



Individual differences in inhibitory control abilities modulate the functional neuroplasticity of inhibitory control

Mo Chen¹ · Yan Jing Wu² · Junjie Wu¹ · Yongben Fu¹ · Shuhua Li¹ · Huanhuan Liu³ · Chunming Lu^{1,4} · Taomei Guo^{1,4} 

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Abstract

Previous research has shown that training on inhibitory control (IC) leads to functional neural plastic changes, although this effect on individuals with different levels of IC abilities has yet to be studied. Here, we examined the individual differences in IC abilities of 85 participants, who performed a Simon task while undergoing a functional magnetic resonance imaging (fMRI) scan. Participants in the experimental group followed an 8-day training session on IC between the pre- and the post-test, whereas the control group did not receive any training. The fMRI results reported that, in comparison to the control group, the training session elicited different patterns of neural adaptation between participants with high- and low-IC abilities in the experimental group. While training reduced activation levels in the supplementary motor area (SMA), bilateral thalamus, and left anterior cingulate cortex (ACC) of individuals with low-IC performance prior to the training, the same pattern was not found in participants with high-IC performance. In addition, individual differences in IC abilities before training also positively correlated with activation reduction in these brain regions after training. These results suggest that individual differences in IC abilities modulate the neural plasticity of IC, and IC training specifically enhanced neural efficiency in individuals with low-IC abilities. Our findings provide a novel perspective for investigating the functional neuroplasticity of the IC system by highlighting the interaction between individual variances in IC abilities and short-term training effects.

Keywords Inhibitory control · Brain plasticity · Individual differences · fMRI

Introduction

Inhibitory control (IC) refers to the ability to suppress inappropriate responses, S–R mappings or task-sets when context changes (Aron et al. 2004b). As a part of the executive

functions, IC plays a crucial role in the everyday life of human beings (Miyake et al. 2000).

Previous studies showed that performing IC-related tasks, including the go/no-go task, activate a fronto-striatal neural network involving the right ventrolateral prefrontal cortex, the bilateral dorsolateral prefrontal cortices, and the pre-supplementary motor area/anterior cingulate cortex (Aron 2010; Aron et al. 2004b, 2014). Thus, it is believed that the prefrontal cortex plays a central role in both top-down control and response inhibition (e.g., Chambers et al. 2009; Chikazoe 2010), while the pre-supplementary motor area/anterior cingulate cortex is generally associated with conflict detection (e.g., Botvinick et al. 2001; Crottaz-Herbette and Menon 2006). Furthermore, several neuroimaging studies on IC training have associated the functional and structural changes observed in both the dorsal anterior cingulate cortex and the right inferior frontal gyrus with the training effects seen on IC performance (Berkman et al. 2014; Chavan et al. 2015, 2017; Lenartowicz et al. 2011). For example, Chavan et al. (2015) trained healthy participants using the go/no-go task for 2 weeks, and found that participants responded

Mo Chen, Yan Jing Wu, and Junjie Wu contributed equally to this study.

✉ Taomei Guo
guotm@bnu.edu.cn

- ¹ State Key Laboratory of Cognitive Neuroscience and Learning, Beijing Normal University, Beijing 100875, People's Republic of China
- ² Faculty of Foreign Languages, Ningbo University, Ningbo, People's Republic of China
- ³ Research Center of Brain and Cognitive Neuroscience, Liaoning Normal University, Dalian, People's Republic of China
- ⁴ IDG/McGovern Institute for Brain Research, Beijing Normal University, Beijing, People's Republic of China

faster to Go trials after training associated with decreased neural activity in the bilateral inferior frontal gyrus and with an increase in both the grey matter and the modulations of the white matter microstructure in the right inferior frontal gyrus (rIFG). However, the effectiveness of IC training is a matter of controversy, given that some studies have reported null effects (e.g., Bowley et al. 2013; Cohen and Poldrack 2008; Enge et al. 2014).

One possible explanation for the contrasting findings in previous training studies is that individual differences in participants' background factors, including IC abilities prior to training, were not taken into consideration. In fact, it has been shown that individual variance in IC abilities is a critical factor determining the neural mechanisms underlying IC performance. For example, Aron and Poldrack (2006) found a negative correlation between stop-signal reaction time (SSRT) and levels of neural activations in the right IFG. In addition, individuals with shorter SSRT also presented enhanced activity in a large-scale network including both the rIFG and the posterior cingulate cortex (Congdon et al. 2010). As compared to young adults, older adults showed enhanced improvement in their ability to implement cognitive control after practice (Paxton et al. 2006). Schizophrenia patients also exhibited substantial cognitive control improvements in their performance after cognitive control training (Edwards et al. 2010). Overall, the evidence suggests that individuals with lower baseline performance prior to training benefit more from training than individuals with higher baseline performance. Therefore, we expect individual variance in IC abilities prior to training to affect the behavioral outcome of training and the underlying neural processes.

To the best of our knowledge, although IC includes the two sub-components, most studies have focused on neural plastic changes as a result of training in response inhibition rather than interference suppression. However, previous studies suggest that the neural mechanisms underlying the two processes are dissociable (e.g., Nigg 2000; Van Boxtel et al. 2001). For example, Blasi et al. (2006) showed that interference suppression induced strong activation in the dorsal anterior cingulate cortex (ACC), while response inhibition activated an extended area in the prefrontal and parietal cortex. In addition, previous studies have not investigated the role of individual variance in IC abilities in training-induced functional neuroplastic changes. Addressing these issues would improve our understanding of the neuroplasticity of the IC system, and also have implications for the treatment of IC deficits. We are particularly interested in the potential plasticity of two brain regions, i.e., the ACC, and the rIFG. This is due to the fact that both these areas play crucial, though distinct, roles in cognitive control.

Specifically, the ACC is associated with conflict monitoring and detection (Botvinick et al. 1999, 2001, 2004; Carter et al. 1998). Previous studies have shown that the activity in

the ACC is greater when processing trials with high levels of conflict as opposed to trials with low levels of conflict (for reviews see Botvinick et al. 2004; Heilbronner and Hayden 2016).

