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► **To cite this version:**

Mario Hervault, Pier-Giorgio Zanone, Jean-Christophe Buisson, Raoul Huys. Hold your horses: Differences in EEG correlates of inhibition in cancelling and stopping an action. *Neuropsychologia*, 2022, 172, pp.108255. 10.1016/j.neuropsychologia.2022.108255 . hal-04782566

**HAL Id: hal-04782566**

**<https://hal.science/hal-04782566v1>**

Submitted on 29 Nov 2024

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## **Title**

Hold your horses: Differences in EEG correlates of inhibition in cancelling and stopping an action.

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## **Abstract**

Behavioral adaptation to changing contextual contingencies often requires the rapid inhibition of planned or ongoing actions. Inhibitory control has been mostly studied using the stop–signal paradigm, which conceptualizes action inhibition as the outcome of a race between independent GO and STOP processes. Inhibition is predominantly considered to be independent of action type, yet it is questionable whether this conceptualization can apply to stopping an ongoing action. To test the claimed generality of action inhibition, we investigated behavioral stop–signal reaction time (SSRT) and scalp electroencephalographic (EEG) activity in two inhibition contexts: Using variants of the stop–signal task, we asked participants to cancel a prepared–discrete action or to stop an ongoing–rhythmic action in reaction to a STOP signal. The behavioral analysis revealed that the discrete and rhythmic SSRTs were not correlated. The EEG analysis showed that the STOP signal evoked frontocentral activity in the time and frequency domains (Delta/Theta range) in a task–specific manner: The P3 onset latency was the best correlate of discrete SSRT whereas N2/P3 peak-to-peak amplitude was the best correlate of rhythmic SSRT. These findings do not support a conceptualization of inhibition as action–independent but rather suggest that the differential engagement of both components of the N2/P3–complex as a function of action type pertains to functionally independent inhibition subprocesses.

## **Keywords**

action inhibition, executive control, ERP, time–frequency, motor control

## Highlights

- Inhibitory control was investigated in two stop–signal tasks that differed in the type of motor response to inhibit.
- Frontocentral EEG markers of inhibition differed between cancelling a prepared–discrete action and stopping an ongoing–rhythmic action.
- STOP–signal ERP components were distinctively related to inhibition in prepared–discrete (P3 onset) and ongoing–rhythmic (N2/P3 peak-to-peak amplitude) actions.
- Inhibitory control is not a generic process; its underpinning neural subprocesses are modulated as a function of action type.

## 1 **1. Introduction**

2 Inhibitory control is generally conceived of as a core executive function involved in the control of  
3 attention, thought, emotion, and action (Bari & Robbins, 2013; Diamond, 2013; Miyake et al.,  
4 2000). In particular, people are apt to cancel actions rapidly when unanticipated events or  
5 contextual changes occur. Some situations call for the inhibition of prepared actions, a case  
6 thoroughly studied by experiments using the stop-signal paradigm: some GO stimuli are  
7 unpredictably followed by a STOP signal requiring the cancellation of the prepared response  
8 (Logan & Cowan, 1984; Verbruggen et al., 2019). Other situations call for the abrupt cessation of  
9 ongoing actions during execution (Alegre et al., 2008; Lofredi et al., 2021). In this regard,  
10 previous experimental findings strongly suggest that the behavioral inhibition latency in the two  
11 situations are unrelated (Hervault et al., 2019). Still, it is unclear whether the neural processing  
12 of action inhibition relies on a single set of processes, that is, whether it is action-independent  
13 or action-specific. The present study addressed this issue by investigating behavioral stopping  
14 latency and scalp neural activity through EEG when participants inhibited either prepared-  
15 discrete or ongoing-rhythmic actions.

16 Based on the prevalent horse-race model (Logan & Cowan, 1984; Verbruggen et al., 2019),  
17 inhibition can be evaluated in stop-signal tasks as the outcome of a race between independent  
18 GO and STOP processes: it fails when the GO process finishes the race before the STOP process  
19 and succeeds otherwise. The grounding statistical model provides an elegant way to estimate  
20 the duration of the covert action inhibition process, the stop-signal reaction time (SSRT). The  
21 model remains silent, however, about the underlying neural processes. The stop-signal  
22 paradigm was developed as a tool to evaluate a presumably action-independent process of  
23 inhibition (Band & van Boxtel, 1999; Logan & Cowan, 1984). Logan and Cowan (1984, p. 318)  
24 could have been hardly more explicit about the presumption of generality when they stated that

25 *“The model developed so far is addressed to discrete tasks with discrete responses. It would*  
26 *seem to be relatively straightforward to generalize the model to continuous responses”*. Yet, for  
27 an ongoing–continuous action, the assumed race between the GO and STOP processes  
28 underlying the stop–signal paradigm cannot apply, since the GO (action initiation) process has  
29 long run to its completion at the time of the STOP Signal occurrence. This argument motivates  
30 our challenging the assumption that action inhibition is generic by comparing cancelling a  
31 prepared–discrete action and stopping an ongoing–rhythmic action. In addition, the  
32 investigation of ongoing–rhythmic action inhibition has been conceived as crucial in establishing  
33 the real–world generalizability of inhibitory control (Hannah & Aron, 2021). Studying ongoing–  
34 rhythmic action inhibition also provides the opportunity to measure the SSRT without relying on  
35 race–model assumptions (Lofredi et al., 2021; Morein-Zamir et al., 2006; Schultz et al., 2021).

36 Inhibitory control is indispensable in everyday life to stop either type of action. Discrete actions,  
37 like grasping, are delimited by moments without movement (i.e., with zero velocity and  
38 acceleration). That is, a discrete action unit refers to a movement that is preceded and followed  
39 by a non-negligible period in which the position of the moving limb does not change. In contrast,  
40 rhythmic actions, like walking, are continuous and periodic and lack such recognizable endpoints  
41 (Hogan & Sternad, 2007). The neural structures associated with controlling discrete and  
42 rhythmic actions differ considerably (Schaal et al., 2004; Spencer et al., 2003; Wiegel et al.,  
43 2020), due to differing timing and initiation mechanisms (Huys et al., 2008; Spencer et al., 2003).

44 Thus, various lines of evidence persuasively suggest that discrete actions require additional  
45 control processes, involving supplemental brain activation as compared to rhythmic actions.  
46 Therefore, since discrete and continuous movements tap into distinct neural mechanisms of  
47 action control, they provides an excellent window to test whether inhibition is the action–

48 independent process that is often claimed to be (Logan & Cowan, 1984; Schall et al., 2017;  
49 Verbruggen & Logan, 2009a).

50 As of yet, only few studies have investigated whether the same inhibition process underwrites  
51 cancelling a prepared action or stopping an ongoing action. Morein-Zamir et al. (2004) found a  
52 correlation between the inhibition latencies (SSRTs) associated with cancelling a prepared action  
53 and stopping an ongoing (yet) isometric action in a rather peculiar task: Participants tracked the  
54 speed of a target that rotated along an imaginary circle on the screen by varying the pressure on  
55 a force sensor. Therefore, while the perceptual task was clearly continuous, there was hardly  
56 any movement to inhibit at all, since there was no limb displacement or trajectory to speak of.  
57 In contrast, a recent investigation showed that the SSRTs associated with cancelling a prepared–  
58 discrete key–pressing action (the classic stop–signal task) and stopping an ongoing–rhythmic  
59 drawing action were unrelated across participants (Hervault et al., 2019). This finding  
60 contradicts the assumption that a single mechanism is involved in inhibiting prepared and  
61 ongoing actions.

62 Regarding the electroencephalographic (EEG) neural activity related to action inhibition in the  
63 stop–signal paradigm, event–related potential (ERP) studies have linked inhibition to  
64 pronounced frontocentral negativity around 200–300 ms after the STOP–signal onset (N2),  
65 followed by a distinct positive activity about 150 ms later (P3) with a frontocentral to  
66 centroparietal topography (Huster et al., 2013). Suggestive of their functional relevance, the  
67 amplitude and latency of both waves differ between successful and failed STOP trials (e.g.,  
68 Bekker et al., 2005; Kok et al., 2004). In addition, P3 onset latency correlates strongly with SSRT  
69 (e.g., Wessel & Aron, 2015). Finally, both N2 and P3 predict individual differences in inhibition  
70 performances (Chikara & Ko, 2019; Vahid et al., 2018). In the frequency domain, STOP trials  
71 reveal augmented power in the Delta (0–3 Hz) and Theta (4–8 Hz) frequency bands compared

72 to GO trials (e.g., Chikara et al., 2014; González-Villar et al., 2016; Lavalley et al., 2014; Wessel &  
73 Aron, 2013). This difference is reliably discernible between 200 and 500 ms post stimulus  
74 presentation, that is, within the time range of the N2/P3-complex (Huster et al., 2013).

75 These ERP and time-frequency patterns correlate significantly with action inhibition and proved  
76 to change with the loss of inhibitory control due to various disorders (Bekker et al., 2005;  
77 Kuzstor et al., 2019; Lansbergen et al., 2007; Tschuemperlin et al., 2019; Van Voorhis et al.,  
78 2019). Whereas such correlations between inhibition performance and N2/P3 ERPs and the  
79 related time-frequency patterns are robust and reproducible, similar correlations have also been  
80 reported between these EEG indices and multiple other behavioral measures, thus questioning  
81 their specificity in indexing inhibitory processes per se (Huster et al., 2020). Hence, EEG  
82 correlates could either truly reflect a, say, “pure” inhibitory process (Hynd et al., 2020; Wessel &  
83 Aron, 2015), or the processing of the conflict required to inhibit action (Enriquez-Geppert et al.,  
84 2010), or a contextual update of the situation requiring an sporadic action revision (Waller et al.,  
85 2019). Thus, studying inhibition-related EEG patterns in tasks known to differ in the engaged  
86 action-control processes, as it is the case for discrete and rhythmic actions, may provide  
87 additional information to a functional interpretation of these EEG correlates.

88 Apart from classical stop-signal experiments, only two studies, to our knowledge, report that  
89 components of the N2/P3 complex are linked to the intentional termination of ongoing actions:  
90 The P3 wave was associated with ending sustained isometric elbow contraction (Hatta et al.,  
91 2003), while the N2 wave was related to the reactive cessation of an ongoing drawing action  
92 (Sosnik et al., 2015). In view of their presumed functional relevance, EEG correlates of inhibition  
93 constitute suitable candidates to address whether inhibitory processes are involved similarly in  
94 cancelling and stopping action. Indeed, EEG correlates of inhibition were modulated in  
95 amplitude and latency when the inhibitory requirement is varied by using stop-signal tasks,



96 stop–change tasks, and GO/NoGO tasks (e.g., Krämer et al., 2011; Raud et al., 2020).  
97 Nonetheless, these studies reported EEG modulations when cancelling one action type only,  
98 again, discrete action, while there is, to our best knowledge, no study investigating modulations  
99 of inhibition EEG for continuous action. Therefore, no comparison across the two action types is  
100 possible in terms of neural inhibition modulations.

101 To sum up, the present study probes the assumption that action inhibition generalizes across  
102 action types by using tasks involving movements associated with distinct kinematics, dynamics,  
103 as well as neural control mechanisms. Therefore, we recorded EEG in participants who had  
104 either to cancel a prepared–discrete action or to stop an ongoing–rhythmic action performed on  
105 a digitizing tablet, while keeping the movement effector and the plane of motion the same. We  
106 investigated the frontocentral N2/P3–complex and Delta/Theta band activity as neural  
107 correlates of inhibitory action control.

108 **2. Materials and Methods**

109 **2.1. Participants**

110 Twenty-three participants volunteered in the experiment. For three participants, behavioral and  
111 EEG artifacts warranted their exclusion from the final analysis, which was therefore restricted to  
112 twenty participants (12 males; 8 females, mean age  $25 \pm 2.4$  (SD) years). All participants were  
113 healthy and had normal or corrected-to-normal vision. Participants' handedness was  
114 determined using the Edinburgh handedness inventory (Bryden, 1977) and only participants  
115 with a homogeneous pattern of right-hand preference were included. The study was conducted  
116 according to the principles stated in the Declaration of Helsinki and the procedures were  
117 approved by the local research ethics committee (ID-RCB: 2020-A03215-34). In order to be  
118 able to detect a difference interpreted as large (Cohen's  $d = 0.80$ , Cohen, 1988; Sawilowsky,  
119 2009) between discrete and rhythmic inhibition latencies, and by accepting a Type 1 risk  $\alpha$  of .05  
120 and a statistical power  $1 - \beta$  of .90, the required number of participants for a repeated measures  
121 t-tests was minimally estimated as 15 (using G\*power software, Faul et al., 2009) .

