Published in final edited form as:

Neuroimage. 2014 December ; 103: 225–234. doi:10.1016/j.neuroimage.2014.09.048.

Inhibitory motor control based on complex stopping goals relies on the same brain network as simple stopping

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Abstract

Much research has modeled action-stopping using the stop-signal task (SST), in which an impending response has to be stopped when an explicit stop-signal occurs. A limitation of the SST is that real-world action-stopping rarely involves explicit stop-signals. Instead, the stopping-system engages when environmental features match more complex stopping goals. For example, when stepping into the street, one monitors path, velocity, size, and types of objects; and only stops if there is a vehicle approaching. Here, we developed a task in which participants compared the visual features of a multidimensional go-stimulus to a complex stopping-template, and stopped their go-response if all features matched the template. We used independent component analysis of EEG data to show that the same motor inhibition brain network that explains action-stopping in the SST also implements motor inhibition in the complex-stopping task. Furthermore, we found that partial feature overlap between go-stimulus and stopping-template lead to motor slowing, which also corresponded with greater stopping-network activity. This shows that the same brain system for action-stopping to explicit stop-signals is recruited to slow or stop behavior when stimuli match a complex stopping goal. The results imply a generalizability of the brain's network for simple action-stopping to more ecologically valid scenarios.

Keywords

Electroencephalography; Independent Component Analysis; Inhibitory control; Stop- signal task; Braking

1. Introduction

The ability to stop ongoing behaviors after they have been initiated is a cognitive mechanism that is part of everyday life. Much research has used the stop-signal task (SST; Logan et al., 1984; Verbruggen and Logan, 2009) to investigate the factors that affect stopping, and how stopping is implemented in the brain. Stopping in the standard SST recruits an interconnected network of fronto-subcortical brain regions (the 'stopping-

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network') including the pre-supplementary motor area (pre-SMA), the right inferior frontal cortex (rIFC), and the basal-ganglia, with downstream effects on M1 (for reviews, see: Aron et al., 2014; Bari and Robbins, 2013; Chambers et al., 2009; Ridderinkhof et al., 2011; Stinear et al., 2009; Wiecki and Frank, 2013). Activity within this stopping network has been found across several brain imaging modalities. In the human scalp electroencephalogram (EEG), time-frequency analyses show a signature of successful action-stopping at fronto-central scalp sites, specifically within the theta- (5–8Hz) and delta-frequency bands (1–4Hz) (Lavalley et al., 2014; Nigbur et al., 2011; Schmiedt-Fehr and Basar-Eroglu, 2011; Wessel and Aron, 2013; Yamanaka and Yamamoto, 2010).

Yet it is important to ask whether this 'stopping network' for the standard SST generalizes to stopping in more realistic scenarios. Arguably, instances in which behaviors need to be cancelled following explicit stop-signals (as in the standard SST) are relatively rare in the real world. Instead, stopping must be exerted in more complex situations such as the one given in the abstract, in which someone has to stop their step into the street when a car is bearing down. The stopping goal in that situation presumably consists of a complex template of features, which include the size of an object, its trajectory, velocity, and its distance. This stopping-template is presumably represented in working memory, and the stopping system is turned on if all or many features of a given situation match it.

Here, we developed a new behavioral paradigm that models action-stopping to more complex, realistic, stopping goals. In this task, participants had to quickly respond to arrow stimuli, just like in the standard SST. However, unlike the standard SST, we now used arrow-stimuli that differed perceptually along five different dimensions: color, position, number of arrows, arrow style, and print (outline or bold). Before every sequence of stimuli, a unique combination of these five features was presented to the participants as a 'stopping-template', which they had to maintain in memory. Participants then had to respond as quickly as possible to a sequence of arrow-stimuli, *unless* all five dimensions of the current stimulus matched the dimensions of the stopping-template. In that case, the action had to stopped.

We hereafter refer to this new task as the 'complex-stopping task' (CST). Note that while the task is more akin to a Go/NoGo task (where the signal to NoGo occurs at the same time as the Go stimulus) than a classic stop-signal test (where the signal to stop occurs later than the Go stimulus), our task is set up to also elicit a clear-cut stopping situation similar to the standard SST. This was done by creating a highly prepotent go-response on all trials, through having relatively few stop/NoGo-trials, and by requiring relatively fast reaction times on go-trials. The prepotency of the go-response was measured by the number of failed stop/nogo-trials that is clearly attributable to failed motor inhibition (see below). Note also that this task is clearly more ecologically valid than the SST. This is because participants now have a more complex, multidimensional stopping goal in mind. As they are about to respond, they must match the features of the stimulus (a proxy for context) to their stopping goal. A partial match does not constitute a stopping scenario. This is similar to the situation in which a car is bearing down on a pedestrian with the correct trajectory to be potentially stopping-relevant, but is not moving fast enough to necessitate a stop. Of course, the CST is

again a laboratory-based model of control that involves sequential trials with relatively simple stimuli, but it is clearly a closer model of realistic situations than the standard SST.

In a behavioral pilot (Experiment 1), we first established that the Go response did have prepotency (similar to the standard SST): participants often failed to successfully stop, despite recognizing that stopping was needed. Interestingly, we further observed that partial matches between the go-stimulus and the stopping-template lead to slowed responding: when some (but not all) of the features of the go-stimulus matched the stopping template, Go RT was increased. While the slowing could relate to many potential factors (Jahfari et al., 2010), we hypothesized that it could reflect partial recruitment of the stopping system, something we have referred to elsewhere as ‘braking’ (Swann et al., 2012b; Wessel et al., 2013).

In the main study (Experiment 2), we used EEG to test whether the observed stopping and ‘braking’ in the CST is subserved by the same motor inhibition network that explains stopping to explicit stop-signals in the standard SST. We recorded scalp EEG during the CST (the main task of interest) and also for the SST (which was used as a functional localizer for the stopping-system). We used independent component analysis (ICA, Jutten and Herault, 1991) to decompose each participant’s observed scalp EEG signal mixture into its underlying temporally independent source signals (independent components, IC). As done previously (Wessel and Aron, 2013), we identified ICs in each subject that represented a typical EEG signature of successful stopping from the SST. We then tested whether this independent network showed increased activity during outright stopping and / or braking in the CST. We predicted that activity within the stopping-ICs identified in the SST should be increased following action-stopping in the CST (stopping hypothesis). Furthermore, if the RT slowing on partial feature match trials is explained by partial recruitment of the brain’s motor inhibition network (i.e., ‘braking’), then the activity within the stopping-ICs should increase when partial matches induce increased RT slowing (braking hypothesis).

2. Materials and methods

2.1. Participants

2.1.2. Experiment 1—17 right-handed participants (mean age: 21y, sem: .37, range: 18–24; 12 female) performed the task in exchange for course credit. They provided written informed consent according to a local ethics protocol. Data from two participants were excluded, one due to high error rates (pressed wrong buttons on 46% of trials), and one due to high miss rates (did not respond to go-stimuli on 16% of trials), leaving a sample of 15 participants.

