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► **To cite this version:**

Mario Hervault, Pier-Giorgio Zanone, Jean-Christophe Buisson, Raoul Huys. Multiple Brain Sources Are Differentially Engaged in the Inhibition of Distinct Action Types. *Journal of Cognitive Neuroscience*, inPress, pp.1-15. 10.1162/jocn_a_01794. hal-03448537

HAL Id: hal-03448537

<https://hal.science/hal-03448537>

Submitted on 27 Nov 2021

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1 **Multiple brain sources are differentially engaged in the inhibition of**
2 **distinct action types**

3

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17 **Acknowledgements:**

18 All authors approved the final submitted version. The authors wish to thank
19 all participants in this research.

20 The authors declare no competing financial interests.

21 **Abstract**

22 Most studies contributing to identify the brain network for inhibitory control
23 have investigated the cancelation of prepared-discrete actions, thus focusing
24 on an isolated and short-lived chunk of human behavior. Aborting ongoing-
25 continuous actions is an equally crucial ability but remains little explored. Al-
26 though discrete and ongoing-continuous rhythmic actions are associated with
27 partially overlapping yet largely distinct brain activations, it is unknown
28 whether the inhibitory network operates similarly in both situations. Thus,
29 distinguishing between action types constitutes a powerful means to investi-
30 gate whether inhibition is a generic function. We, therefore, used indepen-
31 dent component analysis (ICA) of EEG data and show that canceling a dis-
32 crete action and aborting a rhythmic action rely on independent brain compo-
33 nents. The ICA showed that a Delta/Theta-power increase generically indexed
34 inhibitory activity, whereas N2 and P3 ERP waves did so in an action-specific
35 fashion. The action-specific components were generated by partially distinct
36 brain sources, which indicates that the inhibitory network is engaged differ-
37 ently when canceling a prepared-discrete action versus aborting an ongoing-
38 continuous action. In particular, increased activity was estimated in precen-
39 tral gyri and posterior parts of the cingulate cortex for action canceling,
40 whereas an enhanced activity was found in more frontal gyri and anterior
41 parts of the cingulate cortex for action aborting. Overall, the present findings
42 support the idea that inhibitory control is differentially implemented accord-
43 ing to the type of action to revise.

44 **Keywords** Response inhibition, cognitive control, motor control, EEG,
45 source separation

46 **Introduction**

47 The ability to stop planned or ongoing actions, crucial in everyday life,
48 depends on an inhibitory executive function. Research based on the stop-
49 signal paradigm (Logan & Cowan, 1984; Verbruggen et al., 2019) linked
50 action stopping to an "inhibitory network" including cortical areas, namely,
51 inferior frontal cortex (IFC), pre-supplementary motor area (pre-SMA) and
52 cingulate cortex, as well as subcortical structures. This inhibitory network
53 acts downstream on the brain "action network" that generates action through
54 cortical and subcortical activations (Apšvalka et al., 2020; Aron et al., 2016;
55 Bari & Robbins, 2013; Stinear et al., 2009). In the stop-signal paradigm, the
56 standard task used in the domain, this inhibitory network underlies the
57 successful cancelation of discrete action, as revealed by EEG evoked N2 and
58 P3 potentials and increasing power of Delta (1-3 Hz) and Theta (4-7 Hz)
59 oscillations (e.g., Huster et al., 2013; Hynd et al., 2021). These frontocentral
60 EEG patterns proved to correlate with action cancelation across various
61 inhibition situations (Dutra et al., 2018; Waller et al., 2019; Wessel & Aron,
62 2014).

63 Notwithstanding, the fundamental distinction between discrete and
64 continuous actions¹ may challenge a unitary apprehension of the inhibitory
65 network, for these action types rely on distinct action control processes (Huys
66 et al., 2008). Effectively, they constitute *homo sapiens'* main movement
67 primitives and are associated with distinct timing (explicit versus implicit for
68 discrete and rhythmic action, respectively) (Huys et al., 2008, 2010; Spencer

5 ¹ Discrete actions, such as grasping an object, are delimited by moments without
6 displacement (i.e., with zero velocity and acceleration). In contrast, continuous
7 actions, such as walking, lack such recognizable endpoints and are typically rhythmic
8 as they constitute (periodic) repetitions of particular events (Hogan & Sternad, 2007).

69 et al., 2003). Discrete actions entail wider brain activation, implicating both
70 cortical (Lacoste et al., 2016; Schaal et al., 2004; Wiegel et al., 2020) and
71 subcortical areas (Habas & Cabanis, 2008; Mink & Thach, 1991), than
72 rhythmic actions. This distinction has been notably shown regarding
73 activations within the primary motor cortex (M1), pre-SMA, cingulate and
74 cerebellar cortices, as well as thalamus and globus pallidus. Part of this
75 differentiated action network (pre-SMA, cingulate cortex) is engaged in
76 generating the "brain's stopping signal" or is targeted (M1, thalamus and
77 globus pallidus) by this signal through the indirect and hyperdirect cortico-
78 basal pathways that implement the inhibitory activity (Aron et al., 2016;
79 Diesburg & Wessel, 2021). These differences in neural implementation,
80 identified as downstream targets of the inhibitory network, might require
81 distinct inhibitory activity. Yet, most previous studies have focused on
82 discrete action canceling, ignoring continuous action aborting, even though
83 both actions are crucial to human adaptive behavior. Studying inhibitory
84 control of both movement types thus constitutes a powerful strategy to
85 examine the generality of executive control and has practical implications for
86 human performances (Everitt et al., 2015).

87 In addition, the exclusive use of discrete actions in investigating action
88 inhibition comes with some limitations. The statistical "horse-race model" on
89 which the classic stop-signal paradigm relies (Logan & Cowan, 1984;
90 Verbruggen et al., 2019) constraints the exploration of its neural
91 underpinnings and their modulation in clinical disorders (Hervault et al.,
92 2019; Morein-Zamir et al., 2006; Schultz et al., 2021). Several authors have
93 proposed that continuous actions should be favored when investigating

94 action inhibition in clinical populations such as attention deficit hyperactivity
95 disorder (Leontyev & Yamauchi, 2019; Morein-Zamir et al., 2008) and
96 Parkinson's disease (Lofredi et al., 2021). Continuous movements also allow
97 for direct comparison of proactive and reactive inhibitory processes (Schultz
98 et al., 2021). However, although action inhibition can be investigated based
99 either on discrete or continuous actions, no study has directly compared the
100 brain mechanisms, hence sources, engaged in the inhibition of these
101 common types of actions. At least, a recent study of Lofredi et al. (2021)
102 showed that subthalamic nucleus (STN) deep brain stimulation impaired
103 ongoing-rhythmic movement abortion through potential modulation of the
104 activity within a "fronto-subthalamic inhibitory triangle" (i.e., connectivity
105 between IFC, pre-SMA and STN, identified from MRI connectome analysis).
106 This first neuroimaging study on aborting ongoing-continuous movement
107 suggests that the engaged brain structures could belong to the same broad
108 inhibitory network involved in discrete action canceling (Aron et al., 2016).
109 Still, whether the same activity is engaged within this network to inhibit
110 discrete and continuous action is a crucial question that remains unresolved.
111 Indeed, the different brain networks engaged in discrete and rhythmic action
112 might require different inhibition processes to be inhibited. In contrast,
113 independently of the network that produces the action, a unitary inhibition
114 process might suppress the corticospinal output for action inhibition.

115 The few studies investigating stopping enduring or continuous movements
116 have documented the implication of neural correlates of discrete-action
117 inhibition when aborting a sustained static contraction of the elbow (P3 ERP
118 wave, Hatta et al., 2003) or a continuous drawing action (N2 ERP wave,

119 Sosnik et al., 2015). Other research showed that ERP correlates of discrete-
120 action inhibition originate from the cingulate cortex in the inhibitory network
121 (Enriquez-Geppert et al., 2010; Huster et al., 2010), which is also engaged,
122 but distinctly so, in discrete and continuous actions (Habas & Cabanis, 2008;
123 Schaal et al., 2004). It is thus plausible that (some) EEG correlates of
124 inhibition would indicate a dissociation of the inhibitory network activity as a
125 function of the two action types.