122 **2.2. Apparatus and Stimuli**

123 Participants were seated in front of a graphic tablet (WACOM Cintiq 15X, 1280×800-pixel  
124 resolution). As an initial position, they put the stylus between two vertical yellow bands (1 mm  
125 wide) plotted at the center of the digitizing black screen (10 mm distant). Stimuli were green or  
126 blue 50 ms flashes displayed on the whole screen. Participants were requested to react to these  
127 stimuli by initiating swiping movements to the right or left direction in the discrete task and by  
128 continuous ongoing swiping oscillations in the rhythmic task. Occasionally, and unexpectedly  
129 (see Procedure), a red 50 ms flash followed the main stimulus, indicating the participants to stop  
130 the action. The  $x$  and  $y$  coordinates of the performed motions were digitized at a sampling

131 frequency of 143 Hz as long as the stylus touched the tablet. The program controlling the tablet  
132 was custom-made. A hook-and-loop fastener was used to fix the participant's forearm to the  
133 table in order to restraint the movement to the wrist articulation, thereby avoiding large  
134 muscular noise in the EEG signal due to an intense contraction of the biceps and deltoid  
135 muscles.

## 136 **2.3. Procedure**

### 137 *2.3.1. Discrete task*

138 The discrete task (**Fig. 1**) was designed following guidelines to capture the ability to inhibit  
139 actions in stop-signal tasks (Verbruggen et al., 2019). The primary task was a two-choice  
140 reaction time task. When a green versus blue flash appeared, participants were instructed to  
141 reach to the right versus left half-side of the tablet screen, respectively, with the stylus (green  
142 and blue stickers were visible on the right and left tablet sides). On 75% of the trials (GO trials),  
143 only this main stimulus was presented, and the participants had to respond to the stimulus as  
144 fast and accurately as they could. On 25% of the trials (STOP trials), a red flash appeared shortly  
145 after the main stimulus as a STOP signal, which indicated to the participants to cancel their  
146 response (secondary task). This experiment consisted of one practice block and 30 experimental  
147 blocks, each consisting of 20 trials. Each trial began when the participant positioned the stylus  
148 between the two centered vertical lines, and the GO stimulus occurred after 1500 ms. The time  
149 interval between GO stimuli of two subsequent trials was randomized between 3500 and 4000  
150 ms. In STOP trials, the GO stimulus was followed by the STOP signal after a delay (SOA; stimulus  
151 onset asynchrony). The SOA, initially set to 200 ms, was dynamically adjusted in 50 ms  
152 increments to achieve a probability of responding  $p(\text{respond}/\text{signal})$  of .50. When the  
153 participant crossed a vertical line, the STOP trial was considered as a stop failure and the SOA

154 was shortened; when the participant kept the stylus between the two lines, the STOP trial was  
155 considered successful and the SOA was prolonged. Participants were asked to focus on the  
156 primary GO task requiring as fast as possible swiping movements while minimizing errors. They  
157 were also instructed that in some STOP trials they would fail to cancel the response but that  
158 they should not be troubled by these failures.

### 159 2.3.2. *Rhythmic task*

160 The apparatus of the rhythmic task was similar, but the task design differed from the discrete  
161 task (**Fig. 1**). As a main task, participants were not engaged in a GO task but in a CONTINUE task.  
162 They were instructed to rhythmically oscillate the stylus between the two sides of the screen at  
163 a spontaneous frequency. A minimal and consistent oscillation amplitude was guaranteed by  
164 imposing the constraint that the oscillations' extrema had to fall outside the two centered  
165 vertical lines. The main task was to pursue the action without interruption when the green or  
166 blue CONTINUE stimuli appeared. In infrequent STOP trials, a red flash signal occurred after the  
167 main stimulus with a fixed SOA (see below). The participants were instructed to stop their  
168 ongoing-rhythmic action as soon as the STOP signal occurred and to wait for the next stimulus  
169 to restart the oscillation. The repartitioning between frequent CONTINUE trials (75%) and  
170 improbable STOP trials (25%) was similar to the discrete task, as well as the inter-trial duration.  
171 The task was also administered in 30 blocks of 20 trials.

172 For both tasks, the main stimulus was randomly a green or blue flash but with an equal  
173 probability within blocks. Participants had to maintain contact between the stylus and the tablet  
174 screen during the whole block. Participants were free to choose the rest time duration between  
175 the blocks (from 20s to 120s). They completed the discrete task in a first session and the  
176 rhythmic task in a second session one week later. This task order was chosen as it allowed us to

177 set the fixed SOA between CONTINUE stimulus and STOP signal in the rhythmic task as the mean  
178 of the SOAs between GO stimuli and STOP signal obtained by each participant in the discrete  
179 task. This procedure ensured that the average delay between the main (GO/CONTINUE)  
180 stimulus and the secondary (STOP) signal were similar in both tasks, and so was the perceptual  
181 neural activity.

#### 182 **2.4. EEG recording and preprocessing**

183 Scalp-EEG signals were acquired with 64 Ag/AgCl active pin electrodes at 2048 Hz (Biosemi  
184 Active Two 10/20 system). Data were referenced online with a CMS/DRL feedback loop and  
185 online low-pass filtered. Electrode offsets (difference in mV of each channel from the CMS  
186 electrode) were examined after electrode application, which was adjusted if the absolute value  
187 exceeded 15 mV. Three additional face electrodes recorded the vertical and the horizontal  
188 electrooculograms. Digital markers (event codes) were inserted into the continuous EEG via a  
189 DB25 cable through a parallel port interface. Continuous EEG data were imported and  
190 preprocessed in bespoke scripts using functions from the EEGLAB Matlab plugin (Delorme &  
191 Makeig, 2004). Data were downsampled to 500 Hz, high-pass filtered at 0.1 Hz, and low-pass  
192 filtered at 50 Hz with a linear finite impulse response filter. EEG epochs corresponding to task  
193 trials were generated by extracting data from -1000 to 2000 ms around each main stimulus  
194 event (GO or CONTINUE stimulus onset for the discrete and rhythmic task, respectively). After  
195 epoching, the recording was visually inspected for non-stereotypical artifacts (such as muscle  
196 activation or intermittent electrode artifacts) and any epoch found to contain an artifact was  
197 removed from the data. EEG was finally re-referenced to the average of all channels.  
198 Independent component analysis (Infomax ICA; Bell & Sejnowski, 1995) was applied to  
199 continuous EEG data (concatenation of the EEG epochs) to identify neural components  
200 contributing to the observed scalp data. In the original scalp 64-channel data, each row of the

201 data matrix represents voltage, summed between source projections to one data channel. After  
202 decomposition (64 components), each row of the data matrix gives the time course of the  
203 activity of one component process spatially filtered from the channel data (Delorme & Makeig,  
204 2004). Using the ICLABEL classifier (Pion-Tonachini et al., 2019) over the 30 first components,  
205 components identified as artifactual, i.e., with less than 5% chance to account for neural activity,  
206 were removed from the EEG data structure, thus removing their contributions to the observed  
207 EEG. Rejection was systematically checked by visual inspection of component properties (time  
208 series, spectra, topography) according to ICLABEL guidelines (ibid.), leading to an average  
209 number of 7 components rejected by participant (min = 4 ICs, max = 9 ICs). Across all  
210 participants, these procedures led to the omission of 8.8 % of the STOP trials in the discrete task  
211 (SD = 1.6 %) and 4.1 % of the rhythmic STOP trials (SD = 1.7 %).

## 212 **2.5. Behavioral measures**

213 Data analyses were performed using Matlab™ software (Mathworks 2013).

### 214 *2.5.1. Discrete stop task*

215 Reaction times (RTs) were computed for both action initiation and inhibition. For each GO trial,  
216 GoRT was calculated as the time between the GO stimulus onset and the response onset, the  
217 latter being defined as the moment the swipe had exceeded 5% of the Euclidean distance  
218 between the initial and furthest (i.e., end) position of the movement response. The discrete  
219 stop–signal reaction time (SSRTd) was computed using the integration method with  
220 replacement of GO omissions, which entails selecting the  $n^{\text{th}}$  GoRT, where  $n$  equals the number  
221 of RTs in the GoRT distribution multiplied by the overall  $p(\text{respond}/\text{signal})$ . The SSRTd is then  
222 obtained by subtracting the mean SOA from the  $n^{\text{th}}$  GoRT (Verbruggen & Logan, 2009a) (**Fig. 2**).  
223 The SSRTd could be estimated for all of the 20 participants as their  $p(\text{respond}/\text{signal})$  did not

224 differ significantly from .50 (see Results). Fail–STOP RT was computed as the reaction time  
225 measured in failed STOP trials (FsRT).

### 226 *2.5.2. Rhythmic stop task*

227 In each STOP trial, the SSRT (SSRTr) was computed using the methodology developed in a  
228 previous study (Hervault et al., 2019). Briefly, stop time was calculated as the latency between  
229 the STOP signal onset and the end of the action, identified when the motion velocity reduced to  
230 null following the STOP signal. Within the stop time, SSRTr was calculated as the latency  
231 between the STOP signal onset and the onset of the response adjustment (**Fig. 2**). This time  
232 point was defined as the moment the ongoing trajectory in phase space (i.e., the space spanned  
233 by  $x$  and  $dx/dt$ ) deviated relative to movements without a STOP signal according to statistical  
234 criteria based on a sample's position in phase space, and the angle and magnitude of its  
235 corresponding velocity vector (Hervault et al., 2019).

### 236 *2.6. Scalp activity measures*

237 The neural network engaged in inhibitory control (Aron, 2007; Lofredi et al., 2021) has been  
238 mainly associated to ERPs and time-frequency power visible at frontocentral sites, when  
239 investigated using EEG (Chikara et al., 2014; González-Villar et al., 2016; Huster et al., 2013;  
240 Lavallee et al., 2014; Wessel & Aron, 2013, 2015). According to this literature, the following  
241 analyses were performed using the three FCz, Cz and CPz EEG channels. In addition, the occipital  
242 Oz channel was added to the ERP analysis to evaluate the potential contribution of the attention  
243 in our task comparison. Especially, the visual N1 wave reflects the operation of a discrimination  
244 process within the focus of attention. Indeed, N1 appears to be larger when participants are  
245 performing discrimination tasks than when they are performing detection tasks (Vogel & Luck,  
246 2000).

247 *2.6.1. Event-related potentials*

248 For the main task's ERP, we averaged the EEG time series locked to the main stimulus onset (GO  
249 or CONTINUE) following the subtraction of a -200 to 0 ms pre-stimulus period as baseline. As  
250 STOP trials contained both the main stimulus (GO or CONTINUE) and the subsequent STOP  
251 signal, the STOP-signal-locked ERP cannot be directly and reliably computed as it may be  
252 contaminated by residual main-stimulus related activity (if not removed by the averaging  
253 process; Woldorff, 1993). For the rhythmic task, this overlapping problem was overcome by  
254 computing the neural activity difference of the main-stimulus locked ERP between STOP trials  
255 and CONTINUE trials, and then re-locking the STOP trial ERP to the STOP signal onset. This  
256 procedure, however, cannot be used for the discrete task, where the SOA delay (duration  
257 between GO stimulus and STOP signal) was adjusted across trials. To assess differences in the  
258 STOP-signal-processing irrespective of differences in the GO-stimulus processing, we  
259 computed, for the discrete task, the difference EEG activity separately for successful and failed  
260 STOP trials, in a similar manner as in previous studies (see Krämer et al., 2011; Ramautar et al.,  
261 2004, 2006). Specifically, GoRTs associated with the GO trials were rank-ordered, and then split  
262 into two parts. According to the horse-race model the fast and slow tails of the GoRTs  
263 distribution corresponded to the proportion of failed and successful STOP trials, respectively  
264 (Verbruggen & Logan, 2009a). Thus, ERPs were averaged across slow GO trials. This "virtual"  
265 GO-trials-ERP was then subtracted from successful STOP trials EEG data. STOP trials ERP was  
266 then computed and re-locked to the STOP-signal-onset accounting for the SOA delay of the  
267 successful STOP trials.