2.1.2. Experiment 2—11 right-handed participants (mean age: 20.9y, sem: .87, range: 18–28; 9 female) provided written informed consent according to a local ethics protocol and performed the task in exchange for \$15/hr. These participants were different participants from Experiment 1.

2.2. Materials and procedure

2.2.1. Experiment 1—Stimuli were displayed on a 17inch iMac personal computer (Apple, Inc., Cupertino, CA) running MATLAB 2009b (TheMathWorks, Natick, MA) and Psychtoolbox 3 (Brainard, 1997). Responses were registered through a standard Apple USB keyboard. Participants performed the complex-stopping task first, then performed a working memory task, and then a standard stop-signal task (results from these latter two tasks are not discussed).

2.2.2. Experiment 2—After the EEG caps were attached and prepared, participants were seated in an electromagnetically-shielded and sound-attenuated room. Stimuli were displayed on an electromagnetically shielded CRT monitor (NEC MultiSync FB2141SB; NEC Corporation, Japan) connected to an IBM-compatible personal computer running MATLAB 2009b and Psychtoolbox 3. Viewing distance was 70cm. Responses were registered through a custom USB keypad. Participants performed the complex-stopping task first, then a working memory task (results not discussed), and then the standard stop-signal task.

2.3. Complex-stopping task (CST), Experiments 1 and 2

Each trial consisted of a template-encoding phase followed by a stop/go phase (Figure 1). The template-encoding phase began with a fixation screen showing the word “MEMORIZE!” and three pairs of horizontal lines, which were arranged on three vertical positions on the screen. After 1000ms of fixation, the stopping-template appeared, which consisted of squares that varied along five perceptual dimensions: color (red, blue, green), vertical position (as indicated by the horizontal lines), number (1, 2, or 3 squares), print (filled or outline), and style (either simple squares or squares with additional horizontal lines on both sides; this feature indicates whether the arrows in the stop/go phase would consist of arrowheads only, or of arrowheads with lines attached to them). This stimulus was on the screen for 3000ms, and the participants were instructed to memorize all five features. Then, the word ‘Go!’ appeared for 500ms, followed by a blank screen of 500ms, which started the stop/go phase.

The stop/go phase consisted of a series of arrow stimuli that were displayed on the screen, which varied along the same five dimensions as the stopping-template. These stimuli could match the stopping-template anywhere between 0 and all 5 dimensions. These stimulus types will henceforth be denoted M0 (0 matches with the stopping-template), M1 (1 match), M2 (2 matches), M3 (3 matches), M4 (4 matches), and MSTOP (all five dimensions match; i.e., those trials are stop-trials). Participants were instructed to respond as fast as possible to the direction of the arrow using a finger of their right hand (right arrow key for right, left arrow key for left), *unless* the stimulus on the screen matched the stopping-template in all five dimensions (MSTOP-stimuli). In that case, the participants had to stop their response. In order to increase the stopping demand of the task, an adaptive deadline algorithm ensured that RT remained fast throughout the experiment¹. After the response was made, the stimulus on the screen disappeared. The ITI was set so that the stimulus-onset asynchrony between two subsequent go-stimuli was exactly 2000ms. Importantly, in order to discern whether a failure to withhold responding following an MSTOP-stimulus was due to failed

stopping (i.e., participants realized they would have had to stop but could not due to the prepotency of the go-response) or due to forgetting of the stopping-template (i.e., participants did not realize that they had to stop), participants were instructed to press a third button with their left hand in case they realized that they had to stop their response but did not do so. This button could be pressed any time during the ITI. That way, MSTOP-stimuli could be divided into successful-stop MSTOP-stimuli (succMSTOP), failed-stop MSTOP-stimuli due to stopping failure (failMSTOP), and failed-stop MSTOP-stimuli due to working memory failure (forgetMSTOP).

On each trial, the stop/go phase consisted of a sequence of 10 to 20 successive stimuli (number drawn from a uniform distribution). The probability of occurrence of an MSTOP-stimulus was set to 15%. Furthermore, an MSTOP-stimulus could appear within the stop/go phase not earlier than on the fifth go-stimulus. This was done in order to further increase the prepotency of the go-response before any MSTOP-stimulus appeared. The features of each individual stimulus (as well as the stopping-template) were drawn from uniform distributions. Participants performed 40 total trials (i.e., sequences of template-encoding followed by stop/go-phase). In addition, trials in which the first MSTOP-stimulus was a forgetMSTOP were immediately cancelled (since the stopping-template was probably inaccurately remembered), and were appended to the end of experiment to be performed again. Trials were divided into 6 blocks.

2.4. Standard stop-signal task (SST), Experiment 2

Each trial began with a fixation cross in the center of the screen. After 500ms, a white arrow pointing either right or left appeared (go-signal). On 25% of all trials (stop-trials), the go-signal was followed by a stop-signal (red exclamation point displayed above the arrow) after a short delay (stop-signal delay, SSD). The SSD was set to 200ms initially, and was then adapted by 50ms following each instance of a stop-trial: following failed stop-trials (response not withheld) it decreased, following successful stop-trials it increased. Response deadline on go-trials was 1500ms. Following the response (or when the deadline was reached), the fixation cross was displayed. The ITI was set so that each trial lasted 2500ms in total. Participants were told that both going quickly and stopping accurately were equally important. They performed 240 trials of the SST spread out over 6 blocks.

2.5. EEG recording (Experiment 2)

EEG data was recorded using a 128-channel BioSemi system (BioSemi Instrumentation, The Netherlands, 1024 Hz sampling rate), with additional electrodes placed on the bilateral mastoids and canthi, as well as below and above each eye. The data were online referenced to the BioSemi CMS-DRL reference. All offsets from the reference were kept below 25 microvolts.

¹This was done as follows: The initial deadline was set to 1000ms. After the first 2 trials (a trial denotes a full sequence of template-encoding followed by the stop/go-phase), it was adapted online, based on the performance on all go-stimuli within the last 2 trials: if the miss rate (no response made before the deadline on go-stimuli) and the error rate (wrong button pressed) were both below 10%, the deadline would decrease by 50ms. If either error rate or miss rate exceeded 10%, the deadline would increase by 50ms. A minimum for the deadline was set at 600ms.

2.6. EEG analysis

2.6.1. Overview—The analysis proceeded in five steps. The EEG data for both tasks together were (1) preprocessed, (2) screened for artifacts, and (3) subjected to an independent component analysis to decompose the scalp EEG mixture into its underlying independent components. [Such independent components correspond to individual cortical domains (usually singular or paired anatomical areas / cortical patches), whose EEG time courses are independent from ongoing processing / independent components in other brain areas (Onton et al., 2006)]. Then, (4) we identified an independent component for each subject that showed increased activity on successful (compared to failed) stop-trials in the standard SST. This was our ‘localizer’ for the stopping network (Wessel and Aron, 2013). Having identified this stopping component from the SST in each person, we then (5) analyzed its activity in the CST data, and tested our predictions that activity would explain complex stopping and braking in this latter task.