In contrast, the rIFG is an important brain region related to response inhibition (Aron et al. 2004b, 2014; Chikazoe 2010). In fact, while patients with lesions in the rIFG report an impaired function in their inhibitory control (Aron et al. 2003, 2004a), participants with stronger activation of the rIFG usually perform better in tasks related to response inhibition (Aron and Poldrack 2006; Chamberlain et al. 2009; Garavan et al. 1999). In addition, it has been proposed that, once the ACC detects conflict, the rIFG is then recruited to inhibit inappropriate responses (Aron et al. 2004a; Gehring and Knight 2000).

In the current study, we aimed to examine the functional plasticity of the neural mechanisms associated with IC training using a modified Simon task (e.g., Liu et al. 2015). Unlike the Go/No-go task and the stop-signal task, which mainly tap into response inhibition, performance in the Simon task reveals participants' ability to suppress interference from task-irrelevant information (e.g., Diamond 2013). Neuroimaging studies have shown the Simon effect (the contrast between the incongruent condition with the congruent condition) induced the strong activity in the dorsal ACC, dorsal premotor cortex as well as the parietal cortex (e.g., Fan et al. 2003; Liu et al. 2004; Peterson et al. 2002), indicating that individuals need to rely on this fronto-parietal network to perform the Simon task. Participants in the present study were divided into an experimental group (i.e., the training group) and a control group (i.e., the non-training group) and were matched by age, gender, and fluid intelligence. All participants performed the Simon task while undergoing a functional magnetic resonance imaging (fMRI) scan in the pre-test. The experimental group then followed an 8-day training using a Simon task highly comparable to the one used in the experiment, while the control group did not receive any training. To examine the effects of individual variance on IC ability, participants were assigned to either the high-IC group or the low-IC group, according to their pre-test performance. Specifically, the median split method was used; the top half of the participants with smaller Simon effects were assigned to the high-IC group, whereas the bottom half with larger Simon effects were assigned to the low-IC group (Duverne et al. 2009; Liu et al. 2014; Wolf and Walter 2005). Our hypotheses were that (1) a difference in performance in the Simon task between the training and the control group prior to training would not be observed; (2) the training would induce functional plastic changes in the neural mechanisms underlying IC performance in the experimental group; (3) individuals with high- and low-IC abilities would present different patterns of training effects at both the behavioral and the neuroanatomical levels. Particularly,

given that the Simon task is more associated with interference suppression (e.g., Diamond 2013), we expect neural plastic changes to be observed as reduced activation levels in the ACC rather than the rIFG. In addition, we also expect to see significant correlations between individual differences in IC abilities before training and neural changes after training.

Materials and methods

Participants

Eighty-five participants (47 females) were recruited in the current study, which was approved by the ethics committee of the State Key Laboratory of Cognitive Neuroscience and Learning at Beijing Normal University. Written informed consent was obtained from each participant prior to the start of the experiment. All participants were right handed, and had normal or corrected-to-normal vision. No participants reported a history of neurological or psychiatric disorders. In addition, participants were randomly assigned to either the experimental group or the control group. Given that five participants were excluded from the analysis due to excessive head movement (i.e., > 3 mm) during the fMRI scanning, the experimental and control groups comprised 40 participants each. To examine the effects of individual differences (in IC ability) on IC training, the experimental group and control group were further divided into the high-IC and the low-IC groups, according to the magnitude of the Simon effect (i.e., the difference in reaction times between incongruent and congruent trials) in the pre-test. For both the experimental and control groups, an independent samples *t* test described the Simon effect for the high-IC group to be significantly smaller than that for the low-IC group (experimental group: $t(38) = 7.083$, $p < 0.001$; control group: $t(38) = 8.695$, $p < 0.001$).

In addition, according to a 2 (group: experimental group/control group) \times 2 (individual differences: high IC/low IC) ANOVA on age, and fluid intelligence, the main effect of group and individual differences was not significant (group for age: $F(1, 75) < 1$; group for fluid intelligence: $F(1, 75) = 1.87$, $p = 0.176$; individual differences for age: $F(1,$

$75) < 1$; individual differences for fluid intelligence: $F(1, 75) = 1.14$, $p = 0.290$). In contrast, the interaction between groups and individual differences for age was significant, $F(1, 75) = 6.15$, $p = 0.015$. However, a significant difference between IC groups was not reported by independent samples *t* tests (experimental group: $t(38) = -1.32$, $p = 0.194$; control group: $t(38) = 1.91$, $p = 0.064$). Overall, the participants were well matched in age and intelligence (see Table 1).

Task

A modified Simon task was used for both test sessions and training sessions. At each trial of the test sessions, a fixation cross was presented for 200 ms at the center of a screen, followed by a blank screen that was shown for 300 ms. Successively, a colored arrow, pointing either to the left or to the right, appeared in the center of the screen for 1 s, during which the participants were asked to respond by pressing a button with either their left or right index finger. The arrow was then replaced by a blank screen for 1, 2, 3, or 4 s (i.e., the inter-stimuli intervals). In contrast, the inter-stimulus interval in the training session varied between 1500 and 2000 ms randomly (rather than 1, 2, 3, or 4 s), while the stimuli appeared on the screen until participants provided a response (rather than for a fixed duration of 1 s). The color (blue or red) of the arrow specified the condition of the trial. Specifically, a blue arrow signified that participants needed to press the button on the same side to which the arrow pointed (i.e., the congruent condition); whereas the red arrow signified that they should press the button on the opposite side to which the arrow pointed (i.e., the incongruent condition). The correspondence between the color of the arrow and the congruency of the trial was counterbalanced across participants.