268 *2.6.2. Time-frequency analysis*



269 The EEG signals were convolved with complex 3 to 8-cycle-long Morlet's wavelets. Their central  
270 frequencies were changed from 0.5 to 50 Hz in 0.5 Hz steps. From the wavelet transformed  
271 signal,  $w_k(t, f)$ , of trial  $k$  at time  $t$  (2 ms time resolution) and with frequency  $f$ , the  
272 instantaneous power spectrum  $p_k(t, f) = R(w_k(t, f))^2 + I(w_k(t, f))^2$  and instantaneous  
273 phase  $\varphi_k(t, f) = \arctan\{I(w_k(t, f))/R(w_k(t, f))\}$  were extracted ( $R$  and  $I$  symbolize the real  
274 and imaginary parts of a complex number, respectively). Using the instantaneous power  
275 spectrum,  $p_k(t, f)$ , the average power spectrum was computed for each participant in the GO,  
276 CONTINUE, and STOP conditions as follow:

277 
$$Power = \frac{1}{N} \cdot \sum_{k=1}^N p_k(t, f), (N = \text{number of trials}).$$

278 As for the ERPs computation, GO and CONTINUE Power was subtracted from its respective STOP  
279 trials results (see above). Power was then normalized with respect to a -500 to -200 ms pre-  
280 stimulus baseline and transformed to decibel scale ( $10 \cdot \log_{10}$  of the signal).

### 281 2.6.3. Statistical analysis

282 To assess differences in the STOP-signal related neural activity between discrete and rhythmic  
283 action stopping, we subjected the individual ERP vectors and Power matrices to a non-  
284 parametric permutation procedure (Maris & Oostenveld, 2007). For this purpose, the 0 to 800  
285 ms time window of the participants' STOP-signal locked ERP was used. Regarding the individuals'  
286 Power matrices, we used a similar time-window associated with a 2 to 8 Hz frequency-window.  
287 These windows contains both the time (N2/P3) and frequency ranges (Delta/Theta) of interest  
288 to investigate inhibitory processes (Huster et al., 2013). A 0 to 300 ms window was used for the  
289 occipital Oz ERP permutation testing.

290 ERPs of a given EEG channel were subjected to a non-parametric permutation procedure, at the  
291 group level (Maris & Oostenveld, 2007). The 20 participants' windowed ERPs were pooled over  
292 the two STOP conditions (20 by conditions). Two sets of 20 ERPs each were then drawn  
293 randomly from this pool, and the differential grand-average ERP was computed between the  
294 two sets. This procedure was repeated 10 000 times, thus producing a distribution of these ERPs  
295 based on shuffled data under the null hypothesis. For each time point, a  $p$ -value was computed  
296 as the proportion of these pseudo-differential ERPs that exceeded the observed participants'  
297 average differential ERP. This  $p$ -value thus indicates at which time point the observed power  
298 distribution for the two conditions is more divergent than expected for random data ( $p = .05$   
299 threshold). To correct for multiple comparisons, we analyzed the resulting distributions of  $p$ -  
300 values to compute  $p$ -thresholds corresponding to the 2.5th percentile of the smallest, and the  
301 97.5th percentile of the largest  $p$ -values distribution (Cohen, 2014). This permutation analysis  
302 was similarly applied to each time-frequency point to assess the Power matrices significance  
303 between the two STOP conditions.

304 Next, brain-behavior correlations were computed. For each of the three frontocentral channel  
305 ERPs, N2 peak amplitude was computed by searching for the local peak with the minimal value  
306 in the 100–300 ms time range. N2 onset latency was defined as the time when half of the N2  
307 peak amplitude value was reached (Lopez-Calderon & Luck, 2014). Peak amplitude and onset  
308 latency were computed in the same way for the P3 wave but now by searching for the maximal  
309 value in the 200–500 ms range. ERP peak and onset detection were visually checked for each  
310 participant and each channel. In addition, the N2/P3 peak-to-peak amplitude was computed.  
311 Each of these five measures was then subject to a Pearson correlation test with the behavioral  
312 SSRT value. Resulting  $p$ -values were corrected using Bonferroni correction, that is, by multiplying

313 each  $p$ -value by the number of tests (i.e., five). Similarly, time-frequency Power peak amplitude  
314 and peak latency were computed and correlated to SSRT using the same correction (two tests).

### 315 3. Results

#### 316 3.1. Behavior

317 Main behavioral results are reported in **Table 1**. Importantly, FsRT (M = 424 ms, SD = 51 ms) was  
318 significantly shorter than GoRT (M = 474 ms, SD = 58 ms) for each single participant (paired-t-  
319 tests,  $p < .01$ ). The participants'  $p(\text{respond}/\text{signal})$  (M = .53, SD = .08) was not significantly  
320 different from .50 ( $t_{(19)} = 0.33$ ,  $p = .74$ ,  $CI_{95} [.44, .61]$ , *Cohen's d* = 0.37), and this probability  
321 increased sigmoidally with the SOA (i.e., the inhibition function). These analyses confirm the  
322 validity of the race-model between GO and STOP processes in the discrete task, validating the  
323 computation of the SSRTd in accordance with the model. In the rhythmic task, the spontaneous  
324 oscillation frequency was 1.60 Hz on average (SD = 0.55 Hz). Previous studies have shown that  
325 movement performed at this frequency were continuously rhythmic (Hermes et al., 2012;  
326 Seeber et al., 2016; Toma et al., 2002) and that the stopping latencies computed in the same  
327 task was not dependent of the movement phase (Hervault et al., 2019). The participants' mean  
328 Stop Time (M = 399, SD = 34 ms) and mean SSRT<sub>r</sub> (M = 268, SD = 24 ms) correlated strongly  
329 (*Pearson*  $r_{(18)} = .78$ ,  $p < .001$ ,  $CI_{95} [-.52, .91]$ ), which underlines the contribution of the inhibition  
330 process in the final stopping performance. Across the 20 participants, the SSRTd (M = 268 ms,  
331 SD = 52 ms) pertaining to the discrete task and the SSRT<sub>r</sub> (M = 270 ms, SD = 24 ms) pertaining to  
332 the rhythmic one did not differ (paired t-test,  $t_{(19)} = -0.08$ ,  $p = .93$ ,  $CI_{95} [-23 \text{ ms}, 21 \text{ ms}]$ ,  $d = 0.05$ ).  
333 Crucially though, the inhibition times of both tasks were unrelated across participants ( $r_{(18)} = -$   
334  $.04$ ,  $p = .86$ ,  $CI_{95} [-.47, .41]$ , **Fig.5**). As a validity check, we next verified whether for the rhythmic  
335 task the SSTR<sub>r</sub> values measured in the first 15 blocks of trials versus the second 15 blocks of  
336 trials were correlated, and found a positive significant correlation ( $r_{(18)} = .81$ ,  $p < .001$ ,  $CI_{95} [-.56,$   
337  $.92]$ ). To ensure that participants did not proactively anticipate the STOP signal occurrence, we  
338 tested whether the rhythmic movement was slow down at the SOA latency (Schultz et al., 2021).

339 Including all the rhythmic STOP trials of our participants, we compared the movement mean  
340 velocity of the 100 ms time window surrounding the STOP signal occurrence (-50 ms to + 50 ms)  
341 to the movement mean velocity of the 100 ms time window surrounding the CONTINUE  
342 stimulus occurrence. This comparison (paired *t*-test) failed to show a significant difference  
343 between the two time windows for both the x-velocity ( $t_{(2709)} = -0.10, p = .92$ ) and y-velocity  
344 ( $t_{(2709)} = 0.47, p = .64$ ), indicating that the participants did not proactively adapt their movements  
345 in either movement dimension.

### 346 **3.2. EEG time domain**

347 The ERP analysis showed that both N2 and P3 waves were evoked in the two stop-signal tasks,  
348 yet with clear differences between the discrete and rhythmic actions (**Fig. 3**). Indeed, the  
349 permutation analysis identified a significantly higher voltage in the discrete STOP condition as  
350 compared to the rhythmic STOP condition in the 218 - 458 ms time window at FCz, in the 204 -  
351 450 ms time window at Cz and in the 202 - 254 ms time window at CPz ( $p < .05$ , corrected). The  
352 topographies of the ERP plotted at N2 and P3 peak latency (**Fig. 3**) confirmed the frontocentral  
353 locus of the evoked activity, excepted for the N2 discrete STOP condition for which the small  
354 amplitude was associated to a less focused activity. In contrast, the Oz site showed a lower  
355 voltage in the discrete STOP condition, as compared to the rhythmic one, which was significant  
356 in the 228 - 280 ms time window. This difference was present after the early, negative, N1 wave  
357 (**Fig. 3**), probably occurring later than perceptual detection of the STOP signal (Hillyard et al.,  
358 1998; Luck, 2014).

359 The N2 and P3 peak values and onset latencies, as well as the N2/P3 peak-to-peak value were  
360 computed for each of the three frontocentral channels (FCz, Cz, CPz) and correlated to behav-  
361 ioral inhibition latencies. Mainly, at FCz site P3 onset latency was significantly correlated to SSRT

362 in the discrete STOP condition ( $r_{(18)} = .69$ , corrected  $p < .01$ ,  $CI_{95} [.35, .87]$ , **Fig. 5**) but not in the  
363 rhythmic one ( $r_{(18)} = .15$ ,  $p = .52$ ,  $CI_{95} [-.31, .55]$ ). In contrast, the N2/P3 peak-to-peak latency was  
364 significantly correlated to SSRT in the rhythmic STOP condition ( $r_{(18)} = -.57$ , corrected  $p = .04$ ,  
365  $CI_{95} [-.81, -.17]$ ) but not in the discrete one ( $r_{(18)} = -.21$ ,  $p = .37$ ,  $CI_{95} [-.59, .25]$ ). SSRT correlation  
366 with P3 onset latency was also significant at the Cz site in the discrete STOP condition ( $r_{(18)} = .50$ ,  
367 corrected  $p = .02$ ,  $CI_{95} [.08, .77]$ ). No other ERP-behavior correlation was significant.

### 368 **3.3. EEG time–frequency domain**

369 The grand average Delta/Theta–window STOP-signal locked power maps are displayed in **Fig. 4**.  
370 A clear frontocentral power increase was present in the Delta/Theta range after STOP signal  
371 occurrence, with differences between discrete and rhythmic conditions. Indeed, the permuta-  
372 tion analysis identified a significantly higher power in the discrete STOP condition as compared  
373 to the rhythmic STOP condition for time-frequency points included in a 3.5 - 6.5 Hz / 128 - 368  
374 window at FCz, in a 4.5 - 7 Hz / 268 - 382 window at Cz and a 4 - 7.5 Hz / 212 - 376 window at  
375 CPz ( $p < .05$ , corrected, **Fig. 4**).

376 The Power peak values and latencies were computed for each of the three frontocentral chan-  
377 nels (FCz, Cz, CPz) and correlated to behavioral inhibition latencies. Power peak latency was  
378 significantly correlated to SSRT in the discrete STOP condition when computed at FCz ( $r_{(18)} = .52$ ,  
379 corrected  $p = .04$ ,  $CI_{95} [.10, .79]$ ) and Cz sites ( $r_{(18)} = .60$ , corrected  $p = .01$ ,  $CI_{95} [.20, .83]$ ), whereas  
380 Power peak amplitude was significantly correlated to SSRT in the rhythmic STOP condition at FCz  
381 ( $r_{(18)} = -.53$ , corrected  $p = .04$ ,  $CI_{95} [-.79, -.09]$ ). No other Power-behavior correlation was signifi-  
382 cant.

383 **4. Discussion**

384 Prior work has investigated the scalp activity evoked by the successful cancellation of a  
385 prepared–discrete action (Huster et al., 2013). As inhibitory processes are supposed to  
386 generalize across action types, we tested whether EEG correlates of action cancellation were  
387 also observed when stopping an ongoing–rhythmic action. Comparing discrete and rhythmic  
388 movements, which are fundamentally distinct in terms of their dynamics (Hogan & Sternad,  
389 2007) and engage distinct brain areas (Schaal et al., 2004), proved a fruitful strategy to  
390 investigate the assumption that action inhibition is an action–independent process.