126 In the present study, we investigated this issue using the inferential tool of
127 EEG independent component analysis, based on the "common independent
128 process identification" (CIPI) approach introduced by Wessel (2018). Blind
129 source separation procedures enable decomposing EEG data into
130 independent components that relate putatively to different psychological
131 processes (Onton et al., 2006). By examining whether stop signals for distinct
132 action types modulate similar brain components, we evaluated the
133 commonality of the inhibitory network activity in canceling prepared-discrete
134 actions and aborting ongoing-continuous (rhythmic) actions. This approach
135 has been previously used to probe inhibitory control (e.g., Castiglione et al.,
136 2019; Wessel et al., 2016) and is adopted here to disentangle the brain
137 sources involved in inhibiting the two types of action.

138 **Methods**

139 ***Participants***

140 Fourteen healthy individuals (8 males, mean age 25 years, SD = 2.2) served
141 as voluntary participants. As assessed by the Edinburgh Handedness
142 Inventory (Oldfield, 1971), all were right-handed and had a normal or
143 corrected-to-normal vision. None of the participants reported a history of
144 psychiatric or neurological disorders. The study was conducted according to
145 the principles stated in the Declaration of Helsinki and the local research
146 ethics committee approved the procedures (ID-RCB: 2020-A03215-34).

147 ***General procedure***

148 Participants performed two experiments involving discrete or rhythmic
149 movements execution. Both experiments were performed on the same
150 WACOM Cintiq 15X tablet (1280×800-pixel resolution). As long as the stylus
151 touched the tablet, the x and y coordinates of the performed movements
152 were digitized at a sampling frequency of 143 Hz. The program controlling
153 the tablet was custom-made.

154 ***Initial state***

155 In the discrete experiment, at the beginning of each trial, participants
156 adopted a static position (i.e., no movement), which consisted in keeping the
157 stylus between two vertical yellow bands (1 mm wide, 10 mm distant) plotted
158 at the center of the digitizing black screen. In the rhythmic experiment,
159 participants were initially instructed to continuously oscillate at a
160 spontaneous frequency (i.e., rhythmic movement) with the stylus between
161 the two sides of the screen but with the oscillation's extrema falling outside
162 the two centered vertical lines. In both experiments, the participant's arm

163 was fixed on the table to restrain the movement to the wrist articulation and
164 avoid large muscular noise in the EEG signal due to intense contraction of the
165 biceps and deltoid muscles.

166 Primary task

167 Primary-task stimuli were green or blue 50 ms flashes displayed on the whole
168 tablet screen. In the discrete experiment, participants were instructed to
169 reach with the stylus to the right versus left half-side of the tablet screen
170 when a green versus blue flash appeared, respectively. As a reminder of the
171 task, green and blue stickers were visible on the right and left sides of the
172 tablet (**Fig.1**). The primary discrete task consisted of a two-choice reaction
173 time involving a discrete-action response (GO_D condition). In the rhythmic
174 experiment, the primary task was to pursue the rhythmic movement without
175 interruption when the green and blue stimuli appeared (CONTINUE_R
176 condition).

177 Secondary task

178 In 25 % of the trials, the primary-task stimulus was followed by a red 50 ms
179 flash, which indicated the participants to suppress their primary-task
180 response. Thus, the secondary task required either to cancel the prepared
181 discrete movement (CANCEL_D condition) or to abort the ongoing rhythmic
182 movement (ABORT_R condition). Following ABORT_R trials, a rhythmic GO_R trial
183 was added to reengage participants in the rhythmic movement. In these GO_R
184 trials, participants were instructed to transit from a static position to an
185 oscillating movement as soon as the GO stimulus (green or blue) was
186 presented. In the discrete experiment, the STOP-signal delay between the
187 primary-task stimulus and the STOP signal (SSD), initially set to 200 ms, was

188 dynamically adjusted in 50 ms increments to achieve a probability of
189 responding $p(\text{respond}|\text{signal})$ of .50. When the participant crossed a vertical
190 line, the CANCEL_D trial was considered a cancel-failure and the SSD was
191 shortened; when the participant kept the stylus between the two lines, the
192 CANCEL_D trial was considered successful and the SSD was prolonged. In the
193 rhythmic experiment, the SSD value was fixed at the value equivalent to the
194 mean SSD obtained by each participant in the discrete experiment. All
195 participants completed the discrete experiment one week prior to the
196 rhythmic one.

197 In both experiments, the participants completed one practice block and 30
198 experimental blocks, each consisting of 20 trials. The discrete experiment
199 was set up according to the standard guideline for STOP-signal experiments
200 (Verbruggen et al., 2019). The rhythmic experiment design was close to the
201 discrete one in terms of sensory stimulations and movement effector in order
202 to limit the difference between the two experiments to the movement type
203 involved in the main task (i.e., discrete or rhythmic).

204 ***EEG recording and preprocessing***

205 EEG recordings were performed using an ActiveTwo system (BioSemi
206 Instrumentation, 64 electrodes) with a sampling rate of 2048 Hz. The EEG
207 electrodes were cautiously positioned based on four anatomical landmarks
208 (i.e., nasion, inion, and preauricular points) in accordance with the 5 % 10/20
209 international system (TCT, 2012). The same experimenter positioned the EEG
210 cap and electrodes, based on the same measures and landmarks, between
211 the two experimental sessions of a single participant. Additional electrodes
212 were placed below and above each eye. The data were online referenced to

213 the BioSemi CMS-DRL reference. All offsets from the reference were kept
214 below 15 mV. The EEG data were filtered online with a band-pass frequency
215 of 0.5-150 Hz. Continuous EEG data were imported and preprocessed in
216 bespoke scripts using functions from the EEGLAB Matlab plugin (Delorme &
217 Makeig, 2004). Visual inspection was used to remove channels with
218 prominent artifacts in the continuous EEG. The EEG data were then re-
219 referenced to a common average. The data were next partitioned into epochs
220 of 1.6 s (locked to the primary stimulus onset; -400 ms to 1200 ms). Epochs
221 containing values exceeding the average of the probability distribution of
222 values across the data segments by 5 SD were rejected.

223 ***Experimental design and statistical analysis***

224 *Behavioral analysis*

225 In both experiments, STOP-signal reaction times (SSRT) were computed in
226 CANCEL_D and ABORT_R conditions to assess the inhibitory performance. For the
227 discrete experiment, the participant's SSRT_D was estimated using the
228 integrative method (Verbruggen & Logan, 2009). This consisted in
229 subtracting the mean SSD from the n^{th} reaction time (RT), where n equals the
230 number of primary-stimulus RTs multiplied by the overall $p(\text{respond}|\text{signal})$.
231 The main-stimulus RT in GO_D condition was calculated as the time between
232 the stimulus onset and the response onset, the latter being defined as the
233 moment the stylus motion had exceeded 5 % of the Euclidean distance
234 between the initial and furthest (i.e., end) position of the discrete-movement
235 response. In the rhythmic experiment, SSRT_R was computed in each ABORT_R
236 trial using the rhythmic movement deviation method (Hervault et al., 2019).
237 Briefly, SSRT_R was calculated as the latency between the STOP signal onset
238 and the onset of the response adjustment. This time point was defined as the
239 moment the ongoing trajectory in phase space (i.e., the space spanned by x
240 and dx/dt) deviated relative to movements without a STOP signal according
241 to statistical criteria based on a sample's position in phase space, and the
242 angle and magnitude of its corresponding velocity vector.

243 *Overall CIPI procedure*

244 EEG analyses were performed to test whether the inhibitory activity triggered
245 by the STOP signal for prepared-discrete action canceling and ongoing
246 rhythmic movement aborting involved the same brain component. A
247 Common Independent Process Identification (CIPI) approach (Wessel, 2018)

248 was carried out according to the following steps (**Fig. 1**). First, the
249 preprocessed EEG data from the two experiments were concatenated and
250 submitted to a single decomposition using the infomax algorithm (Bell &
251 Sejnowski, 1995; Makeig et al., 1996) for each participant. In the original
252 scalp 64-channel data, each row of the data matrix represents voltage,
253 summed between source projections to one data channel. After
254 decomposition (63 components), each row of the data matrix gives the time
255 course of the activity of one component process spatially filtered from the
256 channel data (Delorme & Makeig, 2004). Second, non-brain components were
257 removed from the data, that is, components with less than 10 % chance to
258 account for neural activity as detected by the ICLABEL algorithm (Pion-
259 Tonachini et al., 2019). The remaining components were fitted with individual
260 inverse dipole-solutions using the DIPFIT algorithm (Oostenveld &
261 Oostendorp, 2002). Components with non-dipolar equivalent dipole solutions
262 usually represent non-brain signals (as defined by a residual variance of their
263 equivalent dipole solution being greater than 15 %, Delorme et al., 2012),
264 and were also removed. Third, one component was selected for each
265 participant (IC-D) as the component which best reflected the STOP-signal-
266 related activity in the discrete experiment (see below). Fourth, the capacity
267 of this IC-D to reflect the STOP-signal-related activity in the rhythmic
268 experiment was tested (see below). Fifth, conversely, an IC-R was similarly
269 identified in the rhythmic experiment for each participant and then tested in
270 the discrete one.

