1   **Unexpected sounds non-selectively inhibit active visual stimulus**

2   **representations**

3   Running title: Inhibition of attention by unexpected events

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17

18 **Abstract**

19 The brain's capacity to process unexpected events is key to cognitive flexibility. The most well-

20 known effect of unexpected events is the interruption of attentional engagement (distraction). We

21 tested whether unexpected events interrupt attentional representations by activating a neural

22 mechanism for inhibitory control. This mechanism is most well-characterized within the motor

23 system. However, recent work showed that it is automatically activated by unexpected events and

24 can explain some of their non-motor effects (e.g., on working memory representations). Here,

25 human participants attended to lateralized flickering visual stimuli, producing steady-state visual

26 evoked potentials (SSVEP) in the scalp-electroencephalogram. After unexpected sounds, the

27 SSVEP was rapidly suppressed. Using a functional localizer (stop-signal) task and independent

28 component analysis, we then identified a fronto-central EEG source whose activity indexes

29 inhibitory motor control. Unexpected sounds in the SSVEP task also activated this source. Using

30 single-trial analyses, we found that sub-components of this source differentially relate to sound-

31 related SSVEP changes: while its N2 component predicted the subsequent suppression of the

32 attended-stimulus SSVEP, the P3 component predicted the suppression of the SSVEP to the

33 unattended stimulus. These results shed new light on the processes underlying fronto-central

34 control signals and have implications for phenomena such as distraction and the attentional blink.

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36 **Keywords:** Attention, Distraction, Inhibitory Control, Steady-state visual evoked potential, Stop-

37 signal task

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39 Unexpected perceptual events, such as sudden sounds, are known to disrupt ongoing

40 thoughts and actions. For example, when a warehouse worker hears an unexpected glass-shattering

41 sound while doing inventory, they may momentarily stop writing on a count sheet and indeed

42 forget their count altogether. This type of stimulus-driven distraction produced by unexpected

43 perceptual events is of key interest to researchers studying attentional capture and its control

44 (Yantis, 1993; Simons, 2000; Corbetta et al., 2008; Awh et al., 2012). Cognitive psychology has

45 generated substantial insights into how distractors affect attentional engagement with the same

46 sensory domain (Bacon & Egeth, 1994; Theeuwes, 2004; Gaspelin et al., 2017; Liesefeld et al.,

47 2017). Moreover, cognitive neuroscience has provided a comprehensive picture of neural activity

48 after unexpected events (Courchesne et al., 1975; Corbetta & Shulman, 2002). However, relatively

49 little is known about the exact neural mechanisms by which task-irrelevant unexpected events

50 disrupt active, task-relevant attentional representations, especially across sensory domains. In a

51 recent theoretical article, we proposed that unexpected sensory events engage an inhibitory brain

52 mechanism that is capable of interrupting active neural representations that support active motor

53 and non-motor (i.e., cognitive) functions (Wessel & Aron, 2017). At the core of this proposal is

54 the assertion that a well-characterized neural mechanism for inhibitory control (which is well-

55 known to be involved in suppressing active motor representations, and which is automatically

56 triggered by unexpected events; Wessel & Aron, 2013; Wessel, 2018), can exert inhibitory

57 influence even on non-motor representations, such as working memory or attention (Chiu & Egner,

58 2014, 2015; Wessel et al., 2016; Castiglione et al., 2019; Tempel et al., 2020).

59 The brain network in question implements its inhibitory function via a fronto-basal-ganglia

60 (FBg) network that involves the pre-supplementary motor area (preSMA), the right inferior frontal

61 cortex (rIFC), and the subthalamic nucleus (STN) of the basal ganglia (Nachev et al., 2007;

62   Ridderinkhof et al., 2011; Schall & Godlove, 2012; Aron et al., 2014; Jahanshahi et al., 2015). Its

63   inhibitory influence on movement is most well-studied in the stop-signal task (SST, Logan et al.,

64   1984), where it implements the 'stop'-component of the purported race between going and

65   stopping (Aron et al., 2014). The temporal dynamics of this network can be non-invasively

66   measured using scalp-electroencephalography (EEG), where its activity is indexed by a

67   pronounced stop-signal induced fronto-central N2/P3 event-related potential (ERP) complex (de

68   Jong et al., 1990; Kok et al., 2004; Huster et al., 2013; Wessel & Aron, 2015). The N2 component

69   of this ERP complex has been proposed to reflect the detection of the stop-signal or the associated

70   conflict (Schröger, 1993; Donkers & van Boxtel, 2004; Azizian et al., 2006; Enriquez-Geppert et

71   al., 2010; Smith et al., 2010), whereas the P3 has been hypothesized to reflect the subsequently

72   implemented inhibitory process (de Jong et al., 1990; Kok et al., 2004; Enriquez-Geppert et al.,

73   2010; Wessel & Aron, 2015).

74   Our past work has shown that unexpected perceptual events automatically engage this same

75   inhibitory mechanism (Wessel et al., 2012; Wessel & Aron, 2013, 2017), even when there is no

76   explicit instruction to engage inhibitory control (Wessel, 2018a). Amongst other findings, this

77   assertion is supported by the fact that unexpected events lead to a slowing of motor responses

78   (Dawson et al., 1982; Parmentier et al., 2008) and elicit a fronto-central N2/P3 ERP complex that

79   is morphologically similar to the fronto-central ERP complex elicited by stop-signals (Courchesne

80   et al., 1975; Squires et al., 1975). Indeed, independent component analysis (ICA) of EEG data

81   recorded in subjects that performed both the stop-signal task and tasks involving unexpected events

82   suggests that both ERP complexes share a common underlying neural generator (Wessel & Aron,

83   2013; Wessel & Huber, 2019). Moreover, local field potential recordings from the human

84   subthalamic nucleus (the key subcortical node of the inhibitory FBg-network) suggest that

4

85   unexpected events engage this subcortical structure as well (Bočková et al., 2011; Wessel et al.,

86   2016). In line with this, optogenetic inactivation of the STN negates the inhibitory influence that

87   unexpected sounds have on behavior in mice. While unexpected sounds typically lead to a

88   premature interruption of ongoing licking bouts, this effect is absent if the STN is inactivated,

89   providing key causal evidence for the role of inhibitory control structures in surprise processing

90   (Fife et al., 2017).

91   An important property of this inhibitory FBg mechanism is that it implements inhibition in

92   non-selective, 'global' fashion, both after stop-signals and unexpected sensory events. This is most

93   evident from experiments that use transcranial magnetic stimulation and electromyography to

94   measure cortico-spinal excitability (for reviews, see Duque et al., 2017; Wessel & Aron, 2017;

95   Derosiere et al., 2020). Such experiments show that the rapid reduction of cortico-spinal

96   excitability that is found after stop-signals (Coxon et al., 2006; Stinear et al., 2009) extends even

97   to task-unrelated motor effectors (Badry et al., 2009; Greenhouse et al., 2011; Cai et al., 2012;

98   Goode et al., 2019). The same non-selective suppression of cortico-spinal excitability can be

99   observed after unexpected events (Wessel & Aron, 2013; Dutra et al., 2018; see also: Novembre

100   et al., 2018; Novembre et al., 2019). As mentioned above, we have proposed that this type of non-

101   selective, 'global' suppression exerted by the inhibitory mechanism could explain why unexpected

102   events have effects even on non-motor, cognitive representations (Wessel & Aron, 2017) –

103   conceivably, if fronto-basal ganglia mediated inhibition is broad and non-selective enough, it may

104   even affect non-motor representations (provided neural underpinnings of those representations are

105   susceptible to this inhibitory circuit).

106   Indeed, some preliminary evidence for this proposal already exists. In a first series of

107   studies, Chiu & Egner (2014) have found that pairing faces with the requirement to rapidly

108   withhold a prepotent action – thereby triggering the inhibitory control network – inhibits the

109   encoding of these face stimuli into memory. In a follow-up study, the strength of this effect related

110   directly to the activation of the inhibitory FBg-network (Chiu & Egner, 2015). Further in line with

111   this, both action-stopping in the stop-signal task and active suppression of memory contents (e.g.,

112   in the Think/NoThink paradigm, Anderson & Green, 2001) are accompanied by activity from the

113   same neural source (Castiglione et al., 2019). Finally, in line with the finding that unexpected

114   events engage the inhibitory network, we have found that the activity of the inhibitory FBg-

115   network also mediates the disruptive effects of unexpected sounds on active verbal working

116   memory representations (Wessel et al., 2016).

117   While these studies lend first preliminary support to the general idea that the FBg-network

118   underlying motor inhibition could also explain the suppression of non-motor representations, all

119   existing work so far is limited to mnemonic processes. Moreover, it has already been found that

120   not all types of memory representations seem to be subject to the purported inhibitory influence of

121   the FBg network (indeed, short-term visual memory representations as operationalized in the

122   classic work of Vogel & Machizawa, 2004; Vogel et al., 2005, seem to be interrupted by other

123   mechanisms, cf., Wessel, 2018a). Therefore, it is hitherto unclear which exact types of non-motor

124   representations are potentially subject to interruption by inhibitory control exerted from the FBg-

125   network, and whether its influence extends beyond the realm of mnemonic representations.

126   In the current study, we tested whether the activity of this mechanism could explain the

127   effects of unexpected events on ongoing attentional representations. A highly influential body of

128   past behavioral work indicates that indeed, attentional regulation may include inhibitory processes

129   (Shapiro & Raymond, 1994; Klein & Taylor, 1994; Tipper et al., 1990). To test whether attentional

130   representations are affected by inhibitory control signals after unexpected events, a novel task was

131   designed in which participants attended to one of two concurrently presented rhythmic flickers.

132   Such flickering visual stimuli are known to produce a steady-state visual evoked potential (SSVEP)

133   – a stimulus-driven entrainment of parieto-occipital EEG activity to the frequency of the rhythmic

134   sensory stimulation (Regan, 1989; Silberstein et al., 1995). Notably, the covert direction of

135   attention towards a specific stimulus leads to an increase in the amplitude of the associated SSVEP

136   (Morgan et al., 1996; Müller et al., 1998; Ding et al., 2006; Walter et al., 2012). In our task, we

137   then presented unexpected sounds on a subset of trials while subjects were attending one of the

138   flickering visual stimuli. We expected the unexpected sounds to rapidly and transiently reduce the

139   amplitude of the SSVEP, reflecting an interruption of attentional engagement. In addition to this

140   task, all participants also performed a stop-signal task, which served as a functional localizer for

141   the FBg-network underlying inhibitory motor control. To test the hypothesis that this same neural

142   mechanism is related to the interruption of attentional representations after unexpected events, we

143   used ICA to identify the neural source signal underlying the N2/P3-complex in the stop-signal task

144   and tested whether this source was also active following unexpected events (as found in prior work,

145   cf. Wessel & Aron, 2013; Wessel & Huber, 2019). Finally, we then tested whether the activity of

146   that EEG source related to the disruption of attention (i.e., the SSVEP) on a trial-to-trial basis.

