participant made an error. The authors interpreted this as an effect of conflict adaptation. Successive trials of the same type (i.e., either compatible or incompatible trials), increases the adaptation to the specific task demands, thus increasing the probability of making errors when task demands change. Eichele and colleagues also observed that the participants' response times to the target stimulus progressively shortened in the five trials preceding a trial with an erroneous response, with an average decrease of 2.5 msec (+/- 0.56 msec) per preceding trial.

Eichele and colleagues (2010) demonstrated the effect of preceding Sequence-Length on the N2 ERP-component and reaction times to the target stimulus in a modified Erikson Flanker Task. Participants sped up their responses in the trials leading up to an error,

demonstrating pre-ES. Modulations of response times to a target stimulus have previously been proposed as a behavioral marker of proactive inhibition. A speeding up of reaction times can be interpreted as a decreased recruitment of proactive inhibition, increasing the probability of making an error.

Eichele and colleagues (2010) found an effect of Sequence-Length on proactive inhibition when investigating data collected from participants performing a variation of the Eriksen Flanker task. In this task, participants must respond in accordance with a target stimulus while inhibiting the influence of distractor stimuli, all presented at the same time. In the SST, however, participants must cancel an already initiated motor response, possibly involving different inhibitory processes than those in the Eriksen Flanker task. These differences might involve different inhibitory processes making it difficult to directly compare data from the two tasks. However, some studies have investigated the effect of Sequence-Length on SST performance (Chang et al., 2017; Hu et al., 2015).

Hu and colleagues (2015) found evidence for an effect of Sequence-Length on estimated probability of a stop-trial called p(stop), and thus GoRT slowing. They applied a dynamic Bayesian model (Yu & Cohen, 2008) to a large fMRI SST data set with the goal of relating control processes with behavioral adjustments by finding the neural networks related with both. Based on the preceding trial history, they could estimate each participant's p(stop) on each trial and investigate how behavioral and neural activity differed based on variations in p(stop). The dynamic Bayesian model assumes that participants update their estimation of the p(stop) after each trial using Bayesian inference based on the current p(stop) and the trial type of the previous trial.

They found that on a trial-to-trial basis, higher probability of stop trials were associated with longer GoRT, indicating that participants slowed down their responses when they estimated the probability of a stop-trial to be higher. Hu and colleagues found that activity in

the anterior pre-supplementary motor area (pre-SMA) was associated with an increase in p(stop). In addition, when participants slowed down their GoRT, activity in the anterior pre-SMA was correlated with activity in the posterior pre-SMA and bilateral anterior insula. This can indicate that the involvement of posterior pre-SMA and bilateral anterior insula is related to the recruitment and implementation of proactive inhibition.

Hu and colleagues showed that the recent trial history influenced proactive inhibition via p(stop) on a trial-by-trial basis, evidenced by slowed down response times on go-trials when p(stop) increased.

1.4. Aim of Study & Hypotheses

The aim of the current study was to investigate the effect of Sequence-Length on GoRT and sensorimotor post-go Beta-Burst Rates in the stop-signal task. Previous studies have shown that participants adjust their response to the go-signal with the purpose of optimizing task performance. Such performance adjustments are a way of proactively increasing the probability of correctly inhibiting a response by increasing the time it takes for a response to be executed.

Single-trial sensorimotor Beta-Burst rates extracted from the post-go time-window have been proposed as a neural marker of proactive inhibition (Soh et al., 2021; Wessel, 2020). However, due to Beta-Burst rates being a relatively novel approach, the relationship between Beta-Bursts and different aspect of response inhibition have yet to be firmly established.

Some studies have shown that Sequence-Length, i.e., the number of consecutive go-trials prior to a stop-trials, affects inhibition. However, there exists only a limited amount of research that has investigated this relationship using the stop-signal task.

In our study, we investigated SST performance on stop-trials preceded by sequences of one to five go-trials. Based on previous literature, we expected longer sequences to be associated with less proactive inhibition. With the premise of sensorimotor post-go Beta-Burst rates reflecting proactive inhibition, we expected to see a similar effect of recent trial history on both GoRT and Beta-Burst rates.

Based on the abovementioned rationale, the following four hypotheses have been formulated:

1. GoRT in go-trials immediately preceding unsuccessful stop-trials will be shorter than GoRT in go-trials immediately preceding successful stop-trials.
2. The number of consecutive go-trials preceding a stop-trial will be negatively correlated with GoRT on go-trials immediately preceding stop-trials.
3. There will be a lower sensorimotor post-go Beta-bursts rates in go-trials immediately preceding unsuccessful stop-trials compared to in go-trials immediately preceding successful stop-trials.
4. Sensorimotor post-go Beta-Burst rates in go-trials immediately preceding stop-trials will be negatively correlated with the number of consecutive go-trials preceding a stop-trial.

2. Methods

2.1. Participants

Thirty-four right-handed participants (n = 34, 18 females) participated in our study. Participants were recruited through social media and posters at the University of Oslo (UiO). All participants reported having normal or corrected-to-normal vision, no previous(?) neurological- or eye-surgery, and no history of psychiatric or neurodevelopmental disorders (e.g. ADHD, migraines). On the day of participation, participants also reported having consumed no more caffeine than they usually do, and no alcohol in the past 24 hours. One participant was excluded due to missing data, and a second participant was excluded due to correctly responding on less than 90% of go-trials. Data from 32 participants, age 19 to 39 (M=24.81, SD=4.714) were included in the analyses.