271 ERP and ERSP computation

272 To later select the components of interest, component-level event-related
273 potentials (ERP) were computed for each participant in the two experiments'
274 primary and secondary tasks. Event-related spectral perturbations (ERSP)
275 were also computed. In the time-domain analysis, and for each of the 63
276 components, time-series locked to the primary stimulus onset were averaged
277 across trials, following the subtraction of a -200 to 0 ms pre-stimulus period
278 as baseline, to calculate ERP for GO_D and $CONTINUE_R$ conditions. For $CANCEL_D$
279 and $ABORT_R$ conditions, the ERP was re-aligned to the STOP-signal onset. For
280 the frequency-domain analysis, the 63 component time series were
281 convolved with complex 3-to-8 cycle-long Morlet's wavelets to compute the
282 ERSP. Spectral power was estimated for each condition (1 to 50 ± 0.5 Hz,
283 linearly spaced) as the absolute of the resulting coefficients for each
284 frequency (normalized with respect to a -200 to 0 ms pre-stimulus baseline
285 and transformed to decibel scale), and next averaged over trials. For the
286 $CANCEL_D$ and $ABORT_R$ conditions, the spectral power locked to the primary-
287 task stimulus was then subtracted from the spectral power in the GO_D and
288 $CONTINUE_R$ conditions, respectively.

289 *IC-D selection from $CANCEL_D$ condition and testing in $ABORT_R$ condition*

290 One single component per participant, representing the inhibitory activity in
291 the discrete experiment, was chosen as the IC-D in order to disentangle this
292 activity from other neural and non-neural activities in the EEG. Whereas
293 multiple components could, in principle, relate to inhibitory activity, the first
294 component is most potent to investigate whether inhibitory activity in the
295 discrete experiment is similarly engaged in the rhythmic experiment, and
296 inversely. In addition, restricting the focus of investigation on one single

297 component significantly increases the signal-to-noise ratio of the underlying
298 process of interest (Wessel, 2018; Wessel & Ullsperger, 2011). In order to
299 select the component that best matched the inhibitory activity evoked by the
300 STOP signal in the discrete experiment, we proceeded in three steps. First,
301 since inhibitory processes are often discriminated by frontocentral activity
302 over the scalp for both discrete (Huster et al., 2013) and continuous actions
303 (Hervault et al., 2021; Sosnik et al., 2015), the topographical representation
304 of the scalp-back projected IC had to reach its extrema at the frontocentral
305 EEG electrodes (F1, Fz, F2, FC1, FCz, F2, C1, Cz or C2). Second, the IC-ERP
306 had to show a significant difference between the successful CANCEL_D
307 condition and the GO_D condition (e.g., Enriquez-Geppert et al., 2010),
308 indicating its functional implication in discrete action canceling. The
309 significance of the ERPs was assessed, for each single participant, by a
310 permutation testing procedure (see below). Third, in the resulting
311 components, IC-D was chosen as the component that maximized the
312 difference between CANCEL_D and GO_D conditions IC-ERPs. To quantify the ERP
313 difference between the two conditions, we took the maximal absolute
314 difference between the two whole-epoch ERPs.

315 In order to evaluate the ability of the chosen IC-Ds to disentangle the
316 CANCEL_D and GO_D conditions' ERPs at the group level, a permutation testing
317 procedure was applied (see below). Then, to assess the functional
318 significance of the IC-D in the rhythmic experiment, we similarly tested
319 whether the IC-D activity could disentangle ABORT_R and CONTINUE_R
320 conditions ERPs by a similar permutation analysis (see below).

321 Reciprocal analysis

322 The same procedure was applied reciprocally to test the ability of the IC-R,
323 selected from ABORT_R versus CONTINUE_R conditions in the rhythmic
324 experiment, to differentiate the EEG activity between CANCEL_D and GO_D
325 conditions in the discrete experiment.

326 Statistical permutation testing

327 To compare components' activity between conditions at the group level, ERPs
328 were subjected to a non-parametric permutation procedure (Maris &
329 Oostenveld, 2007). The 14 participants' whole-epoch IC-ERPs were pooled
330 over the two conditions (14 by conditions). Two sets of 14 ERPs each were
331 then drawn randomly from this pool, and the differential grand-average ERP
332 was computed between the two sets. This procedure was repeated 10 000
333 times, thus producing a distribution of these ERPs based on shuffled data
334 under the null hypothesis. For each time point, a p -value was computed as
335 the proportion of these pseudo-differential ERPs that exceeded the observed
336 participants' average differential ERP. This p -value thus indicates at which
337 time point the observed power distribution for the two conditions are more
338 divergent than expected for random data ($p = .05$ threshold). To correct for
339 multiple comparisons, we analyzed the resulting distributions of p -values to
340 compute p -thresholds corresponding to the 2.5th percentile of the smallest,
341 and the 97.5th percentile of the largest p -values distribution (Cohen, 2014).
342 The same procedure was applied between trials of each participant in the IC
343 selection procedure (see above). This permutation analysis was similarly
344 applied to each time-frequency point to assess the ERSP significance in the
345 different conditions.

346 Note that the CIPI approach has previously been used mainly to identify
347 common neural mechanisms between task contexts (e.g., Castiglione et al.,
348 2019; Wessel et al., 2016; Wessel & Aron, 2014), whereas here it was to show
349 a disjunction between neural networks implicated in two different inhibition
350 tasks. In this context, the disjunction approach requires appropriate
351 precautions to avoid type-2 errors (Wessel, 2018). In order to show that a
352 component related to one task is not involved in another, Wessel and Aron
353 (2014) used a more liberal significance threshold of $p = .20$ to account for the
354 type-2 error probability. Accordingly, we used a threshold of $p = .05$ to assess
355 ERP significance when testing IC-D in the discrete task, whereas we used a
356 threshold of $p = .20$ when testing IC-D in the rhythmic experiment and
357 reciprocally for IC-R analysis.

358 ***Brain sources reconstruction***

359 The CIPI procedure tests for the presence of IC-D activity in the rhythmic
360 experiment and, inversely, for IC-R activity in the discrete one. Following this
361 analysis, we compared the estimated brain areas recruited in the two
362 experiments based on their corresponding representative ICs (i.e., IC-D for
363 the CANCEL_D condition and IC-R for the ABORT_R one). For the source
364 localization of the ICs, the inverse ICs weight projections onto the original
365 EEG channels were exported to the sLORETA (standardized low-resolution
366 brain electromagnetic tomography) data processing module (Pascual-Marqui,
367 2002). sLORETA provides a unique solution to the inverse problem (Marco-
368 Pallarés et al., 2005; Pascual-Marqui, 2002; Sekihara et al., 2005). For
369 sLORETA, the intracerebral volume is partitioned into 6239 voxels with a 5
370 mm spatial resolution. Then, the standardized current density at each voxel

371 has been calculated in a realistic head model (Fuchs et al., 2002) based on
372 the MNI152 template due to the unavailability of individual participants' MRI
373 scans.