147

148   **Materials and Methods**

149   *Participants*

150   In Experiment 1, 21 healthy adult college students (mean age: 19.05 years; SD: 1.12; three left-

151   handed; 14 females) participated the experiment for course credit. In Experiment 2, 21 healthy

152   adult college students (mean age: 20.52; SD: 2.14; one-left-handed; 11 females) participated. Six

153   of those participants received course credit and the rest were compensated with $15 per hour. All

154   participants had normal or corrected-to-normal vision. None of participants performed both
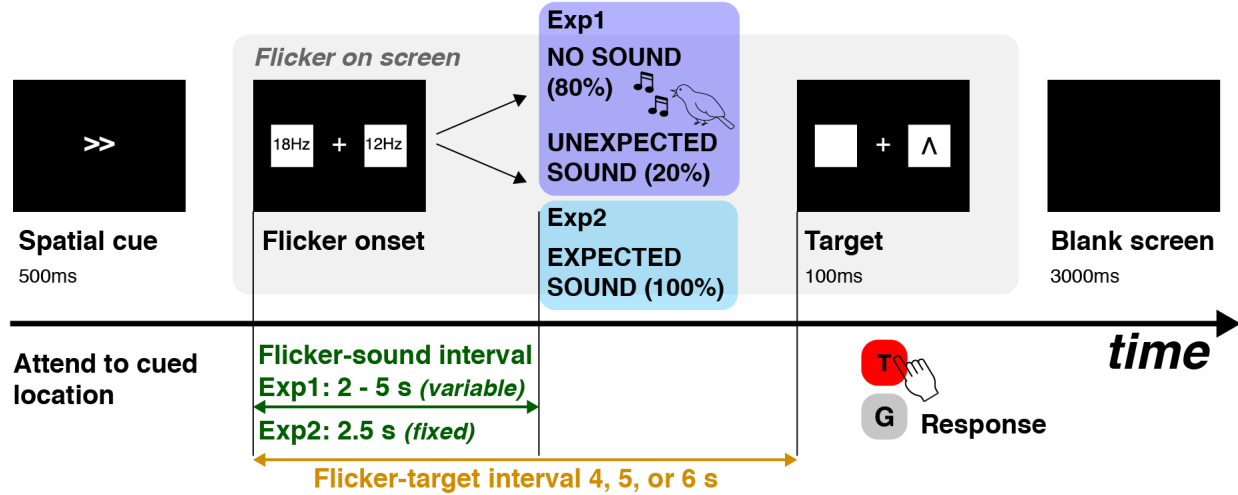
155   experiments.

156

157   ***Stimulus presentation***

158   All stimuli were presented on a BenQ XL2420B 120Hz gaming monitor with 1ms response time,

159   connected to an IBM compatible PC running Fedora Linux and MATLAB 2015b. Stimuli were

160   presented using Psychtoolbox 3 (Brainard, 1997) at the monitor's native resolution of 1920 x 1080

161   pixels. Responses were made using a standard QWERTY USB keyboard. Viewing distance was
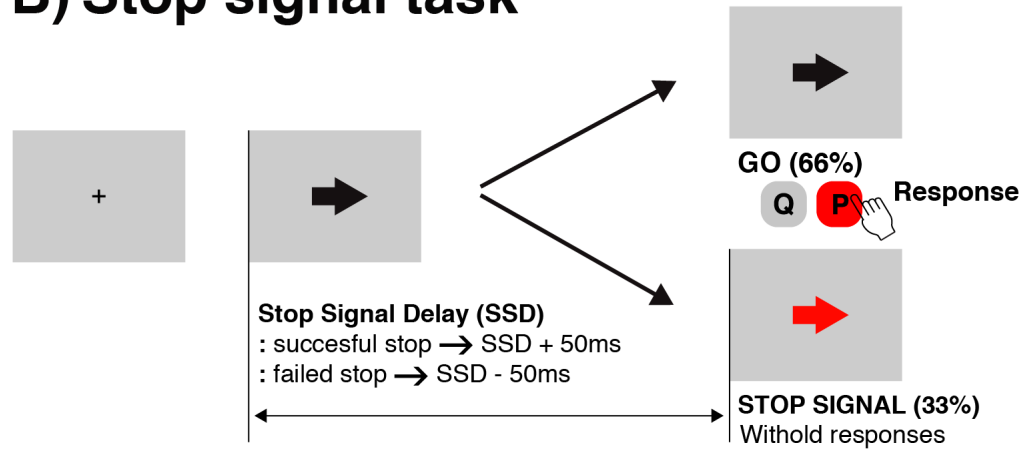
162   kept constant at 90 cm.

163

A) Cross-modal SSVEP oddball task

B) Stop signal task

Figure 1. *Task design. A) In Experiment 1, participants attended to a spatially cued rhythmic flicker (12 or 18 Hz) in order to detect a visual target that was superimposed on the cued flicker after a variable delay interval. On 20% of trials, unexpected sounds were presented in the delay interval, prior to target appearance. In Experiment 2, expected sounds were played 2.5 seconds after the flicker onset in all trials. B) In the stop signal task (performed by all subjects after the crossmodal SSVEP oddball task), participants made speeded responses to black arrows (go stimulus). On 33% of trials, the color of arrows changed into red (stop signal) after an adaptive delay, after which they were instructed to attempt to stop their response.*

173

174   ***Experimental paradigms.***

175   <u>Experiment 1: Cross-modal SSVEP oddball task.</u> The task (from here onwards referred to as the

176   "SSVEP task") was designed to induce sustained active perceptual / attentional visual

177   representations during which unexpected sounds were presented on a subset of trials. All stimuli

178   were presented in white color on a black background. A task diagram can be found in ***Figure 1***.

179   Each trial began with a centrally presented white double-arrow (<< or >>, 500ms duration,

180   font size: 100) that informed the subjects which side of the display to covertly attend to. The initial

181   double-arrow cue was followed by the SSVEP display, which consisted of a central fixation cross

182   (+) flanked by two white flickering boxes (size: 9.66 x 9.66° of visual angle) that were presented

183   to the left and right (offset 8.44° of visual angle laterally from center), with one box flickering at

184   a frequency of 12Hz (f12) and the other flickering with a frequency of 18Hz (f18, half of the trials

185   consisted of 12Hz left / 18Hz right displays, and the other half of 18Hz left / 12Hz right, presented

186   in pseudorandom order). After a variable delay period, a visual target (/\ or \/, font size 100) would

187   appear in the center of the flickering box on the cued side of the display for 100ms. Participants

188   were instructed to press either the 't' (up, /\) or 'g' (down, \/) key on the keyboard to indicate the

189   direction of the target stimulus. All responses were made using index or middle finger of the

190   dominant hand (responses were made in vertical direction using the same hand to prevent potential

191   cue-target spatial incompatibility effects that could result from the lateralized stimulus display).

192   The flicker onset-to-visual-target delay was either 4, 5, or 6 seconds long, pseudo-randomly chosen

193   from a uniform distribution of values. Participants were instructed to keep fixating on the central

194   fixation cross while covertly attending to the cued flicker and monitoring it for the visual target.

195   Horizontal eye movements were monitored by the experimenter between blocks (using the

10

196   recording from the HEOG electrode on each trial) to ensure that the cued flickers were covertly

197   attended and no overt saccades were made (trials with saccades to either side of the screen were

198   removed during the analysis, see below). A blank screen was displayed for three seconds between

199   trials.

200   On 20% of trials, unexpected sounds (unique bird song segments of 290 ms length) were

201   played in the delay period between the flicker and the target onset (UNEXPECTED condition)

202   through speakers positioned to either side of the computer screen. Subjects were not instructed to

203   expect any sounds during the experiment, nor were sounds presented in the practice block. The

204   inter-stimulus-interval between flicker onset and unexpected sound was drawn from a uniform

205   distribution ranging from 2,000 to 5,000 msec, with the constraint that the chosen delay could not

206   exceed the duration of the SSVEP display (i.e., the flicker onset-to-visual-target delay). In the

207   remainder of trials (80%), no sounds were presented (NO SOUND condition). Sound volume was

208   set at conversational level, which reliably evokes an orienting response without inducing a startle

209   reflex. After one block of practice (24 blocks), participants performed 360 trials in total (36 trials

210   per block, 10 blocks) with self-paced resting periods between blocks. Across the experiment, all

211   conditions were counterbalanced (i.e., f12/f18 positions were equally distributed between left and

212   right, as well as between UNEXPECTED and NO SOUND trials).

213

214   Experiment 2: Control SSVEP task. Experiment 1 was designed to test whether unexpected sounds

215   disrupted the SSVEP by comparing the UNEXPECTED to the NO SOUND condition. We used

216   Experiment 2 to confirm that the reduction of the SSVEP on UNEXPECTED trials in Experiment

217   1 was due to the unexpected nature of the sounds, and not due to the presence of a sound itself.

218   The task was identical to Experiment 1, with the exception that *every* trial included a sound (a 600

11

219   Hz sine wave tone of 200ms duration), which was presented at the same time on each trial (exactly

220   2.5 seconds after flicker onset). Contrary to Experiment 1, participants were instructed to expect

221   these sounds before the task, and their practice block (18 trials) included these sounds as well

222   (hence, we will refer to this as the EXPECTED condition). To collect a matching number of trials

223   in relation to the UNEXPECTED condition in Experiment 1 (and thereby equate the signal-to-

224   noise ratio of the SSVEP), Experiment 2 contained only 72 trials (all within the EXPECTED

225   condition), split evenly across two blocks.

226

227   Stop-signal task (functional localizer). To evoke the neural signature of the inhibitory control

228   mechanism, we used the same version of the SST that we used in prior work (Wessel, 2020). At

229   the beginning of each trial, a black central fixation cross appeared for 500ms on a grey background.

230   Then, a black arrow pointing to either the left or right was presented at the center of the display

231   (go trials). Participants made speeded bimanual responses using either 'q' (left) or 'p' (right) key

232   on the keyboard that spatially matched the go stimuli (e.g., 'q' for left arrow). In 33% of trials, the

233   black arrows were replaced by red arrows (stop-signal) after certain amount of stop signal delay

234   (SSD). The initial delay was set at 200ms. Participants were instructed to withhold responses on

235   trials in which stop-signals appear. The SSD was adaptively adjusted in accordance with the

236   stopping performance to ensure about 50% of probability in successful stopping [P(stop)], which

237   is optimal for estimating the stop signal reaction time (SSRT) and guarantees motor prepotency on

238   most successful stop-trials. The SSD was increased by 50ms after every successful stop; and

239   decreased by 50ms after every failed stop. Experimenters instructed that making fast responses to

240   the go stimuli and stopping responses to the stop-signals are both equally important. Verbal

241 feedback was given after each block. Participants performed 300 trials (200 go, 100 stop), split

242 evenly across five blocks.

243

244 <u>Procedure.</u> The SSVEP tasks in both experiments were always performed before the SST. The

245 tasks were performed in this fixed order to avoid biasing participants towards using inhibitory

246 control in the SSVEP task.

247

248 ***Behavioral Analysis***

249 All behavioral data from the SST were examined to check whether each subjects' data conformed

250 to the prediction of the race model of the stop-signal task (Logan et al., 1984). Specifically, we

251 checked whether failed-stop trial reaction time was faster than Go-trial reaction time for each

252 subject. We also checked whether the SSD algorithm converged around p(stop)=.5 by ensuring

253 that the final p(stop) for each subject was between .4 and .6. SSRT was then computed using the

254 revised version of the integration method with replacement of errors and misses, as suggested by

255 Verbruggen et al. (2019).

256

257 ***EEG Recording***

258 We used a 62-channel electrode cap connected to Brain Vison MRplus amplifiers (BrainProducts)

259 to record EEG at a sampling rate of 500 Hz. The reference electrode was Pz and ground was Fz.