The logic underlying this analysis method is as follows: If stopping / braking in the CST is explained by a different network than successful stopping in the SST, ICA will disentangle these components, and the SST stopping-ICs will show no activity in the CST. Conversely, if the same network explains action-stopping in both tasks, the same ICs that explain successful stopping in the SST will show activity when actions are stopped or braked in the CST (for other studies using this method, see e.g., Gentsch et al., 2009; Roger et al., 2010; Torrecillos et al., 2014; Wessel and Aron, 2013; Wessel et al., 2012).

2.6.2. EEG preprocessing, artifact rejection, and ICA (Steps 1–3)—Data were preprocessed using custom routines in MATLAB 2012a (TheMathWorks, Natick, MA). ICA and dipole fitting (DIPFIT 2.2) were performed using functions from the EEGLAB toolbox (Version 9, Delorme and Makeig, 2004). On import into MATLAB, the data were re-referenced to CP1 (according to the 10–20 system). The continuous time-series (which included both the CST and SST data) was resampled to 512Hz, and filtered using symmetric two-way least-squares finite-impulse-response (FIR) filters (.5 Hz high-pass, 50 Hz low-pass). The time-series were then visually inspected for bad channels and segments with non-stereotyped artifact activity (e.g. from gross movement or spurious muscle activity). Such data were removed. Then, the remaining data were re-referenced to common average, reduced to 50 principle components, and subjected to a temporal infomax ICA algorithm (Bell and Sejnowski, 1995; with extension towards sub-gaussian sources, Lee et al., 1999). The data was reduced to principle components prior to ICA in order to avoid over-fitting of the ICA model, which can happen due to the high dimensionality of the sensor space (in this case, 128+8 channels), and can cause considerable estimation errors. PCA prior to ICA can circumvent this problem (Hyvarinen et al., 1999). 50 components were chosen based on the experience of the authors. The resulting ICA component matrix was screened for ICs representing stereotypic artifacts (blinks, saccades, and electrode artifacts) using outlier statistics (procedure as described in Wessel et al., 2012). Such components were removed. The remaining components were fitted with individual inverse dipole-solutions using the DIPFIT 2.2 algorithm. Components with non-dipolar equivalent dipole solutions usually represent non-brain signals (as defined by a residual variance of their equivalent dipole solution of greater than 15%, Delorme et al., 2012), and were also removed. The automatic

classifications based on these criteria were visually screened for inaccurate classifications and manually rectified if necessary. The remaining non-artifact components were subjected to further analyses.

2.6.3. EEG analysis – deriving the stopping IC from the standard stop signal task (Step 4)

2.6.3.1. Time-frequency analysis: In order to perform stopping-IC selection based on the SST EEG data, the SST time-series of each independent component was first filtered using two-way least-squares FIR filters in the theta (4–8Hz) and delta (1–4Hz) frequency ranges. These were the frequencies of interest for the contrast of successful vs. failed stopping in the SST, based on previous literature (reviewed by Huster et al., 2013). The filtered delta-/theta-band data were transformed into frequency space using a Hilbert-transform, whose absolute value represents an analytic signal for the frequency band in question. Analytic amplitudes were then averaged separately for successful- and failed stop-trials in the SST, time-locked to stop-signal onset.

2.6.3.2. Stopping-IC selection: The selection of independent components that explained successful stopping in the SST was almost identical to our previous study, which used 64 rather than 128 electrodes (Wessel and Aron, 2013). We defined selection criteria based on previous literature using EEG data of the SST (as reviewed by Huster et al., 2013). In order to be able to qualify as a stopping component, an IC needed to show both:

- A.** A fronto-central radial topography (maximum IC weight at fronto-central electrodes (see Figure 2; topographical criterion), as well as
- B.** Increased activity for successful compared to failed stop trials in the theta and/or delta frequency bands for least a successive stretch of 100ms within the first 500ms following the stop-signal (functional criterion).

At least one component was found in every participant. Out of the eleven participants, eight had one component that was selected. Two had two components that met both criteria, and one participant had three such components. In the latter cases (which can be the result of residual ICA over-fitting), the IC timecourses were averaged for all subsequent analyses. A group average of SST-related activity (average weight matrices as well as successful vs. failed stop-difference delta-/theta-band timecourses) can be found in Figure 2.

2.6.4. Step 5: EEG analysis testing hypotheses in the complex stop task

2.6.4.1. Time-frequency analysis: For full-spectrum event-related perturbation (ERSP) analyses of the stopping-ICs in the CST, the CST-timecourses of those ICs were filtered into 50 linearly spaced individual frequencies ranging from 1 to 50 Hz (with a frequency-window of $\pm .5$ Hz around the center frequency), then Hilbert-transformed and converted into analytic amplitudes. ERSP amplitudes were then averaged separately for each feature match condition (i.e., M0, M1, M2, M3, M4, and MSTOP-stimuli), time-locked to stimulus onset (time-range: –300ms to 1000ms relative to event). All time-frequency data were converted from arbitrary units into percent-change-from-baseline, by subtracting the average

activity from 300ms preceding the event in question to the event itself, then dividing by that baseline, and then multiplying by 100.

2.6.4.2. Stopping hypothesis: The first analysis tested whether outright stopping of action in the CST was explained by activity within the stopping-ICs identified based on the standard SST. The ERSPs for succMSTOP-stimuli (all features matched the stopping template) were compared to the ERSPs for M0-stimuli (no features matched). Full-spectrum ERSPs were tested for significant differences using t-tests for each time-point and each frequency individually, with a two-sided p-value of $p < .05$ (FDR-corrected, Benjamini and Hochberg, 1995).

2.6.4.3. Braking hypothesis: The second analysis tested whether braking of RT on partial match trials in the CST was explained by activity within the stopping-ICs identified in the standard SST. To test this, a single-trial GLM was constructed for each participant, which investigated which one of three regressors predicted the activity at each point in the EEG time-frequency response following a given stimulus. The regressors were:

1. The number of matches between a stimulus and the stopping template (MATCHES).
2. The response slowing following a stimulus S, defined as $100 * (\text{RT}_S - \text{mean}(\text{RT}_{M0})) / \text{mean}(\text{RT}_{M0})$, i.e., expressed as percentage of RT slowing on a given trial compared to the average RT following M0-stimuli (SLOWING).
3. The SLOWING x MATCHES INTERACTION, i.e., the amount of relative RT slowing caused by the number of feature matches between a given stimulus and the stopping template.

Similar general linear model analyses of EEG data have been used in the time-domain (Fischer and Ullsperger, 2013) and time-frequency domain (Cohen and Cavanagh, 2011). Both the MATCHES and SLOWING regressors were centered (by subtracting the mean), and the INTERACTION was computed based on the centered regressors. This was done in order to reduce potential intercorrelations between the three regressors, which could lead to multicollinearity. This measure was effective, as none of the GLMs for any of the participants showed any multicollinearity (all variance inflation factors for each regressor in each participants' GLM were smaller than 1.3). The regressions were computed for each sampling point within the time-frequency space (512 sampling points in the time domain [corresponding to 1000ms of data], 50 sampling points in the frequency domain [corresponding to each center-frequency from 1–50 Hz]). The standardized beta-weights from each participants' individual regression analyses were then tested at the group-level. This was done using z-tests against 0 for each time-frequency sampling point, at a two-sided significance level of $p < .05$ (FDR-corrected). Note that this analysis is analogous to a voxel-wise GLM analysis in fMRI, only that the regressands in this analysis were not individual voxels, but power values for each individual frequency at each individual EEG sample point within the first 1000ms following stimulus onset.