391 ***4.1. About unitary of action inhibition***

392 The behavioral analysis indicated that the SSRTs did not differ between the discrete and  
393 rhythmic tasks and were within the range found in previous studies, including various discrete  
394 responses (e.g., Boucher et al., 2007; Kok et al., 2004; Krämer et al., 2011; Montanari et al.,  
395 2017). Critically, the absence of a correlation between the 20 participants' inhibitory  
396 performances in the discrete and rhythmic tasks, a finding consistent with previous work  
397 (Hervault et al., 2019), challenges the notion that the processes inhibiting actions are  
398 fundamentally task–independent. This finding is strengthened by the strong positive correlation  
399 between blocks of rhythmic tasks, emphasizing that the absence of a correlation between the  
400 discrete and rhythmic task does not simply reflect weak performance reproducibility. It rather  
401 signifies that genuinely distinct processes, at least partly so, are implicated in the inhibition of  
402 discrete and rhythmic actions. The EEG analysis corroborated this conclusion: Although the  
403 STOP signals evoked a distinctive N2/P3–complex in the time domain and, concurrently, a  
404 Delta/Theta power increase in the frequency domain in both tasks, these inhibition–related EEG  
405 patterns differed significantly between tasks and, crucially, did so in a functionally relevant  
406 manner.

407 N2 and P3 waves were both evoked in the two tasks. Strikingly though, a later P3 onset latency  
408 was associated with a longer SSRT in the discrete task, but not in the rhythmic task. In contrast,  
409 a larger N2/P3 peak-to-peak amplitude was associated with a shorter SSRT in the rhythmic task,  
410 but not in the discrete one. For the discrete task, these findings confirm the P3 – SSRT  
411 correlation previously reported, with an earlier SSRTs being associated with higher P3s (e.g.,  
412 Huster et al., 2014), as well as earlier P3s (Anguera & Gazzaley, 2012; Wessel, 2018; Wessel &  
413 Aron, 2015); the reported correlation values were more inconsistent for the N2 – SSRT relation  
414 (e.g., Anguera & Gazzaley, 2012; Raud & Huster, 2017; Senderecka, 2016). Thus, N2 and P3 may  
415 act as partially dissociated correlates of inhibition processes, their importance varying as a  
416 function of the type of movement to revise. Such functional partial dissociation between  
417 discrete–task conditions was previously advocated by isolating the neural generators underlying  
418 the occurrence of two ERP waves in a combined GO/NoGO – stop–signal task. The anterior part  
419 of the mid–cingulate cortex was identified as the origin of the N2 differences between  
420 conditions (GO, NoGO, STOP), whereas the inferior frontal gyrus as well as the posterior part of  
421 the mid–cingulate cortex were identified as at the origin of the P3 differences (Enriquez–  
422 Geppert et al., 2010; Huster et al., 2010). In another framework, an fMRI frontocentral  
423 dissociation between discrete and continuous action generation has been reported in the mid–  
424 cingulate area (Schaal et al., 2004). While further investigation is needed to interpret these  
425 activations in the context of action inhibition, this dissociation may play a role in the differential  
426 involvement of N2 and P3 waves in action inhibition.

427 Our results also indicate that time–frequency elicited by the STOP signal in the Delta/Theta  
428 frequency range differed between the two tasks, this power increase being significantly related  
429 to inhibitory performance in both situations. Therefore, a STOP–signal–related Delta/Theta  
430 power increase can be viewed as more generically engaged in inhibitory processes. Indeed, a



431 Delta/Theta power increase has been associated with the conflictual/decisional requirement of  
432 tasks in various situations (Cavanagh et al., 2012; Harmony, 2013). Our findings are thus in line  
433 with the idea that action inhibition pertains to a conflict management between the regular main  
434 stimulus (GO or CONTINUE) and the occasional STOP signal. Taken together, the present findings  
435 demonstrate that SSRTs and related frontocentral EEG patterns differed between cancelling a  
436 prepared–discrete action and stopping an ongoing–rhythmic action, suggesting that the neural  
437 processes underlying inhibitory control are modulated as a function of action type.

#### 438 ***4.2. Different inhibitory subprocesses***

439 The STOP–related Delta/Theta power increase was visible in the two STOP conditions whereas  
440 the N2 and P3 ERP waves were more specifically engaged in rhythmic action stopping and  
441 discrete action cancelling, respectively. This dissociation between the STOP–signal evoked ERPs  
442 allows for different interpretations following different conceptualizations of cognitive  
443 functioning in inhibitory control.

444 First, this dissociation may indicate differences in conflict processing between the two tasks. N2  
445 and P3 waves are classically observed in conflictual situations, such as action inhibition  
446 (Ramautar et al., 2004), interference monitoring (Groom & Cragg, 2015), or task switching (Kopp  
447 et al., 2020). In GO/NoGO and stop–signal tasks, conflict arises whenever infrequent responses  
448 are required in the face of frequent responses (Braver et al., 2001; Mirabella, 2014). More  
449 specifically, high conflict occurs when infrequent STOP responses have to overcome the  
450 prepotency of frequent responses. In discrete inhibition tasks, previous work has shown that N2  
451 indicates the occurrence of the conflict in information processing, whereas P3 relates to the  
452 resolution of the conflict by inhibiting the action (Enriquez–Geppert et al., 2010; Randall &  
453 Smith, 2011). Interpreting our results along this line, conflict detection might be more crucially

454 engaged in stopping an ongoing action (as reflected by a larger N2 negativity), while conflict  
455 resolution would be so in cancelling a preplanned action (as reflected by a higher P3 wave). Still,  
456 the overall balance between the two conflict-related subprocesses may result in similar  
457 inhibition latencies, as reflected by the statistically indistinguishable SSRTs. The analysis of the  
458 occipital Oz channel revealed no significant difference between the tasks regarding the early  
459 visual N1 wave, suggesting that the two tasks did not differ regarding early attentional  
460 processes, but did so for later cognitive control engagement. The higher P3 amplitude observed  
461 in the discrete task may reflect the allocation of more resources in resolving the conflict, when  
462 the GO vs. STOP response is more conflictual than the CONTINUE vs. STOP response. This  
463 functional dissociation can find support in prior work requiring prepared-discrete actions  
464 (Enriquez-Geppert et al., 2010; Randall & Smith, 2011). To extend its plausibility in the context  
465 of ongoing-continuous actions, further investigation is needed manipulating the requirement  
466 associated to the infrequent signal (i.e., STOP, CONTINUE).

467 Second, the observed EEG dissociation between discrete movement cancellation and rhythmic  
468 movement stopping may reflect task-specific differences in brain predictive processing.  
469 Assuming that the brain is a predictive organ, cortical responses can be seen as transient  
470 expressions of prediction error (Friston, 2005). The P3 wave, which commonly follows low-  
471 probability signal occurrence, would index the update of a mental representation of the  
472 environment (Polich, 2007). Comparably, motor control has been modeled as a dual-  
473 representation system comparing the predicted and the actual states of the effector (Synofzik et  
474 al., 2008). Thus, P3 would reflect prediction error processing that is common to both  
475 sensorimotor and cognitive functions. In the case of movement generation, multiple studies  
476 have distinguished discrete and rhythmic continuous actions on the basis of the engaged  
477 representation of time (Ivry & Spencer, 2004; Zelaznik et al., 2002). Briefly, the timing control of

478 discrete actions requires an explicit process, that is, it depends on an explicit representation of  
479 the passage of time. In contrast, the timing of continuous actions is implicit as the temporal  
480 properties of the action are presumably emergent (i.e., an explicit representation of time does  
481 not directly guide performance). This fundamental distinction between the two action types  
482 may, in the present experiment, result in distinct mental predictions accompanying the  
483 processing of the GO and the CONTINUE stimuli (e.g., related to the temporal dimensions of the  
484 movement, Krigolson et al., 2008). The unexpected STOP signal causes a prediction error being  
485 indexed by P3 occurrence. Given that the STOP signal probability (25 %) was the same between  
486 the two tasks, as was the mean SOA, the differences in P3 measures may imply a prediction  
487 error modulation due to the discrete or continuous type of the action. Indeed, the explicit  
488 representation of time in the discrete task is plausibly accompanied by a “strong” prediction  
489 related to the movement execution. In contrast, the implicit timing processing in the rhythmic  
490 task can be accompanied by a “weak” prediction. Thus, the prediction violation evoked by the  
491 STOP signal might be extended in the discrete STOP trial as compared to the rhythmic STOP trial,  
492 as suggested by a higher P3 amplitude in the discrete condition. To further investigate this  
493 hypothesis, future EEG studies might manipulate the “prediction level” by varying the  
494 probability of the STOP signal (Dimoska & Johnstone, 2008) and/or by varying the SOA between  
495 the main stimulus and the STOP signal, in the two action types.

496 Third, another account on inhibitory control posits that performance in a stop–signal task  
497 encompasses both “reactive inhibition” and “proactive inhibition”. Thus, participants may adjust  
498 the response speed to the main stimulus when anticipating the occurrence of the improbable  
499 STOP signal (Verbruggen & Logan, 2009b). In the discrete task, these strategic adaptations allow  
500 adjusting the preparation of the response to the GO stimulus. In the rhythmic task, such  
501 adaptation appears unlikely, as there is no response preparation to adjust when the CONTINUE

502 stimulus appears while the movement is ongoing. In addition, no proactive slowing of the  
503 ongoing rhythmic movement was shown in the present study. Thus, action inhibition might  
504 involve both reactive and proactive processes in the discrete cancellation, whereas only reactive  
505 processes would be engaged in the rhythmic stopping. The proactive process, which modifies  
506 the neural implementation of inhibitory control (Kenemans, 2015; Leunissen et al., 2016), could  
507 thus cause the observed differences in brain activity engaged in the two tasks. To examine this  
508 tentative explanation, further EEG studies should manipulate cued-based proactive  
509 engagement (Verbruggen & Logan, 2009b) or capture the proactive/reactive balance through a  
510 kinematic movement analysis (Benedetti et al., 2020; Schultz et al., 2021).

#### 511 **4.3. Implications**

512 Inhibitory control is known to rely on a fronto-basal ganglia brain network (Aron, 2011; Lofredi  
513 et al., 2021). Recent studies found this network to become active when facing various  
514 unexpected events, leading to a “*unified theory*” of inhibition (Wessel & Aron, 2017). Our  
515 findings are incompatible with the view of action inhibition as a unitary construct but are readily  
516 accommodated in a framework holding that different components of a unique fronto-basal  
517 ganglia inhibition network are implicated in inhibition in an action-type dependent manner.  
518 Thus, given a potential dissociation within the cingulate cortex enrollment (see also section  
519 4.1.), the relative engagement of different parts of the inhibitory network may vary as a function  
520 of the action type. Along this line, the fronto-basal ganglia brain network was recently shown to  
521 be differentially engaged depending on the task constraints, such as facing STOP or NoGO  
522 signals (Raud et al., 2020).

523 The activation of different neural subprocesses depending on action type has two important  
524 implications. The first is technological. Devices such as a wheelchair, a robotic arm, or a drone

525 can be controlled through different kinds of movements via brain–computer interfaces (BCI).  
526 Chikara and Ko (2019) suggested that the neural activity related to inhibition, particularly the P3  
527 wave, can be used as a stop signal. However, given the assumption of dissociate processes of  
528 action inhibition, cancelling a prepared–discrete response (e.g., grasping the wrong object) and  
529 stopping an ongoing–continuous action (e.g., wheelchair displacement) might require dissociate  
530 neural command based on P3 and N2/P3 peak-to-peak amplitude, respectively. The second is  
531 methodological and clinical. While the “discrete” procedure for SSRT estimation leads classically  
532 to a single across-trials SSRT per participant, ongoing–continuous actions offer a way of  
533 measuring single–trial SSRTs that would not depend on an inhibition function inferred  
534 statistically. This within–trial variability would be of particular interest for assessing (action)  
535 inhibition in troubles such as Attention–Deficit / Hyperactivity Disorder (ADHD) (Morein–Zamir  
536 et al., 2008). This disorder has been associated with longer SSRT and specific ERP signatures in  
537 discrete–response cancellation tasks (Bekker et al., 2005). Nonetheless, given the temporal  
538 variability characteristic of ADHD, a more variable inhibition latency might be a singular  
539 characteristic of the ADHD population, rather than a longer latency (Castellanos et al., 2006;  
540 Lijffijt et al., 2005). Consequently, tasks allowing for within–trial SSRT determination, such as the  
541 rhythmic task used here, could be instrumental in revealing an inhibitory deficiency in ADHD.