260 Two additional eye electrodes were placed beside and below the left eye to monitor for saccades

261 and blinks, respectively.

262

263 ***EEG preprocessing***

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264   EEG data were preprocessed using custom MATLAB scripts written in Version 2015b

265   (TheMathWorks, Natick, MA). For each experiment, raw EEG data from the SSVEP task and the

266   SST were imported into MATLAB and concatenated (i.e., the SST timeseries data were appended

267   to the SSVEP task data). The merged timeseries were bandpass filtered (High-pass cutoff: .5 Hz;

268   Low-pass cutoff: 50 Hz) using a Hamming-windowed sinc finite-impulse response filter (the

269   default FIR filter in EEGLAB). All timeseries were visually inspected and non-stereotypical

270   artifacts (muscle artifacts, transient electrode artifacts, etc.) were removed. Segments including

271   saccades were manually removed and excluded from the further analyses to exclude trials in which

272   attention was shifted overtly. Then data were re-referenced to the common average and entered

273   into an infomax Independent Component Analysis (ICA) decomposition algorithm. Specifically,

274   three different trial selections were performed prior to ICA, depending on which hypothesis was

275   tested. Separate ICA solutions were generated for each of the three datasets.

276   1.   To test the primary hypotheses (i.e., that unexpected sounds and stop-signals produce

277   N2/P3 complexes from the same neural source, and that the activity of that source after

278   unexpected sounds predicts the interruption of the SSVEP), all UNEXPECTED trials from

279   the SSVEP task in Experiment 1 were combined with a matched amount of randomly

280   selected NO SOUND trials, as well as the entire SST data. This was done to equate the

281   signal-to-noise ratio of the SSVEP between UNEXPECTED and NO SOUND trials in the

282   SSVEP task. Specifically, for each UNEXPECTED trial, a pseudo-event was generated

283   within a randomly paired NO SOUND trial at the same after flicker onset at which the

284   unexpected sound was played in the UNEXPECTED trial. Data from the SSVEP trials was

285   included starting from 60ms prior to cue onset to 60ms following the response to the target.

14

2. To test whether EXPECTED sounds influence the SSVEP as well, data from the SSVEP portion of Experiment 2 were combined with the SST data for each of those subjects.

3. To test the attentional tuning of the SSVEP (i.e., to perform a manipulation check on the efficacy of the attentional cue), a dataset that only included the 288 NO SOUND trials from the SSVEP task (for the subjects in Experiment 1) or the 72 EXPECTED trials (for the subjects in Experiment 2) was generated.

Each of the resulting IC matrices for every subject was separately screened for stereotypic artifacts (e.g., blinks, EKG, channel noise), which were excluded prior to further analysis.

Independent Component selection

Motor inhibition component selection. In line with previous work from us and others (Wessel, 2018b; Castiglione et al., 2019; Waller et al., 2019), one IC was selected from each participants' ICA solution using the SST portion of the data as a functional localizer (this was only done for the ICA solutions generated to test the influence of the sounds on the SSVEP, and not on the ICA solution generated to test the SSVEP for attentional tuning effects, which did not include the SST data). In the following, this component will be referred to as motor inhibition independent component (MI-IC). The MI-IC shows four primary characteristics in the SST that have been demonstrated in our previous work (Wessel & Aron, 2015; Wessel et al., 2016; Wessel, 2017). First, the MI-IC shows maximal weights around fronto-central electrodes (FCz, Cz). Second, the MI-IC shows a pronounced positive deflection in its ERP, which peaks around 250-300 ms after stop-signals (the stop-signal P3), which is not present during matched time periods on Go-trials. Third, the onset of this ERP in the MI-IC occurs significantly earlier in successful stop-trials compared to failed stop-trials. This characteristic reflects a key prediction of the in the independent

309   race model of the SST (Logan & Cowan, 1984), which holds that a faster stop process will lead to

310   successful stopping. Fourth and finally, the onset of stop-related P3 is positively correlated to the

311   behavioral measure of stopping speed (SSRT) across subjects, such that subjects with an earlier

312   onset of the P3 component in the MI-IC have a shorter SSRT (for details, cf. Wessel & Aron,

313   2015).

314   To extract the IC for each subject that most closely corresponded to these criteria, we first

315   selected those ICs that showed scalp topographies with maximal weights at fronto-central

316   electrodes (F1, Fz, F2, FC1, FCz, FC2, C1, Cz, C2). Second, the resulting ICs were individually

317   backprojected into channel space and their fronto-central stop-trial ERP was plotted to ensure that

318   they showed a fronto-central N2/P3 complex following stop-signals. The relationship between the

319   activity of these components and stopping behavior was then validated as follows.

320

321   Motor inhibition component validation. To identify the onset of the stop-signal P3 feature of the

322   MI-IC, four types of trials in the SST portion of each subjects' data were investigated: successful

323   stop (SS) and matched go (SGo); failed stop (FS) and matched go trials (FGo). Go-trials were

324   matched to stop-trials by selecting one go-trial per stop-trial in which the SSD staircase was at the

325   same point (i.e., for a stop trial with an SSD of 200ms, we selected a go trial on which a stop-

326   signal would have appeared at 200ms, had there been one). We then compared the mean sample-

327   to-sample difference in MI-IC activity between stop and matched go-trials (SS vs. SGo; FS vs.

328   FGo) within each subject using label-switching permutation testing (10,000 iterations, p = .01,

329   corrected for multiple comparisons using the false-discovery rate method, FDR, Benjamini et al.,

330   2006) . The onset of the P3 was then defined as the first sample at which stop and matched go-trial

331   MI-IC ERPs significantly diverged prior to the peak of the P3 (in essence, the peak of the P3 was

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332   identified, and the algorithm then worked 'backwards' towards the stop-signal until the stop-vs-

333   go difference was no longer significant). The thusly identified P3 onset was then compared

334   between successful and failed stop-trials across subjects using a paired-samples t-test. Moreover,

335   the onset of the P3 on successful stop-trials was correlated to each subjects' SSRT estimate using

336   Pearson's correlation coefficient. These procedures are identical to our first report of these

337   properties (Wessel & Aron, 2015).

338

339   <u>SSVEP component selection.</u> Independent components reflecting the SSVEP were identified

340   based on topographical and frequency criteria for all three ICA solutions for each subject. To be

341   selected as an SSVEP component, an IC had to fulfill the following criteria: First, it had to show

342   weight matrix maximum at parieto-occipital electrodes (PO8, PO7, PO4, PO3, P8, P7, P6, P5, P4,

343   P3, P2, P1, O2, and O1). Second, it had to be among the top eight ICs in terms of explained

344   variance of the whole-scalp 12 and 18 Hz response (identified by EEGLAB's built-in spectopo()

345   function). This resulted in an average of 3.24 components per subject that were selected as SSVEP

346   components (range: 2-6).

347

348   <u>Manipulation check: Unexpected events and stop-signals elicit N2/P3 complexes in the same IC.</u>

349   After selecting the MI-IC and confirming its properties in the SST, we then aimed to replicate prior

350   findings showing that unexpected events evoke an N2/P3 complex within that same neural source

351   (Wessel & Aron, 2013; Wessel & Huber, 2019). To this end, the MI-IC was back-projected into

352   channel-space, and the fronto-central ERP (average at FCz and Cz) of that back-projection was

353   time-locked to the onsets of UNEXPECTED sounds in the SSVEP task and the above-mentioned

354   'pseudo-events' on NO SOUND trials in Experiment 1 (-500 to 1000ms), as well as to the

355   EXPECTED sounds in Experiment 2. We then compared the subject-average activity time-course

356   using sample-to-sample t-tests in the post-event period. Specifically, a paired-samples test was

357   used to test the difference between UNEXPECTED sounds and the NO SOUND condition in

358   Experiment 1, and an independent samples t-test was used to test the difference between the

359   UNEXPECTED sounds in Experiment 1 and the EXPECTED sounds in Experiment 2. Both

360   resulting vectors of p-values were corrected for multiple comparisons using the FDR-method to a

361   critical alpha-level of .05.

362

363   ***Time-frequency analysis***

364   To convert the time-domain EEG signal to the time-frequency domain, the entire EEG timeseries

365   were bandpass filtered with 30 linearly spaced center frequencies spanning $1 - 30$ Hz with a range

366   of 1 Hz around the respective center frequencies. The analytical amplitude of the signal at each

367   center frequency was then computed using the square of the absolute of the Hilbert coefficients,

368   identified using MATLAB's hilbert() function.

369

370   ***Steady-state visual evoked potential analysis***

371   SSVEP extraction. All SSVEP activity was quantified from the time-frequency time series at

372   electrodes PO7 (left hemisphere) and PO8 (right hemisphere). For frequency and attentional tuning

373   analyses, EEG data with all ICs were used to match prior studies. The remainder of SSVEP

374   analyses used the backprojection of the EEG data produced using the SSVEP ICs because our

375   main hypothesis tested how neural activities from two statistically independent neural sources (MI-

376   IC and SSVEP IC) interact with each other after unexpected sounds. The IC-based source-signal

377   approach not only avoids cross-contamination of channel-space activity due to volume conduction,

18

378   but it also increases the single-trial signal-to-noise ratio of both the SSVEP and the MI-IC activity.

379   Five participants from Experiment 2 were excluded from the SSVEP analyses because after artifact

380   rejection, at least one of their SSVEP conditions included fewer than 10 trials (Experiment 2

381   contained only 18 trials per each of the four SSVEP conditions).

382

383   Manipulation check: frequency tuning. To identify whether there was an SSVEP entrained to the

384   visual stimuli, the data were segmented from -300 to 3,000 ms relative to flicker onset. Each trial

385   was then baseline corrected by converting the amplitude to a z-score relative to the 300ms pre-

386   stimulus period. For each trial, we then computed the median amplitude of the z-scored time-

387   frequency amplitude at both 12 and 18Hz from the contralateral hemisphere to the location of the

388   f12 or f18 flicker. These values were then averaged to produce the trial-average SSVEP amplitude

389   for each frequency (12/18Hz ERSP) contralateral to each flicker type. We then tested whether the

390   SSVEP at either hemisphere was entrained more strongly to the frequency of the flicker in the

391   contralateral visual field using paired-samples t-tests.

392

393   Manipulation check: Attentional tuning. We then investigated whether instructed shifts in covert

394   attention increased the amplitude of the SSVEP, in line with previous literature (e.g., Regan, 1989;

395   Müller et al., 1998; Ding et al., 2006). To this end, four SSVEP time series were investigated: 12

396   Hz attended, 12 Hz unattended, 18Hz attended, 18Hz unattended. These analyses were performed

397   on the contralateral electrode only. To investigate the effect of attentional tuning over time, the z-

398   scored single-trial data described above was binned into consecutive segments of 200ms, and the

399   attended condition was tested against the unattended condition for each frequency using paired-

400   samples t-tests.