3. Results

3.1. Behavior: CST

3.1.1. Experiment 1—Average RT for correct go-responses was 436ms (sem: 8.1). Error- and miss rates were low (3.1% and 4.3%, respectively). Following MSTOP-stimuli, responses were withheld successfully 70.0% of the time (sem: 5.4). Of the remaining MSTOP-stimuli on which responses could not be withheld, 66.5% (sem: 7.8) were due to failed stopping (participants pressed the third button to indicate they recognized that they were supposed to stop but failed to do so). An outlier contaminated this measurement, as one participant exhibited a failed-stop signaling-rate of 0%. Without this outlier, 71.3% of all failed MSTOP-trials were due to failed stopping (sem: 6.6%). Importantly, these results show that there is a prepotency to respond to the arrow-stimuli within this paradigm, which oftentimes leads to failures of stopping.

RT to the non-MSTOP-stimuli increased parametrically with the number of feature matches with the stopping-template: Reaction times were 426ms for M0-stimuli, and 427ms, 431ms, 443ms, and 461ms, for increasing number of matches, respectively (Figure 3A). A repeated-measures ANOVA showed a significant main effect of the number of matches, with a large effect size ($F(4,44) = 43.2$; $p < 10^{-13}$, $\eta^2 = .8$).

3.1.2. Experiment 2—Average RT for correct go-responses was 428ms (sem: 4.9). Error- and miss rates were low (1.8% and 3.9%, respectively). Following MSTOP-stimuli, responses were withheld successfully 74.9% of the time (sem: 3.6). Of the MSTOP-stimuli on which responses could not be withheld, 76.3% (sem: 4.9) were due to failed stopping (i.e., participants pressed the third button to indicate they recognized stopping was needed).

As in Experiment 1, RT to the non-MSTOP-stimuli increased parametrically with the number of feature matches with the stopping-template: Reaction times were 417ms for M0-stimuli, and 419ms, 424ms, 441ms, and 460ms, for increasing number of matches, respectively (Figure 3B). A repeated-measures ANOVA showed a significant main effect of the number of matches, with large effect size ($F(4,40) = 37.8$; $p < 10^{-12}$, $\eta^2 = .79$).

3.2. Behavior: SST (Experiment 2)

Performance was exemplary. RT on go-trials was 488ms (sem: 14.2), RT on failed stop-trials was 413ms (sem: 9.8), and RT on failed stop-trials was faster than RT on go-trials for all participants, which shows the data are in accordance with the race model of the SST (Verbruggen and Logan, 2009). The probability of stopping was 51.8% (sem: .8), demonstrating the effectiveness of the SSD staircase-algorithm at achieving roughly equal proportions of successful and failed stop trials. Stop-signal reaction time was estimated using the mean method, and was 246ms on average (sem: 6.3), with an average SSD of 241ms (sem: 13.8). Error and miss rates were low (1% and .1%, respectively).

3.3. EEG (Experiment 2)

3.3.1. CST stopping hypothesis—Activity in the stopping-ICs on succMSTOP-stimuli in the CST was dominated by theta- and delta- band activity that was increased after

stimulus onset (Figure 4). Importantly, when compared to M0-stimuli, both delta- (4Hz) and theta-band (7Hz) activity was significantly increased within the first 1000ms following stimulus onset (Figure 4A). This is consistent with our stopping hypothesis, i.e., that the same brain system underlying outright stopping in the SST is also active when actions are stopped based on the more complex stopping goals in the CST.

3.3.2. CST braking hypothesis—Both delta- and theta-activity within stopping-ICs correlated positively with both regressors (MATCHES and SLOWING), as well as with their interaction. For the MATCHES regressor (Figure 4C, left panels), there were significant positive correlations between the number of feature matches and activity within the stopping-ICs at 2–4Hz (delta-band), as well as 5–6Hz (theta-band). This shows that stronger theta- and delta-band activity within the stopping-ICs reflected increasing numbers of feature matches between a given stimulus and the stopping-template (independent of the amount of RT slowing this induced). For the SLOWING regressor (Figure 4C, middle panels), there were significant positive correlations between RT slowing and activity within the stopping-ICs at 1 Hz (delta-band), as well as 5–8Hz (theta-band). This shows that stronger theta- and delta-band activity within the stopping-ICs also reflected increased RT slowing on a given trial compared to M0-stimuli (independent of how many features of the stimulus matched the stopping-template). There was also a significant negative correlation with some late beta-band (18–19Hz) activity, which occurred after mean RT. For the interaction between MATCHES and SLOWING (Figure 4C, right panels), there was a significant positive correlation with activity of the stopping-ICs at 4Hz. This shows that increased activity within the stopping-ICs reflected an increased amount of RT slowing due to the number of feature matches between a given stimulus and the stopping-template.

3.3.3. Supplementary Analysis—The main hypotheses tests of our study concerned fronto-central independent components (ICs) that reflect the implementation of motor inhibition. In a supplementary analysis, we show that it is possible to dissociate independent component activity between the non-overlapping components of the task, i.e., components that are related to the success of stopping in one (but not both) of the tasks, and do not reflect the motor inhibition process. To this end, we tested whether posterior visual alpha activity, which reflects early perceptual processing that can be influenced by attention (Yamagishi et al., 2003), is differentially related to stopping success depending on the task. Our prediction was that, in line with a prior study (Greenhouse and Wessel, 2013), early posterior visual activity will relate to the success of stopping in the standard SST (since the detection of the salient visual stop-signal is a key part of the stopping process). However, since there is no salient stop-signal in the CST, the components that reflect posterior visual alpha activity should not be related to stopping-success in the CST. Our analysis shows that this is indeed the case (see Supplementary Materials). Hence, even though the motor inhibition process is explained by the same brain process (i.e., independent component) in both SST and CST, stopping-related components that are not directly related to motor inhibition differ in accordance with the differential demands of both tasks.