All participants received information about the aim of the study, electrophysiological methods, and the SST. Before starting the experiment, each participant provided a written consent and received a gift card worth 300 NOK as compensation. The study was conducted in line with the Declaration of Helsinki and approved by the local ethics committee at the University of Oslo.

2.2. Design

The study was designed as a within-subject experiment using a bimanual stop-signal task with visual stimuli presented on a computer screen. Prior to starting the task, demographical data was collected by having each participant answer a short online questionnaire. All participants performed two consecutive sessions of the stop-signal task. The two sessions differed only in the approach used to determine the stop-signal delay, using

either the fixed or the tracked SSD approach. To avoid a differential influence of fatigue on the two conditions, the order between the conditions was counterbalanced across subjects.

The full experiment lasted for approximately 60 minutes with each session lasting for approximately 30 min. The participants were free to take a short break between the two sessions, but they had to remain seated during the break due to being connected to the EEG and EMG equipment. The effect of SSD-approach on the participants performance was beyond the scope of this thesis and will therefore not be analyzed or discussed further.

To investigate the effect of local trial history on performance adjustments in the stop-signal task, the number of consecutive go-trials prior to each stop-trial per block was predetermined. This was done to ensure that a sufficiently high number of each Sequence-Length was present in the data used in the analyses.

	nGo-trials prior to stop-trial	n cequences per block
Sequence 0	0	8
Sequence 1	1	6
Sequence 2	2	8
Sequence 3	3	8
Sequence 4	4	10
Sequence 5	5	10

The participants' response times and stop accuracy were recorded while they did the SST. In addition, electrophysiological measurements of brain activity and hand-muscle activity were recorded using EEG and EMG respectively. Due to practical concerns, the EMG data was not analyzed or discussed in this thesis.

2.3. Materials

2.3.1. Setup

The experiment was conducted using a Dell Precision T5500 computer (Dell, Inc., Texas, USA) running Windows 10. The stop-signal task was programmed in MATLAB v.R2021b scripts (The MathWorks, Inc., Massachusetts, USA) and presented using PsychToolBox-3 (version 3.06.16). The task was presented to the participants via a Eizo Flexscan S2411W monitor (Eizo, Inc.), with all participants seated approximately 70 cm from the screen. The participants' responses (button-press) were collected using a SuperLab RB-740 button box (Cedrus corporation, 2006).

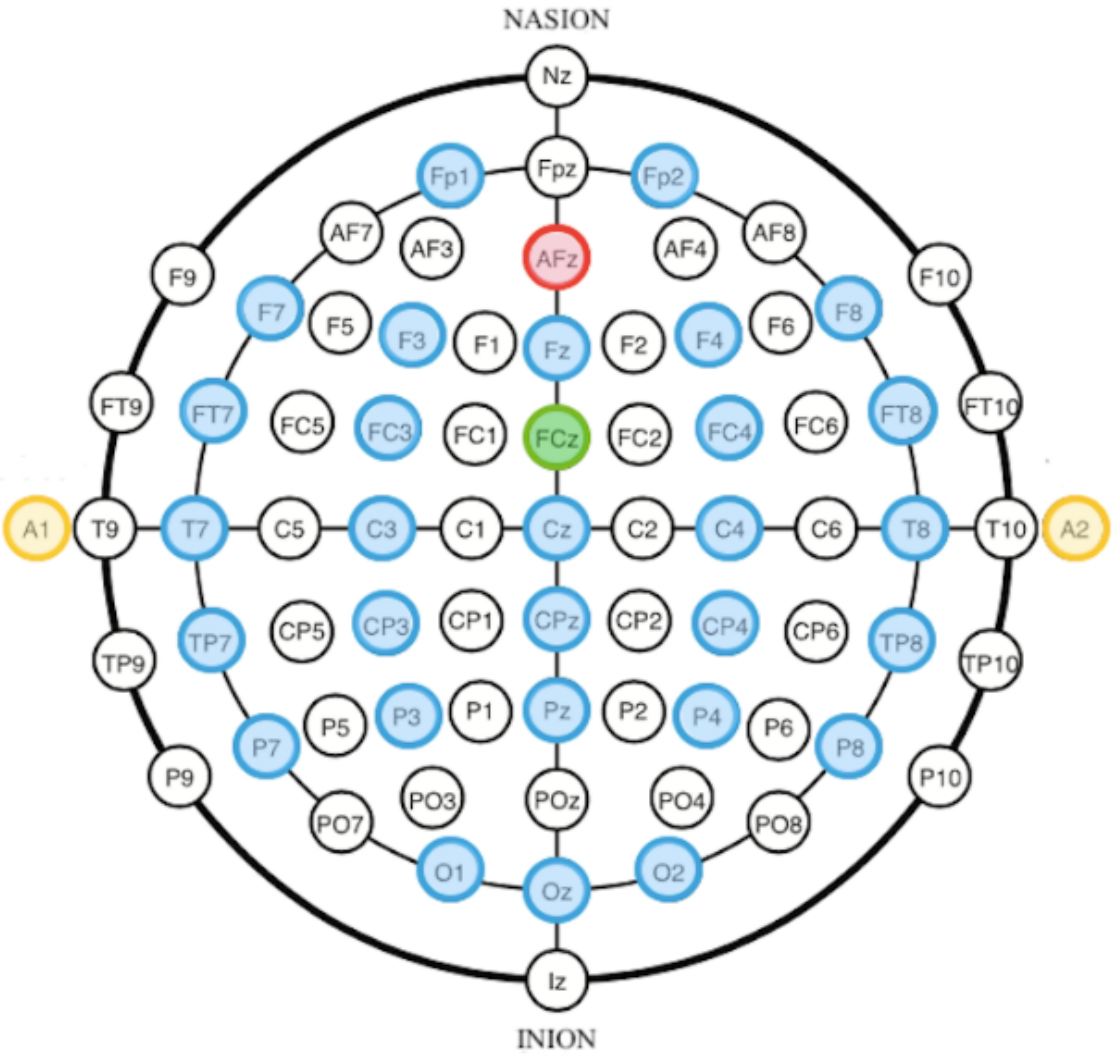
The timing-accuracy of the computer-based SST was tested using The Black Box Tool Kit v2. The timing of the presentation of visual stimuli on the computer screen was compared to the timing of the triggers in the EEG-data that marks the occurrence of experimental events. The test showed that the delay between trigger and presentation of visual stimuli was constant, indicating high timing-accuracy in the computer-based SST used in the current study.