19

401

402   Hypothesis test: SSVEP change after unexpected sounds. For all Experiment 1 datasets, each

403   UNEXPECTED trial was paired with a matching NO SOUND trial as described above. Trials were

404   then epoched into -500 to 1000ms segments around the sound for the UNEXPECTED trials, and

405   around the same time point for the matching NO SOUND trial. For all Experiment 2 datasets, the

406   data were time-locked to the EXPECTED sound. For all three trials types, both the attended

407   (contralateral to cued location) and the unattended (ipsilateral) SSVEP were averaged across trials,

408   and the resulting data were z-scored relative to the 500ms period prior to sound onset (or the

409   'pseudo'-sound in case of the matched NO SOUND trials). Differences between the resulting

410   average time-courses were then tested for significance using a sample-to-sample 2x2 ANOVA.

411   Specifically, to test whether UNEXPECTED sounds reduced the SSVEP compared to the NO

412   SOUND condition, we analyzed the data from Experiment 1 using the repeated-measures factors

413   SOUND (unexpected vs. no sound) and ATTENTION (attended vs. unattended). Furthermore, to

414   test whether any change after the UNEXPECTED sound was due to the expectancy violation,

415   rather than presence of the sound itself, we compared the UNEXPECTED sound condition from

416   Experiment 1 with the EXPECTED sound condition from Experiment 2 using the between-subject

417   factor SOUND (Exp1: unexpected vs. Exp2: expected sound) crossed with the within-subject

418   factor ATTENTION (attended vs. unattended). Both ANOVAs were applied to each sample point

419   individually, resulting in three vectors of p-values (main effect of SOUND, main effect of

420   ATTENTION, SOUND * ATTENTION interaction) for each analysis. These p-values were then

421   corrected for multiple comparisons using the FDR-method to a critical alpha level of .05.

422

423

Single trial general linear model: N2/P3 to SSVEP relationship

424

425   We tested our main hypothesis that interruptions of the SSVEP by unexpected sounds will

426   be related to MI-IC activity triggered by those sounds using a single-trial GLM. To do so, we

427   quantified the peak amplitude of the N2 and P3 portions of the N2/P3 complex in the MI-IC on in

428   each trial with an UNEXPECTED sound in Experiment 1, as well as the reduction of the SSVEP

429   on the same trial. The N2 peak amplitude was quantified by measuring the activity-minimum in

430   the MI-IC backprojection at fronto-central electrodes FCz and Cz between 140 and 300ms

431   following the time-locking event. The P3 peak amplitude was quantified by measuring the activity-

432   maximum within a 150ms window starting from the peak latency of the N2 within that same time

433   course.

434   To identify the change in trial-to-trial SSVEP after an unexpected sound on the same trials,

435   we first conducted a group-level ANOVA on the trial averages. Specifically, in order to find a

436   common time window for this analysis regardless of the type of SSVEP frequency, and to provide

437   an independent contrast to identify this window, we conducted repeated-measures 2-way ANOVA

438   with factor SOUND (unexpected vs. no sound) and FREQUENCY (12 Hz vs. 18Hz). As above,

439   this was repeated for all samples and then FDR-corrected to reach a critical alpha level of .05. The

440   time window in which the main effect of SOUND was significant was used to quantify the degree

441   of SSVEP disruption at the single trial level. The SSVEP reduction was quantified as the change

442   from baseline within that time window (38-744 ms).

443   Based on these values for the SSVEP ICs and the MI-ICs, four single-trial GLMs were

444   generated for each participant, relating the single-trial amplitude of the MI-IC (N2 and P3) to the

445   single-trial amplitude in the SSVEP (attended and unattended). Both predictors and DVs were

446   standardized prior to the calculation of the coefficients. The resulting regression beta coefficients

21

447   were Fisher's z-transformed to ensure a normal distribution prior to statistical testing. The thusly-

448   transformed beta-weights for each subject were then tested for significant differences from 0 using

449   paired-samples t-tests.

450

451   *Temporal order of N2, P3, and SSVEP reduction*

452   Finally, we compared the latencies of the ERP peaks in the MI-IC and the SSVEP reduction

453   in the SSVEP-IC. These latencies were quantified on each UNEXPECTED trial and collapsed

454   across all conditions to be averaged, and then compared across subjects using paired-samples t-

455   tests. We predicted that the timing of N2 and P3 would reliably precede the SSVEP suppression.

456

457   **Results**

458   *Stop-signal task (functional localizer)*

459   <u>Behavior</u>. Consistent with the assumption of the independent race model (Logan & Cowan, 1984),

460   all participants across both experiments showed slower go-trial RT (mean: 557.16 ms; SEM: 13.37)

461   compared to failed stop-trial RT (mean: 482.53 ms; SEM: 11.30). The SSD staircase algorithm

462   successfully kept the probability of stopping around .5, with a range from 0.47 to 0.57. Average

463   SSRT was 216 ms (SEM: 5.14) and average SSD was 336 ms (SEM: 16.77). Average error rate

464   was 0.38% and miss rate was 3.37%. Overall, these results represent a typical parameter range for

465   healthy young adults.

466

467   <u>MI-IC validation</u>. Validating the MI-IC included testing whether 1) the MI-IC P3 onset on

468   successful stop trials occurred reliably earlier than on failed stops; and 2) the P3 onset in successful

469   stops was correlated to the behavioral measure of stopping speed (SSRT) across subjects. The

470   N2/P3 complex in the MI-IC in the SST is depicted in ***Figure 2A***. ***Figure 2B*** shows that P3 onset

471   on successful stop-trials (mean: 258.43 ms; SEM: 6.51) was significantly earlier compared to

472   failed stop-trials (mean: 284.71 ms; SEM: 7.59), $t_{(41)}$=-6.26, p<.001. Finally, ***Figure 2C*** shows that

473   the onset of the successful stop-trial P3 was positively correlated with SSRT (r = 0.52, p<.001).

474   These results suggest that the P3 ERP component of the MI-IC index the activity of the inhibitory

475   control process in the SST, as described in prior work.

476

477
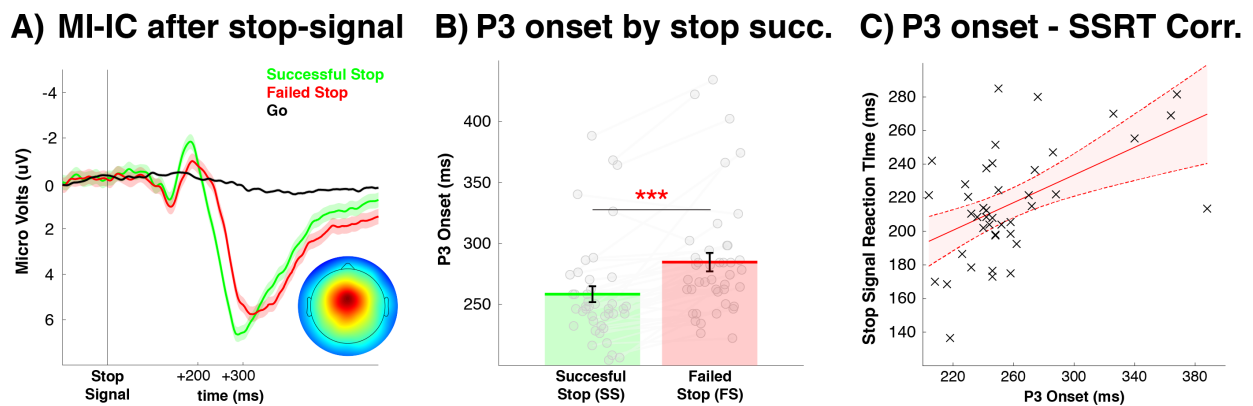
478   ***Figure 2.*** *MI-IC activity after stop-signals. A) MI-IC fronto-central ERP time-locked to stop-*

479   *signals in the SST data. Scalp topography inset represents averaged MI-IC weights from all*

480   *participants. B) P3 onsets in successful (SS) vs. failed stop (FS) trials. C) Cross-subject correlation*

481   *between SS P3 onset and SSRT.*

482

483   <u>Manipulation check: MI-IC activity following unexpected sounds.</u> In line with our hypotheses and

484   prior work, the neural source underlying the MI-IC also showed a pronounced N2/P3 following

485   unexpected sounds in the SSVEP task (***Figure 3***).
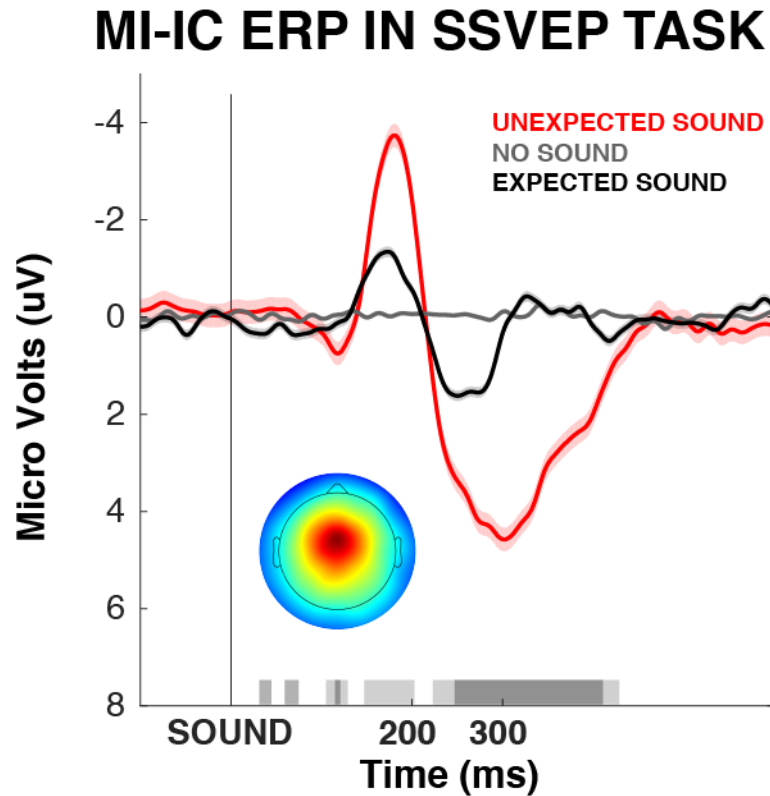
Figure 3. *MI-IC fronto-central ERP activity after UNEXPECTED/EXPECTED/NO sound stimuli in the crossmodal SSVEP oddball task. Bright grey shade at the bottom of the figure indicates significant difference between UNEXPECTED vs. NO SOUND trials. Dark grey shade indicates significant difference between UNEXPECTED vs. EXPECTED sound trials.*

Steady-state visual evoked potentials

Manipulation check: frequency tuning. The hemisphere contralateral to f_{12} showed reliably higher $ERSP_{12Hz}$ compared to $ERSP_{18Hz}$ ($t_{(36)}$=3.29, p<.01, Fig.4A), indicating that the 12Hz flickers successfully entrained a contralateral SSVEP . In turn, the hemisphere contralateral to f18 showed significantly higher $ERSP_{18Hz}$ compared to $ERSP_{12Hz}$ ($t_{(36)}$=3.81, p<.001, Fig.4A), indicating that the 18Hz flickers successfully entrained a contralateral SSVEP as well.

