

Leveling the Field for a Fairer Race between Going and Stopping: Neural Evidence for the Race Model of Motor Inhibition from a New Version of the Stop Signal Task

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Abstract

■ The stop signal task (SST) is the gold standard experimental model of inhibitory control. However, neither SST condition-contrast (stop vs. go, successful vs. failed stop) purely operationalizes inhibition. Because stop trials include a second, infrequent signal, the stop versus go contrast confounds inhibition with attentional and stimulus processing demands. While this confound is controlled for in the successful versus failed stop contrast, the go processes is systematically faster on failed stop trials, contaminating the contrast with a different noninhibitory confound. Here, we present an SST variant to address both confounds and evaluate putative neural indices of inhibition with these influences removed. In our variant, stop signals occurred on every trial, equating the noninhibitory demands of the stop versus go contrast. To entice participants to respond despite the impending stop signals, responses produced before

stop signals were rewarded. This also reversed the go process bias that typically affects the successful versus failed stop contrast. We recorded scalp electroencephalography in this new version of the task (as well as a standard version of the SST with infrequent stop signal) and found that, even under these conditions, the properties of the frontocentral stop signal P3 ERP remained consistent with the race model. Specifically, in both tasks, the amplitude of the P3 was increased on stop versus go trials. Moreover, the onset of this P3 occurred earlier for successful compared with failed stop trials in both tasks, consistent with the proposal of the race model that an earlier start of the inhibition process will increase stopping success. Therefore, the frontocentral stop signal P3 represents a neural process whose properties are in line with the predictions of the race model of motor inhibition, even when the SST's confounds are controlled. ■

INTRODUCTION

Motor inhibition is a process that enables humans to rapidly stop an already initiated action. This critical function allows humans to adapt ongoing behavior to an unpredictable environment. The stop signal task (SST) is regarded as the gold standard experimental of motor inhibition in the laboratory (Logan & Cowan, 1984). On each trial, participants initiate a response to a first signal (the go signal). A subset of trials then includes a second signal (the stop signal) that is presented with some delay after the go signal, prompting participants to attempt to cancel their impending response.

One appealing feature of the SST is that stopping behavior can be appropriately described by process models that conceptualize action stopping as a race between the go process (triggered by the go signal) and the stop process (triggered by the stop signal; Boucher, Palmeri, Logan, & Schall, 2007; Logan, Cowan, & Davis, 1984). A key proposition of these race models is that an earlier

onset of the stop process will lead to more successful stopping, as an earlier onset makes it more likely for the stop process to complete before the go process.

Such race models also allow the calculation of stop signal RT (SSRT), which is used to estimate the duration of the latent stopping process. SSRT is often the primary variable of interest in studies of motor inhibition (for a review, see Verbruggen & Logan, 2009). For example, clinicians use SSRT to quantify inhibitory deficits in disorders like attention-deficit/hyperactivity disorder (e.g., Nigg, 1999; Oosterlaan, Logan, & Sergeant, 1998) and Parkinson's disease (Obeso et al., 2011). The SST has also proven useful in studies of general behavioral/impulse control, including studies of excessive eating (Nederkorn, Houben, Hofmann, Roefs, & Jansen, 2010) and alcohol consumption (Houben, Nederkorn, Wiers, & Jansen, 2011).

Despite widespread use, the SST is plagued by a key design problem that hampers its use as a pure measurement of motor inhibition, particularly at the neural level. Per the standard design, stop signals occur only on a subset of trials (typically 1/4 or 1/3). This ensures that a motor response is initiated on most trials, including stop trials, which is a prerequisite to engaging inhibitory

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control (Wessel, 2018). However, the fact that stop signals are infrequent also implies that the success of action stopping does not purely depend on inhibitory control. Indeed, the detection of infrequent, salient events, such as stop signals, involves an additional set of psychological and neural processes that are independent of inhibition. Such processes, for example, stimulus perception or attentional orienting, can account for performance differences in the SST because their fidelity and timing affect the speed and accuracy of stopping. This issue is especially important because many of the populations in which the SST has been used to identify purported inhibitory deficits also show marked deficits in processes related to signal detection or attention (Huster, Enriquez-Geppert, Lavalée, Falkenstein, & Herrmann, 2013; Kanai & Rees, 2011; Drew & Vogel, 2008; Martens, Munneke, Smid, & Johnson, 2006; Bekker, Kenemans, Hoeksma, Talsma, & Verbaten, 2005; Kenemans et al., 2005; Ratcliff, Thapar, & McKoon, 2001). At its most severe interpretation, this could mean that many findings of impaired cognitive control in such populations could potentially be accounted for by attentional deficits and may not reflect impaired inhibitory control at all. In addition to such population-based studies on inhibitory deficits, the confound between inhibitory and noninhibitory processes in the SST is also problematic for studies of the neural indices of the motor inhibition process (for reviews, cf. Aron, Robbins, & Poldrack, 2014; Ridderinkhof, Forstmann, Wylie, Burle, & van den Wildenberg, 2011). Indeed, recent debates in the neuroscientific literature focus on the fact that differences in brain activation between stop and go trials could be attributable to noninhibitory processes, rather than inhibitory control (Waller, Hazeltine, & Wessel, 2019; Erika-Florence, Leech, & Hampshire, 2014; Hampshire, Chamberlain, Monti, Duncan, & Owen, 2010).

Consequently, recent studies have attempted to illustrate that action stopping in the SST consists of a cascade of psychological processes, with motor inhibition being only the final subprocess in a chain of processes that also involves stimulus perception and attentional detection—all of which contribute to SSRT (Matzke, Hughes, Badcock, Michie, & Heathcote, 2017; Verbruggen, Stevens, & Chambers, 2014; Verbruggen, Aron, Stevens, & Chambers, 2010). Some researchers have used Bayesian modeling to disentangle attentional processes from “pure” motor inhibition (e.g., Matzke, Dolan, Logan, Brown, & Wagenmakers, 2013). However, even if post hoc methods can disentangle the contributions of inhibitory and noninhibitory processes in the SST, such approaches will not address the fact that the infrequent nature of stop signals will invariably affect behavior. Studies have shown that when stop signals are infrequent, participants’ expectation of stop signals varies from trial to trial, leading to differential proactive recruitment of both inhibitory and noninhibitory processes depending on that expectation (Ramautar, Kok, & Ridderinkhof, 2006; Vink et al., 2005). This further complicates these comparisons in the SST.

The comparison of successful and failed stop trials (instead of stop vs. go trials) circumvents this confound, because both conditions contain the infrequent stop signal. However, this contrast suffers from a different flaw. Failed stop trials predominately represent the fast part of the go RT distribution, that is, trials on which the go process started earlier (Logan et al., 1984), whereas successful stop trials represent the slow part of the go RT distribution. Thus, the go process differs significantly between the two conditions in this comparison. Hence, any condition difference between successful versus failed stop trials (such as differences in brain activity) could theoretically be attributed to go process differences, rather than differences in motor inhibition: In other words, successful stop trials may only be successful because of the (relatively) slower go process and may not differ in inhibitory control activity at all.