#### 542 **4.4. Conclusion**

543 The present article raises a key question about inhibitory control: Are the neural mechanisms  
544 underlying inhibition action–independent or action–specific? We addressed this issue by  
545 comparing cancelling a prepared–discrete response and stopping an ongoing–rhythmic action.  
546 Behavioral analysis provided a first indication that inhibition of the two action types is controlled  
547 differently. Consistent therewith, EEG analysis further revealed that the components of the  
548 frontocentral N2/P3–complex are implicated in inhibition in an action–type dependent manner.

549 Extending our understanding of inhibition requires the identification of the task constraints that  
550 set inhibition “types” apart versus those that do not, and to spell out principles allowing for an  
551 unambiguous delineation of such types in neural space.

552 **Acknowledgements**

553 All authors gave approval of the final submitted version. The authors wish to thank all  
554 participants in this research.

555 **Declaration of conflicting interests**

556 There are no conflicting interests.

557 **Funding**

558 This research did not receive any specific grant from funding agencies in the public, commercial,  
559 or not-for-profit sectors.

560 **References**

- 561 Alegre, M., Alvarez-Gerriko, I., Valencia, M., Iriarte, J., & Artieda, J. (2008). Oscillatory changes  
562 related to the forced termination of a movement. *Clinical Neurophysiology*, *119*(2), 290–  
563 300. <https://doi.org/10.1016/j.clinph.2007.10.017>
- 564 Anguera, J. A., & Gazzaley, A. (2012). Dissociation of motor and sensory inhibition processes in  
565 normal aging. *Clinical Neurophysiology*, *123*(4), 730–740.  
566 <https://doi.org/10.1016/j.clinph.2011.08.024>
- 567 Aron, A. R. (2007). The neural basis of inhibition in cognitive control. *The Neuroscientist: A*  
568 *Review Journal Bringing Neurobiology, Neurology and Psychiatry*, *13*(3), 214–228.  
569 <https://doi.org/10.1177/1073858407299288>
- 570 Aron, A. R. (2011). From reactive to proactive and selective control: Developing a richer model  
571 for stopping inappropriate responses. *Biological Psychiatry*, *69*(12), e55-68.  
572 <https://doi.org/10.1016/j.biopsych.2010.07.024>
- 573 Band, G. P., & van Boxtel, G. J. (1999). Inhibitory motor control in stop paradigms: Review and  
574 reinterpretation of neural mechanisms. *Acta Psychologica*, *101*(2–3), 179–211.
- 575 Bari, A., & Robbins, T. W. (2013). Inhibition and impulsivity: Behavioral and neural basis of  
576 response control. *Progress in Neurobiology*, *108*, 44–79.  
577 <https://doi.org/10.1016/j.pneurobio.2013.06.005>
- 578 Bekker, E. M., Overtoom, C. C. E., Kooij, J. J. S., Buitelaar, J. K., Verbaten, M. N., & Kenemans, J. L.  
579 (2005). Disentangling deficits in adults with attention-deficit/hyperactivity disorder.  
580 *Archives of General Psychiatry*, *62*(10), 1129–1136.  
581 <https://doi.org/10.1001/archpsyc.62.10.1129>

582 Bell, A. J., & Sejnowski, T. J. (1995). An information-maximization approach to blind separation  
583 and blind deconvolution. *Neural Computation*, *7*(6), 1129–1159.  
584 <https://doi.org/10.1162/neco.1995.7.6.1129>

585 Benedetti, V., Gavazzi, G., Giovannelli, F., Bravi, R., Giganti, F., Minciacchi, D., Mascalchi, M.,  
586 Cincotta, M., & Viggiano, M. P. (2020). Mouse Tracking to Explore Motor Inhibition  
587 Processes in Go/No-Go and Stop Signal Tasks. *Brain Sciences*, *10*(7), 464.  
588 <https://doi.org/10.3390/brainsci10070464>

589 Boecker, M., Gauggel, S., & Druke, B. (2013). Stop or stop-change—Does it make any difference  
590 for the inhibition process? *International Journal of Psychophysiology*, *87*(3), 234–243.  
591 <https://doi.org/10.1016/j.ijpsycho.2012.09.009>

592 Boucher, L., Stuphorn, V., Logan, G. D., Schall, J. D., & Palmeri, T. J. (2007). Stopping eye and  
593 hand movements: Are the processes independent? *Perception & Psychophysics*, *69*(5),  
594 785–801. <https://doi.org/10.3758/BF03193779>

595 Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate  
596 cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex*  
597 (*New York, N.Y.: 1991*), *11*(9), 825–836. <https://doi.org/10.1093/cercor/11.9.825>

598 Bryden, M. P. (1977). Measuring handedness with questionnaires. *Neuropsychologia*, *15*(4–5),  
599 617–624. [https://doi.org/10.1016/0028-3932\(77\)90067-7](https://doi.org/10.1016/0028-3932(77)90067-7)

600 Castellanos, F. X., Sonuga-Barke, E. J. S., Milham, M. P., & Tannock, R. (2006). Characterizing  
601 cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences*, *10*(3),  
602 117–123. <https://doi.org/10.1016/j.tics.2006.01.011>

603 Cavanagh, J. F., Zambrano-Vazquez, L., & Allen, J. J. B. (2012). Theta lingua franca: A common  
604 mid-frontal substrate for action monitoring processes. *Psychophysiology*, *49*(2), 220–  
605 238. <https://doi.org/10.1111/j.1469-8986.2011.01293.x>



606 Chikara, R. K., & Ko, L.-W. (2019). Neural Activities Classification of Human Inhibitory Control  
607 Using Hierarchical Model. *Sensors*, *19*(17), 3791. <https://doi.org/10.3390/s19173791>

608 Chikara, R. K., Perumal, R., Ko, L.-W., & Chen, H. (2014). EEG dynamics in inhibition of left-hand  
609 and right-hand responses during auditory stop-signal task. *2014 IEEE Symposium on*  
610 *Computational Intelligence, Cognitive Algorithms, Mind, and Brain (CCMB)*, 115–120.  
611 <https://doi.org/10.1109/CCMB.2014.7020703>

612 Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2. ed., reprint).  
613 Psychology Press.

614 Cohen, M. X. (2014). *Analyzing Neural Time Series Data – Theory and Practice* (1st ed.). MIT  
615 Press.

616 Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG  
617 dynamics including independent component analysis. *Journal of Neuroscience Methods*,  
618 *134*(1), 9–21. <https://doi.org/10.1016/j.jneumeth.2003.10.009>

619 Diamond, A. (2013). Executive Functions. *Annual Review of Psychology*, *64*(1), 135–168.  
620 <https://doi.org/10.1146/annurev-psych-113011-143750>

621 Dimoska, A., & Johnstone, S. J. (2008). Effects of varying stop-signal probability on ERPs in the  
622 stop-signal task: Do they reflect variations in inhibitory processing or simply novelty  
623 effects? *Biological Psychology*, *77*(3), 324–336.  
624 <https://doi.org/10.1016/j.biopsycho.2007.11.005>

625 Enriquez-Geppert, S., Konrad, C., Pantev, C., & Huster, R. J. (2010). Conflict and inhibition  
626 differentially affect the N200/P300 complex in a combined go/nogo and stop-signal task.  
627 *NeuroImage*, *51*(2), 877–887. <https://doi.org/10.1016/j.neuroimage.2010.02.043>

628 Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using  
629 G\*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*,  
630 41(4), 1149–1160. <https://doi.org/10.3758/BRM.41.4.1149>

631 Friston, K. (2005). A theory of cortical responses. *Philosophical Transactions of the Royal Society*  
632 *B: Biological Sciences*, 360(1456), 815–836. <https://doi.org/10.1098/rstb.2005.1622>

633 González-Villar, A. J., Bonilla, F. M., & Carrillo-de-la-Peña, M. T. (2016). When the brain simulates  
634 stopping: Neural activity recorded during real and imagined stop-signal tasks. *Cognitive*,  
635 *Affective, & Behavioral Neuroscience*, 16(5), 825–835. [https://doi.org/10.3758/s13415-](https://doi.org/10.3758/s13415-016-0434-3)  
636 016-0434-3

637 Groom, M. J., & Cragg, L. (2015). Differential modulation of the N2 and P3 event-related  
638 potentials by response conflict and inhibition. *Brain and Cognition*, 97, 1–9.  
639 <https://doi.org/10.1016/j.bandc.2015.04.004>

640 Hannah, R., & Aron, A. R. (2021). Towards real-world generalizability of a circuit for action-  
641 stopping. *Nature Reviews Neuroscience*, 22(9), 538–552.  
642 <https://doi.org/10.1038/s41583-021-00485-1>

643 Harmony, T. (2013). The functional significance of delta oscillations in cognitive processing.  
644 *Frontiers in Integrative Neuroscience*, 7. <https://doi.org/10.3389/fnint.2013.00083>

645 Hatta, A., Nishihira, Y., Kaneda, T., Wasaka, T., Kida, T., Kuroiwa, K., & Akiyama, S. (2003).  
646 Somatosensory event-related potentials (ERPs) associated with stopping ongoing  
647 movement. *Perceptual and Motor Skills*, 97(3 Pt 1), 895–904.  
648 <https://doi.org/10.2466/pms.2003.97.3.895>

649 Hermes, D., Siero, J. C. W., Aarnoutse, E. J., Leijten, F. S. S., Petridou, N., & Ramsey, N. F. (2012).  
650 Dissociation between Neuronal Activity in Sensorimotor Cortex and Hand Movement

651 Revealed as a Function of Movement Rate. *Journal of Neuroscience*, 32(28), 9736–9744.  
652 <https://doi.org/10.1523/JNEUROSCI.0357-12.2012>

653 Hervault, M., Huys, R., Farrer, C., Buisson, J. C., & Zanone, P. G. (2019). Cancelling discrete and  
654 stopping ongoing rhythmic movements: Do they involve the same process of motor  
655 inhibition? *Human Movement Science*, 64, 296–306.  
656 <https://doi.org/10.1016/j.humov.2019.02.010>

657 Hillyard, S. A., Vogel, E. K., & Luck, S. J. (1998). Sensory gain control (amplification) as a  
658 mechanism of selective attention: Electrophysiological and neuroimaging evidence.  
659 *Philosophical Transactions of the Royal Society B: Biological Sciences*, 353(1373), 1257–  
660 1270.