24

499   Manipulation check: Attentional tuning. As can be seen in **Figure 4B**, we found a significant

500   attentional enhancement effect in the time window from 2.2 to 2.8 seconds after flicker onsets

501   ($t_{(36)} > 2.1$, $p < .05$), but only in the 12Hz condition. No such effect was found for the 18Hz condition.

502   While this was not the expected outcome of this manipulation, this is in line with prior reports

503   showing that attentional tuning of the SSVEP is often limited to the alpha range (Ding et al., 2006).
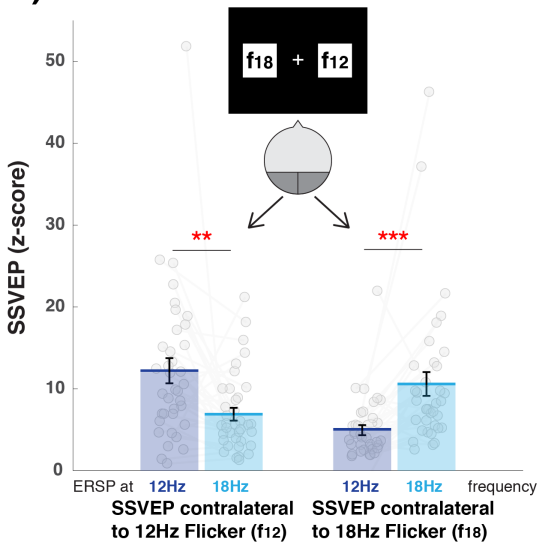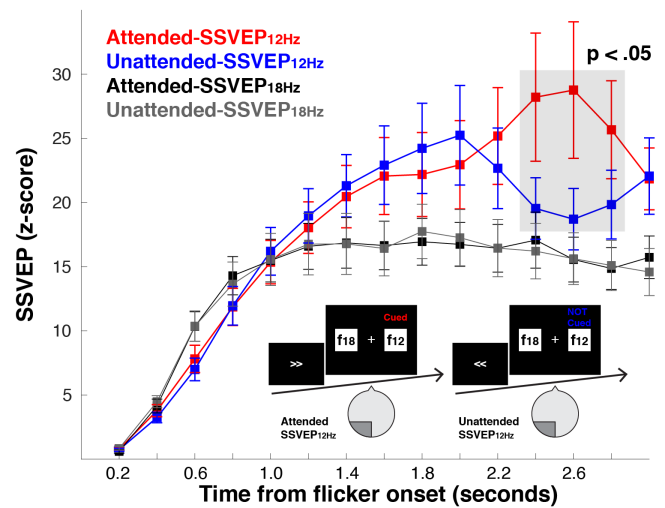

504

505   **Figure 4**. *Frequency entrainment and attentional tuning effects in the SSVEP. A) Cross-frequency*

506   *comparison of the SSVEP within the contralateral hemisphere. B) Binned time series after the*

507   *flicker onset. Grey shade indicates significant difference between attended vs. ignored in the*

508   *SSVEP to 12Hz flickers.*

509

510   **Main hypotheses**

511   SSVEP disruption by unexpected sounds. We then tested if unexpected sounds interrupted the

512   ongoing SSVEP. A repeated-measures 2-way ANOVA with factor SOUND (unexpected vs. no

513   sound) and ATTENTION (attended vs. unattended) showed main effects of SOUND from 0 to

514   770 ms in the 12Hz SSVEP (**Figure 5A**). Despite a visible reduction of the unexpected-sound

25

515   SSVEP in the 18Hz SSVEP, there was no main effect of SOUND in the 18Hz SSVEP that survived

516   corrections for multiple comparisons (*Figure 5B*). A mixed-model 2-way ANOVA with factor

517   SOUND (Exp1: unexpected vs. Exp2: expected sound) and ATTENTION showed main effects of

518   SOUND from 0 to 752 ms in the 12Hz SSVEP (*Figure 5C*); and from 148 to 616 ms in the 18Hz

519   SSVEP (*Figure 5D*). There was no significant main effect of ATTENTION or

520   SOUND*ATTENTION interaction for either ANOVA. This indicates that after unexpected

521   sounds, the SSVEPs to both attended and unattended stimuli were significantly disrupted

522   compared to no sound and expected sound trials.
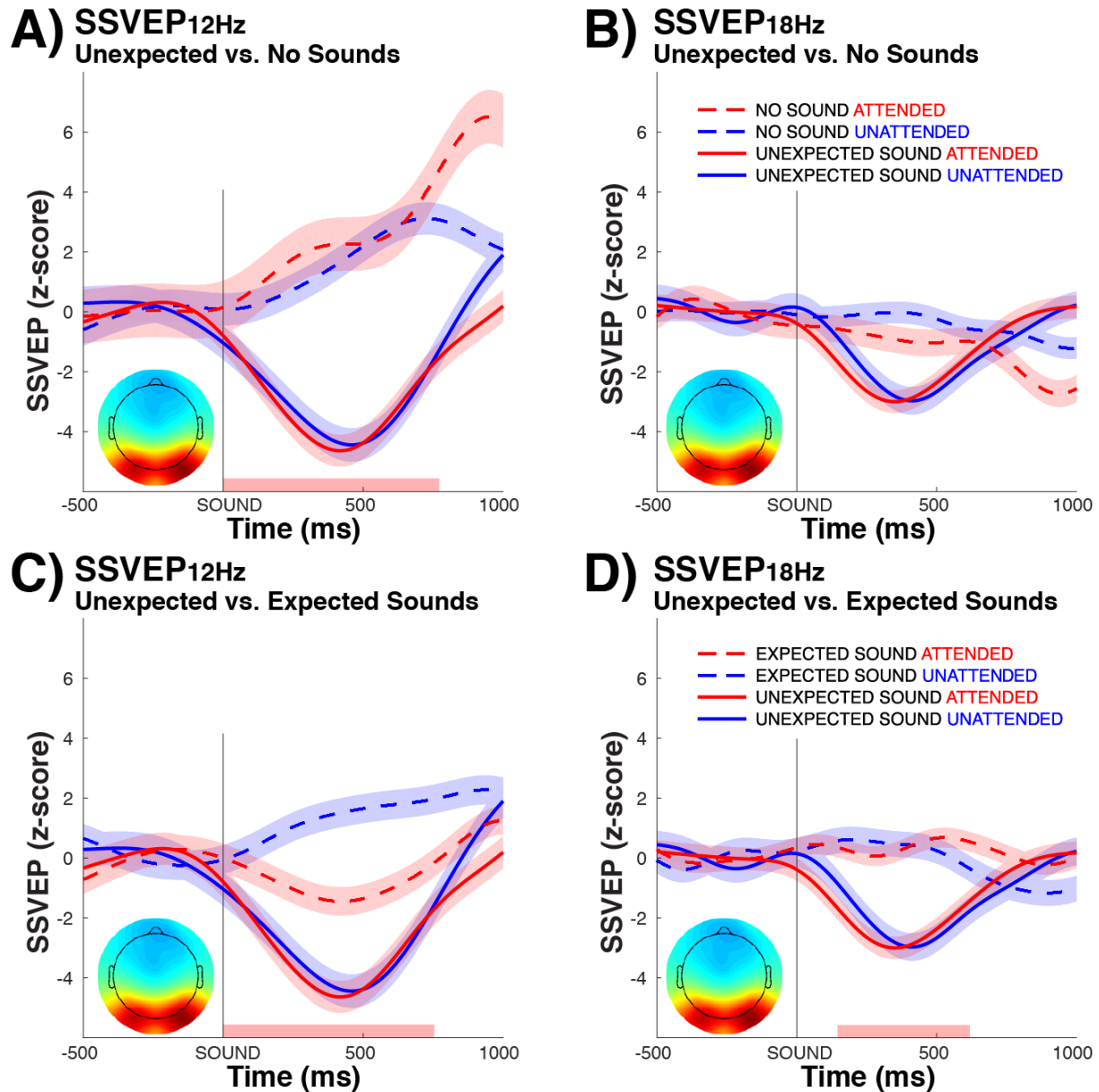
Figure 5. *Suppressive effects of unexpected sounds on the active SSVEP. Time course of UNEXPECTED vs. NO SOUND trials in A) 12 Hz SSVEP; and B) 18Hz SSVEP. Time course of UNEXPECTED (Experiment 1) vs. EXPECTED sound (Experiment 2) trials in C) 12Hz SSVEP; and D) 18Hz SSVEP.*

529   N2/P3 to SSVEP relationship. Our main hypothesis investigated the relationship between the

530   activity of the MI-IC following unexpected sounds and the modulation of the SSVEP by those

531   same sounds on the same trial. Since both N2 amplitudes and SSVEP reductions are negative-

532   signed variables (-), greater N2 amplitudes leading to greater SSVEP decrements would result in

533   positive beta weights, whereas the opposite would be true for the positive-valued P3 component

534   (see *Figure 6B* for direction of each activity). We found that the MI-IC N2 and P3 were

535   differentially related to the two components (attended and unattended) of the SSVEP IC.

536   Specifically, the N2 amplitude reliably predicted the degree of suppression in the attended SSVEP

537   ($t_{(20)}$=2.67, p=.014, *Figure 6A*), where the P3 amplitude reliably predicted the surprise-related

538   decrement in the unattended SSVEP ($t_{(20)}$=-2.44, p=.023, *Figure 6A*).

A) Single-trial analysis
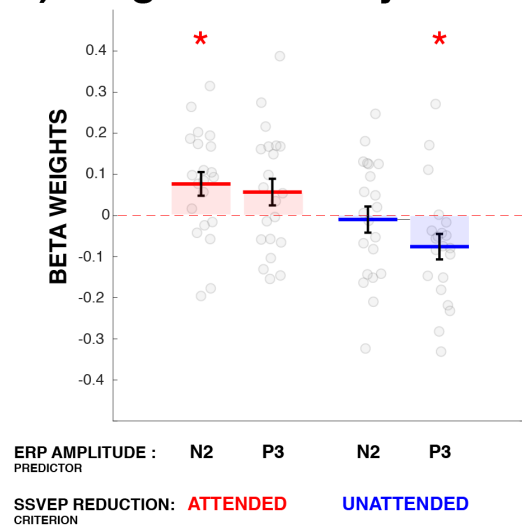
B) N2 & P3 precede SSVEP dip
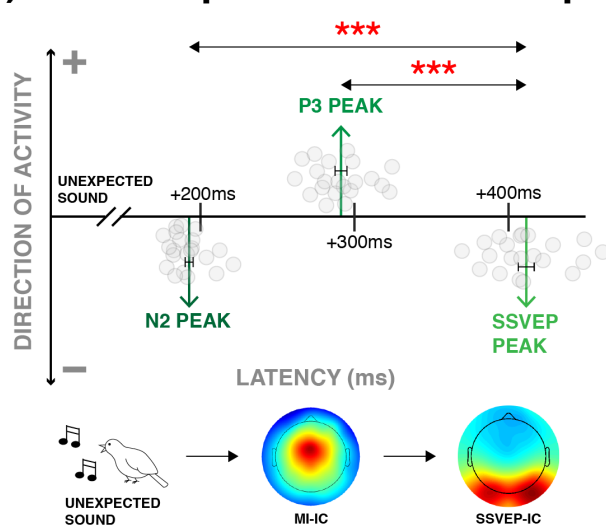
540   *Figure 6. Single trial GLM results and peak onset comparison between MI-IC and SSVEP IC. A)*

541   *Trial-to-trial relationship between N2/P3 amplitudes and SSVEP reduction to attended and*

542   *unattended stimuli after unexpected sounds. B) The N2 and P3 peak latencies in the MI-IC*

543   *backprojection and SSVEP suppression latencies in the SSVEP IC following unexpected sounds.*

544

28

545   <u>N2, P3, SSVEP reduction latencies.</u> The average N2 peak latency was 194.98 ms (SEM: 2.56),

546   whereas the average P3 peak latency was 292.71 ms (SEM: 3.82). The average latency of the

547   SSVEP interruption was at 411.55 ms (SEM: 5.16). Both N2 ($t_{(20)}$=-34.46, p<.0001) and P3 ($t_{(20)}$=-

548   17.73, p<.0001) latency were significantly earlier than the SSVEP latency (**Figure 6B**). These

549   findings demonstrate that MI-IC activity following the unexpected sound preceded the suppression

550   of the SSVEP.

