

Enhancing frontal top-down inhibitory control with Go/NoGo training

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Abstract Whether and how the capacity to inhibit cognitive and motor processes can be trained and the underlying neuroplastic mechanisms remain unclear. Using electrical neuroimaging methods, we investigated how inhibitory control training regimens can be designed to enhance frontal top-down inhibition processes. We trained participants with a Go/NoGo task in which the stimulus–response mapping rules were systematically varied. This task parameter has indeed be hypothesized to determine the extent to which top-down frontal inhibition processes are involved and thus ultimately reinforced during the training. The effects of training on inhibitory control were assessed by analyzing the event-related potentials (ERPs) measured during the Go/NoGo task with a data-driven time- and electrode-wise 2×2 ANOVA with factors Session (beginning; end of the training) and Stimuli (Go; NoGo). To localize the sources of the ERP effects in the brain, the same statistical design was applied to distributed electrical source estimations averaged over the periods of ERP modulations. The training improved inhibitory control performance. Electrophysiologically, we found a significant Session \times Stimulus interaction at 300–400 ms post-stimulus onset over centro-occipital electrodes. Statistical

parametric mapping on the brain source estimations revealed an interaction within right inferior frontal cortices driven by a decrease in response strength to NoGo but not to Go trials in this region. Our collective results demonstrate that frontal top-down inhibition processes can be enhanced with specifically designed inhibitory control training regimens.

Keywords Inhibitory control · Plasticity · Frontal · EEG · Source estimations

Introduction

Inhibitory control (IC), the ability to suppress ongoing or planned cognitive or motor processes, can be improved with short- (50 min in Verbruggen and Logan 2008; Manuel et al. 2010; Manuel et al. 2013) to medium-term training (ca. 10–15 h in Thorell et al. 2009; Johnstone et al. 2012; Berkman et al. 2014; Chavan et al. 2015). For example, training IC with stop-signal tasks (SST) or Go/NoGo tasks has been found to decrease stop-signal reaction times (SSRT; Guerrieri et al. 2012; Manuel et al. 2013), or to decrease false alarm rate and/or response time in the trained task, respectively (Schapkin et al. 2007; Manuel et al. 2010; Johnstone et al. 2012; Benikos et al. 2013). A few studies, however, did not find such a decrease (e.g., Cohen and Poldrack, 2008), or only in some conditions (Ditye et al. 2012). These patterns of behavioral improvements have been hypothesized to follow from improvements in the speed of inhibition processes (White et al. 2014; Chavan et al. 2015).

Functional neuroimaging studies revealed that training-induced improvements in IC were supported by plastic modifications within the cortico-subcortical inhibitory

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control brain network (inferior frontal gyri, pre-supplementary motor area and basal ganglia; IFG, SMA, and BG, respectively; Manuel et al. 2013; Berkman et al. 2014; Chavan et al. 2015) according to two distinct, non-exclusive neurocognitive mechanisms.

First, IC performance improvement may result from the development of automatic forms of inhibition: repeated associations between stop stimuli and stopping goals develop automatic forms of inhibition. The controlled engagement of frontal inhibitory areas is then bypassed and inhibition speeded up (Manuel et al. 2010; Spierer et al. 2013). In such training conditions, IC becomes directly triggered by the Stop stimuli via the brain areas implementing stimulus–response (S–R) mapping rules (e.g., parietal cortices 100 ms after the onset of NoGo stimuli in Manuel et al. 2010). In the same vein, training-induced improvements in conditions where Stop stimuli are predictable could follow from shifts from reactive to proactive control. Such modifications in response strategy would explain the pre-stimulus activation of frontal inhibition networks and the SSRT improvements (e.g., Berkman et al. 2014).

Second, inhibitory control performance improvement can be achieved via the enhancement of controlled top-down inhibition processes (Manuel et al. 2013; Chavan et al. 2015): when the task used to train IC is designed to prevent automatic inhibition to develop, i.e., with varying S–R mapping rules, controlled fronto-basal IC neurocognitive mechanisms are repeatedly solicited during the training and thus ultimately enhanced (Manuel et al. 2013; Spierer et al. 2013; Chavan et al. 2015). Neurophysiologically, such optimizations of frontal inhibition processes have been shown to manifest as decreases in the activity of right inferior frontal cortices around 200 ms (Manuel et al. 2013) to 300 ms after the onset of stop stimuli (Benikos et al. 2013).