374 **[Fig. 1]**

375 **Results**

376 ***Behavioral results***

377 In the discrete experiment, basic requirements for the race-model application
378 (Verbruggen et al., 2019) were fulfilled as main-stimulus (i.e., GO_D) RT was
379 significantly longer for GO_D trials than for failed CANCEL_D trials (paired t-test, t
380 = 9.30, $p < .001$) and the 14 participants' overall $p(\text{respond}|\text{signal})$ was not
381 significantly different from .50 ($t = 1.85$, $p > .05$). These results validate the
382 computation of SSRT_D ($M = 269$ ms, $SD = 45$ ms) in accordance with the
383 model. In the rhythmic experiment, the spontaneous oscillation frequency
384 was 1.62 Hz on average ($SD = 0.49$ Hz). To ensure that participants did not
385 proactively anticipate the STOP signal occurrence, we tested whether the
386 movement was slow down at the SSD latency (Schultz et al., 2021). Including
387 all the ABORT_R trials of our participants, we compared the movement mean
388 velocity of the 100 ms time window surrounding the STOP signal occurrence
389 (-50 ms to + 50 ms) to the movement mean velocity of the 100 ms time
390 window surrounding the CONTINUE stimulus occurrence. This comparison
391 (paired t-test) failed to show a significant difference between the two time
392 windows for both the x-velocity ($t_{(2004)} = 0.28$, $p > .70$) and y-velocity ($t_{(2004)} =$
393 0.07 , $p > .90$), indicating that the participants did not adapt the movement,
394 proactively, in either movement's dimension. Measured SSRT_R values ($M =$
395 268 ms, $SD = 24$ ms) did not differ from the SSRT_D estimates (one-way
396 ANOVA, $F = .02$, $p > .05$). The inhibition times of both experiments were
397 unrelated across the 14 participants (Pearson correlation, $r = .02$, $p > .05$).

398 ***IC-D selection in the discrete experiment***

399 According to the above-described procedure, one component per participant
400 was retained; the grand-average characteristics of these IC-D components
401 are shown in **Fig.2**.

402 The differential ERSP (CANCEL_D minus GO_D trials) showed a significant post-
403 STOP-signal increase in the Delta/Theta frequency range (**Fig.2 B**). The ERPs
404 of the chosen IC-Ds demonstrated clear P3 wave evoked by the STOP signal,
405 which significantly distinguish GO_D and CANCEL_D conditions in the 318 - 559
406 ms time window (permutation test, $p < .05$, corrected for multiple
407 comparisons, **Fig.2. A**) but did not show any N2 activity. The differential ERP
408 was further tested for correlation across participants with the behavioral
409 inhibition latency. The P3 peak amplitude was computed by looking for the
410 maximal value in the 250-550 ms time range. P3 onset latency was defined
411 as the time when half of the P3 peak amplitude value was reached. Pearson
412 correlation across the 14 participants showed a significant correlation
413 between SSRT_D and both and P3 peak amplitude ($r = -.79$, $p < .001$, **Fig.3. A**)
414 and P3 onset latency ($r = .78$, $p < .001$, **Fig.3. B**), indicating that an
415 extensive and early P3 wave was associated with a shorter SSRT_D. P3 peak
416 amplitude was also positively correlated to the ERSP peak (maximal value of
417 the ERSP map within the significant region; $r = .62$, $p < .05$), indicating that a
418 large evoked P3 amplitude was associated with an evoked high power in the
419 Delta/Theta frequency range.

420 In addition to the functional relevance of the chosen IC-D, assessed by the
421 correlations between the IC-D ERP measures and SSRT_D, we investigated IC-D
422 relevance at the scalp-channel level. First, the EEGLAB *pval* function
423 indicated that IC-D was, on average, explaining 11.88 % (SD = 5.40 %) of the

424 total scalp (64-channel) variance in the 600ms following the STOP signal
425 occurrence in the CANCEL_D condition and 54.48 % (SD = 15.20 %) when
426 considering the frontocentral FCz site solely. Second, following Waller et al.
427 (2019) logic, we compared the frontocentral channel-ERP (FCz site) between
428 GO_D and CANCEL_D conditions after back-projecting all non-artifact ICs, IC-D
429 solely, or all ICs but IC-D. The results (**Fig.4 A**) suggested that most of the
430 scalp-frontocentral difference between the two conditions was accounted for
431 by the IC-D.

432

433 **[Fig. 2]**

434 **[Fig. 3]**

435 ***IC-D testing in the rhythmic experiment***

436 Each participant's chosen IC-D component was then analyzed in the rhythmic
437 experiment, with the same statistical procedure. The grand-average
438 characteristics of these IC-D components are also shown in **Fig.2**. The
439 differential ERSP (ABORT_R minus CONTINUE_R trials) showed a significant
440 increase in the Delta/Theta frequency band in a similar time range as in the
441 discrete experiment (**Fig.2 C**). However, the IC-D ERP was unable to
442 significantly disentangle CONTINUE_R and ABORT_R conditions (permutation
443 test, $p > .20$, **Figure 2.D**), even at a liberal threshold of $p = .20$. Moreover,
444 the differential ERP (ABORT_R minus CONTINUE_R condition) did not show any
445 significant correlation between P3 measures and the 14 participant's SSRT_R
446 (P3 onset latency: $r = -.38$, $p > .20$, peak amplitude: $r = .03$, $p > .90$) nor
447 with time-frequency values (P3 peak amplitude - ERSP peak: $r = .05$, $p > .80$).

448 This indicates that the discrete IC-D activation was significantly related
449 neither to the rhythmic SSRT_R nor to the Theta oscillations.

450

451 **[Fig. 4]**

452

453 ***IC-R selection in the rhythmic experiment***

454 As for the discrete experiment, one component per participant was chosen in
455 the rhythmic experiment according to the reciprocal procedure; the grand-
456 average characteristics of these IC-R components are shown in **Fig.5**. The
457 differential ERSP (ABORT_R minus CONTINUE_R trials) showed a significant post-
458 STOP-signal increase in the Delta/Theta frequency range (**Fig.5 C**). The ERPs
459 of the chosen IC-Rs showed both clear N2 and P3 waves evoked by the STOP
460 signal, which significantly separated the ABORT_R and CONTINUE_R conditions in
461 a 192 - 308 ms and a 406 - 556 ms time window, respectively (permutation
462 test, $p < .05$, corrected for multiple comparisons, **Fig.5 D**). The differential
463 ERP (ABORT_R minus CONTINUE_R trials) was further tested for correlation with
464 behavioral inhibition latency. To this end, P3 peak amplitude and P3 onset
465 latency were computed as previously described. N2 features were similarly
466 computed, looking for the minimal value in a 150-350 ms time window.
467 Pearson correlation across the 14 participants showed a significant relation
468 between SSRT-R and P3 peak amplitude ($r = -.65$, $p < .01$, **Fig.3. C**),
469 indicating that an extensive P3 wave was associated with a shorter SSRT_R. In
470 contrast SSRT_R values did not correlate with P3 onset latency ($r = -.25$, p
471 $> .30$) or N2 features ($|r| < .20$, $p > .30$). P3 peak amplitude was also
472 positively correlated, across participants, to the ERSP peak value ($r = .60$, p

473 < .05), indicating that an evoked high P3 peak amplitude was associated with
474 an evoked high power in the Delta/Theta frequency range. ERSP peak value
475 did not correlate with N2 peak amplitude ($r = -.07, p > .80$).

476 As for IC-D, we investigated IC-R relevance at the scalp-channel level. IC-R
477 was, on average, explaining 8.36 % (SD = 3.72 %) of the total scalp (64-
478 channel) variance in the 600ms following the STOP signal occurrence in the
479 ABORT_R condition and 32.00 % (SD = 16.51 %) when considering the
480 frontocentral FCz site solely. Next, we compared the frontocentral channel-
481 ERP (FCz site) between CONTINUE_R and ABORT_R conditions after back-
482 projecting all non-artifact ICs, IC-R solely, or all ICs but IC-R. The results
483 (**Fig.4 B**) suggested that most of the scalp-frontocentral difference between
484 the conditions was accounted for by the IC-R.

485

486

[Fig. 5]

487

488 ***IC-R testing in the discrete experiment***

489 Each participant's chosen IC-R component was next analyzed in the discrete
490 experiment with the same statistical procedure. The grand-average
491 characteristics of these IC-R components are shown in **Fig.5**. The differential
492 ERSP (CANCEL_D minus GO_D condition) showed a significant increase in the
493 Delta/Theta frequency band in a similar time range as in the rhythmic
494 experiment (**Fig.5 B**). However, the IC-R ERP was unable to significantly
495 disentangle GO_D and CANCEL_D conditions (permutation test, $p > .20$, **Fig.5**
496 **A**). Moreover, the differential ERP (CANCEL_D minus GO_D condition) did not
497 show any significant correlation between ERP measures and SSRT_D (P3

498 measures: $|r| < .10$, $p > .80$; N2 measures: $|r| < .20$, $p > .60$) or time-
499 frequency values (P3 peak amplitude - ERSP peak: $r = .50$, $p = .07$; N2 peak
500 amplitude - ERSP peak: $r = .06$, $p > .80$). This indicates that the IC-R
501 activation was not significantly related to SSRT_D or Theta oscillations in the
502 discrete experiment.