Procedure

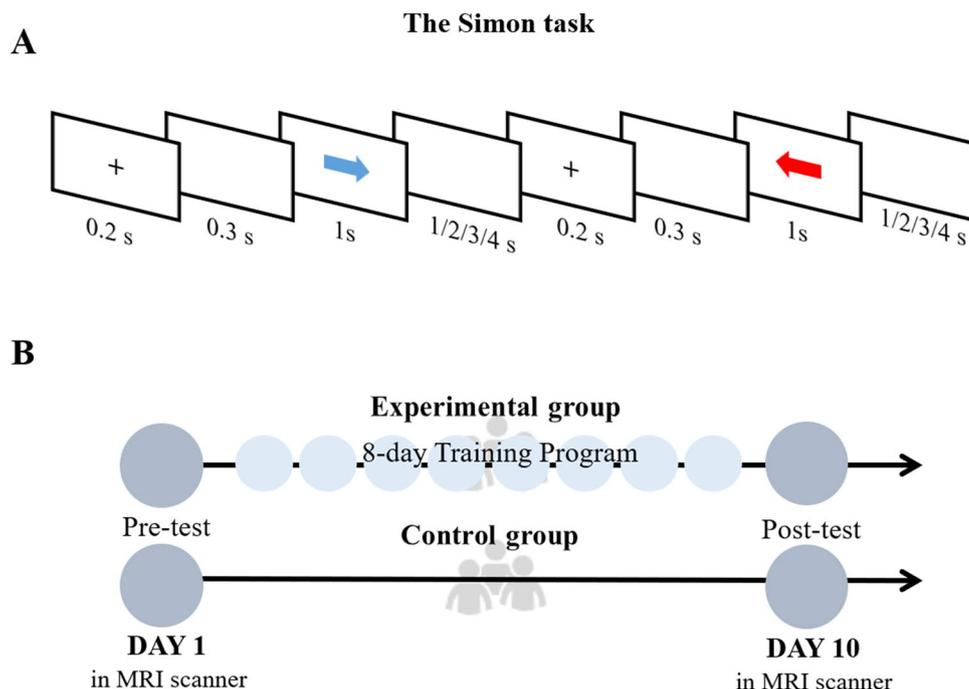
As shown in Fig. 1, all participants completed identical pre- and post-test sessions. The experimental group followed an 8-day training between the two test sessions, while the control group did not receive any training. In each session, participants completed a short practice (16 trials) using a

Table 1 Descriptive characteristics for participants of the two groups (standard deviations in parentheses)

	Experimental group		Control group	
	High IC ($N=20$)	Low IC ($N=20$)	High IC ($N=20$)	Low IC ($N=20$)
Gender	10 Females	9 Females	13 Females	15 Females
Age (years)	22.90 (2.45)	22.05 (1.50)	21.90 (1.97)	23.25 (2.47)
Intelligence (0–60)	57.00 (2.47)	56.05 (4.32)	55.79 (4.53)	54.90 (3.73)
Simon scores in the pre-test (ms)	-7 (17)	27 (14)	0 (14)	34 (10)

IC inhibitory control

Fig. 1 **a** The schematic display of the Simon task design. **b** Protocols for experimental and control groups



Simon task identical to the experimental task. The formal experiment included 2 runs, each containing 41 congruent trials and 41 incongruent trials, which were presented in pseudorandomized orders, and lasted for 5 min and 28 s in total. Owing to the unstable magnetic field of the scanner, two trials were added at the beginning of each run as filler trials, but were not considered for statistical analysis. Following the functional scanning, the anatomical images (8 min long) were obtained and, finally, all participants completed Raven's Progressive Matrices to measure their fluid intelligence.

Participants in the experimental group underwent an 8-day training on inhibitory control. Specifically, each training session consisted of 6 blocks with 40 congruent and 40 incongruent trials presented in a random order. One trial, not considered for statistical analysis, was added at the beginning of each block. Notably, the correspondence between the color and the congruency was reversed from one block to the other to increase both the difficulty and the novelty of the training. Every training session lasted for around half an hour.

Data collection

All images were obtained using a 3-T Siemens Sonata whole-body MRI scanner. During scanning, participants laid on the scanner table with their heads fixed with a coil to minimize head motion. For each participant, functional scans were acquired using a single shot T2*-weighted gradient echo planar imaging (EPI) sequence at 164 time points. Contiguous

axial slices were procured to cover the entire brain (including the cerebellum). Each functional scan contained 33 slices. The following scanning parameters were used: TR (repeated time) = 2000 ms, TE (echo time) = 20 ms, flip angle = 90°, field-of-view (FOV) = 200 × 200 mm², matrix size = 64 × 64, resolution within slices = 3.1 × 3.1 mm², and slice thickness/gap = 4 mm/0.8 mm. In addition, high-resolution T1-weighted anatomical images were acquired. The following scanning parameters were used: TR = 2530 ms, TE = 3.39 ms, flip angle = 7°, FOV = 256 × 256 mm², matrix size = 256 × 256, resolution within slices = 1.0 × 1.0 mm², slice thickness = 1.33 mm, and the number of slices = 144. All images were scanned in an interleaved fashion.

Behavioral data analysis

Reaction times either below 200 ms or above 1500 ms were considered outliers and excluded from our analysis. In addition, we omitted reaction times 2.5 standard deviations either below or above each participant's mean value. Statistical analyses were then performed on the remaining correct trials.

A two (individual differences: high-IC versus low-IC) by eight (training session) by two (trial type: congruent versus incongruent) repeated measures ANOVA was performed for training results.

Furthermore, a two (group: experimental versus control) by two (individual differences: high-IC versus low-IC) by two (test session: pre-test versus post-test) by two (trial type:

congruent versus incongruent) repeated measures ANOVA was performed for pre- and post-test results.

Imaging data analysis

Imaging preprocesses and whole-brain analyses were performed using SPM8 (Wellcome Department of Cognitive Neurology, London, UK) implemented in MATLAB. The first four scans were discarded to allow magnetization to reach the equilibrium state for each participant. For the rest of the images, slice-timing correction was used to reduce differences in acquisition time between slices. Subsequently, the images were realigned to the first scan and the realignment parameters were examined to correct for head motion. The inclusion criterion was that any given participant did not show an absolute motion greater than 3 mm. Accordingly, five participants were excluded due to large head motions. The remaining images were normalized to the T1 template provided by SPM8 to minimize cerebral differences between participants and then were re-sampled with $3 \times 3 \times 3$ mm voxels. The images were smoothed with a cubic Gaussian filter (6-mm full width at half-maximum).