However, while a reinforcement of frontal inhibitory mechanisms has been demonstrated following short training with a stop-signal task, only one study found corresponding effects after training with a Go/NoGo task with varying S–R mapping rules (Chavan et al. 2015). These authors showed that 2 weeks of such training yielded a decrease in the neural activity within the left and right IFG, as well as changes in both grey and white matter frontal microstructure. These modifications were, however, measured after medium-term training and with fMRI, which does not provide information on the temporal dynamics of the effects.

We addressed this question by training participants during 50 min with a Go/NoGo task involving systematically varying S–R mapping rules. We compared behavioral and electrical neuroimaging responses to Go and NoGo stimuli between the beginning and the end of

the training session to assess the plastic changes induced by the training. We hypothesized that behavioral improvements should be accompanied by a decrease in right frontal electrophysiological responses to NoGo stimuli over inhibitory processing phases ca. 250–300 ms post-stimulus onset, without changes in responses to Go stimuli.

Materials and methods

Participants

Twenty-two right-handed male volunteers (university students) participated in this study. All had normal or corrected to normal vision. None of the participants reported a history of neurological or psychiatric disorder. Three participants were excluded from the analyses because of artifactual EEG recording; improper calibration phase (see the “Procedure” section); and part of the task performed with the left hand. A total of 19 participants (mean age \pm SD = 25.4 \pm 4.8 years) were thus eventually included in the data analyses. All our procedures were approved by the local ethics committee.

Stimuli

Visual stimuli were blue, cyan, green, red, white or yellow ‘A’, ‘E’, ‘M’, ‘O’, ‘S’ or ‘T’ letters, presented in the center of a black screen. Each possible combination of the letter and color were used, for a total of 36 different stimuli. In a given block, NoGo stimuli were either all letters of a given color or all colors of a given letter (total 12 different NoGo stimuli); Go trials were all the remaining stimuli. For example, in a block where the letter “M” was the NoGo stimulus, a total of 30 “M” were presented, 5 in each of the 6 possible colors (5 red “M”, 5 yellow “M”, 5 blue “M”, etc.) and the 30 Go stimuli were the 30 remaining color/letter combinations: 5 “A” (one of each color), 5 “E”, 5 “O”, etc., so that in a block, the letter M was presented 5 times more than the other letters, while each colors were equally represented.

Procedure and task

Participants were seated in a sound-attenuated booth and completed a Go/NoGo task in which they were instructed to respond as fast as possible to Go stimuli by pressing a button on a response box with their right index finger, while withholding their responses to NoGo stimuli. Stimulus presentation and response recording were controlled by the E-Prime 2.0 software (Psychology Software Tools, Inc., Sharpsburg, PA).

There were 12 blocks of 60 trials separated by 2 min breaks. Each block consisted of 60 trials: 30 Go and 30 NoGo trials presented randomly. The NoGo stimuli (i.e., a given color or letter) were pseudorandomly determined for each block and across participants, so that there was never two times the same NoGo for a given participant and that the order of the NoGo was different for each participant.

Before each block, participants were presented with written instructions and told which stimuli were the NoGo for the block. Participants then completed a calibration block of 12 trials (6 Go; 6 NoGo) during which the mean response time (RT) to Go trials was calculated. This mean RT (RT threshold, RTt) was then used as a threshold during the following experimental block: if the RT to a Go trial was below 90 % of the mean RTt, a feedback ‘Too late!’ was presented at the end of the trial. This procedure enabled maintaining the same level of time pressure across participants and blocks, i.e., independently on any initial inter-individual differences in Go/NoGo performance and on performance improvement with training (for similar procedures: Vocat et al. 2008; Manuel et al. 2010). No feedback was given on performance during the calibration block and participants were kept naive to the aim of the calibration block.