503 **Brain sources reconstruction**

504 The voxel-based sLORETA images were compared across the two
505 experimental tasks using voxel-wise randomization paired *t*-tests with 5000
506 permutations, based on statistical non-parametric mapping. Estimated voxels
507 with significant differences ($p < .01$, corrected for multiple comparisons)
508 between contrasted conditions were located in the MNI-brain. Differences
509 between ICs-D and ICs-R on statistical maps of the source analyses are
510 displayed in **Table 1** and **Fig.6**. These analyses revealed a significantly
511 higher current density estimated for IC-D in the frontocentral region,
512 including the midcingulate cortex (MCC) and the medial portion of the frontal
513 gyrus, with maximum current source density estimate at MNI [(x,y,z) -10, 0,
514 40; $t = 3.66$]. In contrast, the IC-R showed higher activation in the insula, the
515 anterior cingulate cortex (ACC), and the anterior part of the frontal cortex
516 (see **Table 1**).

517

518

[Fig. 6]

519

520

[Table 1]

521 **Discussion**

522 Prior work has established distinctive EEG patterns correlated to the activity
523 of a brain inhibitory network when canceling a prepared discrete action. Here,
524 we tested whether these EEG correlates indicate a similar inhibitory activity
525 in the case of aborting an ongoing-continuous (rhythmic) action. We recorded
526 EEG while participants performed two experiments calling either for discrete
527 action canceling or for rhythmic action aborting. In both situations, behavioral
528 inhibition latencies fell within the range observed in previously used stop-sig-
529 nal tasks (e.g., Castiglione et al., 2019; Enriquez-Geppert et al., 2010; Havas
530 et al., 2020; Hervault et al., 2019). The SSRT values from the two experi-
531 ments were not significantly correlated. Although our current sample size
532 prohibits to firmly conclude the absence of a behavioral relation, this result
533 replicates one previously reported in inhibitory tasks with a comparable de-
534 sign (Hervault et al., 2019). The CIPI analysis of the EEG data (Wessel, 2018)
535 corroborated these results, as independent brain components engaged when
536 canceling and aborting action revealed a partial dissociation in the brain
537 sources engaged in the two experiments.

538 The component that best represented the inhibitory activity in the discrete
539 experiment exhibited the by-now classic correlates of action inhibition,
540 namely, increasing Delta/Theta power and a large evoked P3 wave. The rele-
541 vance of this IC-D for inhibition was confirmed by the significant $SSRT_D/P3$ la-
542 tency correlation across participants, in line with previous stop-signal task
543 studies (Huster et al., 2020; Hynd et al., 2021; Wessel & Aron, 2015). Cru-
544 cially, its inability to disentangle ERPs pertaining to the rhythmic $ABORT_R$ and
545 $CONTINUE_R$ conditions revealed that the IC-D is functionally irrelevant in

546 aborting ongoing rhythmic movements. Thus, the brain component that was
547 most involved in discrete-action canceling turned out to be irrelevant for
548 rhythmic-action aborting. At the same time, the IC-R identified in the rhythmic
549 task also exhibited a stop-signal related Delta/Theta increase, as well as
550 N2 and P3 waves, but failed to separate the ERPs for discrete CANCEL_D and
551 GO_D conditions (in either N2 or P3 time ranges). Therefore, the brain component
552 that was most involved in (rhythmic) action stopping was irrelevant for
553 (discrete) action canceling. Based on the inferential logic of the CIPI analysis
554 (Wessel, 2018; Wessel & Aron, 2014), the present study thus demonstrates
555 that distinct brain components are involved in discrete-action canceling and
556 rhythmic-action aborting.

557 In terms of ERPs, both IC-D and IC-R contained a stop signal-related P3, which
558 correlated to their respective SSRT in the discrete and rhythmic experiments.
559 The absence of a significant P3 when evaluating the IC-D activity in the rhythmic
560 experiment or, inversely, the IC-R activity in the discrete one suggests
561 that the P3 signature is specific to the engaged inhibition brain component.
562 This pattern of findings indicates that P3 is a clear-cut neural marker of action
563 inhibition in the context of stop-signal reactions (Fine et al., 2020; Hynd et al.,
564 2021; Tatz et al., 2021; Wessel & Aron, 2015). In addition, an N2 wave was
565 exclusively evoked by the IC-R in the rhythmic ABORT_R but not in any other
566 condition. This N2 exclusivity allows for two interpretations. First, if N2 is considered
567 for indexing an inhibitory process (see Huster et al., 2013, 2020), it
568 sets apart discrete-action canceling and rhythmic-action aborting. Second, if
569 N2 is thought to reflect another cognitive process, such as the allocation of
570 attention or conflict detection, this process is differentially involved in the two

571 experiments. Regarding the time-frequency correlates of inhibition, the stop
572 signal-related Delta/Theta power increase pertained to the P3 amplitude of
573 both IC-D in the discrete task and IC-R in the rhythmic one. However, a sig-
574 nificant Delta/Theta power increase was also observed when testing IC-D in
575 the rhythmic experiment and, inversely, IC-R in the discrete one, corroborat-
576 ing the non-specificity of this power correlate. Similarly, a theta power en-
577 hancement is known to relate to the recruitment of cognitive control across
578 different types of conflicts in the stream of information processing (Nigbur et
579 al., 2011). Taken together, our EEG analyses suggest that stop signal-related
580 inhibitory activity is exerted by different brain components for discrete and
581 rhythmic actions, leading to distinct scalp activities (N2/P3 ERP complex) be-
582 tween the two situations.

583 The dissociation, at least partial, suggested by the CIPI analysis raises the
584 question of whether the brain sources involved in discrete-action canceling
585 and rhythmic-action aborting reflect a comparable engagement of the in-
586 hibitory network. Several studies have successfully localized the generators
587 underlying discrete-action inhibition EEG correlates. They identified N2 gener-
588 ators in the IFC and MCC regions (Bekker et al., 2005; Enriquez-Geppert et
589 al., 2010; Huster et al., 2010; Nieuwenhuis et al., 2003). P3 was also gener-
590 ated by a deep source in MCC, as well as by precentral and midfrontal gyri
591 (Huster et al., 2010; Kok et al., 2004; Ramautar et al., 2006). Theta power in-
592 crease could also originate, in several cognitive control tasks, from the frontal
593 lobe and MCC sources (Mitchell et al., 2008). Here, the significant differences
594 between IC-D and IC-R estimated neural sources tap well into the inhibition
595 generators commonly reported in EEG signatures, suggesting that the two sit-

596 uations might differ in how inhibition is implemented. Notably, IC-D showed
597 higher activity in the precentral gyrus (BA 6, likely corresponding to the pre-
598 SMA), while IC-R sources revealed higher ACC activity. These separately esti-
599 mated activations in the inhibitory network echo previous fMRI dissociations
600 found in the action network, with higher pre-SMA activations in discrete
601 movements (Habas & Cabanis, 2008; Schaal et al., 2004) and specific ACC
602 activation in continuous movements (Habas & Cabanis, 2008). One interpre-
603 tation is that, rather than a clear-cut delineation of anatomical action and in-
604 hibition networks, action execution and inhibition are controlled by networks
605 with partially identical brain areas. Consequently, these common denomina-
606 tors (the precentral gyrus versus ACC for discrete and rhythmic actions, re-
607 spectively) are either engaged in a functionally different, context-dependent
608 manner (action control, inhibition) or act as "final pathway" to exert the in-
609 hibitory control. Other parts of the inhibitory network showed higher activity
610 related to either IC-D (in MCC) or IC-R (in anterior regions of the frontal gyri)
611 brain sources. Taken together, these results suggest that distinct action types
612 associated with distinct cognitive and cerebral activity require, to be inhib-
613 ited, an action-specific engagement of the inhibitory network. To further de-
614 lineate how the brain action network modulation has consequences in an ac-
615 tion-dependent involvement of the inhibitory network and its EEG correlates,
616 future investigations might combine EEG-fMRI acquisition of multiple action
617 types.