In this study, we address these two concerns and evaluate a neural index of inhibition in this “cleaner” comparison. We do not intend to create a superior version of the SST but rather to check the validity of an electrophysiological marker of inhibition without some confounds of the original task. The goal was to create task demands that encourage participants to respond as quickly as possible while expecting the stop signal on every trial. This equates the stimulus processing demands of the stop versus go comparison. In other words, we matched the stimulus layout of successful stop trials and go trials. In the standard SST, stimulus processing requirements differ, depending on the presence of a stop signal (i.e., the stop signal represents an additional stimulus that must be detected and attended to before inhibition can begin while this requirement is absent in go trials). To eliminate this issue, we introduce a task that includes the same number of signals on every trial. Thus, stimulus processing related to the stop signal is equated in this cascade of events for each trial type.

To ensure that participants initiate a response on most trials despite the guaranteed stop signal, we differentially rewarded stopping and going by instructing participants that they could gain higher reward by making a go response before the stop signal. Stop signal delays (SSDs) varied from trial to trial and could be as long as 900 msec, making it possible for participants to realistically beat the stop signal on many trials. These instructions reliably produced both successful (no response made) and failed (response made after the stop signal) stop trials. Importantly, this incentive structure also reversed the usual bias toward faster go RTs in failed stop trials. We acknowledge that there is likely no “perfect” task to compare go processes when stop signals are present to when they are absent, yet we believe our new SST offers a cleaner comparison. The new task differs from the classic version not only in that stop signals occur on every trial but also in the distribution of SSDs. In the classic task, SSDs are typically restricted in range based on performance for each participant. Critically, this contrasts with a new variant in which SSD range is much wider, from 50 to 900 msec. As a consequence, the stop signal may appear at a time that the

response cannot be stopped. In this way, within-subject variation in the duration of the go process has little opportunity to influence successful versus failed stopping. This differs from the classic task, where an adaptive SSD procedure makes stopping and failing equally likely. In other words, this case maximizes the role of the go process duration in determining successful versus failed stops.

In this way, any differences in successful versus failed comparisons can no longer be solely explained by a faster go process on failed stop trials and can instead be attributed to true differences in inhibitory control. Because the stop signal is expected on every trial, there is no reason to delay the response to determine whether the stop signal will occur. Thus, RTs during the task should approach those observed on blocks performed without stop signals, and failed stop RTs should no longer be shorter than those observed during go trials.

We measured EEG from healthy humans while they performed this variant of the SST, as well as a standard SST with an adaptive SSD and infrequent stop signals. We aimed to investigate the properties of frontocentral P3 ERP following the stop signal using the new paradigm. For this study, we consider an independent component (IC) that is elicited by the stop signal in the classic SST and indexes the latency of the inhibition process in that task (e.g., Wessel & Aron, 2015; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004). This signal is therefore named the frontocentral stop signal P3. This component is topographically similar to the frontocentral P3a after unexpected events (e.g., Polich, 2007; Courchesne, Hillyard, & Galambos, 1975). Indeed, we have argued before that the P3(a) after such unexpected events may indeed reflect the activity of an inhibitory process (Waller et al., 2019; Wessel & Huber, 2019; Wessel & Aron, 2013). However, we would caution the reader against “reverse inference” interpretations that take the presence of a frontocentral P3(a) after a specific event as automatic evidence for the presence of an inhibitory process. Frontocentral P3-type waveforms have been observed after many different psychological events across several ERP paradigms (e.g., Barcelo, Escera, Corral, & Periáñez, 2006; Nieuwenhuis, Aston-Jones, & Cohen, 2005; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). However, inferences about potential common processes underlying ERPs in different task contexts can only be made under very narrow circumstances (e.g., when their neural generators cannot be disentangled and/or the amplitude of the ERP relates to behavior in both task contexts in a way that is consistent with commonalities in processing). Therefore, our present observations deal with the specific frontocentral P3 waveform elicited by signals to stop an already initiated action.

This ERP has been proposed as a marker of motor inhibition, because the timing of its onset conforms to a core prediction from the race model in the standard SST: On stop trials in which the frontocentral P3 occurs with faster latency after the stop signals, successful stopping is more likely (Wessel & Aron, 2015; Kok et al., 2004). However, that

interpretation hinges on the same confounds as all other studies using the standard SST (laid out above).

To determine whether the timing P3 conforms to the race model even when these confounds are eliminated, we tested two core hypotheses. First, we investigated whether its amplitude is increased in the stop versus go contrast (just as in the standard SST), even when the stop signal is no longer infrequent. This would show that the stop signal related increase in P3 amplitude increase cannot be attributed to the infrequency of the stop signal. Second, we tested whether the onset of this ERP retains the same difference between successful and failed stop trials, even when failed stop trials are no longer faster than go trials produced before a stop signal. If the onset of the stop signal P3 shows an earlier onset on successful versus failed stop trials (just like in the standard SST), it would firmly establish this neural index as a marker of the inhibitory control process in the brain.

METHODS

Participants

Data were collected from 35 healthy adult undergraduate (mean age = 19.61 years, 19 female) at the University of Iowa. Students volunteered through the Department of Psychological and Brain Science research participant pool to fulfill a course requirement. The University of Iowa Institutional Review Board approved all study procedures.

Exclusions

Participants were excluded from analysis if their behavioral performance in the newly designed task met either of the following criteria:

- fewer than 20% successful stops at SSDs of 200 msec or less and
- more than 70% successful stops at SSDs of 300 msec or longer.

Hence, participants were excluded if they were unable to inhibit their responses even at very short SSDs (suggesting that they did not effectively use inhibitory control), or if they seldom responded even at very long SSDs (suggesting the absence of a prepotent motor response). These criteria ensured that comparisons made in our analyses of stop trials indexed inhibition, not a choice on the participant's part to not initiate a response. Such exclusions were determined a priori by a pilot study and are common practice in analyses using the classic SST when participants fail to follow task instructions (Verbruggen et al., 2019). Four participants were excluded because they met at least one of these criteria.

Materials

Experimental data were collected using an IBM-compatible desktop computer running Fedora Linux and MATLAB R2014a (The MathWorks) with PsychToolbox Version 3

(Brainard, 1997) used to display stimulus material and collect responses.

Procedure

Participants first performed a baseline go RT task to determine their RTs when no inhibitory control demand was present. They next performed the novel SST variant designed for this study, followed by a standard version of the SST.

Experimental Paradigms

Baseline Go RT Task

Trials began with a 500-msec fixation cross in the center of the screen. Immediately following this fixation, a left- or right-facing arrow indicating the appropriate responses was presented, which remained on the screen for 100 msec. Responses were made using the p or q keys on the computer keyboard. Following the response, an intertrial interval comprised the balance of 3000 msec for the trial. Participants completed one block of 50 trials for this task.