Each trial consisted in the presentation of a grey fixation cross during 1500–1900 ms, followed by the stimulus (500 ms) and a response window (1000 ms) terminating as soon as the participant responded, but with a minimal duration of 250 ms. Then, a feedback on the performance was given for 500 ms: a happy smiley icon after Hits (response after a Go stimulus); a feedback “Too late!” replaced the happy smiley after hits with a $RT > RTt$; a happy smiley after correct rejections (no response after a NoGo stimulus); and an unhappy smiley after misses (no response after a Go stimulus) or false alarms (response after a NoGo trial).

Behavioral analyses

We recorded response times (RT) to Go stimuli and accuracy to NoGo stimuli (as indexed by the false alarm rate (FA): the percentage of inaccurately responded NoGo trials). Response times were averaged for each block separately after having excluded RT higher or lower than two standard deviations from the individual’s mean, as well as the first RT of each block. In order to enable comparisons between the behavioral and EEG results, RT and FA rate were averaged for the three first blocks (condition “beginning”, BEG) and the three last blocks of the session separately (condition “end”, END). The BEG and END conditions were then statistically compared with two-tailed paired t tests. Significance threshold was set at $p < 0.05$.

EEG recording

The 64-channel electroencephalogram (EEG) was recorded at a sampling rate of 1024 Hz with a Biosemi ActiveTwo system referenced to the common mode sense-driven right leg (CMS-DRL) ground and processed using the Cartool software by Denis Brunet (brainmapping.unige.ch/cartool) and the STEN toolbox developed by Jean-François Knebel (<http://www.unil.ch/line/home/menuinst/infrastructure/software-analysis-tools.html>).

For each participant, we extracted and averaged EEG epochs from 100 ms pre- to 500 ms post-stimulus onset, separately for successful Go (hits) and NoGo stimuli (correct rejections), for the first three blocks (Beginning condition, BEG) and the three last blocks of the Go/NoGo training (End condition, END). Before the event-related potential (ERP) averaging, epochs with at least one time frame at $\pm 80 \mu V$ were automatically rejected, and a 0.18–40 Hz band-pass filter was applied. After the ERP averaging, data at artifact electrodes from each participant were interpolated using 3D splines before the averaging (mean 0.3 % interpolated electrodes; Perrin et al. 1987), and the data were recalculated against the average reference. The average number [\pm standard error of the mean (SEM)] of accepted epochs was for the condition Go BEG: 87.2 ± 3.4 ; Go END: 88.2 ± 2.1 ; NoGo BEG: 85.4 ± 4.9 and NoGo END: 87.4 ± 2.9 . These values did not differ statistically on our Stimulus (Go; NoGo) \times Session (BEG; END) interaction term of interest ($p > 0.2$), ensuring that the observed ERP effects did not follow from differences in signal-to-noise ratio.

EEG analyses

Event-related potentials

We computed a Stimulus (Go; NoGo) \times Session (BEG; END) repeated measure ANOVA at each time-frame of the ERP and for each electrode. Temporal and spatial autocorrelation were corrected by considering only effects with a p value < 0.05 for at least 11 continuous time points (i.e., here 11 ms @ 1024 Hz EEG sampling rate) on at least 10 % of the electrodes (Guthrie and Buchwald 1991). We focused only on the Stimulus \times Session interaction term because we were interested in the modification of inhibitory processes with training; the main effect of Stimulus and the main effect of Session would, respectively, reveal well-established inhibitory processes and unspecific effects of exposure to the stimuli, two questions beyond the scope of our study.

This first ERP analyses allowed us to identify the time periods showing sustained significant Stimulus \times Session

interactions at the electrode level. Sources estimations were then analyzed over these periods of interest.