661 Hogan, N., & Sternad, D. (2007). On rhythmic and discrete movements: Reflections, definitions  
662 and implications for motor control. *Experimental Brain Research*, 181(1), 13–30.  
663 <https://doi.org/10.1007/s00221-007-0899-y>

664 Huster, R. J., Enriquez-Geppert, S., Lavallee, C. F., Falkenstein, M., & Herrmann, C. S. (2013).  
665 Electroencephalography of response inhibition tasks: Functional networks and cognitive  
666 contributions. *International Journal of Psychophysiology: Official Journal of the*  
667 *International Organization of Psychophysiology*, 87(3), 217–233.  
668 <https://doi.org/10.1016/j.ijpsycho.2012.08.001>

669 Huster, R. J., Messel, M. S., Thunberg, C., & Raud, L. (2020). The P300 as marker of inhibitory  
670 control – Fact or fiction? *Cortex*. <https://doi.org/10.1016/j.cortex.2020.05.021>

671 Huster, R. J., Plis, S. M., Lavallee, C. F., Calhoun, V. D., & Herrmann, C. S. (2014). Functional and  
672 effective connectivity of stopping. *NeuroImage*, 94, 120–128.  
673 <https://doi.org/10.1016/j.neuroimage.2014.02.034>

674 Huster, R. J., Westerhausen, R., Pantev, C., & Konrad, C. (2010). The role of the cingulate cortex  
675 as neural generator of the N200 and P300 in a tactile response inhibition task. *Human*  
676 *Brain Mapping, 31*(8), 1260–1271. <https://doi.org/10.1002/hbm.20933>

677 Huys, R., Studenka, B. E., Rheaume, N. L., Zelaznik, H. N., & Jirsa, V. K. (2008). Distinct Timing  
678 Mechanisms Produce Discrete and Continuous Movements. *PLoS Computational*  
679 *Biology, 4*(4), e1000061. <https://doi.org/10.1371/journal.pcbi.1000061>

680 Huys, R., Studenka, B. E., Zelaznik, H. N., & Jirsa, V. K. (2010). Distinct timing mechanisms are  
681 implicated in distinct circle drawing tasks. *Neuroscience Letters, 472*(1), 24–28.  
682 <https://doi.org/10.1016/j.neulet.2010.01.047>

683 Hynd, M., Soh, C., Rangel, B. O., & Wessel, J. R. (2020). Latency and amplitude of the stop-signal  
684 P3 event-related potential are related to inhibitory GABA<sub>A</sub> activity in primary motor  
685 cortex. *BioRxiv*, 2020.09.15.298711. <https://doi.org/10.1101/2020.09.15.298711>

686 Ivry, R. B., & Spencer, R. M. C. (2004). The neural representation of time. *Current Opinion in*  
687 *Neurobiology, 14*(2), 225–232. <https://doi.org/10.1016/j.conb.2004.03.013>

688 Kenemans, J. L. (2015). Specific proactive and generic reactive inhibition. *Neuroscience and*  
689 *Biobehavioral Reviews, 56*, 115–126. <https://doi.org/10.1016/j.neubiorev.2015.06.011>

690 Kok, A., Ramautar, J. R., De Ruiter, M. B., Band, G. P. H., & Ridderinkhof, K. R. (2004). ERP  
691 components associated with successful and unsuccessful stopping in a stop-signal task.  
692 *Psychophysiology, 41*(1), 9–20. <https://doi.org/10.1046/j.1469-8986.2003.00127.x>

693 Kopp, B., Steinke, A., & Visalli, A. (2020). Cognitive flexibility and N2/P3 event-related brain  
694 potentials. *Scientific Reports, 10*(1), 9859. <https://doi.org/10.1038/s41598-020-66781-5>

695 Krämer, U. M., Knight, R. T., & Münte, T. F. (2011). Electrophysiological evidence for different  
696 inhibitory mechanisms when stopping or changing a planned response. *Journal of*  
697 *Cognitive Neuroscience, 23*(9), 2481–2493. <https://doi.org/10.1162/jocn.2010.21573>

698 Krigolson, O. E., Holroyd, C. B., Van Gyn, G., & Heath, M. (2008). Electroencephalographic  
699 correlates of target and outcome errors. *Experimental Brain Research*, *190*(4), 401–411.  
700 <https://doi.org/10.1007/s00221-008-1482-x>

701 Kuzstor, A., Raud, L., Juel, B. E., Nilsen, A. S., Storm, J. F., & Huster, R. J. (2019). Sleep deprivation  
702 differentially affects subcomponents of cognitive control. *Sleep*, *42*(4).  
703 <https://doi.org/10.1093/sleep/zsz016>

704 Lansbergen, M. M., Böcker, K. B. E., Bekker, E. M., & Kenemans, J. L. (2007). Neural correlates of  
705 stopping and self-reported impulsivity. *Clinical Neurophysiology: Official Journal of the*  
706 *International Federation of Clinical Neurophysiology*, *118*(9), 2089–2103.  
707 <https://doi.org/10.1016/j.clinph.2007.06.011>

708 Lavallee, C. F., Meemken, M. T., Herrmann, C. S., & Huster, R. J. (2014). When holding your  
709 horses meets the deer in the headlights: Time-frequency characteristics of global and  
710 selective stopping under conditions of proactive and reactive control. *Frontiers in*  
711 *Human Neuroscience*, *8*. <https://doi.org/10.3389/fnhum.2014.00994>

712 Leunissen, I., Coxon, J. P., & Swinnen, S. P. (2016). A proactive task set influences how response  
713 inhibition is implemented in the basal ganglia. *Human Brain Mapping*, *37*(12), 4706–  
714 4717. <https://doi.org/10.1002/hbm.23338>

715 Lijffijt, M., Kenemans, J. L., Verbaten, M. N., & van Engeland, H. (2005). A meta-analytic review  
716 of stopping performance in attention-deficit/hyperactivity disorder: Deficient inhibitory  
717 motor control? *Journal of Abnormal Psychology*, *114*(2), 216–222.  
718 <https://doi.org/10.1037/0021-843X.114.2.216>

719 Lofredi, R., Auernig, G. C., Irmen, F., Nieweler, J., Neumann, W.-J., Horn, A., Schneider, G.-H., &  
720 Kühn, A. A. (2021). Subthalamic stimulation impairs stopping of ongoing movements.  
721 *Brain*, *144*(1), 44–52. <https://doi.org/10.1093/brain/awaa341>

722 Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an  
723 act of control. *Psychological Review*, *91*(3), 295–327. [https://doi.org/10.1037/0033-](https://doi.org/10.1037/0033-295X.91.3.295)  
724 [295X.91.3.295](https://doi.org/10.1037/0033-295X.91.3.295)

725 Lopez-Calderon, J., & Luck, S. J. (2014). ERPLAB: An open-source toolbox for the analysis of  
726 event-related potentials. *Frontiers in Human Neuroscience*, *8*.  
727 <https://doi.org/10.3389/fnhum.2014.00213>

728 Luck, S. J. (2014). *An Introduction to the Event-Related Potential Technique*. MIT Press.

729 Maris, E., & Oostenveld, R. (2007). Nonparametric statistical testing of EEG- and MEG-data.  
730 *Journal of Neuroscience Methods*, *164*(1), 177–190.  
731 <https://doi.org/10.1016/j.jneumeth.2007.03.024>

732 Matzke, D., Verbruggen, F., & Logan, G. D. (2018). The Stop-Signal Paradigm. In *Stevens’*  
733 *Handbook of Experimental Psychology and Cognitive Neuroscience* (pp. 1–45). American  
734 Cancer Society. <https://doi.org/10.1002/9781119170174.epcn510>

735 Mirabella, G. (2014). Should I stay or should I go? Conceptual underpinnings of goal-directed  
736 actions. *Frontiers in Systems Neuroscience*, *8*, 6.  
737 <https://doi.org/10.3389/fnsys.2014.00206>

738 Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000).  
739 The Unity and Diversity of Executive Functions and Their Contributions to Complex  
740 “Frontal Lobe” Tasks: A Latent Variable Analysis. *Cognitive Psychology*, *41*(1), 49–100.  
741 <https://doi.org/10.1006/cogp.1999.0734>

742 Montanari, R., Giamundo, M., Brunamonti, E., Ferraina, S., & Pani, P. (2017). Visual salience of  
743 the stop-signal affects movement suppression process. *Experimental Brain Research*,  
744 *235*(7), 2203–2214. <https://doi.org/10.1007/s00221-017-4961-0>

745 Morein-Zamir, S., Chua, R., Franks, I., Nagelkerke, P., & Kingstone, A. (2006). Measuring online  
746 volitional response control with a continuous tracking task. *Behavior Research Methods*,  
747 38(4), 638–647. <https://doi.org/10.3758/BF03193896>

748 Morein-Zamir, S., Hommersen, P., Johnston, C., & Kingstone, A. (2008). Novel measures of  
749 response performance and inhibition in children with ADHD. *Journal of Abnormal Child*  
750 *Psychology*, 36(8), 1199–1210. <https://doi.org/10.1007/s10802-008-9243-7>

751 Morein-Zamir, S., Nagelkerke, P., Chua, R., Franks, I., & Kingstone, A. (2004). Inhibiting prepared  
752 and ongoing responses: Is there more than one kind of stopping? *Psychonomic Bulletin*  
753 *& Review*, 11(6), 1034–1040. <https://doi.org/10.3758/BF03196733>

754 Pion-Tonachini, L., Kreutz-Delgado, K., & Makeig, S. (2019). ICLabel: An automated  
755 electroencephalographic independent component classifier, dataset, and website.  
756 *NeuroImage*, 198, 181–197. <https://doi.org/10.1016/j.neuroimage.2019.05.026>

757 Polich, J. (2007). Updating P300: An Integrative Theory of P3a and P3b. *Clinical*  
758 *Neurophysiology : Official Journal of the International Federation of Clinical*  
759 *Neurophysiology*, 118(10), 2128–2148. <https://doi.org/10.1016/j.clinph.2007.04.019>

760 Ramautar, J. R., Kok, A., & Ridderinkhof, K. R. (2004). Effects of stop-signal probability in the  
761 stop-signal paradigm: The N2/P3 complex further validated. *Brain and Cognition*, 56(2),  
762 234–252. <https://doi.org/10.1016/j.bandc.2004.07.002>

763 Ramautar, J. R., Kok, A., & Ridderinkhof, K. R. (2006). Effects of stop-signal modality on the  
764 N2/P3 complex elicited in the stop-signal paradigm. *Biological Psychology*, 72(1), 96–  
765 109. <https://doi.org/10.1016/j.biopsycho.2005.08.001>

766 Randall, W. M., & Smith, J. L. (2011). Conflict and inhibition in the cued-Go/NoGo task. *Clinical*  
767 *Neurophysiology*, 122(12), 2400–2407. <https://doi.org/10.1016/j.clinph.2011.05.012>

768 Raud, L., & Huster, R. J. (2017). The Temporal Dynamics of Response Inhibition and their  
769 Modulation by Cognitive Control. *Brain Topography*, 30(4), 486–501.  
770 <https://doi.org/10.1007/s10548-017-0566-y>

771 Raud, L., Westerhausen, R., Dooley, N., & Huster, R. J. (2020). Differences in unity: The go/no-go  
772 and stop signal tasks rely on different mechanisms. *NeuroImage*, 210, 116582.  
773 <https://doi.org/10.1016/j.neuroimage.2020.116582>

774 Sawilowsky, S. (2009). New Effect Size Rules of Thumb. *Theoretical and Behavioral Foundations*  
775 *of Education Faculty Publications*. [https://digitalcommons.wayne.edu/coe\\_tbf/4](https://digitalcommons.wayne.edu/coe_tbf/4)

776 Schaal, S., Sternad, D., Osu, R., & Kawato, M. (2004). Rhythmic arm movement is not discrete.  
777 *Nature Neuroscience*, 7(10), 1136–1143. <https://doi.org/10.1038/nn1322>

778 Schall, J. D., Palmeri, T. J., & Logan, G. D. (2017). Models of inhibitory control. *Philosophical*  
779 *Transactions of the Royal Society of London. Series B, Biological Sciences*, 372(1718).  
780 <https://doi.org/10.1098/rstb.2016.0193>

781 Schultz, K. E., Denning, D., Hufnagel, V., & Swann, N. (2021). Stopping a Continuous Movement:  
782 A Novel Approach to Investigating Motor Control. *BioRxiv*, 2021.04.08.439070.  
783 <https://doi.org/10.1101/2021.04.08.439070>

784 Seeber, M., Scherer, R., & Müller-Putz, G. R. (2016). EEG Oscillations Are Modulated in Different  
785 Behavior-Related Networks during Rhythmic Finger Movements. *The Journal of*  
786 *Neuroscience*, 36(46), 11671–11681. <https://doi.org/10.1523/JNEUROSCI.1739-16.2016>

787 Senderecka, M. (2016). Threatening visual stimuli influence response inhibition and error  
788 monitoring: An event-related potential study. *Biological Psychology*, 113, 24–36.  
789 <https://doi.org/10.1016/j.biopsycho.2015.11.003>



790 Sosnik, R., Chaim, E., & Flash, T. (2015). Stopping is not an option: The evolution of unstoppable  
791 motion elements (primitives). *Journal of Neurophysiology*, *114*(2), 846–856.  
792 <https://doi.org/10.1152/jn.00341.2015>

793 Spencer, R. M. C., Zelaznik, H. N., Diedrichsen, J., & Ivry, R. B. (2003). Disrupted timing of  
794 discontinuous but not continuous movements by cerebellar lesions. *Science (New York,*  
795 *N.Y.)*, *300*(5624), 1437–1439. <https://doi.org/10.1126/science.1083661>