551

552   **Discussion**

553   In the current study, we investigated whether the interruption of visual attention after

554   unexpected events is related to the activity of a well-known brain mechanism for inhibitory control.

555   In a newly-developed paradigm, we first found that unexpected sounds lead to a suppression of

556   SSVEP amplitudes to both attended and unattended visual stimuli. Moreover, a control experiment

557   confirmed that this was not true following expected sounds. Using a functional localizer task to

558   elicit the neural signature of a well-characterized brain mechanism for motor inhibition, we then

559   replicated the finding that this EEG source (the MI-IC) was indeed active following unexpected

560   sounds. Then, using a single-trial analysis of the independent components underlying inhibitory

561   control and the SSVEP, we found that specific parts of the MI-IC response to unexpected sounds

562   related to specific changes in the SSVEP on the same trial. Namely, the amplitude of the N2

563   potential of the fronto-central N2/P3-complex related to the suppression of the SSVEP to the

564   attended stimulus, whereas the P3 potential related to the suppression of the SSVEP to the

565   unattended stimulus.

566   These results provide new empirical evidence for the proposal that the brain's inhibitory

567   control mechanism is even broader than previously thought. Indeed, instead of solely affecting

29

568   motor representations (e.g., during action-stopping in the stop-signal task), attentional

569   interruptions after unexpected events appear to potentially result from inhibitory control as well.

570   Indeed, the proposal that inhibitory control could affect non-motor representations goes back to

571   the original work on the stop-signal task and the underlying race model, in which it was already

572   proposed the stop-signal task invokes a mechanism that serves to "inhibit thought and action"

573   (Logan et al., 1984). Notably, though, the vast majority of the subsequent work on this paradigm

574   has focused on the stopping of action. In cognitive neuroscience, this work on action-stopping has

575   firmly established a neural mechanism for motor inhibition, which serves to suppress ongoing

576   motor representations when necessary (for review, cf. Verbruggen & Logan, 2009; Levy &

577   Wagner, 2011; Ridderinkhof et al., 2011; Aron et al., 2014; Verbruggen et al., 2019). Only recently

578   have cognitive neuroscience studies begun to harken back to the "thought" part of Logan and

579   colleagues original proposal, thereby extending the effective range of this inhibitory mechanism

580   to non-motor representations. However, these studies have so far exclusively focused on

581   mnemonic representations, including short-term memory for face stimuli (Chiu & Egner, 2014,

582   2015), verbal working memory (Wessel et al., 2016; Castiglione et al., 2019), and motor sequence

583   memory (Tempel et al., 2020). Expanding on this work, our study is the first of its kind to

584   demonstrate that active attentional representations could be subject to the same type of inhibition

585   as mnemonic and motor representations, mediated via the same neural pathway.

586   In our previous theoretical work on this topic (cf., Wessel & Aron, 2017), we have argued

587   that the neuroanatomy of the neural pathway underlying inhibitory control could offer an

588   explanation as to why non-motor representations like memory and attention could be subject to

589   the same type of inhibition as the motor representations. The mechanism underlying inhibitory

590   control involves a well-specific network of cortical and basal ganglia regions (Aron et al., 2007;

30

591   Wiecki & Frank, 2013; Jahanshahi et al., 2015; Chen et al., 2020). Mechanistically, it is thought

592   that the cortical areas of this network (which include the areas that produce the N2/P3 complex;

593   Enriquez-Geppert et al., 2010; Huster et al., 2012) signal the need to initiate inhibitory control to

594   the basal ganglia; specifically, to the subthalamic nucleus (Swann et al., 2011; Ray et al., 2012;

595   Schmidt et al., 2013). In turn, the subthalamic nucleus is then thought to interrupt the thalamo-

596   cortical loops that are underlying active motor representations (via the output nuclei of the basal

597   ganglia, most notably the globus pallidus, Alexander & Crutcher, 1990; Nambu, 2008; Tanibuchi

598   et al., 2009; Goldberg et al., 2013). Within that same framework, we propose that such fronto-

599   subthalamic-pallidal-thalamocortical inhibition could potentially extend to *any* type of active

600   neural representation that is maintained via thalamocortical loops (Wessel & Aron, 2017). Indeed,

601   of core relevance to the current finding is the fact that the nuclei of the thalamus are a key nodes

602   in the maintenance of not just motor representations, but also of active attentional representations

603   (e.g., Desimone et al., 1990; McAlonan et al., 2008). In fact, while classic conceptualizations

604   thought of the thalamus as merely a relay of sensory information, more recent work has found that

605   thalamic activity exerts gain control over attentional representations, especially in the visual

606   system (e.g., Saalmann & Kastner, 2009; Wimmer et al., 2015; Mease et al., 2016) and that lesions

607   to the thalamus crucially interfere with attentional selection (Snow et al., 2009). If sustained visual

608   attention, such as the type that is operationalized in our current paradigm, is indeed dependent on

609   thalamocortical loops, it is conceivable that the same type of inhibitory influence from the basal

610   ganglia that regulates motor behavior could also function to rapidly inhibit these active attentional

611   representations.

612   In addition to this hypothesized subcortical overlap between the neural networks that

613   regulate motoric and attentional representations, it is notable that the *cortical* areas of the fronto-

614   basal ganglia inhibitory control network also overlap substantially with the wider networks

615   implicated in attentional control in general. Indeed, Corbetta & Shulman's seminal account of the

616   ventral attention network – which ostensibly functions as a 'circuit breaker' that is triggered by

617   suddenly appearing, behaviorally relevant stimuli (Corbetta & Shulman, 2002; Corbetta et al.,

618   2008) – includes both cortical areas of the proposed fronto-basal ganglia inhibitory control network

619   (the preSMA and the rIFG). Notably, however, in its original conceptualization, this purported

620   ventral attention network does not include any specific areas in the basal ganglia, which we would

621   propose based on our circuit model of inhibitory control. However, the absence of prominent basal

622   ganglia involvement in the work on the ventral attention network may be a consequence of the fact

623   that most of the work on that network has been performed using functional magnetic resonance

624   imaging at field strengths that lack a sufficient amount of signal to noise ratio in small subcortical

625   structures (Forstmann et al., 2017), especially in the subthalamic nucleus (de Hollander et al.,

626   2017). Therefore, while it is still unclear how attention may be regulated using subcortical circuitry

627   outside of the thalamus, it is possible that the type of attentional orienting implemented by the

628   ventral attention network is indeed aided by an active inhibitory effort that suppresses ongoing

629   attentional representations, implemented by the specific regions that form the inhibitory FBg-

630   network (Wessel & Aron, 2017). Hence, future studies could use the current paradigm to study the

631   involvement of the basal ganglia in the interruption of active attentional representations by

632   unexpected sensory events.

633   In this vein, it is important to mention that the scalp-EEG methods used here do not allow

634   any inferences about such underlying specific cortical or subcortical circuitry (though notably, the

635   trial-to-trial variance of N2/P3 complex is correlated with BOLD activity in cortical areas that

636   belong to both the fronto-basal ganglia inhibitory control network and the ventral attention network,

637   Enriquez-Geppert et al., 2010; Huster et al., 2012). However, scalp-EEG does provide a temporally

638   precise picture of the activity of the overall network. In this respect, the fronto-central N2/P3

639   complex is well-studied during both action-stopping and surprise processing (for reviews, see

640   Polich, 2007; Folstein & Van Petten, 2008; Huster et al., 2013; Kenemans, 2015). However, in

641   both literatures, the respective interpretation of the two constituent components of this complex

642   waveform (the N2 and the P3) is still subject of controversial debate. Before we offer an

643   interpretation that situates the current set of findings within these literatures, we will briefly

644   describe the predominant interpretations of the N2/P3 complex in both stopping and unexpected-

645   event processing. In the realm of action-stopping, there is relatively widespread agreement on the

646   fact that stopping involves a sequence of attentional detection of the stop-signal, followed by the

647   implementation of motor inhibition (Matzke et al., 2013; Verbruggen et al., 2014). The earliest

648   neuroscientific studies of the SST have proposed that the fronto-central P3 could index the

649   inhibitory process of this cascade (de Jong et al., 1990, cf. Huster et al., 2013, for a review). Indeed,

650   the P3 shows several features that reflect straightforward predictions regarding the inhibitory

651   process that are directly derived from the race model of the stop-signal task. Both its peak and its

652   onset occur earlier on successful compared to failed stop-trials (Kok et al., 2004; Wessel & Aron,

653   2015) and its timing indexes stop-signal reaction time across subjects (Wessel & Aron, 2015;

654   Huster et al., 2019). In line with this, much subsequent work has focused on the proposal that the

655   N2, which precedes the P3, could reflect a process that relates to the attentional processing of the

656   stop-signal itself, or the detection of the associated conflict between the initiated response and the

657   requirement to stop (Donkers & van Boxtel, 2004; Azizian et al., 2006; Enriquez-Geppert et al.,

658   2010; Smith et al., 2010; Groom & Cragg, 2015). In the realm of unexpected-event processing, the

659   exact nature of the mental processes reflected in the N2 and P3 events has been subject to intense

33

660   debate as well, with the entirety of the literature too numerous to discuss (see Folstein & Van

661   Petten, 2008, and Polich, 2007, for reviews). However, the dominant view of the fronto-central N2

662   (specifically, the N2b) is similar to that found in the stop-signal literature, in that it is commonly

663   assumed to reflect the overt attentional processing of the event or the associated conflict (Näätänen

664   & Gaillard, 1983; Folstein & Van Petten, 2008; Larson et al., 2014). The fronto-central P3 (also

665   known as the P3a) after unexpected results has more heterogeneous interpretations, which range

666   from working memory updating (Polich, 2007) to the evaluation of stimulus novelty (Friedman et

667   al., 2001) to the mobilization for action following significant stimuli (Nieuwenhuis et al., 2011).