4. Discussion

We designed a novel paradigm to investigate whether motor inhibition is subserved by the same brain network, regardless of whether stopping is triggered by an explicit stop signal (as in the standard SST) or by a more complex process. Here, that process involved matching the properties of a go-stimulus with the features of a multidimensional stopping-template. In a behavioral pilot (Experiment 1), we found that this new task was successful in creating a race-like situation between a prepotent go-process and a complex stop-process. This is evident by the fact that participants failed to stop a considerable proportion of their responses on stop-trials (i.e. following MSTOP-stimuli) even though they realized that the stimulus matched the stopping template and that they should have stopped (which they indicated by pressing a key subsequently). The behavioral pilot furthermore revealed a parametric increase in RT as more features matched the stopping-template (i.e., for M1–4-stimuli compared to M0-stimuli). We hypothesized that this reflects a partial recruitment of the stopping system, something we have referred to as ‘braking’ (Swann et al., 2012b; Wessel et al., 2013). In Experiment 2, we then measured EEG for both the CST and a standard SST task. We used the standard SST as a functional localizer for the brain network for rapid stopping of action following explicit stop-signals. We then investigated the activity within the independent components that reflected this successful stopping process during stopping and braking in the CST. Theta- and delta-band activity in the stopping-ICs was increased when behavior was stopped outright. Furthermore, a single-trial regression analysis revealed that low-frequency activity within the stopping network accounted for variance in the number of feature matches, in the amount of RT slowing, and importantly, in the interaction of these. We interpret the interaction in particular as evidence for the braking hypothesis.

4.1. Extending the role of the brain network for stopping & braking to a more realistic setting

The most important implication of these findings is that they extend the brain network for motor inhibition in outright stopping situations beyond the standard SST. Stopping in the CST is more akin to scenarios in which actions need to be stopped in the real world. Here, this involved a comparison of a go-stimulus (which not only told people what response to make, but also represented the features of the ‘context’ in which that response was made) to a multidimensional stopping-template that was maintained in working memory while the go-command was being executed. This captures a key aspect of real-world control, which is that people have a complex ‘stopping-template’ in mind (such as a car going down the street with a certain velocity, path, etc.) and monitor their environment for potential matches.

We also showed that even when a current stimulus is only similar (but not identical) to the stopping template, the stopping network was activated by the number of feature matches, the amount of RT slowing, and also the interaction. We interpret the interaction in particular as showing that the stopping network can delay the execution of a motor command (instead of cancelling it outright); i.e., it can act as a brake. This is consistent with several brain imaging studies showing that the stopping-network can be recruited proactively in order to increase the chance of successful stopping (Chikazoe et al., 2009; Jahfari et al., 2010; Swann et al.,

2012b; Wessel et al., 2013; Zandbelt et al., 2012). While there is some controversy about whether the ‘stopping-network’ truly reflects inhibitory control rather than mere attention (Chatham et al., 2012; Erika-Florence et al., 2014), our results show that the stopping-network (at least as identified by scalp-EEG) does both. It is activated by the number of feature matches (indicative of an attentional detection process), and also the amount of motor slowing (which, since this is the same network that explains outright stopping in the SST, is indicative of motor inhibition). And, consistent with the view that inhibitory control needs to be triggered by an attentional process (Aron et al., 2014), the activity within this network related to the interaction of number of matches and RT slowing.

Our showing that the stopping-network also relates to slowing when a stimulus partially matches the stopping template is also consistent with several studies showing it is recruited in situations of response- or decision conflict to “hold your horses” (Brittain et al., 2012; Cavanagh et al., 2011; Frank et al., 2007; Zavala et al., 2014). However, it is unlikely that response-conflict alone can account for our results. Although we did observe theta-band activity from a fronto-medial source (a well-known signature of response-conflict (for a review, see: Cohen, 2014)) in our feature-overlap induced slowing, this activity was based on independent components that were identified based on successful or failed stopping in the standard SST. Hence, the independent components selected here represent a process that differentiates successful from failed stop trials. We believe this process to be related to motor inhibition, and it is hard to explain how different levels of response-conflict would result in activity differences between successful and failed stop-trials. Similarly, our results show that the activity within the independent components we extracted cannot be solely explained by attentional processes. While increased attentional processing could (in principle) explain the difference between successful and failed stop trials (in the sense that better attentional detection of the stop-signal could lead to more successful stopping), the results from the CST show that activity within this brain network is also related to the amount of reaction time slowing (a measurement of motor inhibition), even when the variance attributable to the number of featural matches (a potential measurement of attentional capture) is accounted for. Hence, we propose that a brain network for motor inhibition that explains the differences between successful and failed stopping in the standard stop-signal task also explains successful stopping and braking in the more realistic stopping situations the CST simulates.

The fact that the standard SST network is also active during these more complex inhibitory motor control situations (both braking and stopping behavior based on a matching procedure with a complex stopping-template) helps validate several decades of psychological and neuroscience research into the standard SST (Aron et al., 2007; Boucher et al., 2007; Chambers et al., 2009; Ridderinkhof et al., 2011; Schall and Godlove, 2012; Verbruggen and Logan, 2009). Thus, while the standard SST is a simple task, it apparently taps into a core brain network that has wider ecological and even clinical relevance. For example, an fMRI study in over 1000 adolescents showed that a key node of the stopping network in the standard SST shows variation related to ADHD and substance use (Whelan et al., 2012). Many other studies have similarly implicated regions of the standard SST stopping-network in ADHD, OCD, urges to smoke, and blink suppression (Berkman et al., 2011; Berman et al., 2012; Durston et al., 2006; Menzies et al., 2007; Tabibnia et al., 2011). All these

instances pertain to wider aspects of inhibitory control, which could be subserved by the common neural network whose activity we measured here.

4.2. Neural correlates

This study used EEG, which has the advantage of being sensitive to the difference between successful and failed stopping (e.g. Greenhouse and Wessel, 2013; Kok et al., 2004; Pliszka et al., 2000; Schmajuk et al., 2006; Wessel and Aron, 2013). By contrast, fMRI does not generally show that difference (Aron et al., 2014), even in well-powered studies (Congdon et al., 2010). Thus, we were able to identify the stopping network based on the successful vs. failed stopping contrast, which is more sensitive to the inhibitory process, rather than mere attentional detection of a stop-signal (i.e., a stop-signal is present in both successful and failed stop-trials). Another advantage of using this ICA based EEG method relative to fMRI is that ICA is designed to disentangle brain processes based on ‘true independence’ of their temporal profiles. Whereas fMRI could show that there is anatomical coactivation between a localizer and a test condition, this does not prove that the same neural process is at work (as anatomically overlapping brain regions can be involved in functionally separate brain networks).

A limitation of the current study is that it cannot point to which exact anatomical node (or nodes) of the brain’s stopping network we are measuring from, as the spatial resolution of EEG is relatively poor. Converging evidence from studies with more spatially precise methods such as fMRI (Curtis et al., 2005; Li et al., 2006; Rubia et al., 2001; Swann et al., 2012a) and intracranial recordings (Swann et al., 2009; Swann et al., 2012b) point towards critical roles of pre-SMA and rIFC in simple stopping, in addition to the basal ganglia (Aron and Poldrack, 2006; Ray et al., 2012; Zandbelt and Vink, 2010). Regarding scalp EEG, the majority of studies of successful stopping in the SST reported activity with a fronto-central voltage distribution, similar to what we report here (Etchell et al., 2012; Greenhouse and Wessel, 2013; Johnstone et al., 2007; Kok et al., 2004; Kramer et al., 2011; Lavalley et al., 2014; Nigbur et al., 2011; Schmiedt-Fehr and Basar-Eroglu, 2011; Wessel and Aron, 2013; Yamanaka and Yamamoto, 2010). However, due to the inverse problem, it is still unclear which exact anatomical brain regions generate this fronto-central activity. Studies that used simultaneous EEG-fMRI, which combines the temporal resolution of EEG with the spatial precision of fMRI, indicate that brain areas in the medial wall / pre-SMA as well as the rIFC could contribute to such activity (Enriquez-Geppert et al., 2010; Huster et al., 2010). Hence, we cannot speak with confidence in the current study about which area of the stopping network we are recording from, and we refrain from making inferences about the differential contributions of pre-SMA and rIFC in the current study. However, we can say with confidence that the same brain network that explains outright stopping in the SST also explains stopping in the more complex CST, as well as partial motor inhibition (braking) when a given stimulus matches parts of the stopping goal in working memory.