Standard SST (SST33)

A fixation cross appeared for 500 msec followed by a go stimulus. A black arrow pointed left or right, and participants were instructed to press the key corresponding to the direction of the arrow (q for left, p for right). On 33% of trials, an additional signal was presented; the arrow turned red after a delay. This SSD was adjusted based on performance. Following a successful inhibition, SSD was increased by 50 msec (making it more difficult to stop successfully). Following a failed inhibition, SSD was decreased by 50 msec (making it easier to stop successfully). Through this adaptive procedure, participants should complete the experiment having stopped successfully on 50% of stop trials with the remaining 50% failed stops. An intertrial interval followed the stop signal, and participants saw a blank screen until the balance of 3000 msec had elapsed for the trial. Five blocks of 60 trials were completed. Stop signals were presented on 33% of trials; thus, each block contained 20 stop trials and 40 go trials. Participants were instructed that responding quickly and withholding the responses following a stop signal were equally important.

Novel SST (SST100)

In this variant, a stop signal was presented on every trial. As in the SST33, a fixation cross was presented for 500 msec. A left- or right-pointing arrow followed and was displayed for 100 msec (go signal). The direction of this arrow indicated the appropriate response (q for left and p for right). On every trial, an auditory stop signal (a 600-Hz sine wave tone) was presented after a variable SSD (uniform distribution of values ranging from 50 to 900 msec in 50-msec increments). The tone played for 100 msec, followed by feedback for the remainder of the trial (until

a fixed trial duration of 3000 msec was reached). Feedback consisted of points earned or lost on the trial, with gains displayed in green and losses displayed in red. Participants completed eight blocks of trials, each containing three trials of each SSD. Thus, each block consisted of 54 trials, and the total experiment comprised 432 trials (24 trials per SSD). Two samples of this task were collected. In 14 participants, the stop signal was not displayed when participants successfully responded before it was slated to occur (go trials). In the other 15 participants, stop signals were presented even if the participants responded before it appeared. We present Samples 1 and 2 together in combined plots because the inhibitory P3 in the SST100 task did not differ between samples on either stop or go trials (Figure 1).

Because participants knew that they could complete a trial successfully by waiting for stop signal presentation without ever initiating a response, we instituted a point system to incentivize fast responding despite the ubiquitous stop signal. These points were arbitrary with no real-world value, which participants knew. Two hundred points were given for responding before the stop signal was presented (“beating” the stop signal). One hundred points were given for successfully inhibiting a response after the stop signal. Participants were penalized 100 points for failing to stop after the stop signal or pressing the incorrect key. At the end of each block, participants saw their cumulative scores.

Behavioral Analysis

For the SST33 task, go trials are those with no stop signal present. Failed stop trials are those with both a stop

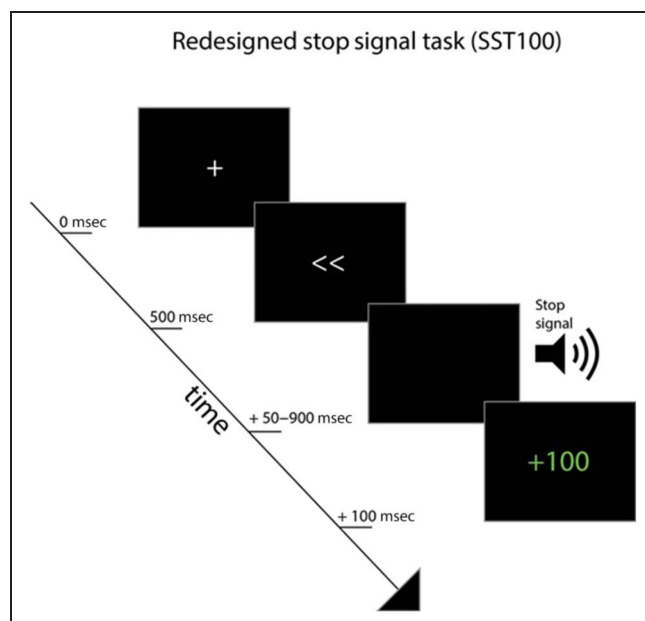


Figure 1. SST100 task diagram. The feedback depicted (+100) reflects points awarded for a successful stop trial.

signal and a response. Successful stop trials are those with a stop signal but no response. For the SST100 task, go trials are those where a response is made before the stop signal is presented. Failed stop trials are those where the response is made after the stop signal. Successful stop trials are those for which no response is made.

For the SST33, mean stopping latency (SSRT) was computed via the integration method. We also computed cumulative distribution functions from behavioral data of each task. For each RT from 0 to 1000 msec, we calculated the proportion of responses given by that time point. Each of these points was fit to a curve using logistic regression and averaged across participants for the purposes of illustration of the RT distribution for both SSTs.

We also explored sequential effects in the behavioral data. For these sequential analyses, all error trials were discarded. We counted totals of each remaining trial type (correct go, successful stop, failed stop) as a function of previous trial type and along varying SSDs. We then transformed these data to proportions of total trials for plotting. To maintain power in this exploratory analysis while adding an additional factor (previous trial type), we collapsed SSDs into 150 msec bins, which yielded similar trial counts as our other behavioral analyses.

EEG Recording

EEG data were recorded using a BrainProducts system with 62 scalp electrodes conforming to the international extended 10/10 system. Two additional electrodes placed below the left eye and on the outer canthus of the left eye recorded blinks and horizontal saccades, respectively. The ground was placed at electrode Fz, and the reference was placed at electrode Pz. EEG was digitized at a sampling rate of 500 Hz.

EEG Preprocessing

Data were preprocessed using custom scripts in MATLAB R2015a (The MathWorks) and the EEGLab toolbox (Version 13.6.5b). All data and analysis scripts used to create results presented in this article may be found on the open science framework (URL to be added after acceptance of manuscript). After import to MATLAB, data were filtered using cutoff criteria of .5 Hz (high pass) and 50 Hz (low pass). The continuous EEG was then divided into 1-sec epochs and visually inspected for bad channels and nonstereotyped artifacts. Examples of these artifacts are gross motor activity, atypical blinks, or saccades. Epochs containing these artifacts were removed for the data set, and the retained intervals were re-referenced to a common average.

Lateralized Readiness Potentials

Epochs were created from -100 to 700 msec around the go stimulus of the SST33 and SST100 tasks. The potentials

were baseline-corrected using a period of 100 msec before the stimulus up to stimulus onset. For each participant, waves were created by computing voltages at electrodes over motor cortex (C3 and C4) contralateral–ipsilateral to the response. This was done separately for left and right response trials. Next, for each participant, the above subtractions were averaged across left and right responses. Finally, these waves were grand-averaged across all participants. This process was applied separately for successful and failed stop trials.