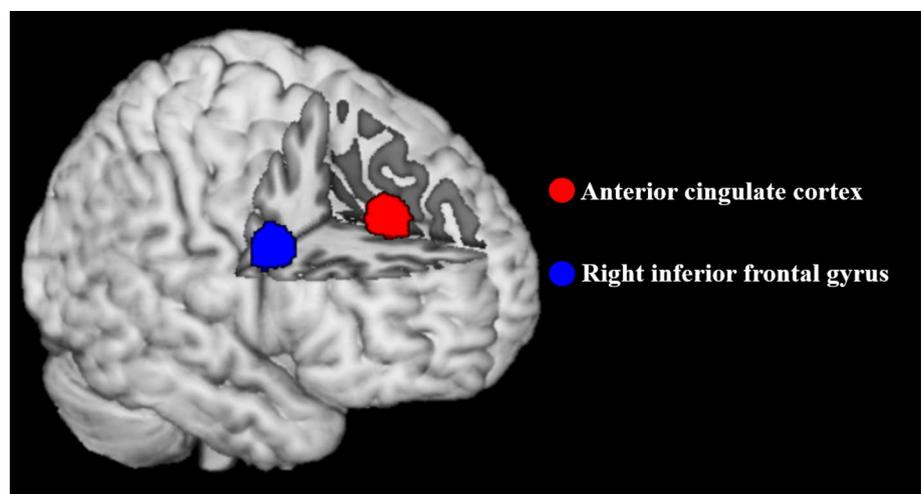
At the individual level analysis, a General Linear Model was used to estimate the hemodynamic parameters of different trial types (i.e., congruent trials and incongruent trials) and to generate statistical contrast maps of interest (incongruent trials relative to congruent trials) for each participant. Statistical analyses were performed by modeling different conditions on a voxel-by-voxel basis.

At the group level, we first performed a two (trial type: congruent/incongruent) by two (group: experimental group/control group) repeated measure ANOVA to examine the neural correlates associated with inhibitory control and any potential group difference in the pre-test session with a threshold of $p < 0.05$ and $k > 10$ at the voxel level using the familywise error (FWE) method implemented in SPM.

To examine how individual differences in IC ability might affect the training effect, the statistical contrast maps, which were derived from the Simon effect in the pre-test session at the group level, were used as a mask to reveal the interaction between individual differences (high-IC/low-IC) and test session (pre-test/post-test) in the experimental and the control group, respectively, with an uncorrected threshold of $p < 0.005$, $k > 10$. Then, we built spherical regions with an 8 mm radius centered at the peak of regions showing significant interactions. The signals for the Simon effect were then extracted to detail the interaction using a 2 (test session: pre-test/post-test) \times 2 (individual differences: high IC/low IC) repeated measure ANOVA. In addition, correlation analyses between Simon scores before training and changes in beta values of significant brain regions were conducted.

In addition, we extracted ROI signals from statistical contrast maps for the Simon effect, and performed ROI analyses to further investigate the impact of individual differences in IC ability on the training effects. The coordinates of the left ACC location (MNI: $-1, 26, 32$) were based on the meta-analysis study by Chein and Schneider (2005), which reported an activity reduction in the ACC following practice of the domain-general control task. In contrast, the coordinates of the rIFG location (MNI: $46, 6, 32$) were based on another meta-analysis on the cognitive control network conducted by Niendam et al. (2012). Subsequently, based on the coordinates of both the left ACC and the rIFG (see Fig. 2), we built spherical regions with an 8 mm radius. The beta values were extracted at the individual level for each region. Furthermore, the experimental and the control participants were separated, respectively, into the high- and low-IC subgroups using the median split method. Thereafter, two (test session: pre-test/post-test) by two (group: experimental group/control group) by two (individual differences: high IC/low IC) ANOVAs were performed to reveal the training effects.

Fig. 2 Graphic display of the left anterior cingulate cortex ($-1, 26, 32$), and right inferior frontal gyrus ($46, 6, 32$). Coordinates are reported in Montreal Neurological Institute (MNI) space



Likewise, correlational analyses between individuals' Simon scores in the pre-test and changes of beta values in each ROI were conducted in the experimental and control groups, respectively. All p values were corrected using the Bonferroni method to avoid alpha error inflation from multiple comparisons within the two predefined ROIs, and were evaluated at a threshold of $p < 0.05$.

Results

Behavioral results

The accuracy rate for the experimental and control groups were 97.1% and 95.7%, respectively. Given the high accuracy rates, a smaller variance in the data may occur. Therefore, we do not report analyses for the accuracy rate data.

Training results

The training results (reaction times) are illustrated in Fig. 3. Analysis of reaction times showed that the main effect of individual differences was not significant, $F(1, 38) = 1.93$, $p = 0.173$, while the main effect of training session was significant, $F(7, 266) = 15.37$, $p = 0.001$, $\eta_p^2 = 0.288$, showing a decrease of global reaction times as a result of training. The main effect of trial type was also significant, $F(1, 38) = 13.36$, $p = 0.001$, $\eta_p^2 = 0.260$, showing longer reaction times in the incongruent trials ($M = 514$ ms, $SD = 55$ ms) as compared to the congruent trials ($M = 502$ ms, $SD = 59$ ms). In contrast, no significant interactions between these variables were found.

Pre-test and post-test results

As shown in Table 2 and Fig. 4, the main effect of group was significant, $F(1, 76) = 8.34$, $p = 0.005$, $\eta_p^2 = 0.099$, whereas

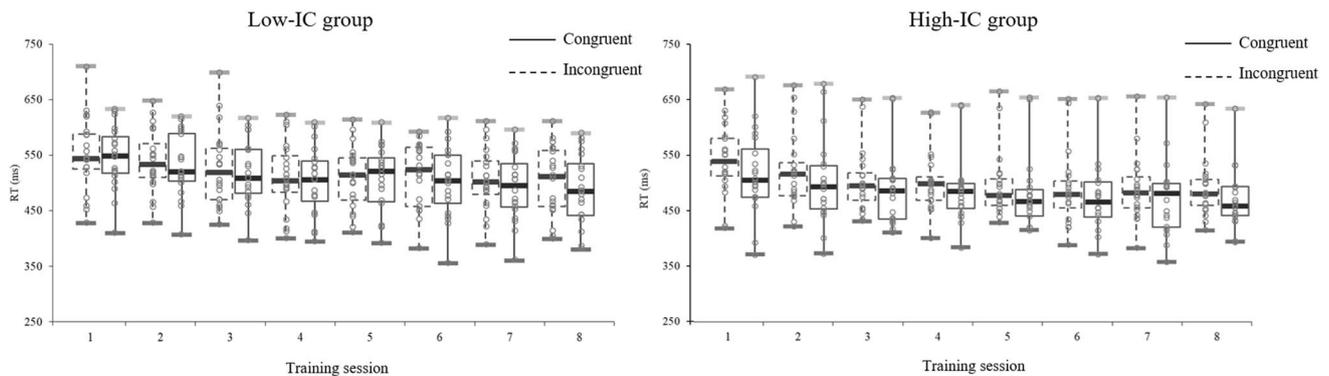


Fig. 3 Mean reaction times (ms) for two trial types (congruent/incongruent) of the Simon task across the low-IC group (left panel) and the high-IC group (right panel) during training