2.3.2. EEG Data Acquisition

Electrophysiological measurements of brain activity were measured with EEG using the BrainAmp system (Brain products GmbH, Germany). Data was recorded using 29 passive Ag/AgCl electrodes placed in accordance with the 10/20 system. The EEG setup also included a ground-electrode (AFz), an online reference-electrode (FCz), and one electrode placed on each earlobe (A1 and A2) intended to be used as the offline reference.

Figure 1.

10-20 System of Electrode Placement



Note. Blue = recording electrodes. Green = online reference electrode. Red = ground electrode. Yellow = offline reference electrodes. Adapted from Luck (2014).

All EEG data was recorded with an online sampling rate of 5000 Hz using the BrainVision software (Brainvision Recorder, Vers. 1.24.0001, Brain Products GmbH, Gilching, Germany) on a second computer. The data was recorded with an online low-pass filter of 250 Hz and an online high-pass filter of 0.015 Hz. The impedances between the electrodes and the scalp were kept below five KΩ.

2.3.3. Stop-signal Task

The task consisted of a total of eight experimental blocks split between two conditions that differed in the method used to determine the time intervals between the presentation of the go-signal and the stop-signal in stop-trials (i.e., SSD).

Each of the four experimental blocks consisted of 186 trials, with the exception of the first experimental block in each condition which consisted of 196 trials. To acquaint the participants with the rules of the task, the task started with 20 training trials with feedback after each trial before the experimental blocks started. The first experimental block of each condition started with ten consecutive go-trials that were later excluded before the analyses. Each condition contained a total of 744 trials (1108 go, 400 stop) with 24% of trials being stop-trials.

The "fixed" condition consisted of four blocks where the SSD in a specific stop trial was randomly extracted from a pool of five SSDs. This pool of SSDs was adjusted block-wise based on the median SSD from the previous block. The median SSD was determined by fitting the participants' stopping performance to the inhibition function (describing the relationship between SSD and stop-accuracy) to find the SSD where the participant would have had 50% stopping accuracy. The pool of five SSDs to be used in the next block was then updated to contain five SSDs diverging from the median SSD from the previous block by -100 msec, -50 msec, +50 msec, and +100 msec. For example, if the median SSD from the previous block was 300 msec, then the updated pool of SSDs would contain five SSDs at 200 msec, 250 msec, 300 msec, 350 msec, and 400 msec.

The "tracked" condition consisted of four blocks where the SSD in a specific stop-trial was determined by a tracking-algorithm which increased or decreased the SSD by one step-size (50 msec) depending on whether the last stop-trial was successful or unsuccessful, respectively. The first SSD in the first block was 200 msec, and the subsequent SSD would