5. Conclusions

We developed a novel paradigm that allowed us to compare stopping following simple, salient stop signals to stopping following a more complex matching process that involves a multidimensional stopping-goal. Using ICA of EEG data, we showed that the same brain

network for successful stopping in the SST also explains stopping in the more complex CST. Furthermore, in the CST, we observed behavioral slowing on stimuli whose features only partially matched the stopping-template, which was also related to activity in this network. This means that this network can implement partial inhibitory control; i.e., it can act as a brake. The fact that activity in this network increased with greater behavioral slowing in the CST even when covarying the number of matching features has an important implication for the interpretation of the EEG signature of the standard SST itself: It strongly suggests that the activity in this network does not merely reflect attentional processes, but is instead related to a motor inhibitory process.

More generally, the current results demonstrate the generality of the brain's network for motor inhibition, by extending it beyond motor situations that merely involve a simple detection of a salient signal. This ratifies several decades of research using the standard SST as a model to investigate the neural basis of stopping in health and the disease. In brief, the SST taps into a brain network that also underlies complex stopping behavior that more closely mimics everyday life.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This work was supported by grants from the Kavli Institute for Brain & Mind (to J.R.W.), the National Institutes of Health (R01DA026452) and the James S McDonnell Foundation (to A.R.A.).

References

- Aron AR, Durston S, Eagle DM, Logan GD, Stinear CM, Stuphorn V. Converging evidence for a fronto-basal-ganglia network for inhibitory control of action and cognition. *J Neurosci.* 2007; 27:11860–11864. [PubMed: 17978025]
- Aron AR, Poldrack RA. Cortical and subcortical contributions to Stop signal response inhibition: role of the subthalamic nucleus. *J Neurosci.* 2006; 26:2424–2433. [PubMed: 16510720]
- Aron AR, Robbins TW, Poldrack RA. Inhibition and the right inferior frontal cortex: one decade on. *Trends Cogn Sci.* 2014; 18:177–185. [PubMed: 24440116]
- Bari A, Robbins TW. Inhibition and impulsivity: behavioral and neural basis of response control. *Prog Neurobiol.* 2013; 108:44–79. [PubMed: 23856628]
- Bell AJ, Sejnowski TJ. An information-maximization approach to blind separation and blind deconvolution. *Neural Comput.* 1995; 7:1129–1159. [PubMed: 7584893]
- Benjamini Y, Hochberg Y. Controlling the False Discovery Rate - a Practical and Powerful Approach to Multiple Testing. *Journal of the Royal Statistical Society Series B-Methodological.* 1995; 57:289–300.
- Berkman ET, Falk EB, Lieberman MD. In the trenches of real-world self-control: neural correlates of breaking the link between craving and smoking. *Psychological Science.* 2011; 22:498–506. [PubMed: 21378368]
- Berman BD, Horowitz SG, Morel B, Hallett M. Neural correlates of blink suppression and the buildup of a natural bodily urge. *Neuroimage.* 2012; 59:1441–1450. [PubMed: 21906689]
- Boucher L, Palmeri TJ, Logan GD, Schall JD. Inhibitory control in mind and brain: an interactive race model of countermanding saccades. *Psychol Rev.* 2007; 114:376–397. [PubMed: 17500631]
- Brainard DH. The Psychophysics Toolbox. *Spat Vis.* 1997; 10:433–436. [PubMed: 9176952]

- Brittain JS, Watkins KE, Joundi RA, Ray NJ, Holland P, Green AL, Aziz TZ, Jenkinson N. A role for the subthalamic nucleus in response inhibition during conflict. *J Neurosci.* 2012; 32:13396–13401. [PubMed: 23015430]
- Cavanagh JF, Wiecki TV, Cohen MX, Figueroa CM, Samanta J, Sherman SJ, Frank MJ. Subthalamic nucleus stimulation reverses mediofrontal influence over decision threshold. *Nat Neurosci.* 2011; 14:1462–1467. [PubMed: 21946325]
- Chambers CD, Garavan H, Bellgrove MA. Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neurosci Biobehav Rev.* 2009; 33:631–646. [PubMed: 18835296]
- Chatham CH, Claus ED, Kim A, Curran T, Banich MT, Munakata Y. Cognitive control reflects context monitoring, not motoric stopping, in response inhibition. *PLoS ONE.* 2012; 7:e31546. [PubMed: 22384038]
- Chikazoe J, Jimura K, Hirose S, Yamashita K, Miyashita Y, Konishi S. Preparation to inhibit a response complements response inhibition during performance of a stop-signal task. *J Neurosci.* 2009; 29:15870–15877. [PubMed: 20016103]
- Cohen MX. A neural microcircuit for cognitive conflict detection and signaling. *Trends Neurosci.* 2014
- Cohen MX, Cavanagh JF. Single-trial regression elucidates the role of prefrontal theta oscillations in response conflict. *Front Psychol.* 2011; 2:30. [PubMed: 21713190]
- Congdon E, Mumford JA, Cohen JR, Galvan A, Aron AR, Xue G, Miller E, Poldrack RA. Engagement of large-scale networks is related to individual differences in inhibitory control. *Neuroimage.* 2010; 53:653–663. [PubMed: 20600962]
- Curtis CE, Cole MW, Rao VY, D'Esposito M. Canceling planned action: an fMRI study of countermanding saccades. *Cereb Cortex.* 2005; 15:1281–1289. [PubMed: 15616130]
- Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods.* 2004; 134:9–21. [PubMed: 15102499]
- Delorme A, Palmer J, Onton J, Oostenveld R, Makeig S. Independent EEG sources are dipolar. *PLoS ONE.* 2012; 7:e30135. [PubMed: 22355308]
- Durston J, Mulder M, Casey BJ, Ziermans T, van Engeland H. Activation in ventral prefrontal cortex is sensitive to genetic vulnerability for attention-deficit hyperactivity disorder. *Biol Psychiatry.* 2006; 60:1062–1070. [PubMed: 16712804]
- Enriquez-Geppert S, Konrad C, Pantev C, Huster RJ. Conflict and inhibition differentially affect the N200/P300 complex in a combined go/nogo and stop-signal task. *Neuroimage.* 2010; 51:877–887. [PubMed: 20188191]
- Erika-Florence M, Leech R, Hampshire A. A functional network perspective on response inhibition and attentional control. *Nat Commun.* 2014; 5:4073. [PubMed: 24905116]
- Etchell AC, Sowman PF, Johnson BW. “Shut up!” An electrophysiological study investigating the neural correlates of vocal inhibition. *Neuropsychologia.* 2012; 50:129–138. [PubMed: 22108442]
- Fischer AG, Ullsperger M. Real and fictive outcomes are processed differently but converge on a common adaptive mechanism. *Neuron.* 2013; 79:1243–1255. [PubMed: 24050408]
- Frank MJ, Samanta J, Moustafa AA, Sherman SJ. Hold your horses: impulsivity, deep brain stimulation, and medication in parkinsonism. *Science.* 2007; 318:1309–1312. [PubMed: 17962524]
- Gentsch A, Ullsperger P, Ullsperger M. Dissociable medial frontal negativities from a common monitoring system for self- and externally caused failure of goal achievement. *Neuroimage.* 2009; 47:2023–2030. [PubMed: 19486945]
- Greenhouse I, Wessel JR. EEG signatures associated with stopping are sensitive to preparation. *Psychophysiology.* 2013
- Huster RJ, Enriquez-Geppert S, Lavalée CF, Falkenstein M, Herrmann CS. Electroencephalography of response inhibition tasks: functional networks and cognitive contributions. *Int J Psychophysiol.* 2013; 87:217–233. [PubMed: 22906815]