618 In stop-signal tasks, conflict arises whenever infrequent stop responses must
619 overcome the prepotency of frequent responses (Braver et al., 2001;
620 Mirabella, 2014). Differences in N2, P3, and the underlying cingulate cortex

621 activity have been shown to reflect the level of conflict existing in distinct in-
622 hibitory contexts (Braver et al., 2001; Enriquez-Geppert et al., 2010). More
623 specifically, the EEG correlates of inhibition were specifically evoked by a go
624 stimulus, a no-go signal, and a stop signal, with underlying modulations in the
625 MCC and the precentral cortices activity (Enriquez-Geppert et al., 2010; Hus-
626 ter et al., 2010). In our case, the finding that IC-R activity is higher in the an-
627 terior part of the cingulate cortex, whereas the IC-D activity is higher in its
628 posterior part, may be linked to the exclusivity of the N2 occurrence in rhyth-
629 mic-movement aborting. Indeed, the anterior region of the MCC has been
630 identified as a major neural generator of N2, whereas its posterior part does
631 so for P3 (Huster et al., 2010). Such modulations of the inhibitory network ac-
632 tivity in discrete actions have been interpreted as differences in the conflict-
633 related stages of the information processing (Enriquez-Geppert et al., 2010;
634 Randall & Smith, 2011). In particular, the discrete and rhythmic actions have
635 been associated with distinct control regimes, in an open-loop versus closed-
636 loop fashion (Jeannerod, 1988) and based on explicit versus implicit time rep-
637 resentations, respectively (Spencer et al., 2003). Thus, the two action types
638 might engage distinct predictive brain activity regarding the expected sen-
639 sory outcomes of the movement and, hence, influence the disruption related
640 to an unexpected stop signal occurrence. This difference in the conflict in-
641 duced by the stop signal might modulate the engagement of the inhibitory
642 network. Further work manipulating both conflict level and action type is
643 needed to clarify the functional relevance of inhibitory-sources dissociation in
644 brain activity, for instance, by varying the requirement associated with the in-
645 frequent signal (i.e., ABORT, CONTINUE).

646 In addition, Schultz et al. (2021) have shown that proactive inhibitory control
647 in aborting a rhythmic action is associated with a slowdown of the movement
648 prior to the stop signal occurrence. We showed that it was not the case in our
649 continuous Abort-task, suggesting that our results suggest reactive inhibition
650 engagement. Still, further studies are necessary to establish how this proac-
651 tive inhibition is implemented in the brain activity when aborting a continu-
652 ous action and whether this activity is common to the brain activity related to
653 the proactive inhibition engaged in canceling a discrete action (Elchlepp et
654 al., 2016).

655 To conclude, the present findings showed that the inhibitory activity involved
656 in discrete action canceling and rhythmic action aborting rely on partially dis-
657 sociated brain sources. This dissociation suggests that the EEG patterns com-
658 monly related to stopping activity may reflect the involvement of distinct
659 brain sources, differentially enrolled in the inhibitory network depending on
660 the action type to revise. Therefore, assessing the commonality of inhibitory
661 control across multiple, fundamentally distinct action types appears instru-
662 mental in providing a complete (neural) model of inhibitory control.

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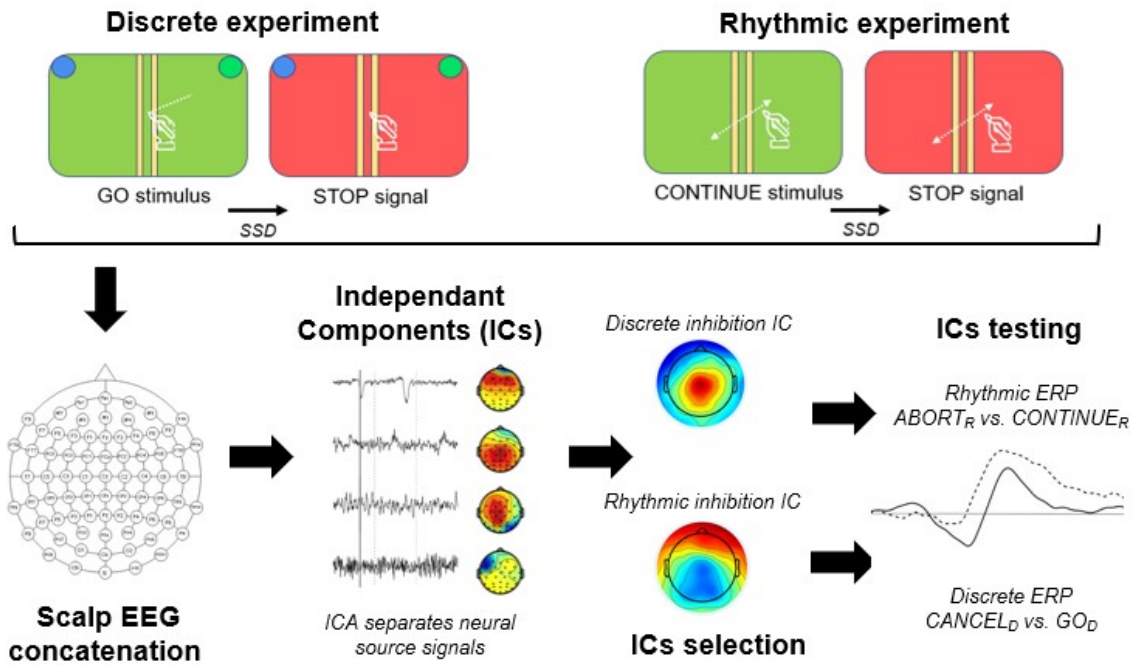
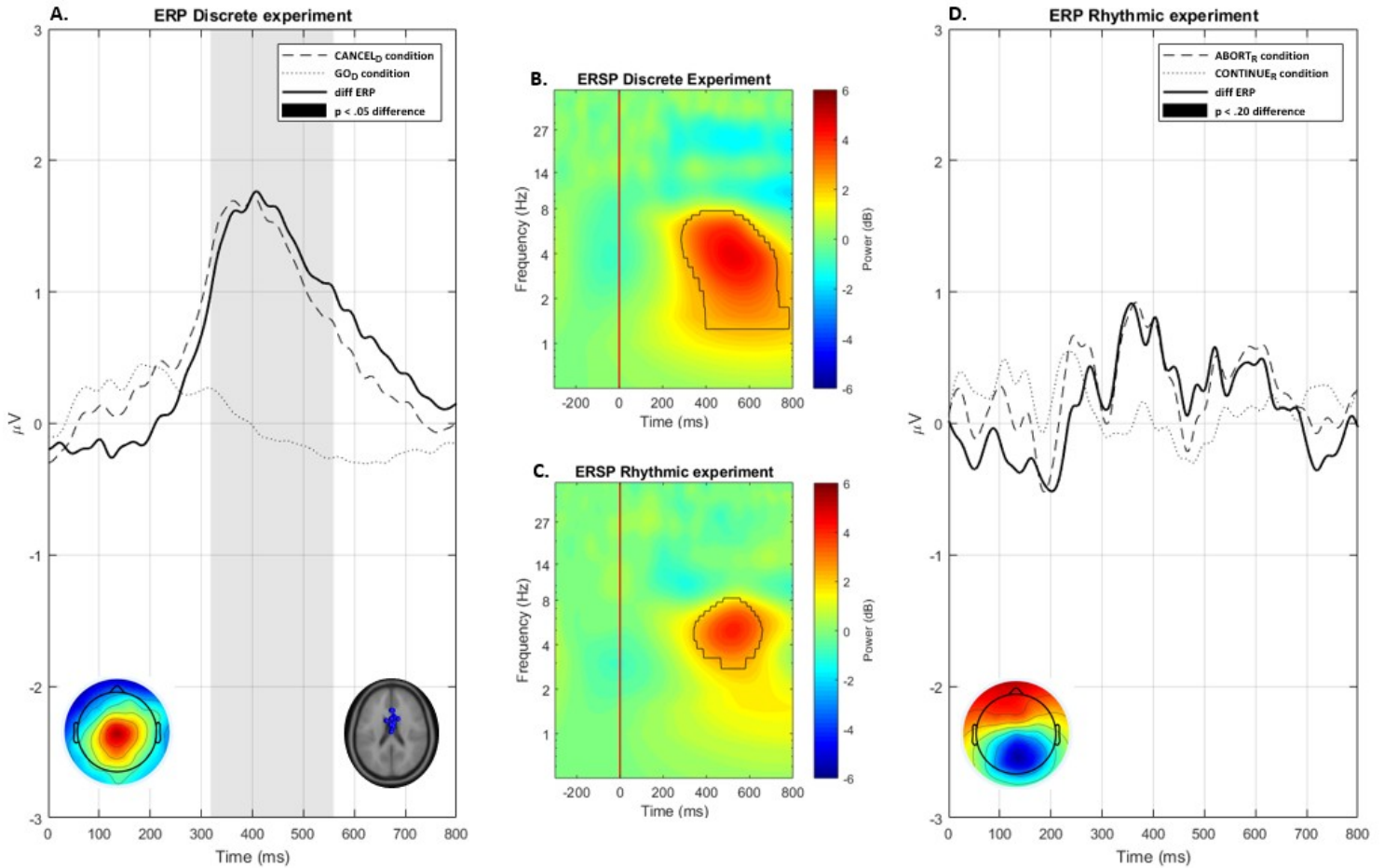