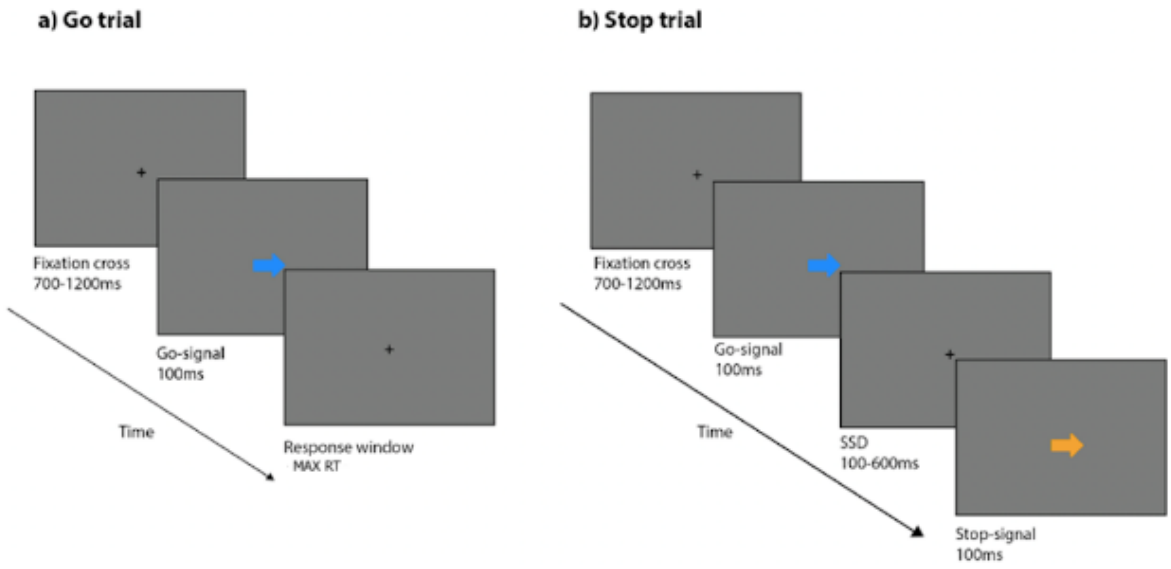
be either 150 msec if the participant failed to stop, or 250 msec if the participant correctly stopped.

In the fixed condition, we added a training-block at the start of the session to adjust the pool of SSDs based on the specific participant's performance. The goal was to make the participants successfully stop on approximately 50% of the stop-trials. To keep the number of blocks between the two conditions equal, a training-block was also added to the start of the tracked-condition. The participants' performance on the two training-blocks were not recorded and thus also excluded from the analyses. We were then left with four blocks in the fixed condition and four blocks in the tracked condition for the analyses.

At the start of each trial, a fixation cross was presented in the middle of the screen for a time interval varying between 700 msec to 1200 msec. The fixation cross was then replaced with an arrow (the go-signal) that was presented for 100 msec. In stop-trials, a second arrow (the stop-signal) was presented after the first arrow, with the SSD (min 200 msec, max 600 msec) separating the two arrows. The arrows were directed either to the left or to the right and all stop-signals were directed in the same direction as the preceding go-signal in that same stop-trial. The go- and stop-signals differed in terms of color. There could either be blue go-signals and orange stop-signals, or orange go-signals and orange stop-signals. To avoid confounding effects of stimulus color, we counterbalanced which of the two signal-color conditions the participants were exposed to. All arrows consisted of an arrowhead that was 3cm long and 4cm high, and an arrow base that was 3cm long and 2cm high.

Figure 2.

Schematic Description of the Go-trials and Stop-trials



Performance-dependent written feedback was presented on a screen after each block. If the go-accuracy in the previous block was below 4%, the participant received feedback telling them to "be more accurate". If the mean GoRT in the previous block was above 600 msec, the participant received feedback telling them to "be faster". If the go-accuracy and the mean GoRT was within the acceptable parameters, the participants received feedback saying "well done".

2.4. Data Processing

2.4.1. Behavioral data

Behavioral data was extracted from the participants' reaction times to the go-signal and based on their performance on go- and stop-trials.

Go-signal reaction time (GoRT) was calculated by averaging the time between the go-signal and the go-response in all correct go-trials. The probability of correctly responding on a go-trial (go-accuracy) was calculated by dividing the total number of go-trials from the number of correct go-trials, and then multiplying by 100. The probability of participants not

responding correctly on a go-trial (go-omission), including cases where they made an incorrect response, was calculated by dividing the number of correct go-trials with the total number of go-trials, and then multiplying by 100. The reaction time of responses to the go-signal made on unsuccessful stop-trials (UsRT) was calculated by averaging the time between the go-signal and the go-response on unsuccessful stop-trials specifically.

The stop-signal reaction time (SSRT) was calculated using the integration method with replacement of omissions (Verbruggen et al., 2019). The percentage of stop-trials where the participants correctly canceled their go-response was calculated by dividing the number of successful stop trials with the total number of stop-trials, and then multiplying by 100.

To assess the presence of pre-error speeding, we compared the GoRT on go-trials immediately preceding unsuccessful stop-trials (US-1) with the mean GoRT on go-trials immediately preceding successful stop-trials (SS-1).

2.4.2. EEG Pre-Processing

The raw EEG data was pre-processed using custom MATLAB v.R2021b scripts (The MathWorks, Inc., Massachusetts, USA) incorporating functions from the EEGLAB toolbox (v2022.0; Delorme & Makeig, 2004). First, for each participant individually, the data collected from each of the two conditions were merged into one dataset, containing all blocks and trials they performed. Next, the channel locations were specified, and data from the two EMG-channels were removed from the dataset. The data was then re-referenced from the online reference (Fcz) to offline reference, which was the average of the earlobes (A1 & A2). The data collected from the online reference Fcz was then added back to the data. A low-pass filter of 30 Hz was applied to the data, before the data was resampled to 500Hz, and high-pass filtered at 0.1 Hz. The triggers marking an event in the data were renamed to more meaningful names. The continuous EEG-data was then epoched based on go-events. An independent component analysis (ICA) was applied to the data, and eye- and muscle related

artifacts were individually removed for each participant after visual inspection of the components.