Electrical source estimations

Electrical source estimations were analyzed over the time periods showing a significant Stimulus \times Session interaction in the sensor space. Source estimations of individual ERP were calculated for each participant and each experimental condition using a local autoregressive average (LAURA) distributed linear inverse solution (Grave de Peralta Menendez et al. 2001; Grave-de Peralta et al. 2004). The solution space (i.e., the lead field matrix) was calculated on a realistic head model that included 3005 nodes, selected from a 6 mm \times 6 mm \times 6 mm grid of voxels equally distributed within the gray matter of the average brain of the Montreal Neurological Institute (MNI). The sources estimations were first averaged over the period of interest (to increase the signal-to-noise ratio), and the current density at each solution node was submitted to the same Stimulus \times Session 2 \times 2 design as for the ERPs analyses. A spatial correction for multiple tests was achieved by considering only clusters of p value < 0.05 of $K_e > 14$ contiguous nodes.

Results

Behavior

Inhibitory control improved with training, as indexed by a decrease in response time (Fig. 1; BEG mean \pm SEM: 375.8 \pm 25.2 ms; END: 363.8 \pm 24.6 ms; $t(18) = 2.522$; $p < 0.05$; $D_z = 0.6$; the same pattern was observed when considering median RT instead of mean RT ($p < 0.05$)), without change in the false alarm rate (BEG:

Fig. 1 Behavioral results. With training, response times to Go stimuli decreased (*asterisk* $p < 0.05$) and false alarms rate to NoGo stimuli did not change

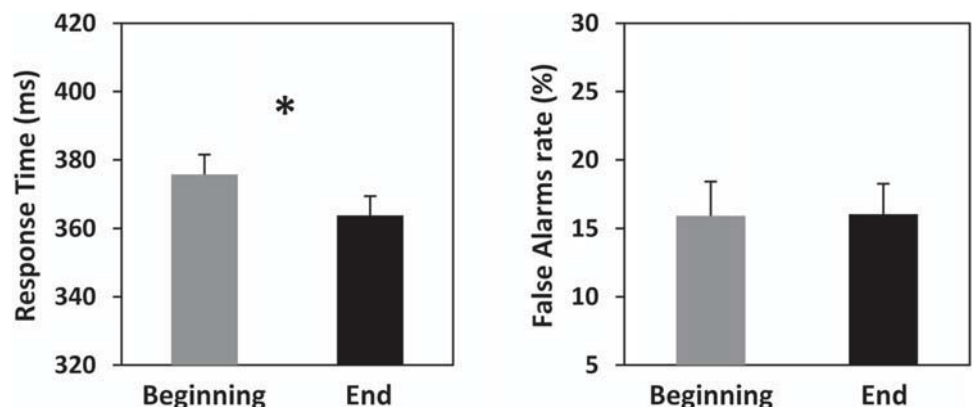


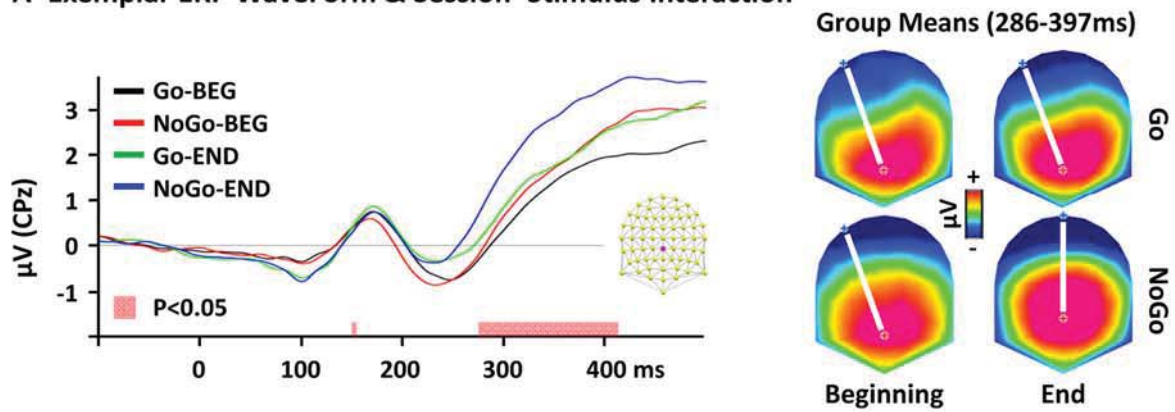
Fig. 2 Electrical neuroimaging results: Session (beginning; end of the training) \times Stimulus (Go; NoGo). **a** Event-related potentials (ERPs) are displayed for an exemplar electrode (CPz) for the four experimental conditions. The result of the Session \times Stimulus interaction is depicted in red ($p < 0.05$) along the X axis. Electric field topographies averaged over the period showing a sustained Session \times Stimulus interaction are depicted nasion upward for the four experimental conditions. Topographies' minima (blue cross) and maxima (red cross) are represented connected by a white bar. **b** Results of the ERP analyses are represented for each time point as the percentage of electrode showing a significant interaction ($p < 0.05$). The period showing a sustained interaction for $> 10\%$ of the electrodes, as well as the electrode showing this interaction are represented in red. **c** Distributed electrical source estimation: the regions showing the Session \times Stimulus interaction over the period of the ERP interaction are represented on a template brain. The mean activity over this period is represented for each experimental condition