668   While these two literatures are historically largely separate, the finding that both N2/P3 complexes

669   originate from the same neural source suggest that they may indeed reflect the same cascade of

670   processing in both situations (i.e., after stop-signals and unexpected events) – i.e., that processes

671   that take place after stop-signals are also automatically engaged by unexpected events. This is

672   backed up by findings from other imaging domains, such as the finding that unexpected events

673   lead to the suppression of the motor system (Wessel & Aron, 2013; Dutra et al., 2018; Novembre

674   et al., 2018; Novembre et al., 2019), and that they engage the subcortical circuitry that is ostensibly

675   underlying inhibitory motor control via the basal ganglia (Bočková et al., 2011; Wessel et al., 2016;

676   Fife et al., 2017). If it is indeed the case that the fronto-central N2/P3 complex reflects the same

677   cascade of processes after both stop-signals and unexpected events, the specific relationships

678   between the N2 and the P3 and the observed suppression of the SSVEP in the current study could

679   provide a potential rejoinder to this literature. Specifically, the fact that the single-trial N2 was

680   related to the interruption of the SSVEP to the *attended* stimulus lends support to the proposal that

681   this potential reflects an attentional orienting to a salient, or, in this case, unexpected stimulus.

682   This is in line with many conceptualizations from both the existing stop-signal and unexpected-

34

683 event literature (see above), which largely converge in their interpretation of the N2. Additionally,

684 the relationship between the trial-to-trial amplitude of the P3 and the observed interruption of the

685 SSVEP to the unattended stimulus suggest that indeed, the P3 may reflect the activity of a 'global',

686 non-selective inhibitory network that interrupts active *motoric and mental* representations when

687 the situational demands call for it (such as after unexpected events). This is in line with our own

688 recent theory about the activity of this network (Wessel & Aron, 2017), as well as with the proposal

689 that the stop-signal P3 in particular reflects the implementation of the inhibitory part of the

690 processing cascade during action-stopping (de Jong et al., 1990; Kok et al., 2004). Finally, it also

691 could provide a hint towards a specific (inhibitory) mechanism by which unexpected events could

692 aid the updating of current working memory contents (Polich, 2007), in line with recent studies of

693 the activity of this inhibitory control mechanism in the suppression of mnemonic representations

694 (Chiu & Egner, 2014, 2015; Wessel et al., 2016; Castiglione et al., 2019).

695 In summary, we have used a newly designed experimental paradigm to demonstrate that

696 unexpected, task-irrelevant sounds lead to a suppression of the neural representation of both

697 attended and unattended stimuli. Moreover, we used independent component analysis and single-

698 trial analyses of EEG to show that these interruptions are related to specific separate aspects of the

699 neural response to unexpected events within a neural system for inhibitory control. These findings

700 provide a crucial potential expansion of the operating range of a well-characterized neural

701 mechanism for cognitive control, and provide key insights into the cascade of neural and

702 psychological processing that leads to distraction.

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References

Alexander GE, Crutcher MD (1990) Functional architecture of basal ganglia circuits: neural

    substrates of parallel processing. Trends in Neurosciences 13:266-271.

Anderson MC, Green C (2001) Suppressing unwanted memories by executive control. Nature

    410:366-369.

Aron AR, Robbins TW, Poldrack RA (2014) Inhibition and the right inferior frontal cortex: one

    decade on. Trends in Cognitive Sciences 18:177-185.

Aron AR, Durston S, Eagle DM, Logan GD, Stinear CM, Stuphorn V (2007) Converging Evidence

    for a Fronto-Basal-Ganglia Network for Inhibitory Control of Action and Cognition. The

    Journal of Neuroscience 27:11860-11864.

Awh E, Belopolsky AV, Theeuwes J (2012) Top-down versus bottom-up attentional control: a

    failed theoretical dichotomy. Trends in Cognitive Sciences 16:437-443.

Azizian A, Freitas AL, Parvaz MA, Squires NK (2006) Beware misleading cues: Perceptual

    similarity modulates the N2/P3 complex. Psychophysiology 43:253-260.

Bacon WF, Egeth HE (1994) Overriding stimulus-driven attentional capture. Perception &

    Psychophysics 55:485-496.

728   Badry R, Mima T, Aso T, Nakatsuka M, Abe M, Fathi D, Foly N, Nagiub H, Nagamine T, Fukuyama

729   H (2009) Suppression of human cortico-motoneuronal excitability during the Stop-signal

730   task. Clinical Neurophysiology 120:1717-1723.

731   Benjamini Y, Krieger AM, Yekutieli D (2006) Adaptive linear step-up procedures that control the

732   false discovery rate. Biometrika 93:491-507.

733   Bočková M, Chládek J, Jurák P, Halámek J, Baláž M, Rektor I (2011) Involvement of the

734   subthalamic nucleus and globus pallidus internus in attention. Journal of Neural

735   Transmission 118:1235-1245.

736   Brainard DH (1997) The Psychophysics Toolbox. 10:433.

737   Cai W, Oldenkamp CL, Aron AR (2012) Stopping speech suppresses the task-irrelevant hand.

738   Brain and Language 120:412-415.

739   Castiglione A, Aron AR, Wagner J, Anderson M (2019) Preventing a Thought from Coming to

740   Mind Elicits Increased Right Frontal Beta Just as Stopping Action Does. Cerebral Cortex

741   29:2160-2172.

742   Chen W, de Hemptinne C, Miller AM, Leibbrand M, Little SJ, Lim DA, Larson PS, Starr PA (2020)

743   Prefrontal-Subthalamic Hyperdirect Pathway Modulates Movement Inhibition in

744   Humans. Neuron.

745   Chiu Y-C, Egner T (2014) Inhibition-Induced Forgetting: When More Control Leads to Less

746   Memory. Psychological Science 26:27-38.

747   Chiu Y-C, Egner T (2015) Inhibition-Induced Forgetting Results from Resource Competition

748   between Response Inhibition and Memory Encoding Processes. The Journal of

749   Neuroscience 35:11936-11945.

37

750   Corbetta M, Shulman GL (2002) Control of goal-directed and stimulus-driven attention in the

751   brain. Nature Reviews Neuroscience 3:201-215.

752   Corbetta M, Patel G, Shulman GL (2008) The Reorienting System of the Human Brain: From

753   Environment to Theory of Mind. Neuron 58:306-324.

754   Courchesne E, Hillyard SA, Galambos R (1975) Stimulus novelty, task relevance and the visual

755   evoked potential in man. Electroencephalography and Clinical Neurophysiology 39:131-

756   143.

757   Coxon JP, Stinear CM, Byblow WD (2006) Intracortical Inhibition During Volitional Inhibition of

758   Prepared Action. Journal of Neurophysiology 95:3371-3383.

759   Dawson ME, Schell AM, Beers JR, Kelly A (1982) Allocation of cognitive processing capacity

760   during human autonomic classical conditioning. Journal of Experimental Psychology:

761   General 111:273-295.

762   de Hollander G, Keuken MC, van der Zwaag W, Forstmann BU, Trampel R (2017) Comparing

763   functional MRI protocols for small, iron-rich basal ganglia nuclei such as the subthalamic

764   nucleus at 7 T and 3 T. Human Brain Mapping 38:3226-3248.

765   de Jong R, Coles MGH, Logan GD, Gratton G (1990) In search of the point of no return: The

766   control of response processes. Journal of Experimental Psychology: Human Perception

767   and Performance 16:164-182.

768   Derosiere G, Vassiliadis P, Duque J (2020) Advanced TMS approaches to probe corticospinal

769   excitability during action preparation. NeuroImage 213:116746.

770   Desimone R, Wessinger M, Thomas L, Schneider W (1990) Attentional control of visual

771   perception: cortical and subcortical mechanisms. In: Cold Spring Harbor symposia on

772   quantitative biology, pp 963-971: Cold Spring Harbor Laboratory Press.

773   Ding J, Sperling G, Srinivasan R (2006) Attentional Modulation of SSVEP Power Depends on the

774   Network Tagged by the Flicker Frequency. Cerebral Cortex 16:1016-1029.

775   Donkers FCL, van Boxtel GJM (2004) The N2 in go/no-go tasks reflects conflict monitoring not

776   response inhibition. Brain and Cognition 56:165-176.

777   Duque J, Greenhouse I, Labruna L, Ivry RB (2017) Physiological Markers of Motor Inhibition

778   during Human Behavior. Trends in Neurosciences 40:219-236.

779   Dutra IC, Waller DA, Wessel JR (2018) Perceptual Surprise Improves Action Stopping by

780   Nonselectively Suppressing Motor Activity via a Neural Mechanism for Motor Inhibition.

781   The Journal of Neuroscience 38:1482.

782   Enriquez-Geppert S, Konrad C, Pantev C, Huster RJ (2010) Conflict and inhibition differentially

783   affect the N200/P300 complex in a combined go/nogo and stop-signal task. NeuroImage

784   51:877-887.

785   Fife KH, Gutierrez-Reed NA, Zell V, Bailly J, Lewis CM, Aron AR, Hnasko TS (2017) Causal role for

786   the subthalamic nucleus in interrupting behavior. eLife 6:e27689.

787   Folstein JR, Van Petten C (2008) Influence of cognitive control and mismatch on the N2

788   component of the ERP: A review. Psychophysiology 45:152-170.

789   Forstmann BU, de Hollander G, van Maanen L, Alkemade A, Keuken MC (2017) Towards a

790   mechanistic understanding of the human subcortex. Nature Reviews Neuroscience

791   18:57-65.

792   Friedman D, Cycowicz YM, Gaeta H (2001) The novelty P3: an event-related brain potential

793   (ERP) sign of the brain's evaluation of novelty. Neuroscience & Biobehavioral Reviews

794   25:355-373.

795   Gaspelin N, Leonard CJ, Luck SJ (2017) Suppression of overt attentional capture by salient-but-

796   irrelevant color singletons. Attention, Perception, & Psychophysics 79:45-62.

797   Goldberg JH, Farries MA, Fee MS (2013) Basal ganglia output to the thalamus: still a paradox.

798   Trends in Neurosciences 36:695-705.

799   Goode C, Cole DM, Bolton DAE (2019) Staying upright by shutting down? Evidence for global

800   suppression of the motor system when recovering balance. Gait & Posture 70:260-263.

801   Greenhouse I, Oldenkamp CL, Aron AR (2011) Stopping a response has global or nonglobal

802   effects on the motor system depending on preparation. Journal of Neurophysiology

803   107:384-392.

804   Groom MJ, Cragg L (2015) Differential modulation of the N2 and P3 event-related potentials by

805   response conflict and inhibition. Brain and Cognition 97:1-9.

806   Huster RJ, Debener S, Eichele T, Herrmann CS (2012) Methods for Simultaneous EEG-fMRI: An

807   Introductory Review. The Journal of Neuroscience 32:6053-6060.

808   Huster RJ, Messel MS, Thunberg C, Raud L (2019) The P300 as marker of inhibitory control – fact

809   or fiction? bioRxiv:694216.

810   Huster RJ, Enriquez-Geppert S, Lavallee CF, Falkenstein M, Herrmann CS (2013)

811   Electroencephalography of response inhibition tasks: Functional networks and cognitive

812   contributions. International Journal of Psychophysiology 87:217-233.

813   Jahanshahi M, Obeso I, Rothwell JC, Obeso JA (2015) A fronto–striato–subthalamic–pallidal

814   network for goal-directed and habitual inhibition. Nature Reviews Neuroscience 16:719.

815   Kenemans JL (2015) Specific proactive and generic reactive inhibition. Neuroscience &

816   Biobehavioral Reviews 56:115-126.

817   Kok A, Ramautar JR, De Ruiter MB, Band GPH, Ridderinkhof KR (2004) ERP components

818   associated with successful and unsuccessful stopping in a stop-signal task.