Independent Components Analysis

Individual participant data sets underwent independent components analysis (ICA) using the infomax algorithm (Bell & Sejnowski, 1995) with extension toward subgaussian sources (Lee, Lewicki, & Sejnowski, 1999). Resultant ICs were screened for components representing stereotypical artifacts (blinks, eye movements, etc.). ICA weights were subjected to Grubbs' test for outliers with alpha set at .0001. Components with outlying weights on vertical or horizontal ocular electrodes were identified as blinks or saccades, respectively, and removed from the data set. Both retained and rejected ICs were visually inspected for accuracy of the automatic classification, which was corrected manually when necessary.

P3 IC Selection

Following ICA decomposition, we conducted an algorithmic analysis to identify the IC for each participant that accounted for the P3 ERP observed following stop signals in the SST33 task. First, each IC was subjected to a topographical criterion test to identify the ICs with maximum weights over frontocentral electrodes (Fz, Cz, FCz). Then, the weights of each of these candidate ICs were applied to channel data to create a component ERP during the time range of interest (100–450 msec) following stop signals in SST33. Finally, each of these component ERPs was correlated to the all-IC channel data during the same time range following stop signals on successful stop trials. The component ERP with the strongest correlation to raw channel data was selected as the prototypical P3 IC. IC selection was performed in a well-established paradigm (SST33) and then extended to our new task design (the SST100 variant). With this IC-based approach (Wessel, 2018, brain topography), it is typical to elicit a “prototype” component using stimuli that are known to produce the signature of interest and then test the activity in that component in another task situation. In this way, we isolated a signal of inhibition in the classic SST33 for comparison to the same signal in SST100.

For several participants, multiple ICs accounted for the stop signal P3 (as revealed by inspection of their channel space reconstruction after removal of the selected component). Ten of the 29 participants had a stop signal P3 that was accounted for by two ICs.

Table 1. RTs by Trial Type: SST 33 versus SST100

	<i>SST33</i>	<i>SST100</i>	<i>Baseline Go</i>
Go Acc	0.95 (0.04)	0.89 (0.07)	0.97 (0.03)
Go RT (msec)	507.25 (121.01)	431.86 (81.47)	406.54 (69.34)
Stop Acc	0.51 (0.04)	0.51 (0.19)	—
Failed stop RT (msec)	430.38 (96.28)	471.42 (87.14)	—
SSRT _i (msec)	245.01 (77.58)	—	—

Values in parentheses represent *SEM*. Acc = Accuracy, SSRT_i = SSRT as computed by the integration method.

ERPs

Stimulus-locked ERPs were created for each event of interest in the SST33 and SST100 tasks. In the SST33 task, where go trials had no stop signals, the ERPs were time-locked to the go signal on go trials and to the stop signal on stop trials. In the SST100, where every trial had a stop signal, ERPs could be extracted with respect to the stop signal on every trial. Epochs were made in windows of 300 msec before stimulus onset to 700 msec following stimulus onset and baseline-corrected using a baseline of 100 msec before stimulus onset to time of stimulus onset. ERPs were tested for significant amplitude differences using sample-to-sample *t* tests, false discovery rate (FDR)-corrected to $p < .05$ (using the procedure presented in Benjamini, Krieger, & Yekutieli, 2006). Any time points where significant differences were found are indicated in ERP figures by gray shading at the bottom of the plot.

P3 Onset Latency Analysis

To test for differences between P3 onset on successful and failed stop trials in both SSTs, we quantified P3 onset using a single-trial analysis, including the following steps (the same steps as in Wessel & Aron, 2015). For each SST, we created four pools of trials: (1) successful stop trials,

(2) go trials matched to successful stop trials by stop signal staircase delay, (3) failed stop trials, and (4) go trials matched to failed stop trials by stop signal staircase delay. The matching go trials were selected from the set of trials at which the current point of the respective SSD staircase matched the selected stop trial. In other words, a left-hand go trial that followed a failed left-hand stop trials with a 200-msec SSD would have a (theoretical) SSD of 150 msec and would be selected to match stop trials with that same SSD (150 msec). That is, stop trials were matched to go trials that would have had a stop signal at the same time point had there have been a stop signal (this matches any implicit expectation effects that could have been built up). The same approach was applied to the SST100 variant.

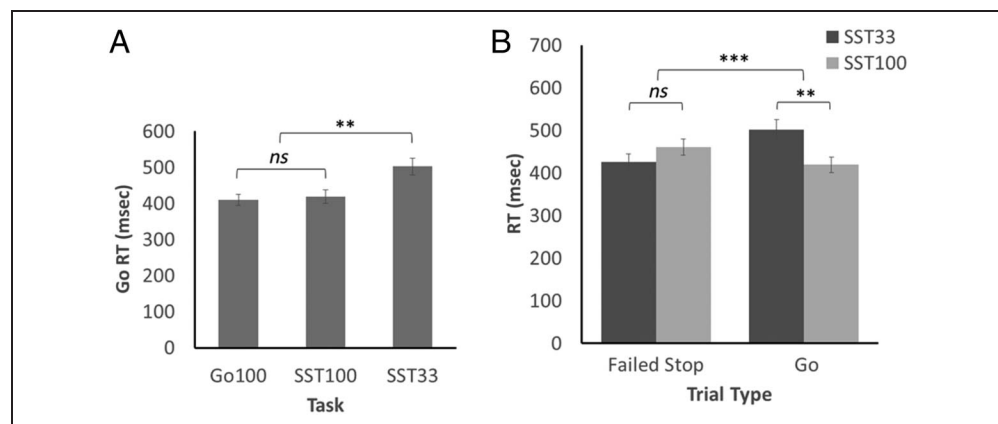
Ten thousand iterations of Monte Carlo *t* tests were performed on these pools of trials and the resulting *t* value vectors used to quantify the onset of P3 in failed stop trials and successful stop trials separately. We identified P3 onset as the maximum *t* value in our search window of interest (200–500 msec after stop signal) and then worked backward through the vector to find the first significant value in that block of significant values. This first significant point ($p < .05$) was quantified as P3 onset. This analysis was corrected for multiple time-point comparisons using an FDR correction. In other words, the point of the P3 onset was defined as the first point of the time period of significant samples that included the peak of the stop signal P3. All analyses were performed on the channel space reconstruction based on the selected P3 ICs, which improves the single-trial signal to noise ratio and enables this single-trial analysis (Wessel & Aron, 2015).

RESULTS

Behavior

Descriptive statistics for all three tasks can be found in Table 1. Results for the SST33 task were as expected for standard versions of the SST. The SST33 task yielded an average stopping success rate of 0.51, showing that

Figure 2. Behavioral results. (A) Go RT for each task. (B) Failed stop and go RTs for each stop signal variant. Error bars represent *SEM*. ns = not significant. ** $p < .01$. *** $p < .001$.