15.9 \pm 10.9 %; END: 16.0 \pm 9.8 %; $t(18) = -0.091$; $p = 0.93$).

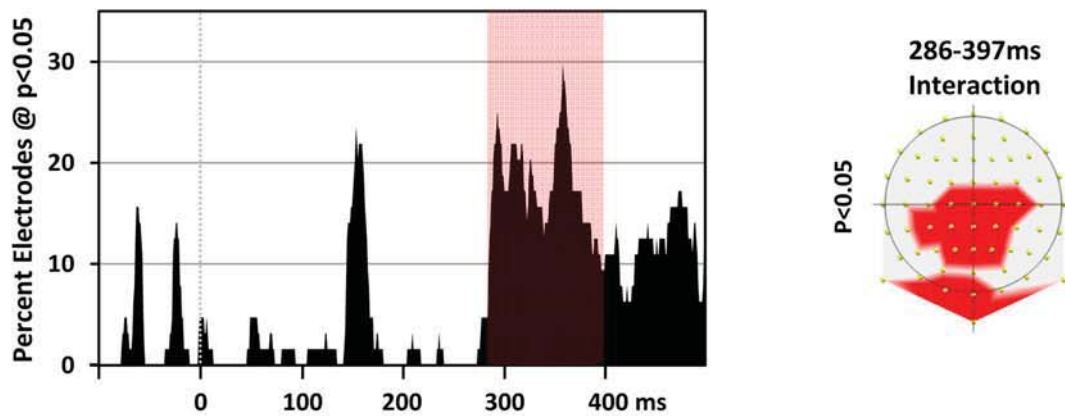
Event-related potentials and source estimations

There was a sustained ($p < 0.05$, > 11 time frame (TF); $> 10\%$ of the electrodes) Stimulus \times Session interaction from 286 to 397 ms over central and occipital electrodes (Fig. 2a, b). While the exemplar waveforms displayed in Fig. 2a indicate that NoGo ERP amplitude increased from the beginning until the end of the training, because the direction of effects in the brain space cannot be directly inferred from modulation in the sensor space (Michel et al. 2004), we interpret the direction of our effects based on the analyses of source estimations. Statistical analyses of source estimations revealed that this ERP modulation stemmed from a Stimulus \times Session interaction ($p < 0.05$, $K_e > 14$) within a right prefrontal network centered on inferior frontal cortices. This interaction was driven by a decrease in response to NoGo but not to Go stimuli within this region (Fig. 2c).