796 Synofzik, M., Vosgerau, G., & Newen, A. (2008). Beyond the comparator model: A multifactorial  
797 two-step account of agency. *Consciousness and Cognition*, *17*(1), 219–239.  
798 <https://doi.org/10.1016/j.concog.2007.03.010>

799 Toma, K., Mima, T., Matsuoka, T., Gerloff, C., Ohnishi, T., Koshy, B., Andres, F., & Hallett, M.  
800 (2002). Movement Rate Effect on Activation and Functional Coupling of Motor Cortical  
801 Areas. *Journal of Neurophysiology*, *88*(6), 3377–3385.  
802 <https://doi.org/10.1152/jn.00281.2002>

803 Tschuemperlin, R. M., Stein, M., Batschelet, H. M., Moggi, F., & Soravia, L. M. (2019). Learning to  
804 resist the urge: A double-blind, randomized controlled trial investigating alcohol-specific  
805 inhibition training in abstinent patients with alcohol use disorder. *Trials*, *20*(1), 402.  
806 <https://doi.org/10.1186/s13063-019-3505-2>

807 Vahid, A., Mückschel, M., Neuhaus, A., Stock, A.-K., & Beste, C. (2018). Machine learning  
808 provides novel neurophysiological features that predict performance to inhibit  
809 automated responses. *Scientific Reports*, *8*(1), 16235. [https://doi.org/10.1038/s41598-](https://doi.org/10.1038/s41598-018-34727-7)  
810 [018-34727-7](https://doi.org/10.1038/s41598-018-34727-7)

811 Van Voorhis, A. C., Kent, J. S., Kang, S. S., Goghari, V. M., MacDonald, A. W., & Sponheim, S. R.  
812 (2019). Abnormal neural functions associated with motor inhibition deficits in

813 schizophrenia and bipolar disorder. *Human Brain Mapping*, 40(18), 5397–5411.  
814 <https://doi.org/10.1002/hbm.24780>

815 Verbruggen, F., Aron, A. R., Band, G. P., Beste, C., Bissett, P. G., Brockett, A. T., Brown, J. W.,  
816 Chamberlain, S. R., Chambers, C. D., Colonius, H., Colzato, L. S., Corneil, B. D., Coxon, J.  
817 P., Dupuis, A., Eagle, D. M., Garavan, H., Greenhouse, I., Heathcote, A., Huster, R. J., ...  
818 Boehler, C. N. (2019). A consensus guide to capturing the ability to inhibit actions and  
819 impulsive behaviors in the stop-signal task. *ELife*, 8, e46323.  
820 <https://doi.org/10.7554/eLife.46323>

821 Verbruggen, F., & Logan, G. D. (2009a). Models of response inhibition in the stop-signal and  
822 stop-change paradigms. *Neuroscience and Biobehavioral Reviews*, 33(5), 647–661.  
823 <https://doi.org/10.1016/j.neubiorev.2008.08.014>

824 Verbruggen, F., & Logan, G. D. (2009b). Proactive adjustments of response strategies in the stop-  
825 signal paradigm. *Journal of Experimental Psychology. Human Perception and*  
826 *Performance*, 35(3), 835–854. <https://doi.org/10.1037/a0012726>

827 Verbruggen, F., Schneider, D. W., & Logan, G. D. (2008). How to stop and change a response:  
828 The role of goal activation in multitasking. *Journal of Experimental Psychology. Human*  
829 *Perception and Performance*, 34(5), 1212–1228. [https://doi.org/10.1037/0096-](https://doi.org/10.1037/0096-1523.34.5.1212)  
830 [1523.34.5.1212](https://doi.org/10.1037/0096-1523.34.5.1212)

831 Vogel, E. K., & Luck, S. J. (2000). The visual N1 component as an index of a discrimination  
832 process. *Psychophysiology*, 37(2), 190–203.

833 Waller, D. A., Hazeltine, E., & Wessel, J. R. (2019). Common neural processes during action-  
834 stopping and infrequent stimulus detection: The frontocentral P3 as an index of generic  
835 motor inhibition. *International Journal of Psychophysiology*.  
836 <https://doi.org/10.1016/j.ijpsycho.2019.01.004>

837 Wessel, J. R. (2018). Prepotent motor activity and inhibitory control demands in different  
838 variants of the go/no-go paradigm. *Psychophysiology*, 55(3), e12871.  
839 <https://doi.org/10.1111/psyp.12871>

840 Wessel, J. R., & Aron, A. R. (2013). Unexpected Events Induce Motor Slowing via a Brain  
841 Mechanism for Action-Stopping with Global Suppressive Effects. *Journal of*  
842 *Neuroscience*, 33(47), 18481–18491. <https://doi.org/10.1523/JNEUROSCI.3456-13.2013>

843 Wessel, J. R., & Aron, A. R. (2015). It's not too late: The onset of the frontocentral P3 indexes  
844 successful response inhibition in the stop-signal paradigm. *Psychophysiology*, 52(4),  
845 472–480. <https://doi.org/10.1111/psyp.12374>

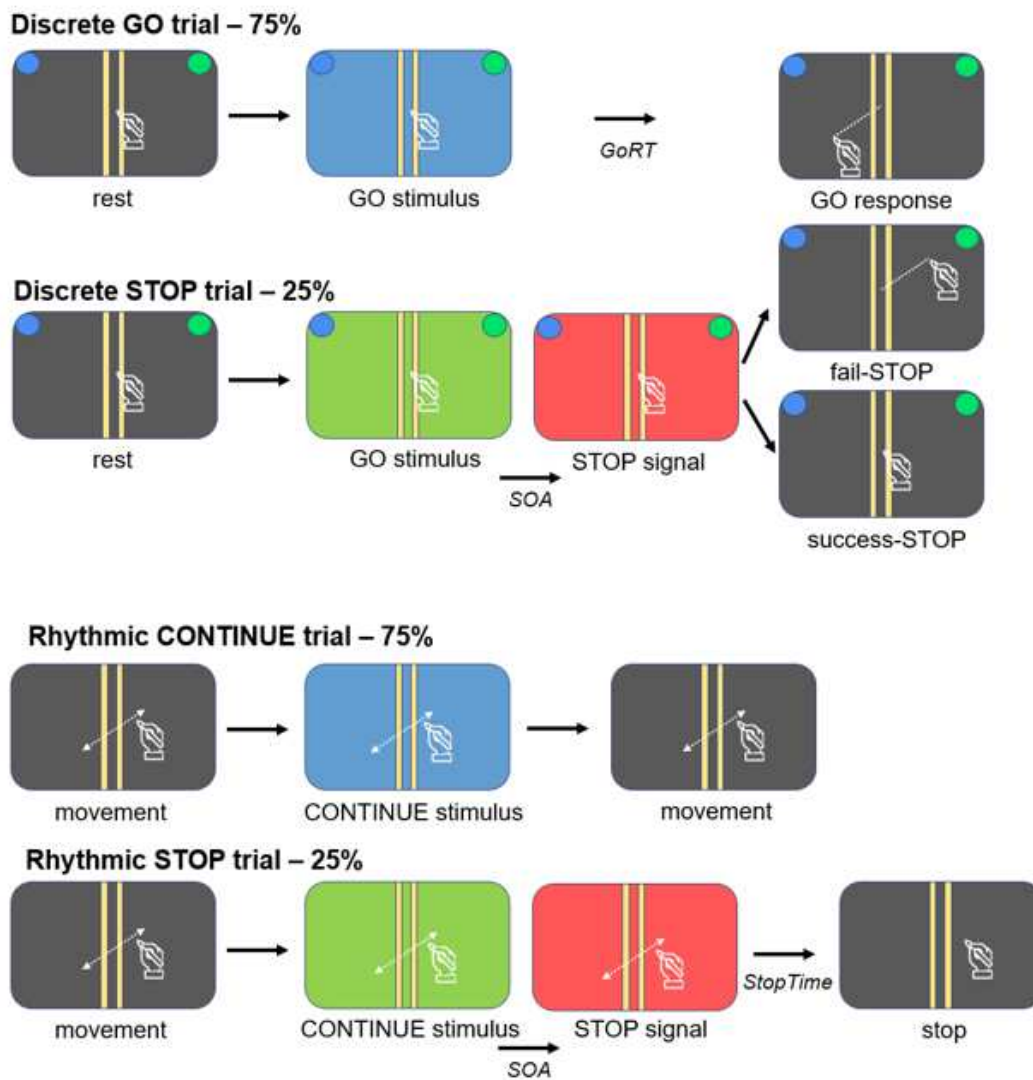
846 Wessel, J. R., & Aron, A. R. (2017). On the Globality of Motor Suppression: Unexpected Events  
847 and Their Influence on Behavior and Cognition. *Neuron*, 93(2), 259–280.  
848 <https://doi.org/10.1016/j.neuron.2016.12.013>

849 Wiegel, P., Kurz, A., & Leukel, C. (2020). Evidence that distinct human primary motor cortex  
850 circuits control discrete and rhythmic movements. *The Journal of Physiology*, 598(6),  
851 1235–1251. <https://doi.org/10.1113/JP278779>

852 Woldorff, M. G. (1993). Distortion of ERP averages due to overlap from temporally adjacent  
853 ERPs: Analysis and correction. *Psychophysiology*, 30(1), 98–119.  
854 <https://doi.org/10.1111/j.1469-8986.1993.tb03209.x>

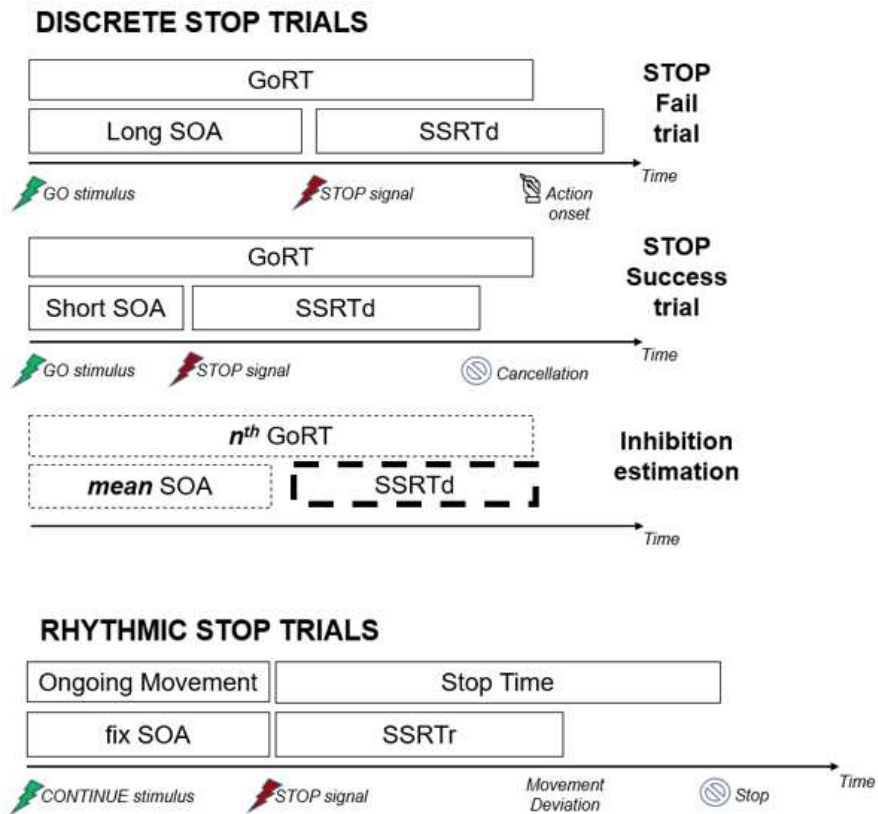
855 Zelaznik, H. N., Spencer, R. M. C., & Ivry, R. B. (2002). Dissociation of explicit and implicit timing  
856 in repetitive tapping and drawing movements. *Journal of Experimental Psychology.*  
857 *Human Perception and Performance*, 28(3), 575–588. [https://doi.org/10.1037//0096-](https://doi.org/10.1037//0096-1523.28.3.575)  
858 [1523.28.3.575](https://doi.org/10.1037//0096-1523.28.3.575)