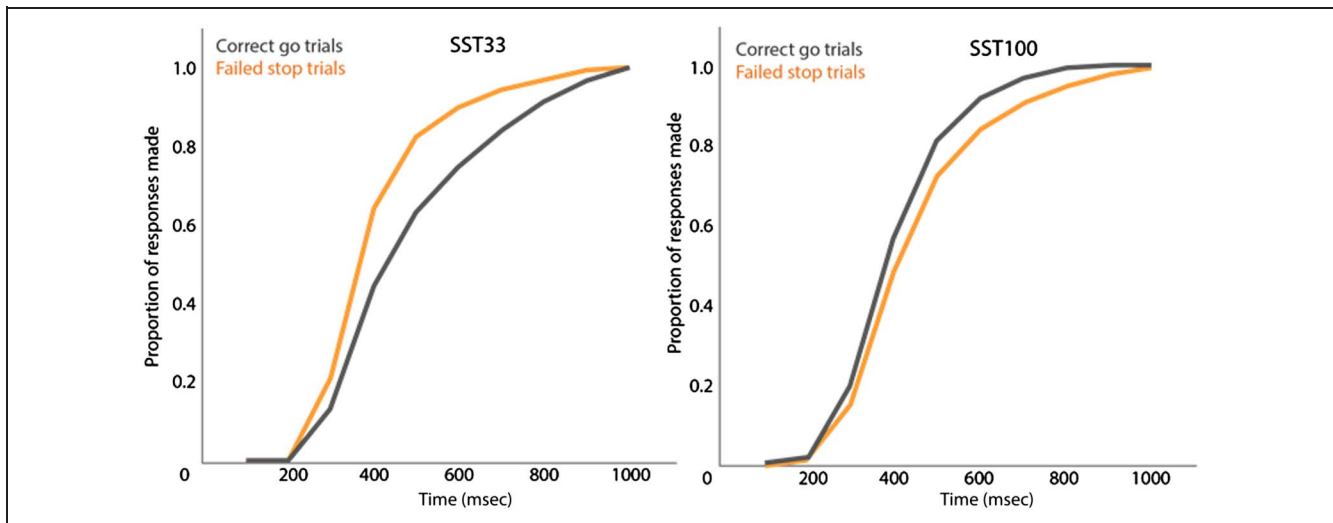


Figure 3. Cumulative distribution functions for both SSTs. Black lines represent correct go RTs; orange lines depict failed stop RTs. The left column depicts SST33, and the right column depicts SST100.

the adaptive SSD algorithm was effective in tracking participants' stopping performance. Coincidentally, the SST100 task on average produced the same stopping success rate, even though no SSD tracking was used.

With regard to go RT across all three tasks, the 1×3 ANOVA showed a significant main effect, $F(2, 46) = 11.82, p < .001$, partial $\eta^2 = .3$. Dunn-Sidak pairwise comparisons showed that the SST33 produced slower RTs compared with both the SST100 and the baseline go RT task ($p < .005$ and $p < .001$, respectively), whereas there was no significant difference between the SST100 and the baseline go RT task ($p = .912$; Figure 2A).

Notably, the two versions of the SST produced different relationships between go and failed stop RTs (Figure 2B). A 2×2 ANOVA comparing go and failed stop trial RT across the two variants of the SST showed a no main effect of task, $F(1, 28) = 1.15, p = .29$, but a main effect of trial type, $F(1, 28) = 17.48, p < .001$, partial $\eta^2 = .38$, and a significant interaction, $F(1, 28) = 209.99, p < .001$, partial $\eta^2 = .88$. Pairwise comparisons showed that, although the SST33 showed the expected bias toward faster RTs in the failed stop condition ($p < .01$), no significant difference was found in the SST100 condition. Numerically, the relationship was even reversed in that task, with the failed stop trials exhibiting nominally slower RTs. This can also be seen from the cumulative RT distribution functions from the SST100 (Figure 3).

Finally, behavior in the SST100 paradigm clearly showed that the point system successfully induced participants to respond rather than wait for a stop signal on every trial. An inspection of trial percentages across SSDs in the SST100 paradigm shows that correct go trials (green line, Figure 4) became more likely as SSD increased, showing that participants were trying to beat the stop signal, resulting in a prepotent go response.

Sequential Effects

We also explored how performance on the current trial was affected by the outcome of the previous trial. Figure 5 depicts these exploratory analyses. We observed significant differences in proportion of responses as a function of the previous trial. Specifically, participants were (1) more likely to go when the last trial was a correct go, (2) more likely to fail stop when they beat the stop signal on the last trial, and (3) more likely to stop successfully when they failed on the last trial.

Lateralized Readiness Potentials

The successful stop rate of 20% at SSDs as long as 800 msec (Figure 4) raises the possibility that participants did not initiate a go response on each trial. Participants may have chosen instead to guarantee 100 points by registering a

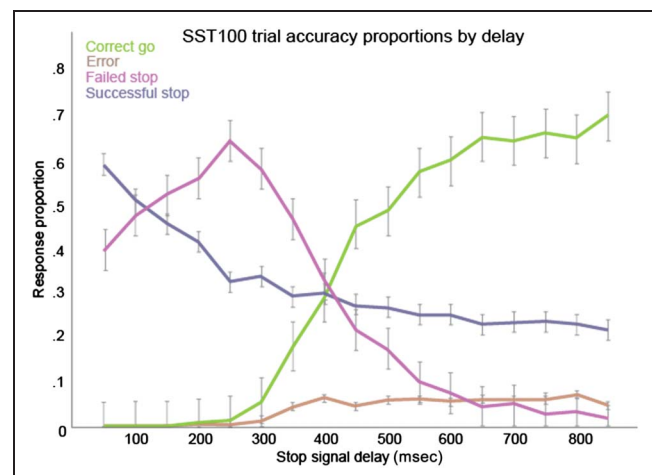


Figure 4. SST 100: Behavior. Probability of each response type as a function of SSD. Error bars represent SEM.

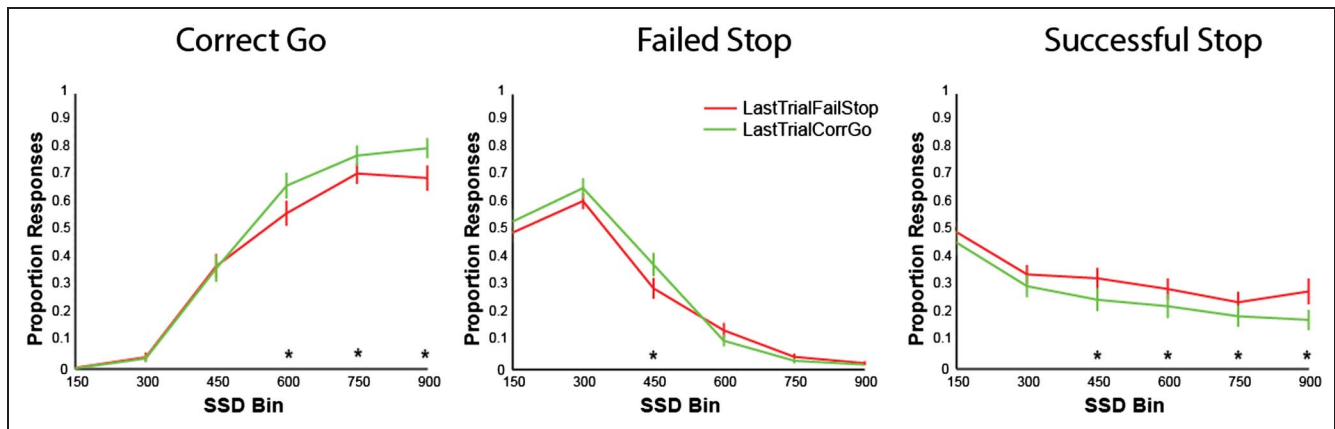


Figure 5. Sequential effects. Proportions of responses in SST100 task as a function of last trial outcome. Correct go trials (left), failed stop trials (center), and successful stop trials (right). Error bars represent *SEM*. Starred bins indicate significant differences.