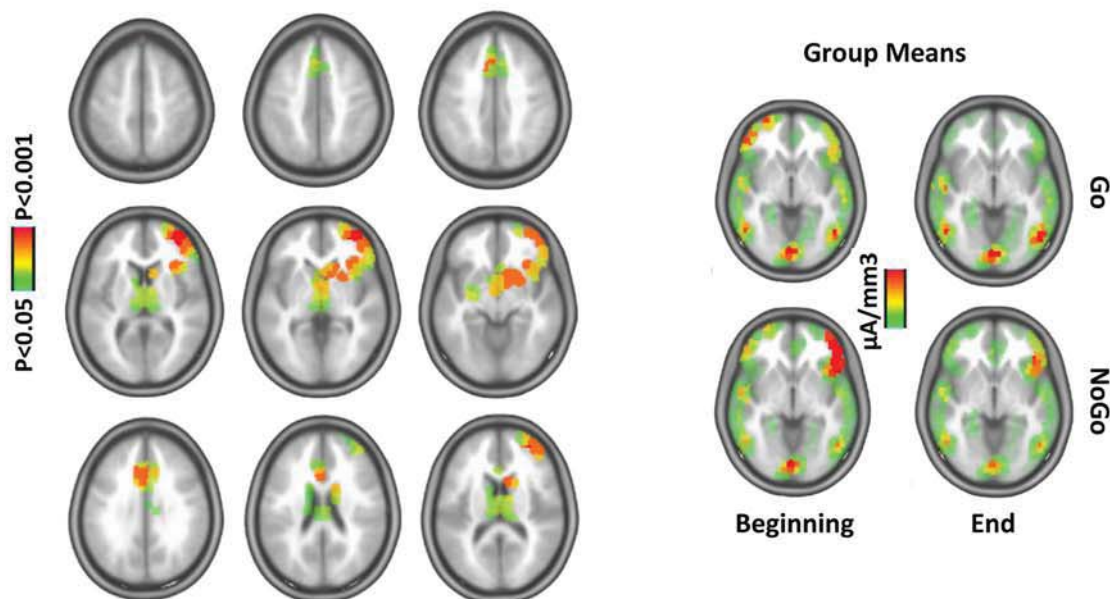
A Exemplar ERP WaveForm & Session*Stimulus Interaction



B Time- and Electrode-Wise Session*Stimulus Interaction



C Distributed Electrical Sources Estimation: Session*Stimulus Interaction (286-397ms)



We would further note that we replicated the usual N2/P3 modulation when contrasting Go to NoGo trials (i.e., main effect of factor Stimulus in our design) as evident from supplementary Fig. 1.

Discussion

Our results reveal that specifically designed Go/NoGo tasks can improve inhibition performance and enhance frontal top-down inhibition processes. After training inhibitory control (IC) with a Go/NoGo task involving systematically varying stimulus–response mapping rules, we observed a decrease in response time (RT) to Go trials with no concomitant change in the rate of false alarms to NoGo trials. These behavioral changes were accompanied by a selective decrease of the right prefrontal responses to NoGo trials.

Our finding for training-induced decrease in response times (RTs) to Go trials with no change in inhibition trials accuracy replicates the patterns of IC behavioral improvements found in previous studies on Go/NoGo training (Benikos et al. 2013; Chavan et al. 2015; though see Manuel et al. (2010) or Enge et al. (2014) for partial speed-accuracy trade-offs and Spierer et al. (2013) for a review).

In line with previous reports, we assume that the participants managed to decrease their RT to Go trials because they became faster at inhibiting their responses when unexpected NoGo stimuli were presented (Manuel et al. 2010; Verbruggen et al. 2012; Manuel et al. 2013; White et al. 2014). According to this hypothesis, IC proficiency was achieved via a speeding up of inhibition processes, but manifested as decreased RTs to Go trials with no change in false alarms rate. Such patterns putatively manifested because emphasis was put on response speed by the negative feedback “too late” given after RTs slower than individuals’ RT thresholds (see the “[Method](#)” section).

Electrophysiologically, the event-related potential (ERP) showed a Stimulus \times Session interaction at 286–397 ms post-stimulus onset, driven by a change in the electrophysiological response to NoGo stimuli with training. This pattern excludes an interpretation of the effects of training in terms of fatigue or of mere exposure to the stimuli because in this case, responses to both Go and NoGo would have been similarly modified.

The timing and the localization of the ERP results further support that the training impacted on inhibition processes. Current behavioral and computational models on action inhibition indicate that action withholding is the result of a sequence of processes, notably including perceptual/discriminative (e.g., Salinas and Stanford 2013; Logan et al. 2014; Verbruggen et al. 2014), decisional (i.e., response selection, memory retrieval; van de Laar et al.