2.4.3. Time-Frequency Analysis and extraction of Beta-bursts

Detection and extraction of beta-bursts were done based on the descriptions in Wessel (2020) and Soh and colleagues (2022). First, the cleaned EEG data was transformed to a reference-free montage using the current source density method (CSD) (Perrin et al., 1989; Tenke & Kayser, 2005). This was done to enable extraction of sensorimotor beta-bursts from specific recording sites (C4 and C3) while reducing the risk of the signal being contaminated by other sites. The data from each electrode were then convolved with a complex Morlet wavelet for each of the 16 frequencies spanning the beta-band (15-30), before a baseline normalization (-200 to -100 msec) of the data was applied. The time-frequency beta-power estimates were calculated based on the squared magnitude of the complex wavelet-convolved data. Beta-Bursts were extracted from 0-200 msec after the go-signal. The specific time-window was based on previous findings in the literature (Wessel, 2020). Beta-Bursts were defined as an increase in the power of the signal equal to more than six times the median power. The Beta-Burst rate was extracted by counting the number of beta-bursts in the post-go time-window in all correct go-trials immediately preceding stop-trials. The post-go time-window was based on Wessel (2020) and defined as the time-window between 0-200 msec after the go-signal. Beta-Burst rates on each individual trial were grouped into separate variables based on the Sequence-Length of the specific trial (1-5).

2.5. Statistical Analyses

IBM SPSS Statistics for Mac, version 27.0, with a two-tailed alpha-level of .05, was used to perform all statistical analyses. Effect sizes were measured using Cohen's d for t-tests and partial eta squared (ηp2) for the repeated measures ANOVAs. For Cohen's d, an effect

size of 0.2 was interpreted as small, 0.5 as medium, and 0.8 as large (Cohen, 1992). For the partial eta squared, an effect size of 0.01was interpreted as small, 0.06 as medium, and 0.14 as large (Richardson, 2011).

2.5.1. Behavioral data

Means, standard deviations (SD) and confidence intervals (CI, 95%) were extracted for all behavioral data. Data from each participant was checked to see if they had to be excluded due to being outliers. The set of exclusion criteria in the current study were based on recommendations from Verbruggen and colleagues (2019). Participants that failed to correctly respond on more than 10% of go-trails were excluded from further analyses. Participants with stop-accuracies less than 25% or more than 75% were also excluded. In addition, any participant deviating by more than three standard deviations (SD) from the mean GoRT, SSRT or SSD, was also excluded from further analyses.

Across subjects, means were extracted for go-signal reaction times (GoRT), percentage of successful stop-trials (stopACC), stop-signal delays (SSD), stop-signal reaction times (SSRT) and percentage of go-trials with no or an incorrect response (go-omission).

To assess the presence of pre-Error-Speeding (pre-ES) a paired samples t-test was performed comparing the mean GoRT on go-trials immediately preceding unsuccessful stop trials (US-1) with the mean GoRT on go-trials immediately preceding successful stop trials (SS-1).

To investigate the effect of trial sequence length on GoRT, a two-way repeated measures ANOVA was performed with StopType (US/SS) and Sequence-Length (1-5) as independent variables, and GoRT on the last go-trial in each sequence as the dependent variable.

2.5.2. Beta-Burst Rates

To investigate whether sensorimotor post-go Beta-Burst rates differ prior to successful compared to unsuccessful stops, a paired samples t-test was performed comparing mean Beta-Burst rates on SS-1 to mean Beta-Burst rates on US-1.

To investigate the effect of trial sequence length on number of beta-bursts, a repeated measures ANOVA was performed with StopType (US/SS) and Sequence-Length (1-5) as independent variables, and mean Beta-Burst rates on the last go-trial in each sequence as the dependent variable.

3. Results

3.1. Behavioral Results

3.1.1. Descriptive Statistics

The mean response time in unsuccessful stop trials (USRT) was significantly shorter than mean GoRT ($t(31) = -12.737$, $p < .001$, $d = -2.252$). This was in accordance with the horse-race-model assumption of independent go and stop-processes. The stop-accuracy was 51,80 % (SD = 6%), meaning that participants successfully inhibited their response on approximately 50% of stop-trials.

Table 1:

Descriptive Statistics of Behavioral Measures (N = 32)

	M	SD	95% CI
goRT (msec)	523	88	492-553
usRT (msec)	462	65	439-484
SSRT (msec)	189	23	180-196
SSD (msec)	320	90	288-350
stopACC (%)	52	6	50-54

Note: goRT = go-signal reaction time, usRT = go-signal reaction time on unsuccessful stop-trials, SSRT = stop-signal reaction time, SSD = stop-signal delay, stopACC = percentage of all stop-trials that were successful stop-trials.

3.1.2. Presence of Pre-Error-Speeding

The paired samples t-test showed a significant difference in GoRT on US-1 (M = 499 msec, SD = 72 msec) compared to SS-1 (M = 537 msec, SD = 94.8) ($t(31) = -7.145$, $p < .001$, $d = -.790$). Participants responded faster to the go-signal on the trials preceding unsuccessful compared to successful stop-trials.