“successful stop” in the absence of an inhibitory control requirement (Wessel, 2018). To assess whether the SST100 fundamentally changed the inhibitory requirements on successful stop trials compared with the SST33 version, we analyzed the lateralized readiness potentials (LRPs), an index of motor activation measured from sites over primary motor cortex (Coles, 1989).

Figure 6 depicts go stimulus-locked LRPs during correct go trials, failed stop trials, and successful stop trials in both tasks. LRPs were calculated for stop trials where the SSD was less than 400 msec, the same subset of trials used in our comparison of the frontocentral P3. Unsurprisingly, we observed motor activity in both SST33 and SST100 during trials where a response was emitted, and we observed activity in both tasks during failed stop trials, where a response was emitted but a stop signal was also present. More importantly, we also observed an LRP on successful stop trials. Moreover, the LRPs between the SST33 and SST100 did not differ significantly from each other, affirming the similarity in motor preparation for these trial types across different versions of the task. Note that, in the SST33, these trials require no reactive inhibition, whereas in the SST100, these trials are those in which the participant “beat” the stop signal. Therefore, we conclude that our proposed neural index of inhibition, the P3, is indeed being measured using trials with prepotent go responses.

ERPs

Frontocentral P3 Amplitude Comparison

In the SST33, the comparison between successful go and stop trials yielded the classic frontocentral P3 ERP. Crucially, the SST100, where stop signals were not infrequent, also yielded a clear frontocentral amplitude increase on successful stop trials compared with go trials (Figure 7). Note that, in the SST33, the go trial ERP is time-locked to the go signal, as no stop signal was present

on go trials. In that sense, the SST100 allows for a cleaner comparison, as the same time-locking event (stop signal) is used to compare stop and go trials. The only condition difference is that, on stop trials, the response was withheld, whereas on go trials, the response was already made by the time the stop signal appeared. Therefore, although go trials and stop trials in the SST100 are identical with regard to their physical properties, the only difference is motor inhibition. The absence of inhibitory demand by the stop signal on successful go trials led to a complete absence of the P3.

For the successful versus failed stop trial amplitude comparison (Figure 8), we found that, in the SST33, sample-to-sample amplitude differences in frontocentral P3 amplitude to the stop signal were accounted for by differences in their onset, with the failed stop P3 yielding a later onset (cf. next section). In the SST100, the increase in P3 amplitude on successful compared with failed stop trials did not seem to be entirely accounted for by differences in onset (cf. Figure 8), with a significant amplitude difference ranging from 182 to 318 msec following the stop signal (FDR-corrected). However, the SST100 also yielded P3 onset differences.

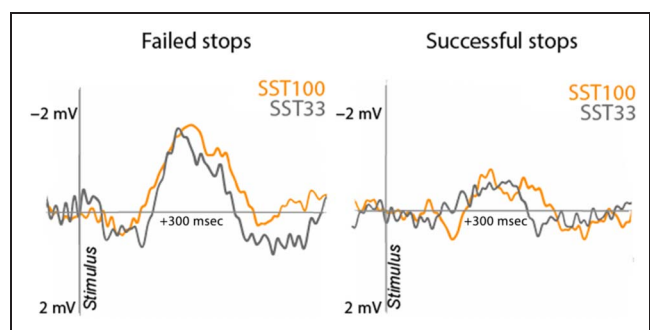
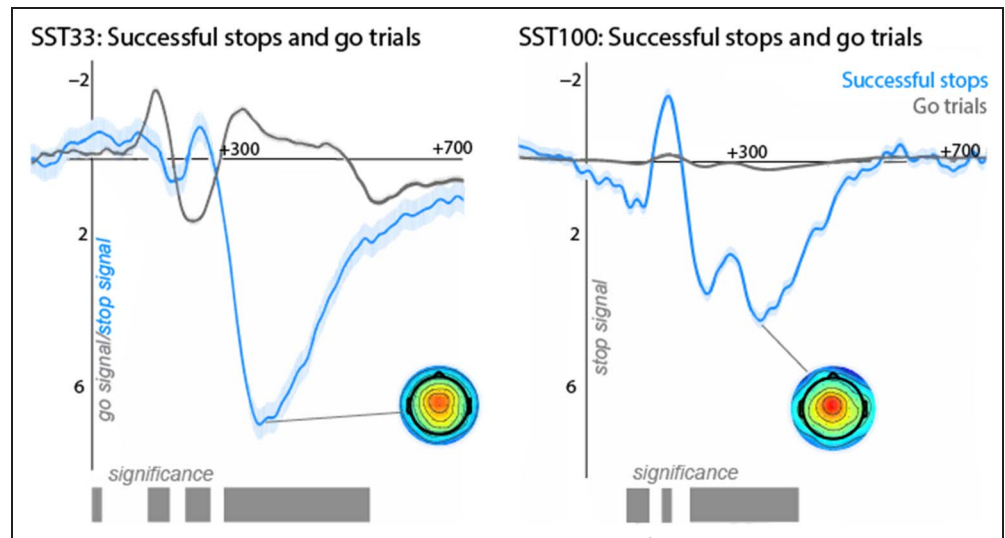


Figure 6. Go stimulus-locked LRPs for trial types: failed stop (left) and successful stop (right). No LRPs significantly differed between task versions.

Figure 7. Stop versus go trial comparison in both SST33 (left) and SST100 (right) variants. A clear frontocentral P3 can be seen on successful go trials in both conditions. Gray solid bar denotes significant amplitude differences at $p < .05$ (FDR-corrected).



Successful versus Failed Stop Signal P3 Onset Comparison

Our ICA-based single-trial comparison of the P3 onset revealed that in both the SST33, $t(28) = -2.80, p = .009, d = .34$, and SST100, $t(28) = -2.17, p = .04, d = .54$, the onset of the stop signal P3 occurred significantly earlier on successful compared with failed stop trials (Figure 8). Although this has been reported for the SST33 paradigm (Wessel & Aron, 2015; Kok et al., 2004), it is notable that the same relationship is found in the SST100, even though failed stop trials did not involve faster RTs compared with

go trials. This shows that the onset of the frontocentral stop signal P3 reflects a key property of the race model of the SST, even when differences in stopping performance cannot be accounted for by difference in go process speed.