Table 2 Behavioral results of 2 (group: experimental group/control group) by 2 (individual differences: high IC/low IC) by 2 (test session: pre-test/post-test) by 2 (trial type: congruent/incongruent) ANOVA analyses for pre-test and post-test across groups

	F	p	η_p^2
Group	$F(1, 76) = 8.34$	< 0.01	0.099
Individual differences	$F(1, 76) < 1$		
Test session	$F(1, 76) = 30.16$	< 0.001	0.284
Trial type	$F(1, 76) = 45.47$	< 0.001	0.374
Group \times test session	$F(1, 76) = 16.11$	< 0.001	0.175
Individual differences \times trial type	$F(1, 76) = 47.09$	< 0.001	0.383
Group \times individual differences	$F(1, 76) < 1$		
Individual differences \times test session	$F(1, 76) < 1$		
Group \times trial type	$F(1, 76) < 1$		
Trial type \times test session	$F(1, 76) < 1$		
Group \times trial type \times test session	$F(1, 76) = 9.05$	< 0.005	0.106
Individual differences \times trial type \times test session	$F(1, 76) = 9.98$	< 0.005	0.116
Individual differences \times trial type \times group	$F(1, 76) < 1$		
Individual differences \times test session \times group	$F(1, 76) < 1$		

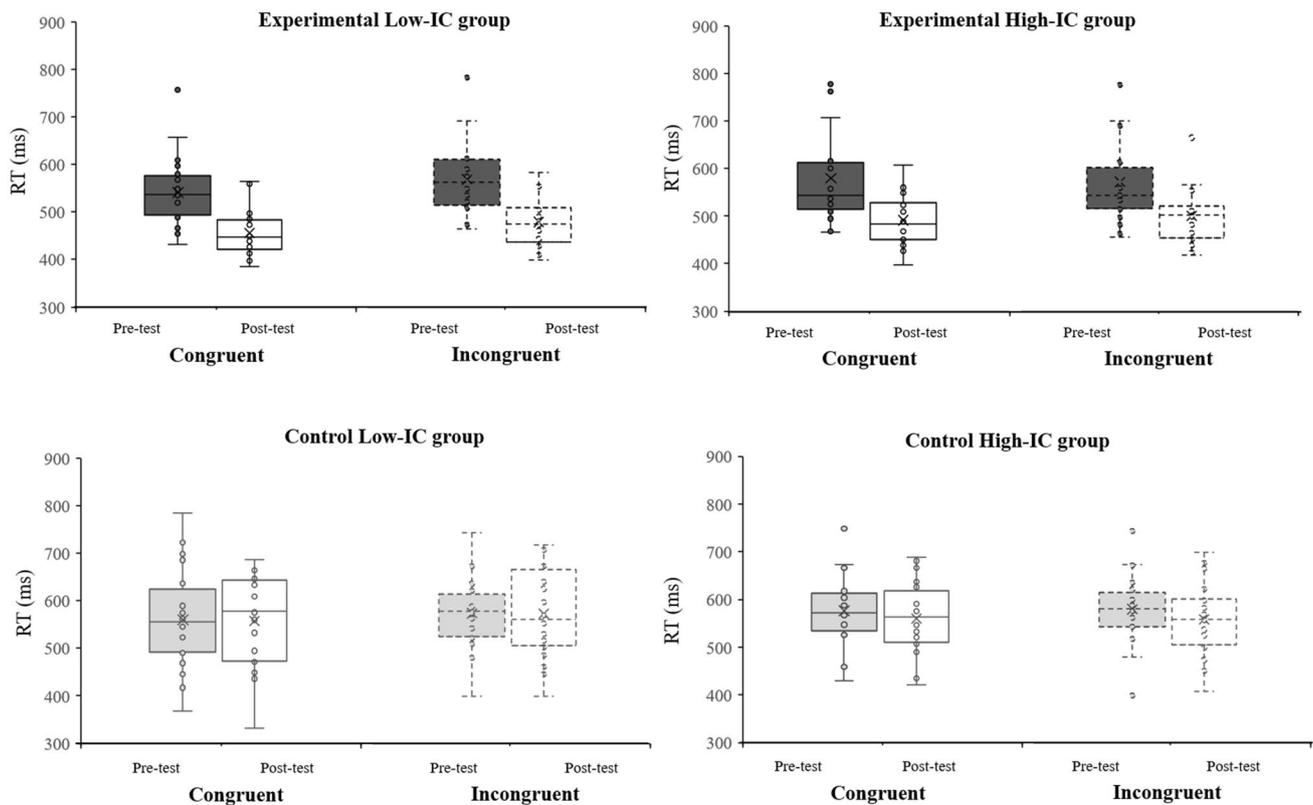


Fig. 4 Mean reaction times (ms) for low-IC participants, and high-IC participants in the experimental and control groups in the pre-test and the post-test sessions

the main effect of individual differences was not found to be significant, $F(1, 76) < 1$. Furthermore, the main effect of test session was significant, $F(1, 76) = 30.16$, $p < 0.001$, $\eta_p^2 = 0.284$, suggesting that participants were faster in the post-test session ($M = 521$ ms, $SD = 85$ ms) as compared to the pre-test session ($M = 570$ ms, $SD = 92$ ms). The main effect of trial type was also significant, $F(1, 76) = 45.47$, $p < 0.001$, $\eta_p^2 = 0.374$, indicating that the responses were slower in incongruent ($M = 552$ ms, $SD = 89$ ms) as compared to congruent trials ($M = 539$ ms, $SD = 94$ ms).