Fig.1: EEG CIPI analysis overview

The concatenated EEG from the discrete and rhythmic experiments were subjected to ICA. One IC that represented the process underlying inhibition in the discrete experiment was selected on a single participant basis. Subsequently, the capacity of that component to disentangle ABORT_R versus CONTINUE_R conditions in the rhythmic experiment was assessed. Reciprocally, ICs underlying rhythmic inhibition were selected and then tested in the discrete experiment to disentangle CANCEL_D versus GO_D conditions.



931

Fig.2: Discrete chosen component (IC-D) analysis

Panel A: IC-D ERP (grand-average) in the discrete experiment, showing the difference between GO_D and $CANCEL_D$ conditions. **Panel D:** IC-D ERP (grand-average) in the rhythmic experiment, showing difference between $CONTINUE_R$ and $ABORT_R$ conditions. $CANCEL_D$ and $ABORT_R$ conditions topographical maps of the IC-D components were computed in the channel space at the P3 peak latency. Equivalent current dipole model of the participants' IC-Ds is also portrayed.

Panel B-C: IC-D ERSP maps computed in the discrete $CANCEL_D$ (B) and rhythmic $ABORT_R$ (C) conditions. Red line: STOP signal occurrence. Black contour line: region of significant activation ($p < .05$, corrected for multiple comparisons).

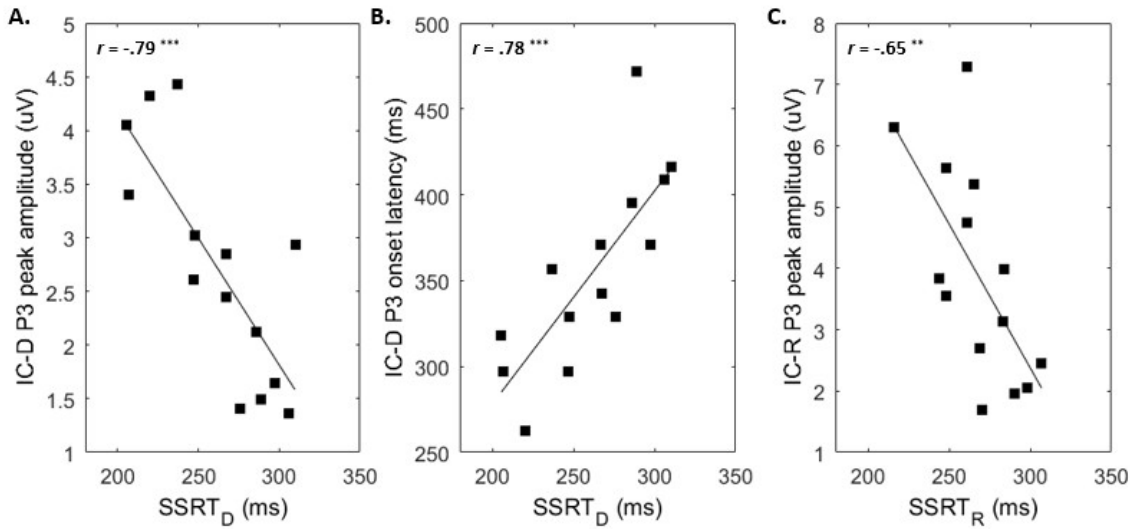


Fig.3: IC ERPs - Behavior correlations

Panel A: IC-D P3 peak amplitude correlation with SSRT_D. **Panel B:** IC-D P3 peak amplitude correlation with SSRT_D. **Panel C:** IC-R P3 peak amplitude correlation with SSRT_R.

*** $p < .001$, ** $p < .01$, * $p < .05$

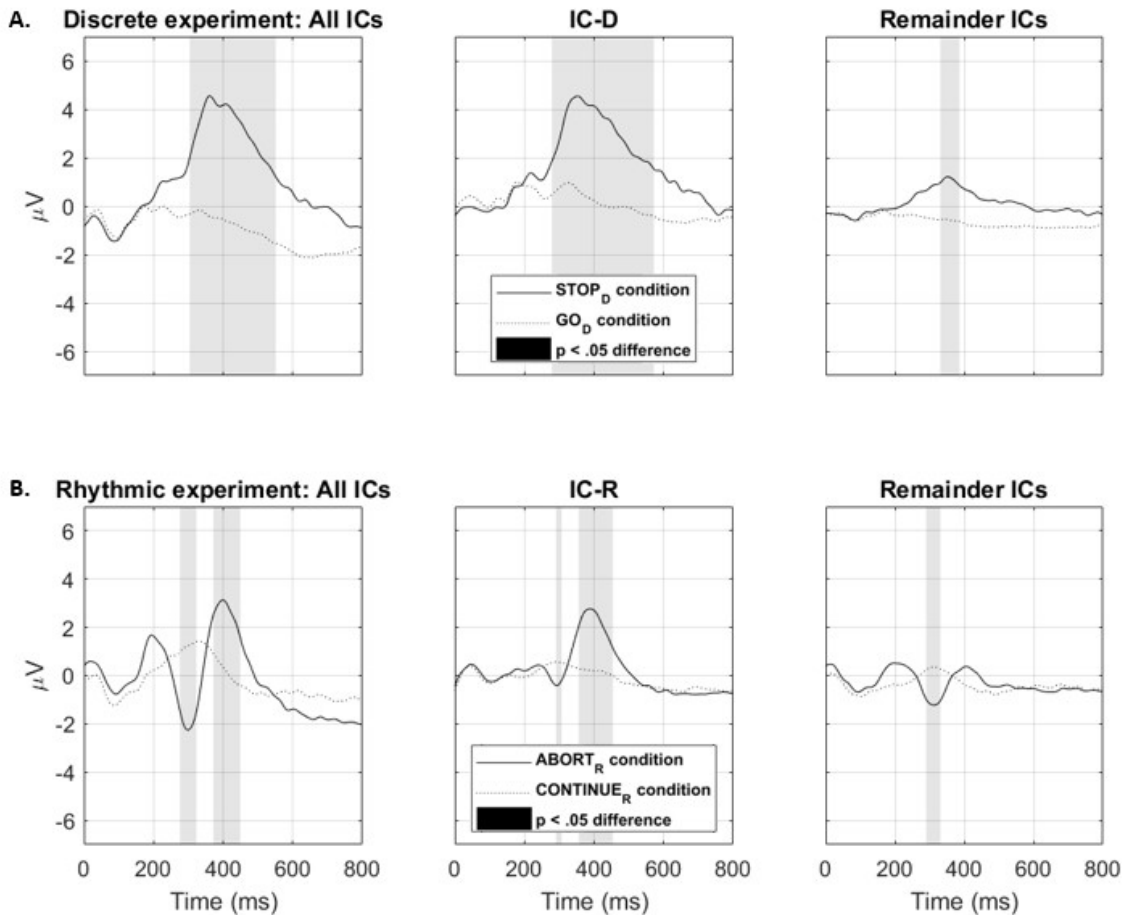


Fig.4: IC-D and IC-R back-projection at the frontocentral channel level

Panel A: Scalp FCz STOP-signal locked ERP in the discrete GO_D and CANCEL_D conditions after back-projecting all non-artefact ICs, IC-D solely, or all ICs but IC-D. The two conditions differed significantly (non-parametric permutation procedure, $p < .05$, corrected for multiple comparisons) for the All-ICs projection (304 to 551 ms), the IC-D solely (275 to 572 ms) and the remaining ICs (331 to 386 ms) projections.