859



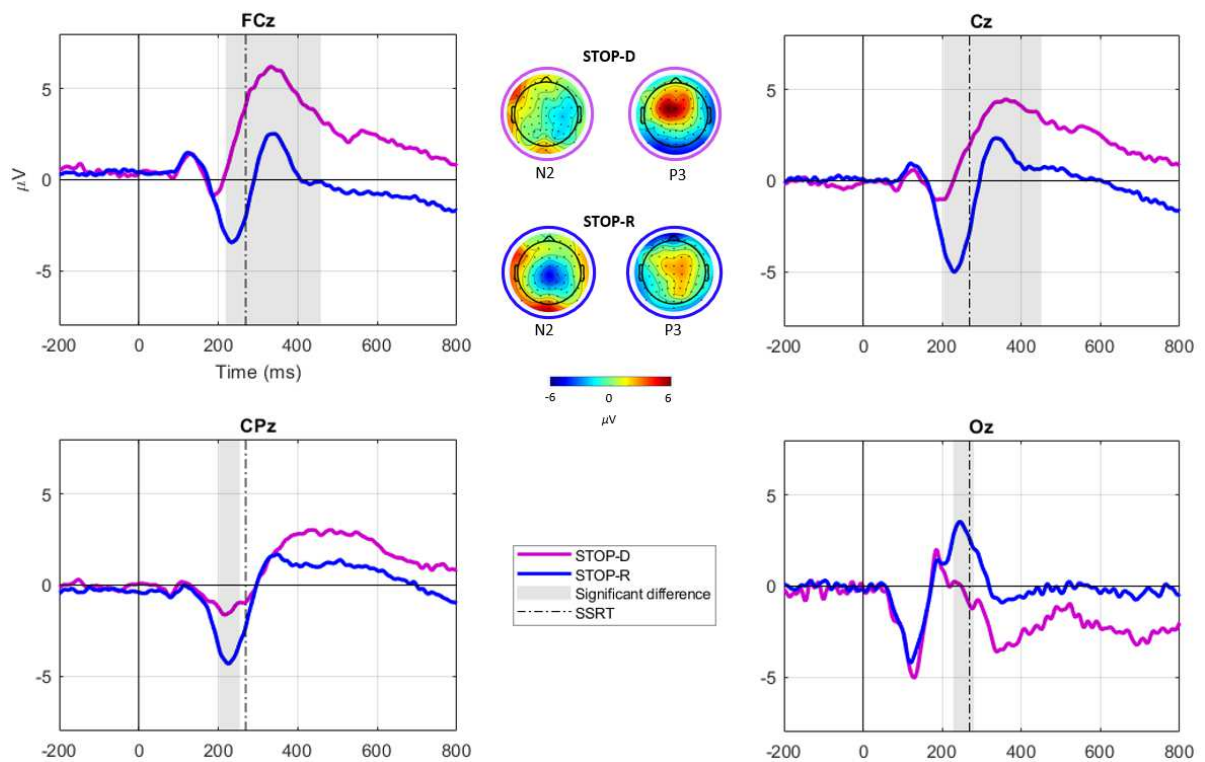
**Fig.1: Discrete and rhythmic tasks design**

Participants responded to the main stimulus by initiating a discrete movement (in the discrete GO condition; *upper panel*) or continuing a rhythmic movement (in the rhythmic CONTINUE condition; *lower panel*). In 25 % of the trials, the main stimulus was unexpectedly followed by a STOP signal after a SOA, which was dynamically adjusted in the discrete task but fixed in the rhythmic one (see text).



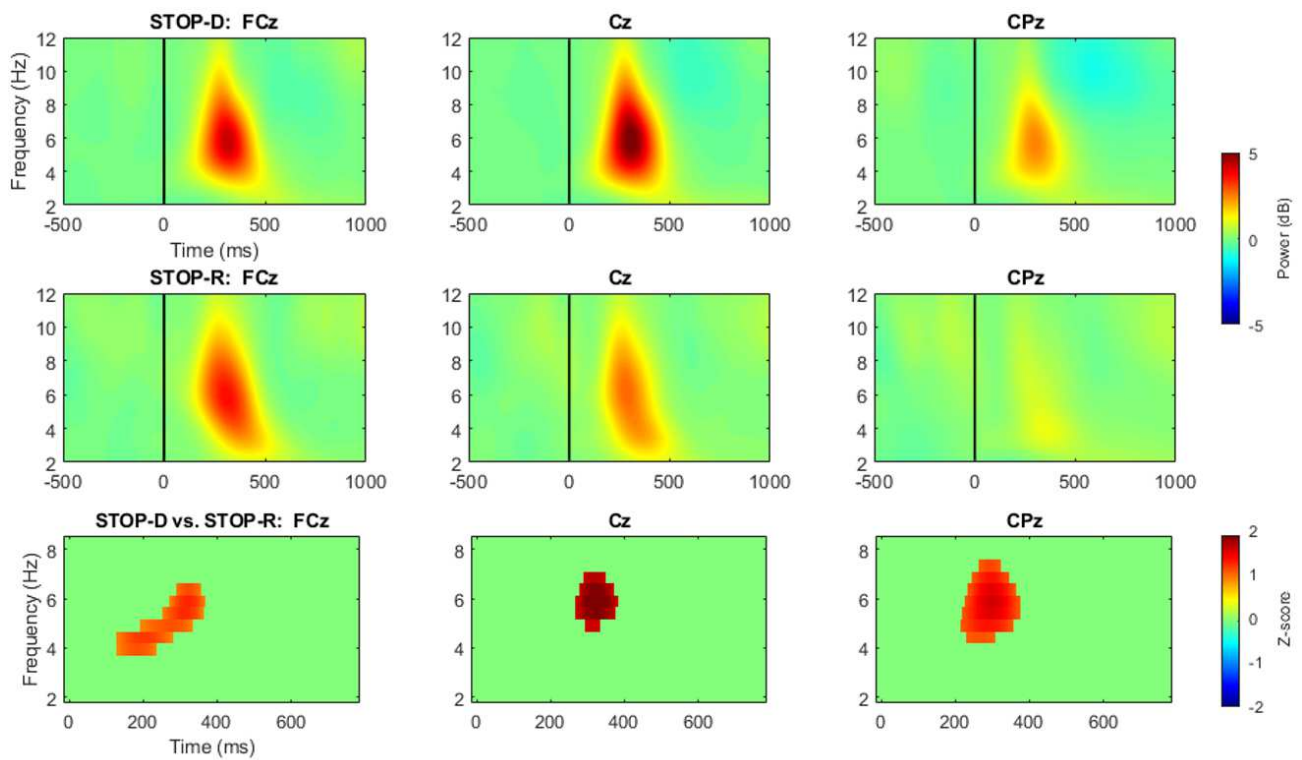
**Fig.2: Stop-signal reaction time (SSRT) computation in the discrete and rhythmic tasks**

Discrete task (*upper panel*): graphic representation of the assumptions of the independent race model, indicating how the probability of responding or cancelling the response depends on the SOA, GoRT and SSRTd. Rhythmic task (*lower panel*): graphic representation of the stopping time course. The SSRTTr is computed in each STOP trial by identifying the onset of the movement deviation relative to movements without a STOP signal (see text).



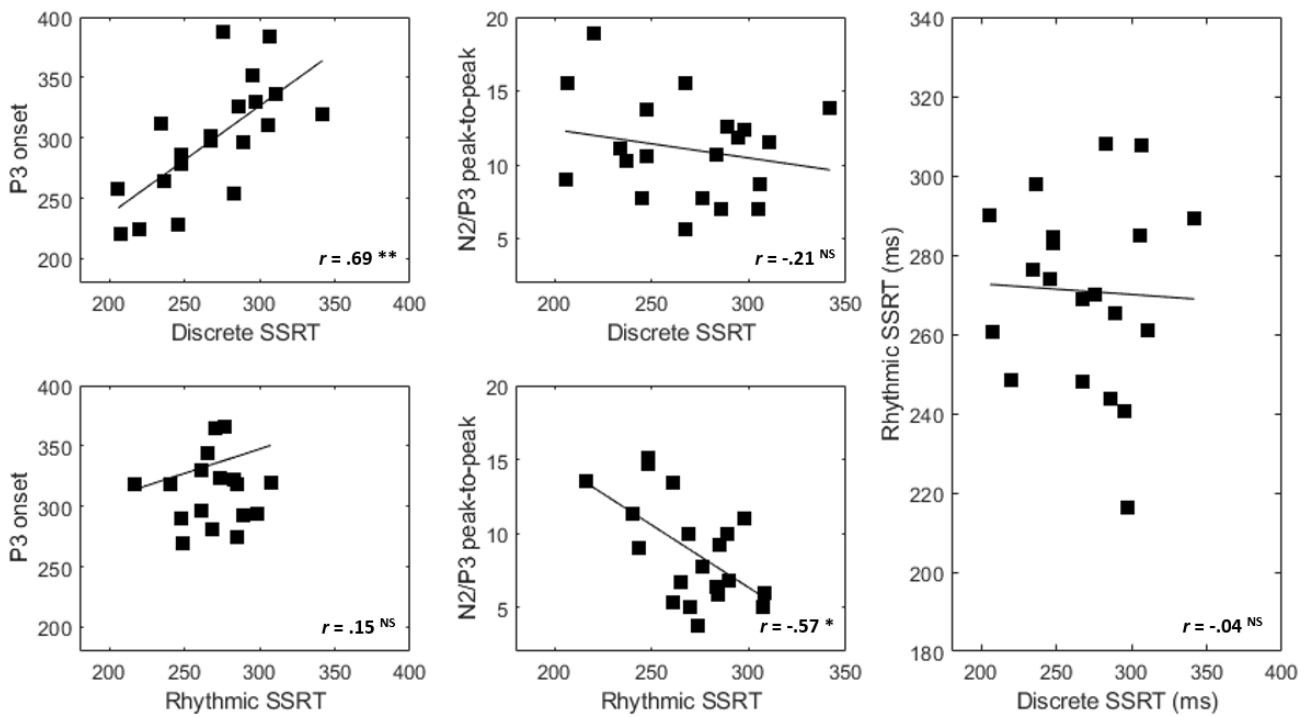
**Fig.3: EEG time-domain analysis of STOP trials**

Grand average STOP-signal locked ERPs in the discrete (STOP-D) and rhythmic (STOP-R) STOP conditions with associated topographies averaged across participants at FCz ERPs peak latencies. ERPs are displayed for three frontocentral (FCz, Cz, CPz) and one occipital (Oz) midline channels. In grey, the regions of significant difference (according to the nonparametric permutation analysis) between discrete (successful) and rhythmic STOP conditions ( $p < .05$ , corrected).



**Fig.4: FCz time–frequency analysis of STOP trials**

Grand averaged STOP–signal locked Power maps for the discrete and rhythmic STOP conditions. The differential Power maps thresholded after the permutation procedure ( $p < .05$ , corrected) are associated to different frequency (2 to 8 Hz) and time (0 to 800 ms) ranges that correspond to the time-frequency window included in the permutation statistical analysis. Red indicates significantly increased activity (two–sided  $p$ , upper tail) in the discrete compared to the rhythmic task.



**Fig.5: Correlation analysis**

Main ERP-behavior correlations reported across participants for the FCz site (see main text for statistical values).

The right panel shows the non-significant correlation between SSRT values computes in the discrete and rhythmic STOP tasks.

\*  $p < .05$ , \*\*  $p < .01$  (after correction for multiple comparisons), <sup>NS</sup>  $p > .05$



	Discrete Task	Rhythmic Task
<b>GoRT</b>	474 ms (58)	/
<b>GO omission rate</b>	.99 (.01)	/
<b>GO error rate</b>	.03 (.05)	/
<b>Movement frequency</b>	/	1.6 Hz (0.55)
<b>SOA</b>	206 ms (73)	206 ms (73)
<b>p(respond signal)</b>	.53 (.08)	/
<b>FsRT</b>	424 ms (51)	/
<b>Stop Time</b>	/	400 ms (31)
<b>SSRT</b>	268 ms (52)	270 ms (24)

**Tab.1: Main Behavioral measures of the two tasks (Mean and SD values)**