Moreover, the interaction between group and test session was significant, $F(1, 76) = 16.11$, $p < 0.001$, $\eta_p^2 = 0.175$. A paired-samples t test showed that for the experimental group, reaction times in the post-test session were significantly shorter than those in the pre-test session, $t(39) = 6.02$, $p < 0.001$, while the same was not true for the control group, $t(39) = 1.25$, $p = 0.220$. Furthermore, the interaction between individual differences and trial type was significant, $F(1, 76) = 47.09$, $p < 0.001$, $\eta_p^2 = 0.383$. A paired-samples t test showed that for the low-IC group, the reaction times in the incongruent condition were significantly longer than those in the congruent condition, $t(39) = 9.38$, $p < 0.001$, while the same was not true for the high-IC group, $t(39) < 1$. In addition, the interaction between group and individual

differences, $F(1, 76) < 1$, between individual differences and test session, $F(1, 76) < 1$, between group and trial type, $F(1, 76) < 1$, and between trial type and test session, $F(1, 76) < 1$, were all not significant.

The three-way interaction among group, trial type and test session was significant, $F(1, 76) = 9.05$, $p = 0.004$, $\eta_p^2 = 0.106$. Accordingly, the two-way ANOVA was performed for both test sessions. In the pre-test session, the main effect of trial type was significant, $F(1, 38) = 259.97$, $p < 0.001$, $\eta_p^2 = 0.870$. In contrast, the main effect of group was not significant, $F(1, 38) < 1$, indicating an absence of significant difference between the two groups in the pre-test session. The interaction between group and trial type was not significant, $F(1, 38) = 3.78$, $p = 0.059$, $\eta_p^2 = 0.090$. In the post-test session, all main effects were significant (trial type: $F(1, 38) = 18.21$, $p < 0.001$, $\eta_p^2 = 0.324$; Group: $F(1, 38) = 17.25$, $p < 0.001$, $\eta_p^2 = 0.312$), suggesting that the participants in the experimental group responded faster than those in the control group in the post-test session. However, the interaction between group and trial type was not significant, $F(1, 38) < 1$.

Similarly, the three-way interaction among individual differences, trial type, and test session was significant, $F(1, 76) = 9.98$, $p = 0.002$, $\eta_p^2 = 0.116$. Accordingly,

two-way ANOVAs were performed for both high-IC and low-IC groups. With regard to the low-IC group, the main effect of test session and trial type were significant (test session: $F(1, 39) = 17.61, p < 0.001, \eta_p^2 = 0.311$; trial type: $F(1, 39) = 88.03, p < 0.001, \eta_p^2 = 0.693$). The interaction between trial type and test session was also significant, $F(1, 39) = 7.33, p = 0.010, \eta_p^2 = 0.158$. The paired-samples t test showed that the reaction times in the incongruent condition were significantly longer than those in the congruent condition (pre-test sessions: $t(39) = 15.58, p < 0.001$; post-test sessions: $t(39) = 4.29, p < 0.001$). Considering the high-IC group instead, only the main effect of test session was significant, $F(1, 39) = 9.77, p = 0.003, \eta_p^2 = 0.200$, as both the main effect of trial type, $F(1, 39) < 1$ and the interaction between trial type and test session, $F(1, 39) = 2.41, p = 0.129, \eta_p^2 = 0.058$, were not significant.

In addition, the three-way interaction among individual differences, trial type, and group was not significant, $F(1, 76) < 1$. Both the three-way interaction among individual differences, test session, and group, $F(1, 76) < 1$, and the four-way interaction among group, individual differences, test session and trial type, $F(1, 76) < 1$, were not significant.

Whole-brain results

For the pre-test session, a two (trial type: congruent/incongruent) by two (group: experimental group/control group) repeated measures ANOVA revealed a significant main effect of trial type in the left middle frontal gyrus, supplementary motor area, bilateral superior parietal lobule, left inferior parietal lobule, left precuneus, bilateral thalamus and bilateral cerebellum (see Table 3; Fig. 5). However, the

main effect of group and the interaction between group and trial type were not significant.

In addition, for experimental group, the significant interaction between individual differences and test session, within the mask of brain regions for the Simon effect in the pre-test, was shown in the supplementary motor area and bilateral thalamus (see Table 4; Fig. 6). Specifically, the main effects were not significant (individual differences: $F(1, 38) = 3.03, p = 0.09, \eta_p^2 = 0.074$; test session: $F(1, 38) < 1$) for the SMA. The interaction between test session and individual differences was significant, $F(1, 38) = 10.50, p = 0.002, \eta_p^2 = 0.216$. Further, paired-samples t test showed the beta values decreased significantly in the post-test session when compared to the pre-test session for the low-IC group, $t(19) = 3.65, p = 0.017$, and no significant changes between test sessions for the high-IC group, $t(19) = -1.93, p = 0.069$. For the left thalamus, the main effect of test session was significant, $F(1, 38) = 5.1, p = 0.030, \eta_p^2 = 0.118$. The main effect of individual differences was not significant, $F(1, 38) < 1$. The interaction between test session and individual differences was significant, $F(1, 38) = 12.31, p = 0.001, \eta_p^2 = 0.245$. Further paired-samples t test showed the beta values decreased significantly in the post-test session when compared to the pre-test session for the low-IC group, $t(19) = 3.79, p = 0.001$, and no significant changes between test sessions for the high-IC group, $t(19) < 1$. For the right thalamus, the main effect of test session was significant, $F(1, 38) = 4.43, p = 0.042, \eta_p^2 = 0.104$. The main effect of individual differences was not significant, $F(1, 38) < 1$. The interaction between test session and individual differences was significant, $F(1, 38) = 14.24, p = 0.001, \eta_p^2 = 0.273$. Further, paired-samples t test showed the beta values decrease significantly in the post-test session when compared to the pre-test session for the low-IC group, $t(19) = 3.65, p = 0.002$, and no significant changes