Although we find earlier onset for successful versus failed stops in the SST100 paradigm, the analysis above does not account for possibly different inhibitory requirements across tasks immediately following the stop signal. Recall that on go trials in the SST100 task, the participant had already responded when the stop signal was played. By contrast, the SST33 go trials contained no stop signal, and participants did not know if it would appear. Thus,

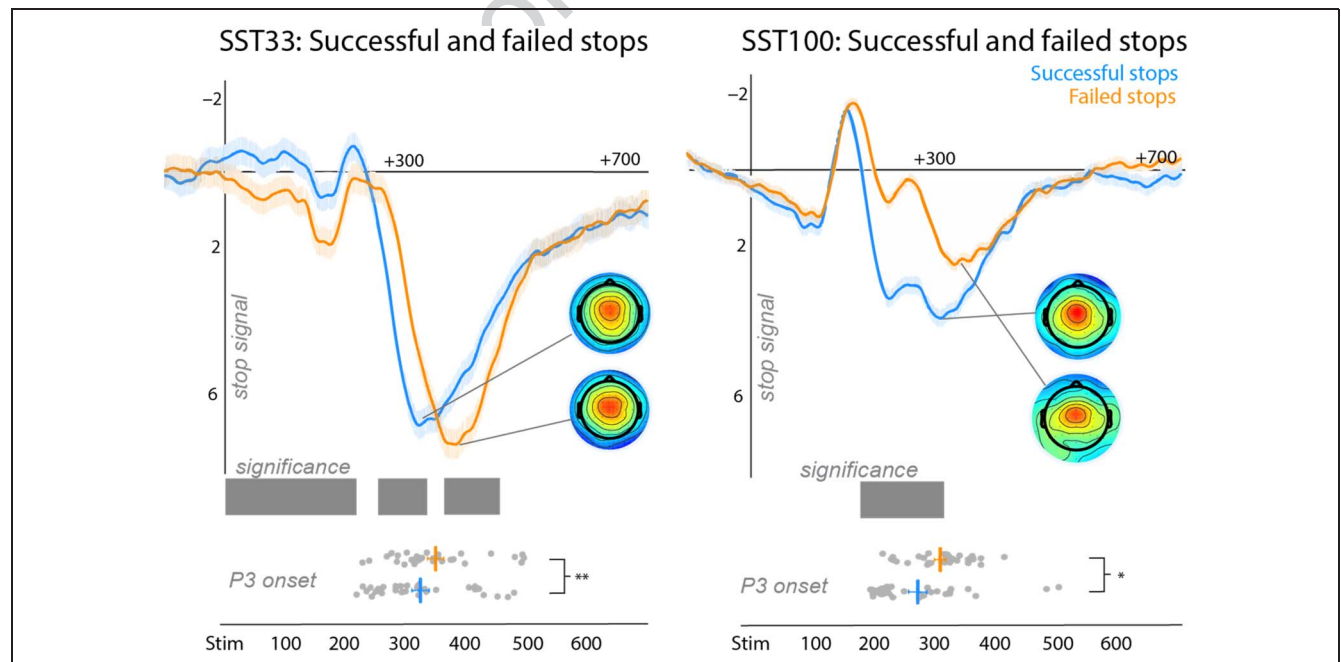


Figure 8. Successful versus failed stop trial comparison in both SST33 (left) and SST100 (right) variants. The frontocentral P3 shows a significantly earlier onset for successful versus failed stop trials in both variants. Gray dots denote individual participants. Orange and blue vertical lines denote condition mean; horizontal lines denote *SEM*. Gray solid bar denotes significant amplitude differences at $p < .05$ (FDR-corrected).

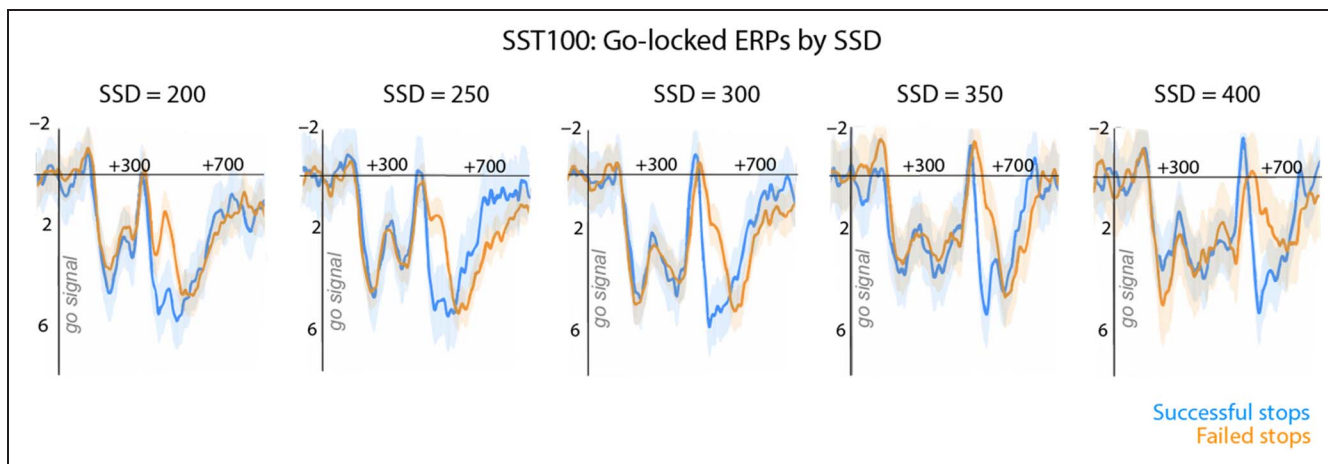


Figure 9. Successful and failed stop ERPs from the SST100 task, time-locked to the go signal and plotted separately for each SSD.

the requirements for inhibition in the time following stop signal onset differs between tasks. To address this issue, we perform the identical analysis but with successful and failed stops locked to the go signal and separating by SSD (Figure 9). We observe the same pattern of earlier onset for successful stop versus failed stop trials with this cleaner comparison.

DISCUSSION

We present EEG data obtained from a novel version of the SST designed to address two confounds in the standard version of this well-established paradigm. By presenting stop signals on every trial, we equated the neural stimulus processing requirements of stop and go trials and therefore excluded any interpretations that attribute condition differences to the presence of a second, infrequent signal on stop trials. We found that the well-established frontocentral P3 ERP to the stop signal showed a significant amplitude increase in the stop versus go contrast, even when stop signals occurred on every trial.