- Huster RJ, Westerhausen R, Pantev C, Konrad C. The role of the cingulate cortex as neural generator of the N200 and P300 in a tactile response inhibition task. *Hum Brain Mapp.* 2010; 31:1260–1271. [PubMed: 20063362]
- Hyvarinen, A.; Sarela, J.; Vigário, R. Spikes and bumps: Artefacts generated by independent component analysis with insufficient sample size. *First International Workshop on Independent Component Analysis and Signal Separation*; 1999.
- Jahfari S, Stinear CM, Claffey M, Verbruggen F, Aron AR. Responding with restraint: what are the neurocognitive mechanisms? *J Cogn Neurosci.* 2010; 22:1479–1492. [PubMed: 19583473]
- Johnstone SJ, Dimoska A, Smith JL, Barry RJ, Pleffer CB, Chiswick D, Clarke AR. The development of stop-signal and Go/Nogo response inhibition in children aged 7–12 years: performance and event-related potential indices. *Int J Psychophysiol.* 2007; 63:25–38. [PubMed: 16919346]
- Jutten C, Herault J. Blind Separation of Sources.1. An Adaptive Algorithm Based on Neuromimetic Architecture. *Signal Processing.* 1991; 24:1–10.
- Kok A, Ramautar JR, De Ruiter MB, Band GP, Ridderinkhof KR. ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology.* 2004; 41:9–20. [PubMed: 14692996]
- Kramer UM, Knight RT, Munte TF. Electrophysiological evidence for different inhibitory mechanisms when stopping or changing a planned response. *J Cogn Neurosci.* 2011; 23:2481–2493. [PubMed: 20849230]
- Lavallee CF, Herrmann CS, Weerda R, Huster RJ. Stimulus-Response Mappings Shape Inhibition Processes: A Combined EEG-fMRI Study of Contextual Stopping. *PLoS ONE.* 2014; 9:e96159. [PubMed: 24763435]
- Lee TW, Girolami M, Sejnowski TJ. Independent component analysis using an extended infomax algorithm for mixed subgaussian and supergaussian sources. *Neural Comput.* 1999; 11:417–441. [PubMed: 9950738]
- Li CS, Huang C, Constable RT, Sinha R. Imaging response inhibition in a stop-signal task: neural correlates independent of signal monitoring and post-response processing. *J Neurosci.* 2006; 26:186–192. [PubMed: 16399686]
- Logan GD, Cowan WB, Davis KA. On the Ability to Inhibit Simple and Choice Reaction-Time Responses - a Model and a Method. *Journal of Experimental Psychology-Human Perception and Performance.* 1984; 10:276–291. [PubMed: 6232345]
- Menzies L, Achard S, Chamberlain SR, Fineberg N, Chen CH, del Campo N, Sahakian BJ, Robbins TW, Bullmore E. Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain.* 2007; 130:3223–3236. [PubMed: 17855376]
- Nigbur R, Ivanova G, Sturmer B. Theta power as a marker for cognitive interference. *Clin Neurophysiol.* 2011; 122:2185–2194. [PubMed: 21550845]
- Onton J, Westerfield M, Townsend J, Makeig S. Imaging human EEG dynamics using independent component analysis. *Neurosci Biobehav Rev.* 2006; 30:808–822. [PubMed: 16904745]
- Pliszka SR, Liotti M, Woldorff MG. Inhibitory control in children with attention-deficit/hyperactivity disorder: event-related potentials identify the processing component and timing of an impaired right-frontal response-inhibition mechanism. *Biol Psychiatry.* 2000; 48:238–246. [PubMed: 10924667]
- Ray NJ, Brittain JS, Holland P, Joundi RA, Stein JF, Aziz TZ, Jenkinson N. The role of the subthalamic nucleus in response inhibition: evidence from local field potential recordings in the human subthalamic nucleus. *Neuroimage.* 2012; 60:271–278. [PubMed: 22209815]
- Ridderinkhof KR, Forstmann BU, Wylie SA, Burle B, van den Wildenberg WPM. Neurocognitive mechanisms of action control: resisting the call of the Sirens. *Wiley Interdisciplinary Reviews-Cognitive Science.* 2011; 2:174–192.
- Roger C, Benar CG, Vidal F, Hasbroucq T, Burle B. Rostral Cingulate Zone and correct response monitoring: ICA and source localization evidences for the unicity of correct- and error-negativities. *Neuroimage.* 2010; 51:391–403. [PubMed: 20152906]
- Rubia K, Russell T, Overmeyer S, Brammer MJ, Bullmore ET, Sharma T, Simmons A, Williams SC, Giampietro V, Andrew CM, Taylor E. Mapping motor inhibition: conjunctive brain activations