Table 3 The brain areas with significant activation for the main effect of trial type revealed by the 2 (trial type: congruent/incongruent) by 2 (group: experimental group/control group) repeated measures ANOVA (voxel-wise $p(\text{FWE}) < 0.05, k > 10$)

Brain regions	Cluster size	BA	MNI coordinates (x, y, z)			F value
Frontal_Mid_L	148	6	-24	0	60	44.65
Frontal_Sup_L			-24	-6	51	44.34
Parietal_Sup_L	670	7	-15	-66	54	59.20
Parietal_Inf_L			-33	-48	45	51.55
Parietal_Sup_R	1539	7	21	-69	54	81.47
Frontal_Sup_R			27	0	57	80.97
Parietal_Inf_R			36	-45	48	61.14
Supp_Motor_Area_L	129	8	-3	15	51	41.60
Temporal_Inf_R	17	20	54	-57	-15	32.93
Thalamus_L	55		-12	-24	9	40.92
Thalamus_R	86		12	-18	0	47.93
Cerebellum_8_L	41		-30	-51	-57	40.73
Cerebellum_8_R	39		27	-57	-51	36.27

BA Brodmann area, L left, R right

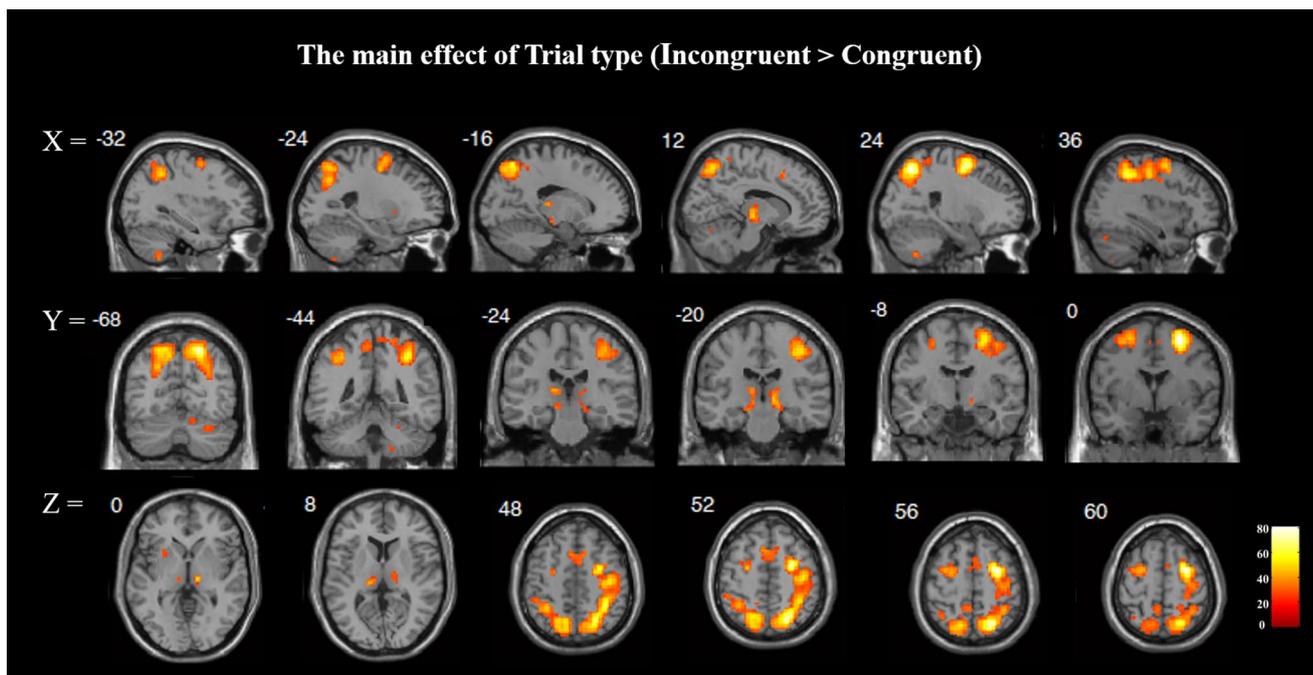


Fig. 5 Activation maps for the main effect of trial type (incongruent > congruent) in the pre-test session ($p < 0.05$, FWE corrected at the voxel level, $k > 10$). Coordinates are reported in Montreal Neurological Institute (MNI) space

Table 4 The brain areas with significant activation for the interaction between individual difference and test session revealed by the 2 (individual differences: high-IC/low-IC) by 2 (test session: pre-test/post-test) repeated measures ANOVA ($p < 0.005$, $k > 10$)

Brain regions	Cluster size	BA	MNI coordinates (x, y, z)			F value
Supp_Motor_Area_L	28	6	0	6	48	14.70
Thalamus_L	18		-18	-24	12	13.99
Thalamus_R	15		12	-15	6	14.74

BA Brodmann area, L left, R right

between test sessions for the high-IC group, $t(19) = -1.41$, $p = 0.347$. Furthermore, individual differences in IC ability before training positively correlated with activation changes between two test sessions (SMA: $r = 0.569$, $p < 0.001$; left thalamus: $r = 0.595$, $p < 0.001$; left thalamus: $r = 0.711$, $p < 0.001$).

As for control group, no brain region showed significant interactions between test session and individual differences within the mask for the Simon effect in the pre-test session, so no further analysis was conducted.

ROI analysis results

The beta values of the Simon effect were extracted within the left ACC and the rIFG for further ROI analyses.