Panel B: Scalp FCz STOP-signal locked ERP in the rhythmic CONTINUE_R and ABORT_R conditions after back-projecting all non-artefact ICs, IC-R solely, or all ICs but IC-R. The two conditions differed significantly (non-parametric permutation procedure, $p < .05$, corrected for multiple comparisons) for the All-ICs projection (276 to 323 ms, 372 to 449 ms), the IC-R solely (291 to 307 ms, 356 to 454 ms) and the remaining ICs (290 to 331 ms) projections.

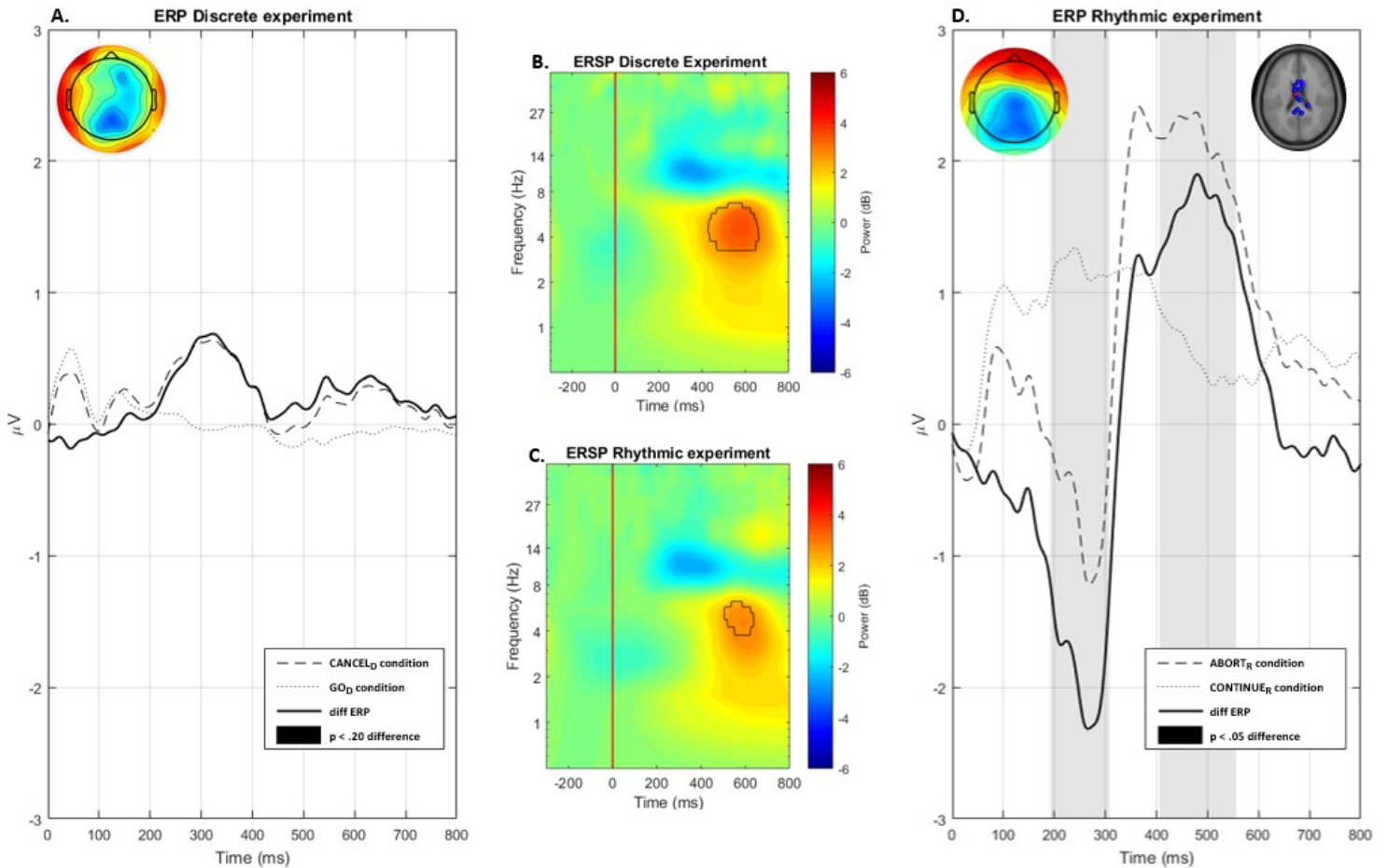
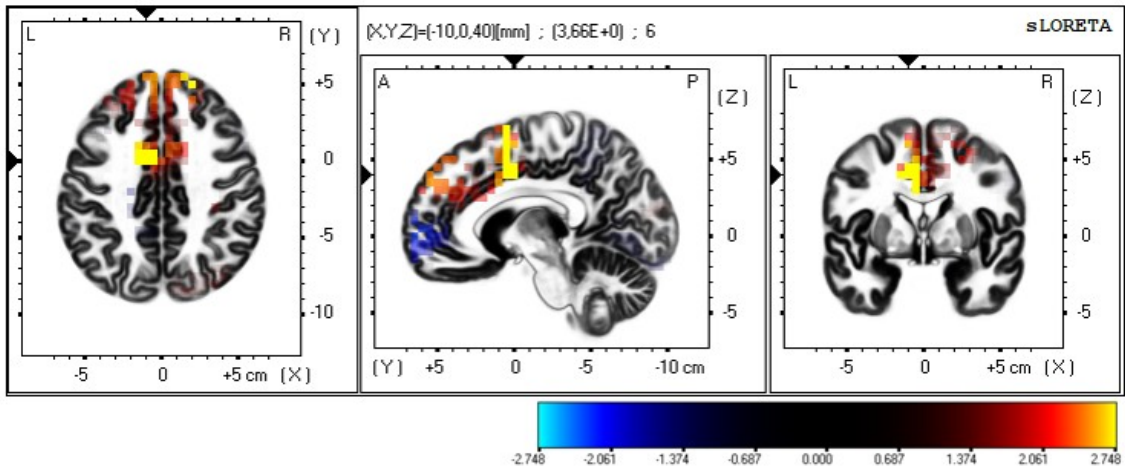


Fig.5: Rhythmic chosen component (IC-R) analysis

Panel A: IC-R ERP (grand-average) in the discrete experiment, showing difference between GO_D and $CANCEL_D$ conditions. **Panel D:** IC-R ERP (grand-average) in the rhythmic experiment, showing difference between $CONTINUE_R$ and $ABORT_R$ conditions. $CANCEL_D$ and $ABORT_R$ conditions topographical maps of the IC-R components were computed in the channel space at the N2 peak latency. Equivalent current model of the participants' IC-Rs is also portrayed.

Panel B-C: IC-R ERSP maps computed in the discrete $CANCEL_D$ (B) and rhythmic $ABORT_R$ (C) conditions. Red line: STOP signal latency. Black line: region of significant activation ($p < .05$, corrected for multiple comparisons).



937 **Fig.6: Source localization analysis: IC-D versus IC-R**
The estimated sLORETA images showing statistical differences (t-values, two-tailed) between IC-D and IC-R for three orthogonal brain slices (horizontal, sagittal, coronal). Maximum current source density voxels are represented with greater IC-D values in yellow and greater IC-R values in blue. Only the voxels that passed the p value threshold ($p < .01$, corrected for multiple comparison) are shown in color.

Table.1: Summary of significant results from whole-brain sLORETA estimation comparisons between IC-D and IC-R

Lobe	Region		BA	X	Y	Z
Discrete experiment						
Limbic	Cingulate Gyrus		24	-10	0	40
	Cingulate Gyrus		32	-15	5	45
Frontal	Medial	Frontal	6	-10	5	55
	Superior	Frontal	6	-10	5	60
Rhythmic experiment						
Sub-lobar	Insula		13	-35	-25	5
Frontal	Middle	Frontal	9	55	15	30
	Medial	Frontal	10	-5	60	5
Limbic	Anterior	cingu-	24	5	30	-5
	Anterior	cingu-	32	5	35	0