- across different versions of go/no-go and stop tasks. *Neuroimage*. 2001; 13:250–261. [PubMed: 11162266]
- Schall JD, Godlove DC. Current advances and pressing problems in studies of stopping. *Curr Opin Neurobiol*. 2012; 22:1012–1021. [PubMed: 22749788]
- Schmajuk M, Liotti M, Busse L, Woldorff MG. Electrophysiological activity underlying inhibitory control processes in normal adults. *Neuropsychologia*. 2006; 44:384–395. [PubMed: 16095637]
- Schmiedt-Fehr C, Basar-Eroglu C. Event-related delta and theta brain oscillations reflect age-related changes in both a general and a specific neuronal inhibitory mechanism. *Clin Neurophysiol*. 2011; 122:1156–1167. [PubMed: 21115266]
- Stinear CM, Coxon JP, Byblow WD. Primary motor cortex and movement prevention: where Stop meets Go. *Neurosci Biobehav Rev*. 2009; 33:662–673. [PubMed: 18789963]
- Swann N, Tandon N, Canolty R, Ellmore TM, McEvoy LK, Dreyer S, DiSano M, Aron AR. Intracranial EEG reveals a time- and frequency-specific role for the right inferior frontal gyrus and primary motor cortex in stopping initiated responses. *J Neurosci*. 2009; 29:12675–12685. [PubMed: 19812342]
- Swann NC, Cai W, Conner CR, Pieters TA, Claffey MP, George JS, Aron AR, Tandon N. Roles for the pre-supplementary motor area and the right inferior frontal gyrus in stopping action: electrophysiological responses and functional and structural connectivity. *Neuroimage*. 2012a; 59:2860–2870. [PubMed: 21979383]
- Swann NC, Tandon N, Pieters TA, Aron AR. Intracranial Electroencephalography Reveals Different Temporal Profiles for Dorsal- and Ventro-lateral Prefrontal Cortex in Preparing to Stop Action. *Cerebral Cortex*. 2012b
- Tabibnia G, Monterosso JR, Baicy K, Aron AR, Poldrack RA, Chakrapani S, Lee B, London ED. Different forms of self-control share a neurocognitive substrate. *J Neurosci*. 2011; 31:4805–4810. [PubMed: 21451018]
- Torreclillos F, Albouy P, Brochier T, Malfait N. Does the processing of sensory and reward-prediction errors involve common neural resources? Evidence from a frontocentral negative potential modulated by movement execution errors. *J Neurosci*. 2014; 34:4845–4856. [PubMed: 24695704]
- Verbruggen F, Logan GD. Models of response inhibition in the stop-signal and stop-change paradigms. *Neurosci Biobehav Rev*. 2009; 33:647–661. [PubMed: 18822313]
- Wessel JR, Aron AR. Unexpected Events Induce Motor Slowing via a Brain Mechanism for Action-Stopping with Global Suppressive Effects. *J Neurosci*. 2013; 33:18481–18491. [PubMed: 24259571]
- Wessel JR, Conner CR, Aron AR, Tandon N. Chronometric electrical stimulation of right inferior frontal cortex increases motor braking. *J Neurosci*. 2013; 33:19611–19619. [PubMed: 24336725]
- Wessel JR, Danielmeier C, Morton JB, Ullsperger M. Surprise and error: common neuronal architecture for the processing of errors and novelty. *J Neurosci*. 2012; 32:7528–7537. [PubMed: 22649231]
- Whelan R, Conrod PJ, Poline JB, Lourdasamy A, Banaschewski T, Barker GJ, Bellgrove MA, Buchel C, Byrne M, Cummins TD, Fauth-Bühler M, Flor H, Gallinat J, Heinz A, Ittermann B, Mann K, Martinot JL, Lalor EC, Lathrop M, Loth E, Nees F, Paus T, Rietschel M, Smolka MN, Spanagel R, Stephens DN, Struve M, Thyreau B, Vollstaedt-Klein S, Robbins TW, Schumann G, Garavan H. Adolescent impulsivity phenotypes characterized by distinct brain networks. *Nature Neuroscience*. 2012; 15:920–925.
- Wiecki TV, Frank MJ. A computational model of inhibitory control in frontal cortex and basal ganglia. *Psychol Rev*. 2013; 120:329–355. [PubMed: 23586447]
- Yamagishi N, Callan DE, Goda N, Anderson SJ, Yoshida Y, Kawato M. Attentional modulation of oscillatory activity in human visual cortex. *Neuroimage*. 2003; 20:98–113. [PubMed: 14527573]
- Yamanaka K, Yamamoto Y. Single-trial EEG power and phase dynamics associated with voluntary response inhibition. *J Cogn Neurosci*. 2010; 22:714–727. [PubMed: 19413474]
- Zandbelt BB, Bloemendaal M, Neggers SF, Kahn RS, Vink M. Expectations and violations: Delineating the neural network of proactive inhibitory control. *Hum Brain Mapp*. 2012
- Zandbelt BB, Vink M. On the role of the striatum in response inhibition. *PLoS ONE*. 2010; 5:e13848. [PubMed: 21079814]

Zavala BA, Tan H, Little S, Ashkan K, Hariz M, Foltynie T, Zrinzo L, Zaghoul KA, Brown P.
Midline Frontal Cortex Low-Frequency Activity Drives Subthalamic Nucleus Oscillations during
Conflict. J Neurosci. 2014; 34:7322–7333. [PubMed: 24849364]

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Highlights

We developed a task that required action-stopping to a complex feature-template

EEG activity in a simple stop task network explained stopping in the complex stop task

EEG activity of this network also explained RT slowing for partial feature matches

A common brain system underlies both simple and more realistic forms of stopping

Figure 1.

Trial diagram for the complex stopping task. A) Every trial consisted of a template-encoding phase, followed by a stop/go phase, of between 10 and 20 individual stimuli. B) Detailed outline of the template-encoding phase. C) Three individual examples of stimuli in the stop/go phase. The first row illustrates an M1-stimulus in the context of the stopping-template in panel B (only the type of arrow matches the template). The second and third rows depict M3- and M5-stimuli, respectively.

Figure 2.

Group-average of the stopping-related independent components, as identified from the standard stop-signal task. A) Topographical distribution of averaged IC weight matrices. B) Mean delta-band power (in units of % change compared to baseline) on successful and failed stop-trials in the standard stop signal task. C) Mean theta-band power (in units of % change compared to baseline) on successful and failed stop-trials in the standard stop signal task. Components were selected based on increases in activity in either one of these two frequency bands following the stop-signal. SSRT = stop-signal reaction time.

Figure 3. Slowing of reaction time as a function of partial feature matches between a given stimulus and the stopping template. A) Experiment 1 (behavioral); B) Experiment 2 (EEG).

Figure 4.

ERSP results. All graphs show activity on the CST task, in the independent components that explained successful stopping in the SST [see Figure 2]. On the x-axis, S marks go-stimulus onset. A) Event-related spectral perturbations associated with stopping action in the complex stopping task. The leftmost panel shows the ERSP (in units of % change from baseline) to succMSTOP-stimuli (all features match the stopping template, and the response was successfully suppressed), on which behavior was successfully stopped. The second panel shows activity following stimuli without any feature matches (M0). The third and fourth panels show the difference between conditions, with and without masking by an FDR-corrected significance threshold of $p < .05$ (two-sided). B) ERSP associated with braking as caused by partial matches in the complex stopping task (see Figure 3). C) The top row depicts group-averaged standardized beta-weights of a single-trial general-linear model investigating the relation of stimulus-locked ERSP-activity in the CST with the amount of feature overlap between the stimulus and the stopping-template (left panel); the amount of reaction time slowing induced on any given trial compared to a M0-stimulus (middle panel); and their interaction (right panel). The bottom row corresponds to the top row, masked by a significance threshold of $p < .05$ (two-sided, FDR-corrected).