2010; Verbruggen and Logan 2015) and eventually motor inhibition processes. In the framework of these models and of previous ERP literature on IC, the 300 ms latency of our effect suggests that the training modified primarily the latest phases of the control process, including late decisional processes and the actual motor inhibition phase. The 286–397 ms latency of the interaction corresponds to the N2/P3 complex, an electrophysiological index observed over fronto-central electrodes at about 200–300 ms (N2) to 300–500 ms post-stimulus onset (P3) and associated with inhibitory processes (Kok et al. 2004; Ramautar et al. 2004). While the N2 ERP component indexes frontal top-down inhibitory mechanisms and conflict detection (Nieuwenhuis et al. 2003), the subsequent P3 component rather indexes the cognitive implementation of the ‘braking’ process within premotor and motor cortices (Kok et al. 2004; Huster et al. 2010).

With respect to these models on the spatio-temporal architecture of inhibition processes, our interaction took place during the transition phase between the initiation of inhibition and its implementation within motor areas. Our training regimen might thus have optimized the interfacing between the inhibition command and its implementation and thus both response selection and actual inhibition.

The only previous ERP study showing a modification of frontal activity after IC training reported slightly earlier effects, at 200 ms (Manuel et al. 2013). However, IC was trained with a stop-signal task in which ongoing and not prepotent motor actions had to be suppressed. In addition, the ERP were time-locked on the Go signals and not on the NoGo. These two important differences with our study could explain the differences in the latency of our results.

The statistical parametric mapping on the brain source estimations revealed that the interaction took place within right frontal areas. These regions constitute the key node of the IC network and are thought to act as a ‘breaking’ mechanism, which inhibits motor commands via basal inhibitory pathways (Aron et al. 2014). Importantly, the right inferior frontal gyrus (rIFG) has also been involved in action selection/updating processes (Erika-Florence et al. 2014; Swick and Chatham 2014). Together with the fact that the 300 ms latency of our effects still entails decisional components, our findings that the training modified the activity of the rIFG thus further support that our training regimen might have also impacted on late response selection processes.

The direction of our effect (i.e., a decrease in the activity of the right frontal regions to NoGo trials) corroborates most of previous studies on IC training, which report that practicing inhibition tasks reduces neural activity within the inhibitory networks to inhibition trials (Manuel et al. 2013; Berkman et al. 2014; Chavan et al. 2015; though see Lenartowicz et al. 2011). Putative accounts for this pattern

of result include a sharpening of the neural populations implementing the inhibition by the exclusion of neurons less specifically involved in the process (Logothetis et al. 2001; Kelly et al. 2006; Johansen-Berg et al. 2012; Zatorre et al. 2012). The resulting decrease in task-irrelevant activity would have in turn enabled a speeding up of the inhibition process and improved IC proficiency.

We advance that the neuroplastic mechanism revealed in our study took place because the stimulus–response mapping rules were systematically varied during the task. Because there were no systematic associations between specific stimuli and inhibition goals, the development of stimulus-driven automatic inhibition could not take place. Furthermore, performance improvements driven by shifts from reactive to proactive forms of inhibition could not take place because the occurrence of the NoGo stimuli remained unpredictable during the training (Berkman et al. 2014). Consequently, reactive controlled frontal top-down inhibition processes were repeatedly involved and ultimately optimized by the training (Spierer et al. 2013).

Importantly, because they are based on associations of inhibition with specific stimuli, the mechanisms of ‘IC automatization’ lead to effects highly specific to the trained NoGo-cueing or NoGo-triggering stimuli (Enge et al. 2014). In contrast, an important consequence of improving the efficiency of top-down controlled inhibition as with the present training regimen is that since it impacts on domain-general inhibition areas, its effects should generalize to untrained IC tasks. This assumption remains however speculative and call for future studies on the pattern of generalization of IC training regimen based on Go/NoGo tasks with systematically varying S–R mapping rules.

As another limitation of our study, we did not assess whether the observed effects of the training persisted in time; it could be that the functional modifications of the frontal IC network were only transient (Verbruggen et al. 2013). Finally, other factors known to influence inhibitory control performance, such a participant’s intelligence quotient (e.g., Yucel et al. 2012), should be controlled in future studies on IC plasticity.

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