3.1.3. Trial Sequence Effects on GoRT

The repeated measures ANOVA investigating the effect of Sequence-Length and StopType on GoRT revealed a significant main effect of StopType ($F(1,32) = 51.056$, $p < .001$, $\eta p2 = .622$), but no significant main effect of Sequence-Length ($F(2.518,32) = 1.454$, $p = .237$, $\eta p2 = .045$) nor a significant interaction effect between StopType and Sequence-Length ($F(4,32) = .108$, $p = .979$, $\eta p2 = .003$).

3.2. Beta-Bursts Results

Table 2:

Descriptive Statistics of Beta-Burst rates prior to US/SS in different Sequence-Lengths (N = 32)

	M	SD	95% CI
burstrate_us1	3.998	0.700	3.761-4.236
burstrate_ss1	3.987	0.538	3.806-4.160
burstrate_us2	4.263	0.652	4.100-4.566
burstrate_ss2	4.125	0.274	3.878-4.376
burstrate_us3	4.043	0.766	3.781-4.303
burstrate_ss3	4.192	0.440	4.053-4.336
burstrate_us4	4.174	0.707	3.929-4.428
burstrate_ss4	4.112	0.5973	3.914-4.315
burstrate_us5	4.225	0.434	4.069-4.362
burstrate_ss5	4.151	0.631	3.946-3.398

*Note: burstrate_**us1** refers to Beta-Burst rate in go-trials immediately preceding unsuccessful stop-trials for all sequences containing only one go-trial before a stop-trial.*

3.2.1. US/SS Differences in Beta-Burst rates

The paired samples t-test showed no significant difference in mean Beta-Burst rates on US-1 compared to SS-1 ($t(31) = .594$, $p = .557$, $d = .105$). Meaning that there were no

significant differences in the Beta-Burst rates prior to a participant unsuccessfully compared to successfully stopping.

3.2.2. Trial Sequence Effects on Beta-Burst rates

The repeated measures ANOVA investigating the effect of Sequence-Length and StopType on Beta-Burst rates revealed no significant main effects of StopType ($F(1,32) = .353$, $p = .557$, $\eta p2 = .011$) or Sequence-Length ($F(1,32) = 1.623$, $p = .189$, $\eta p2 = .050$), as well as no significant interaction effect between StopType and Sequence-Length ($F(1,32) = .727$, $p = .575$, $\eta p2 = .023$).

4. Discussion

The aim of this study was to investigate the effect of Sequence-Length on behavioral and neural measures of proactive inhibition in the stop-signal task. The purpose was to explore how response times and bursts of activity in the beta frequency-band was influenced by the preceding trial-sequence. The analyses of the data showed that, on average, participants had faster response times on the go-trial immediately preceding an unsuccessful compared to successful stop trials, demonstrating pre-error-speeding. However, there was no significant effect of Sequence-Length, indicating that the number of preceding go-trials prior to a stop-trial does not influence response times. The current study also aimed to investigate the relationship between proactive inhibition and single-trial bursts of activity in the beta-frequency band (15-30 Hz) as measured by EEG. The novel approach of analyzing beta-frequencies in the EEG-signal, counting the number of bursts of activity on each trial, have been proposed to reflect inhibition of the motor system (Soh et al., 2021; Wessel, 2020). However, the current study did not find a significant difference in the mean number of Beta-Bursts recorded immediately preceding an unsuccessful compared to successful stop-trial. Additionally, there were no significant main effect of Sequence-Length nor a significant interaction effect between Sequence-Length and StopType (US/SS).

4.1. Association between Stopping Performance, Sequence-Length and GoRT

Across all participants, the mean GoRT on US-1 was shorter than the mean GoRT on SS-1. Meaning that participants responded more quickly on go-trials preceding a stop-trial where they failed to inhibit their response. This finding is in line with the first hypothesis of this study, which predicted shorter mean GoRT on US-1 compared to SS-1. This supports the existing literature describing the pre-ES effect (Eichele et al., 2010; Ramautar et al., 2006; Rawji et al., 2022; Steinhauser et al., 2012).

4.1.1. No Association Between Sequence-Length and GoRT

Sequence-Length had no significant effect on the GoRT on the last trial before a stop-trial. This does not support the second hypothesis of this study which predicted a negative correlation between GoRT and sequence-length.

A potential explanation for the absence of preceding sequence effects on GoRT in the current study could be that previous literature demonstrating the effect did not use the SST. Although these are all tasks that test inhibition, the way inhibition is operationalized differs between the tasks, potentially leading to different effects when being influenced by the same experimental manipulation. For example, Eichele and colleagues (2010) used a variation of the Eriksen Flanker task which requires the participants to respond to a target stimulus while simultaneously inhibiting the influence of incongruent peripheral ("flanker") stimuli. The Stop-Signal task, however, requires the participants to occasionally inhibit an already initiated motor response. Preceding sequence-length might have different effects on the process of selecting a motor action compared to the process of canceling a motor action. Differences in the tasks used and how they measure inhibition, might explain why Eichele and colleagues (2010) found an effect of sequence-length on GoRT. However, some studies have observed effects of trial-sequences on SST-performance (Chang et al., 2017; Hu et al., 2015).