819   Psychophysiology 41:9-20.

820   Larson MJ, Clayson PE, Clawson A (2014) Making sense of all the conflict: A theoretical review

821   and critique of conflict-related ERPs. International Journal of Psychophysiology 93:283-

822   297.

823   Levy BJ, Wagner AD (2011) Cognitive control and right ventrolateral prefrontal cortex: reflexive

824   reorienting, motor inhibition, and action updating. Annals of the New York Academy of

825   Sciences 1224:40-62.

826   Liesefeld HR, Liesefeld AM, Töllner T, Müller HJ (2017) Attentional capture in visual search:

827   Capture and post-capture dynamics revealed by EEG. NeuroImage 156:166-173.

828   Logan GD, Cowan WB (1984) On the ability to inhibit thought and action: A theory of an act of

829   control. Psychological Review 91:295-327.

830   Logan GD, Cowan WB, Davis KA (1984) On the ability to inhibit simple and choice reaction time

831   responses: A model and a method. Journal of Experimental Psychology: Human

832   Perception and Performance 10:276-291.

833   Matzke D, Love J, Wiecki T, Brown S, Logan G, Wagenmakers E-J (2013) Release the BEESTS:

834   Bayesian Estimation of Ex-Gaussian STop-Signal Reaction Time Distributions. Frontiers in

835   Psychology 4.

836   McAlonan K, Cavanaugh J, Wurtz RH (2008) Guarding the gateway to cortex with attention in

837   visual thalamus. Nature 456:391-394.

838   Mease RA, Metz M, Groh A (2016) Cortical Sensory Responses Are Enhanced by the Higher-

839   Order Thalamus. Cell Reports 14:208-215.

840   Morgan ST, Hansen JC, Hillyard SA (1996) Selective attention to stimulus location modulates the

841   steady-state visual evoked potential. Proceedings of the National Academy of Sciences

842   93:4770.

843   Müller MM, Picton TW, Valdes-Sosa P, Riera J, Teder-Sälejärvi WA, Hillyard SA (1998) Effects of

844   spatial selective attention on the steady-state visual evoked potential in the 20–28 Hz

845   range. Cognitive Brain Research 6:249-261.

846   Näätänen R, Gaillard AWK (1983) 5 The Orienting Reflex and the N2 Deflection of the Event-

847   Related Potential (ERP). In: Advances in Psychology (Gaillard AWK, Ritter W, eds), pp

848   119-141: North-Holland.

849   Nachev P, Wydell H, O'Neill K, Husain M, Kennard C (2007) The role of the pre-supplementary

850   motor area in the control of action. NeuroImage 36:T155-T163.

851   Nambu A (2008) Seven problems on the basal ganglia. Current Opinion in Neurobiology 18:595-

852   604.

853   Nieuwenhuis S, De Geus EJ, Aston-Jones G (2011) The anatomical and functional relationship

854   between the P3 and autonomic components of the orienting response.

855   Psychophysiology 48:162-175.

856   Novembre G, Pawar VM, Kilintari M, Bufacchi RJ, Guo Y, Rothwell JC, Iannetti GD (2019) The

857   effect of salient stimuli on neural oscillations, isometric force, and their coupling.

858   NeuroImage 198:221-230.

859   Novembre G, Pawar VM, Bufacchi RJ, Kilintari M, Srinivasan M, Rothwell JC, Haggard P, Iannetti

860   GD (2018) Saliency Detection as a Reactive Process: Unexpected Sensory Events Evoke

861   Corticomuscular Coupling. The Journal of Neuroscience 38:2385.

862   Parmentier FBR, Elford G, Escera C, Andrés P, Miguel IS (2008) The cognitive locus of distraction

863   by acoustic novelty in the cross-modal oddball task. Cognition 106:408-432.

864   Polich J (2007) Updating P300: An integrative theory of P3a and P3b. Clinical Neurophysiology

865   118:2128-2148.

866   Ray NJ, Brittain J-S, Holland P, Joundi RA, Stein JF, Aziz TZ, Jenkinson N (2012) The role of the

867   subthalamic nucleus in response inhibition: Evidence from local field potential

868   recordings in the human subthalamic nucleus. NeuroImage 60:271-278.

869   Regan D (1989) Evoked potentials and evoked magnetic fields in science and medicine. Human

870   brain electrophysiology:59-61.

871   Ridderinkhof K, Forstmann BU, Wylie SA, Burle B, van den Wildenberg WPM (2011)

872   Neurocognitive mechanisms of action control: resisting the call of the Sirens. Wiley

873   Interdisciplinary Reviews: Cognitive Science 2:174-192.

874   Saalmann YB, Kastner S (2009) Gain control in the visual thalamus during perception and

875   cognition. Current Opinion in Neurobiology 19:408-414.

876   Schall JD, Godlove DC (2012) Current advances and pressing problems in studies of stopping.

877   Current Opinion in Neurobiology 22:1012-1021.

878   Schmidt R, Leventhal DK, Mallet N, Chen F, Berke JD (2013) Canceling actions involves a race

879   between basal ganglia pathways. Nature Neuroscience 16:1118-1124.

880   Schröger E (1993) Event-related potentials to auditory stimuli following transient shifts of

881   spatial attention in a Go/Nogo task. Biological Psychology 36:183-207.

882   Silberstein RB, Ciorciari j, Pipingas A (1995) Steady-state visually evoked potential topography

883   during the Wisconsin card sorting test. Electroencephalography and Clinical

884   Neurophysiology/Evoked Potentials Section 96:24-35.

885   Simons DJ (2000) Attentional capture and inattentional blindness. Trends in Cognitive Sciences

886   4:147-155.

887   Smith JL, Smith EA, Provost AL, Heathcote A (2010) Sequence effects support the conflict theory

888   of N2 and P3 in the Go/NoGo task. International Journal of Psychophysiology 75:217-

889   226.

890   Snow JC, Allen HA, Rafal RD, Humphreys GW (2009) Impaired attentional selection following

891   lesions to human pulvinar: Evidence for homology between human and monkey.

892   Proceedings of the National Academy of Sciences 106:4054-4059.

893   Squires NK, Squires KC, Hillyard SA (1975) Two varieties of long-latency positive waves evoked

894   by unpredictable auditory stimuli in man. Electroencephalography and Clinical

895   Neurophysiology 38:387-401.

896   Stinear CM, Coxon JP, Byblow WD (2009) Primary motor cortex and movement prevention:

897   Where Stop meets Go. Neuroscience & Biobehavioral Reviews 33:662-673.

898   Swann N, Poizner H, Houser M, Gould S, Greenhouse I, Cai W, Strunk J, George J, Aron AR

899   (2011) Deep Brain Stimulation of the Subthalamic Nucleus Alters the Cortical Profile of

900   Response Inhibition in the Beta Frequency Band: A Scalp EEG Study in Parkinson's

901   Disease. The Journal of Neuroscience 31:5721.

902   Tanibuchi I, Kitano H, Jinnai K (2009) Substantia Nigra Output to Prefrontal Cortex Via Thalamus

903   in Monkeys. I. Electrophysiological Identification of Thalamic Relay Neurons. Journal of

904   Neurophysiology 102:2933-2945.

905   Tempel T, Frings C, Pastötter B (2020) EEG beta power increase indicates inhibition in motor

906   memory. International Journal of Psychophysiology 150:92-99.

907   Theeuwes J (2004) Top-down search strategies cannot override attentional capture.

908   Psychonomic Bulletin & Review 11:65-70.

909   Verbruggen F, Logan GD (2009) Models of response inhibition in the stop-signal and stop-

910   change paradigms. Neuroscience & Biobehavioral Reviews 33:647-661.

911   Verbruggen F, Stevens T, Chambers CD (2014) Proactive and reactive stopping when distracted:

912   An attentional account. Journal of Experimental Psychology: Human Perception and

913   Performance 40:1295-1300.

914   Verbruggen F et al. (2019) A consensus guide to capturing the ability to inhibit actions and

915   impulsive behaviors in the stop-signal task. eLife 8:e46323.

916   Vogel EK, Machizawa MG (2004) Neural activity predicts individual differences in visual working

917   memory capacity. Nature 428:748-751.

918   Vogel EK, McCollough AW, Machizawa MG (2005) Neural measures reveal individual differences

919   in controlling access to working memory. Nature 438:500-503.

920   Waller DA, Hazeltine E, Wessel JR (2019) Common neural processes during action-stopping and

921   infrequent stimulus detection: The frontocentral P3 as an index of generic motor

922   inhibition. International Journal of Psychophysiology.

923   Walter S, Quigley C, Andersen SK, Mueller MM (2012) Effects of overt and covert attention on

924   the steady-state visual evoked potential. Neuroscience Letters 519:37-41.

925   Wessel JR (2017) Perceptual surprise aides inhibitory motor control. Journal of Experimental

926   Psychology: Human Perception and Performance 43:1585-1593.

927   Wessel JR (2018a) A Neural Mechanism for Surprise-related Interruptions of Visuospatial

928   Working Memory. Cerebral Cortex 28:199-212.

929   Wessel JR (2018b) Testing Multiple Psychological Processes for Common Neural Mechanisms

930   Using EEG and Independent Component Analysis. Brain Topography 31:90-100.

931   Wessel JR (2020) β-Bursts Reveal the Trial-to-Trial Dynamics of Movement Initiation and

932   Cancellation. The Journal of Neuroscience 40:411.

933   Wessel JR, Aron AR (2013) Unexpected Events Induce Motor Slowing via a Brain Mechanism for

934   Action-Stopping with Global Suppressive Effects. The Journal of Neuroscience 33:18481.

935   Wessel JR, Aron AR (2015) It's not too late: The onset of the frontocentral P3 indexes successful

936   response inhibition in the stop-signal paradigm. Psychophysiology 52:472-480.

937   Wessel JR, Aron AR (2017) On the Globality of Motor Suppression: Unexpected Events and Their

938   Influence on Behavior and Cognition. Neuron 93:259-280.

939 Wessel JR, Huber DE (2019) Frontal cortex tracks surprise separately for different sensory

940   modalities but engages a common inhibitory control mechanism. PLOS Computational

941   Biology 15:e1006927.

942 Wessel JR, Danielmeier C, Morton JB, Ullsperger M (2012) Surprise and Error: Common

943   Neuronal Architecture for the Processing of Errors and Novelty. The Journal of

944   Neuroscience 32:7528.

945 Wessel JR, Jenkinson N, Brittain J-S, Voets SHEM, Aziz TZ, Aron AR (2016) Surprise disrupts

946   cognition via a fronto-basal ganglia suppressive mechanism. Nature Communications

947   7:11195.

948 Wiecki TV, Frank MJ (2013) A computational model of inhibitory control in frontal cortex and

949   basal ganglia. Psychological review 120:329.

950 Wimmer RD, Schmitt LI, Davidson TJ, Nakajima M, Deisseroth K, Halassa MM (2015) Thalamic

951   control of sensory selection in divided attention. Nature 526:705-709.

952 Yantis S (1993) Stimulus-driven attentional capture and attentional control settings. Journal of

953   Experimental Psychology: Human Perception and Performance 19:676-681.

954