Furthermore, our new SST variant changed the usual RT selection bias found in the comparison of successful and failed stop trials, where failed stop trials systematically oversample the fast part of the go RT distribution (Osman, Kornblum, & Meyer, 1986). The reason for this change in the pattern of RT is likely that the SST100 differs from SST33 not only in that stop signals occur on every trial but also in the distribution of SSDs. In the SST33, SSDs are restricted in range based on each participant's performance. In contrast, the SST100 uniformly samples SSDs spanning the entire go RT range. Consequently, the stop signal may appear at any given time that the response cannot be stopped, regardless of the duration of the go process on each trial. Hence, within-subject variation in the duration of the go process has reduced influence in determining a successful versus failed stop. We found that,

even though the go RT bias was now reversed, the properties of the frontocentral P3 ERP still reflected a key proposition made in race model conceptualization of the SST (Verbruggen & Logan, 2009; Boucher et al., 2007; Logan et al., 1984): When inhibitory control processes start earlier, stopping is more likely to be successful. Therefore, P3 onset differences between successful and failed stop cannot be explained by differences in the go process, making it more likely that the P3 onset indexes the timing of the inhibition process.

Our findings have two key core implications. First, they firmly establish the frontocentral P3 ERP as a reliable, millisecond precision index of motor inhibition. The onset of this component fulfills the core prediction made regarding the nature of the motor inhibition process in the race model of the SST: If it occurs earlier, successful stopping is more likely. This finding has been reported in prior studies of the standard variants of the task (e.g., Wessel & Aron, 2015; Kok et al., 2004). However, as outlined above, these prior findings are affected by the substantial confounds regarding the condition contrasts in the standard version of the SST. Here, we show that even when stop versus go processing demands are equated and even when successful versus failed stop differences cannot be attributed to a faster go RT process in failed stop trials, the frontocentral P3 onset still behaves like the inhibition process should under the race model.

Second, we introduce a new version of the SST that is unaffected by the confounds of the standard version of the task. Given the frequency with which the SST is used to probe or induce inhibitory control functions across all fields of psychology, this is of wide-reaching importance. For example, clinical researchers use the SST to measure inhibitory control in the context of movement disorders like Parkinson's disease (Obeso et al., 2011) or Huntington's disease (Rao et al., 2014), attention-deficit/hyperactivity disorder (Kenemans et al., 2005; Nigg, 1999), substance use disorders (Nigg et al., 2006), or OCD (Boisseau et al., 2012). Developmental researchers use the task to relate

inhibitory control to the development of externalizing disorders (Albrecht, Banaschewski, Brandeis, Heinrich, & Rothenberger, 2005) or other developmental issues (Schachar & Logan, 1990). Others attempt to induce “self-control” demands via the SST to see if such demands can transfer to affect impulse control problems, unhealthy dieting (Nederkoorn et al., 2010), or alcohol intake (Houben et al., 2011). These researchers, and everyone else looking to use the SST as a “pure” index of inhibitory control, may benefit from this alternative design, which addresses the noninhibitory confounds of the standard version of the task.

One additional confound found in the standard SST condition contrasts remains unaddressed in our current paradigm: Go trials (and failed stop trials) per definition contain a motor response, whereas (successful) stop trials do not. Hence, any condition difference (e.g., in neural activity or in effects that either condition has on other psychological processes) could either be due to motor inhibition in the successful stop condition or due to motor activation in the go/failed stop condition. Here, we addressed this by using ICA (which successfully disentangled response-related activity from the rest of the EEG signal) and by demonstrating that our proposed index of motor inhibition showed a property that is sensible in the context of the race model of the SST (earlier onset on successful compared with failed stopping) but is invariant to condition differences in motor activity between the trial types across both versions of the task. In other words, the onset of the stop signal P3 was earlier on successful versus failed stop trials, whether failed stop trials contained faster (SST33) or slower (SST100) RTs compared with the overall RT distribution. This makes it unlikely that the P3 can be attributed to response-related processes. However, it would be prudent for future work to develop a version of the SST that addresses this residual confound (e.g., by combining our novel SST with a stop change paradigm; cf. Verbruggen & Logan, 2009; Logan & Burkell, 1986).

One shortcoming of the current paradigm compared with the standard SST is that it is impossible to calculate SSRT using standard methods of derivation from our data. The calculation of SSRT assumes that the likelihood of successful stopping increases at slower portions of the go RT distribution (Verbruggen et al., 2019; Logan et al., 1984). Because our instruction has reversed this relationship compared with the standard version of the task, SSRT calculation does not yield sensible values in our task. There are, however, several conceivable ways to calculate a subject-level index of the quality of task performance in this task. For example, one could compute a ratio of RT to stopping percentage. Because every participant had been presented with the same set of SSDs, such a relationship would quantify both the speed with which (correct) go responses are made as well as the numerical success of stopping. However, it is unclear how to validate such a measurement vis-à-vis the race model-based predictions derived from the standard version of

the task (and specifically, with regard to SSRT). Because SSRT is contaminated by attentional demands (Matzke et al., 2017; Verbruggen et al., 2010, 2014), it is unclear how informative the presence or absence of a correlation between SSRT derived from the standard task and any performance metric extracted from our current paradigm would be. One solution to this could be the recent approach of Matzke and colleagues (e.g., Matzke et al., 2013), who proposed a Bayesian effort to disentangle inhibitory components of SSRT from attention-related “trigger failures.” Such validations could be the participant of future study, using an optimized version of the standard variant of the SST used to quantify trigger failures and residual SSRT and relating them to behavioral indices of performance in our version of the task.

Another limitation rests in the fixed nature of SSDs in the SST100 task. Further studies using this paradigm should validate our results using an SSD procedure that adapts to participant go RT, as recommended by a recent consensus of inhibitory researchers (Verbruggen et al., 2019).

Finally, one may wonder whether behavior in the SST100 could be explained by a strategy in which no response is ever initiated on successful stop trials, whereas no inhibition is ever initiated on failed stop trials, leading these trials consisting of pure go activations that were too slow to beat the stop signal. We can rule out the first explanation regarding the successful stop trials, as the LRP (Figure 6) clearly shows that motor activations on successful stop trials in the SST100 version matched the SST33. Regarding failed stop trials, we propose that the ERP results speak against this, as there was clearly a (delayed) frontocentral P3 on failed stop trials (Figures 8 and 9).

Taken together, we present a new version of the classic SST, in which we addressed two well-known confounds with that version of the task. In doing so, we have developed a task in which stop signals are not infrequent, the processing demands of stop and go trials are matched, the only difference between both trial types is overt behavior, and the usual RT bias in the comparison of successful versus failed stop trials is reversed. We found that, even under these conditions, the frontocentral stop signal P3, a well-established purported index of motor inhibition in the standard version of the SST, reflects a core prediction of the race model: Earlier onset means more successful stopping. Therefore, the frontocentral P3 can be seen as a reliable estimator of the latency of the stopping side of the race underlying action stopping, and our novel paradigm can be used to test the neural mechanism underlying motor inhibition process in the absence of the confounds associated with the standard SST.

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