Using the Stop-Signal task, Chang and colleagues (2017) and Hu and colleagues (2015) found that trial-by-trial variations in GoRT were associated with the estimated probability of a trial being a stop-trial (p(stop)). This estimate is based on a dynamic Bayesian model which assumes that participants continuously estimate p(stop), updating their estimates of p(stop) after each trial. They showed that on a trial-by-trial basis, higher estimates of p(stop) were associated with longer GoRT, which were interpreted as indicating a relationship between trial sequence and adjustments in GoRT.

A potential explanation for the different results in the current study compared to the work of Chang and colleagues (2017) and Hu and colleagues (2015), is in how trial sequences are analyzed in relation to GoRT. In the current study, go-trials immediately preceding a stop-trial (S-1) were grouped (1-5) based on the number of preceding go-trials. The mean GoRT were calculated for each group for each participant and analyzed. Hu and colleagues, however, do not investigate a direct relationship between sequence-length and adjustments in GoRT. In their study, preceding sequence length (1-3) was part of the calculation of p(stop), which they then relate to adjustments in GoRT. Thus, a potential explanation of the differing results could be that Hu and colleagues (2015) indirectly investigated the relationship between preceding Sequence-Length and GoRT, while the current study directly investigated how GoRT varied in different sequences.

4.2. Association between stopping performance, Sequence-Length and Beta-Bursts

4.2.1. No Difference in Beta-Burst rates on US-1 compared to SS-1

The results showed no significant difference in Beta-Burst rates on US-1 compared to SS-1. This does not support the third hypothesis of the current study, which predicted that there would be higher Beta-Burst rates in SS-1 compared to US-1. In addition, the repeated measures ANOVA revealed no significant main effect of Sequence-Length, nor a significant interaction effect between StopType and Sequence-Length.

4.2.2. No Effect of Sequence-length on mean Number of Beta-Bursts

The current study found no effect of Sequence-Length on the Beta-Burst rates recorded on a go-trial immediately preceding a stop-trial. The repeated measures ANOVA revealed no significant main effect of Sequence-Length, nor a significant interaction effect between StopType Sequence-Length. This does not support the fourth hypothesis of the current study which predicted a negative correlation between Beta-Burst rates and Sequence-Length.

The relationship between Sequence-Length and Beta-Burst rates has not been investigated previously in the literature. This makes direct predictions about the relationship non-feasible. However, Wessel (2020) and Soh and colleagues (2022) observed a relationship between Beta-Burst rates and proactive adjustments in GoRT. Wessel specifically proposed that sensorimotor post-go Beta-Bursts reflect an inhibited motor system. Based on this background, we aimed to explore the relationship between Beta-Burst rates and adjustments in GoRT by observing how they both varied under the same experimental manipulation.

4.3. No Support for Post-go Beta-Burst Rates reflecting Proactive Inhibition

One of the aims of the current study was to explore the relationship between proactive performance adjustments and sensorimotor post-go Beta-Burst rates. First, if Beta-Burst rates truly reflect inhibition of the motor system, they would be expected to differ prior to unsuccessful compared to successful stop-trials. However, the current study found no significant differences in the mean number of Beta-Bursts prior to US compared to SS. The absence of a relationship between Beta-Burst rates and stopping performance, does not support the view of Beta-Burst rates reflecting proactive inhibition, contrary to what was proposed by Wessel (2020) and Soh and colleagues (2022).

4.3.1. Beta-Burst and Pre-Error-Speeding might relate to Inhibition of the Motor System

Both adjustments in GoRT (Rawji et al., 2022) and sensorimotor post-go Beta-Burst rates (Soh et al., 2021; Wessel, 2020) have been proposed to reflect proactive inhibition of the motor system. Wessel (2020) proposed that post-go Beta-Bursts reflect inhibition of the motor system. He based this on the fact that the beta-bursts were measured in the time interval between the go-signal and the go-response, meaning that the process that Beta-Bursts reflect should take place in this time-window. Wessel also observed that Beta-Burst rates predicted stopping performance, meaning that the process Beta-Bursts reflect should be

involved in the execution or cancellation of a motor response. The proposal of Beta-Bursts reflecting inhibition of the motor system fits Wessel's observations, as well as potentially relating Beta-Bursts to proactive inhibition.

Rawji and colleagues (2022) observed that conditions promoting the use of proactive inhibition were associated with a delayed rise in corticospinal excitability (CSE) leading up to the execution of a motor action. They also observed that the rate of increase in CSE was not different based on whether the task promoted the use of proactive inhibition. Based on these observations, Rawji and colleagues (2022) proposed that proactive inhibition acts on the motor system by delaying the start of the rise in CSE rather than slowing down the rate of increase in CSE. In the context of the horse-race model, proactive inhibition could then influence when the go-process is initiated, increasing the time it takes for the go-process to initiate the go-response, thus increasing the probability of the stop-process winning the race.

If adjustments in GoRT and Beta-Burst rates both reflect proactive inhibition of the motor system, it is possible to indirectly investigate the relationship between Beta-Bursts and adjustments in GoRT by observing how they both vary in different conditions thought to promote varying degrees of proactive inhibition. In the current study, variations in GoRT and Beta-Burst rates were investigated in different sequences of go-trials (1-5 consecutive go-trials). We expected longer trial-sequences to be associated with shorter GoRT and lower Beta-Burst rates. However, the analyses revealed no significant effect of Sequence-Length, neither on GoRT nor on Beta-Burst rates, giving little information about the potential relationship between the two.

The findings of the current study contradict findings from the literature (Hannah et al., 2020; Jana et al., 2020; Soh et al., 2021; Wessel, 2020) showing differences in Beta-Burst rates prior to US compared to SS. However, other studies (Enz et al., 2021; Errington et al., 2020) failed to find an association between Beta-Burst rates and response inhibition. This has

resulted in some researchers questioning what it is that produces these sudden increases of power in the EEG-signal (Errington et al., 2020), while others investigate whether measures of Beta-Bursts other than Beta-Burst *rate* can more reliably predict stopping performance (Enz et al., 2021).

4.3.2. Can Beta-Bursts Qualify as a Marker of Inhibition?

The relationship between Beta Bursts and inhibition can be further explored by investigating whether beta-bursts can qualify as a neural marker of inhibition. Huster and colleagues (2020) conducted a meta-analysis, investigating whether the P3 ERP could act as a neural marker of inhibition. They pointed out that there were no formal set of criteria established that described what qualifies as a direct neural marker of inhibition. However, the authors noted some criteria that seemed relevant in their review of the literature.

The first criterion is that the neural marker should show stronger activity in conditions of higher inhibitory load. Soh and colleagues (2022) found that Beta-Bursts were more likely to occur in regular SST-blocks compared to pure-go-trial-blocks, supporting the role of Beta-Bursts as a neural marker of inhibition. The second criterion is that the neural marker should be temporally delayed in unsuccessful compared to successful stop. Hannah and colleagues (2020) showed that the timing of frontal post-stop Beta-Bursts were positively correlated with EMG-measures of stopping latency, indicating that trials with delayed Beta-Bursts were more likely to be unsuccessful stop-trials. The third criterion is that the neural marker should correlate with SSRT which is the current gold-standard of measuring response inhibition. Wessel (2020) found that sensorimotor post-go Beta-Burst rates negatively correlated with SSRT. However, Huster and colleagues (2020) pointed out that SSRT is an indirect measure of response inhibition computed based on GoRT and SSD. This means that a correlation between a neural marker and SSRT could be partially explained by unknown third variables rather than by inhibition.

In sum, some evidence exists supporting the role of Beta-Bursts as a direct neural marker of inhibition. However, due to the conflicting results in the literature, as well as the results of the current study, it is difficult to support the view of Beta-Bursts as directly reflecting inhibition.

4.4. Limitations of the Current Study, and Future Directions

The current study only investigated potential effects of sequence-length apparent on the group-level, not accounting for individual differences in GoRT or Beta-Burst rates. It might be that the Sequence-Length differentially influenced participants and their task performance. Future research could investigate the effect of Sequence-Length on Beta-Burst rates, while accounting for individual differences.

The design of the current study did not include control-conditions. Future research can include a no-sequences control condition when investigating the effect of Sequence-Length. Alternatively, pure-go blocks that can act as a contrast to conditions requiring response inhibition could be included. This has been done previously in the literature (Rawji et al., 2022; Soh et al., 2021).

Future research should continue the search for standardized neural and behavioral markers of inhibition. When it comes to Beta-Bursts, researchers can explore how different Beta-Burst parameters influence the results. Researchers can also explore how Beta-Bursts stand up to the general requirements of neuroscientific measures by for example investigating the reliability of Beta-Bursts.

4.5. Implications

To the best of our knowledge, the current study is the first to investigate the effect of preceding Sequence-Length on Beta-Burst rates. We aimed to investigate whether Beta-Burst

rates can act as a neural marker of response inhibition. Impairments in the ability to inhibit a response have been associated with several disorders such as ADHD (Wodka et al., 2007) and schizophrenia (Enticott et al., 2008). By identifying a reliable neural marker of inhibition, it could be possible to target this marker with interventions such as neurofeedback in hopes of treating the inhibition-related symptoms. The current study aimed to contribute in this regard, by investigating the proposed neural marker of inhibition that is Beta-Burst rates.

4.6. Concluding Remarks

In the current study, we observed that participants responded faster to the go-signal in the go-trial immediately preceding unsuccessful compared to successful stop-trials, demonstrating pre-error-speeding. The sequence of preceding trials, however, was not related to changes in response times. The current study also aimed to shed light on the relationship between Beta-Burst rates and proactive inhibition by investigating the relationship between Beta-Burst rates and a behavioral measure of proactive inhibition, pre-error-speeding. The results showed that Beta-Burst rates were not significantly different prior to unsuccessful compared to successful stop-trials. In addition, Beta-Burst rates did not significantly differ based on the preceding Sequence-Length. In sum, the current study did not find evidence indicating a relationship between sensorimotor post-go Beta-Burst rates and response inhibition.

5. References

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