As shown in Fig. 7, for the left ACC, the main effects were not significant (group: $F(1, 76) < 1$; test session: $F(1, 76) = 2.42$, $p = 0.248$, $\eta_p^2 = 0.031$; individual differences:

$F(1, 76) < 1$). Moreover, the interaction between test session and individual differences, $F(1, 76) = 3.89$, $p = 0.104$, $\eta_p^2 = 0.049$, between test session and group, $F(1, 76) < 1$, and between group and individual differences, $F(1, 76) < 1$, were not significant. Instead, the three-way interaction was significant, $F(1, 76) = 7.81$, $p = 0.014$, $\eta_p^2 = 0.093$. For both the experimental and the control groups, two-way ANOVAs between test session and individual differences were further performed to examine the training effects in terms of different IC abilities. With regard to the experimental group, the main effect of test session and individual differences was not significant, $F_s(1, 38) < 1$, whereas the interaction between test session and individual differences was significant, $F(1, 38) = 10.51$, $p = 0.008$. Further paired-samples t test showed the beta values to decrease significantly in the post-test session when compared to the pre-test session for the low-IC group, $t(19) = 3.11$, $p = 0.012$, suggesting that training decreased the activation levels in the left ACC for

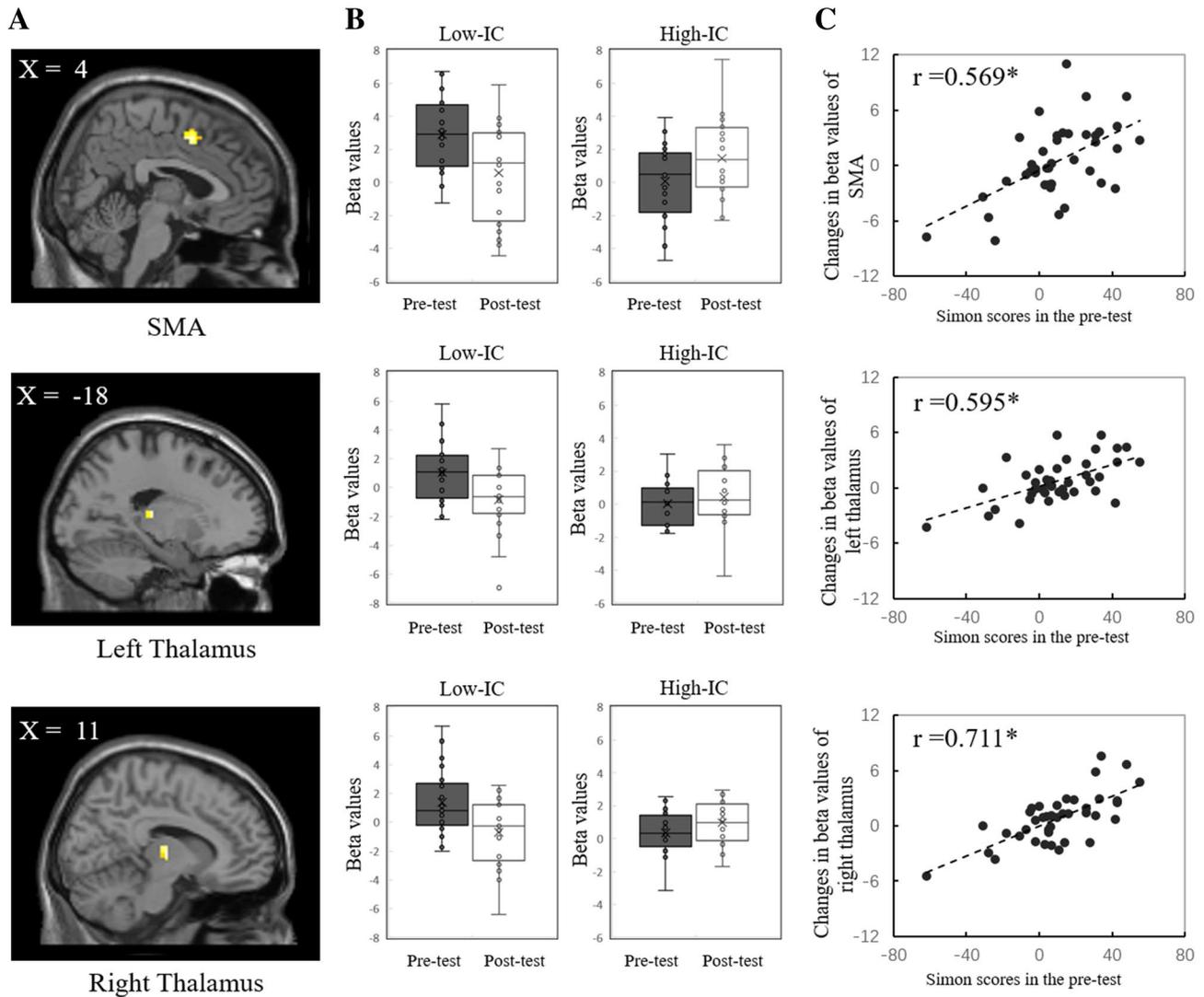


Fig. 6 Results for the experimental group. **a** Active regions for the interaction between test session and individual differences within mask for the Simon effect in the pre-test. **b** The beta values of each region. **c** The correlation results between Simon scores before train-

ing and changes in beta values of each region. *SMA* supplementary motor area. Coordinates are reported in Montreal Neurological Institute (MNI) space

the low-IC group. Considering the high-IC group instead, the beta values in the post-test session did not vary from those in the pre-test session, $t(19) = -1.68$, $p = 0.44$. In contrast, in the control group, none of the main effects were significant (test session: $F(1, 38) = 2.68$, $p = 0.440$, $\eta_p^2 = 0.066$; individual differences: $F(1, 38) < 1$), while neither the interaction between test session and individual differences was significant, $F(1, 38) < 1$.

For the right IFG, although the main effect of both group and individual differences were not significant, $F(1, 76) < 1$, the main effect of test sessions was significant, $F(1, 76) = 6.95$, $p = 0.020$, $\eta_p^2 = 0.084$, suggesting that activation of the right IFG in the post-test session was reduced significantly relatively to the pre-test session. In contrast,

the interaction between test session and individual differences, $F(1, 76) = 3.37$, $p = 0.140$, $\eta_p^2 = 0.042$, between test session and group, $F(1, 76) < 1$, and between group and individual differences, $F(1, 76) < 1$, were all not significant. The three-way interaction was also not significant, $F(1, 76) < 1$.

Furthermore, correlational analyses between activation level changes of the left ACC and rIFG (between pre-test and post-test session) and individual differences were conducted separately for the experimental and the control group. For the experimental group (see Fig. 8), activation level changes in the ACC were positively correlated with individuals' Simon scores in the pre-test ($r = 0.527$, $p < 0.001$). In contrast, there was not

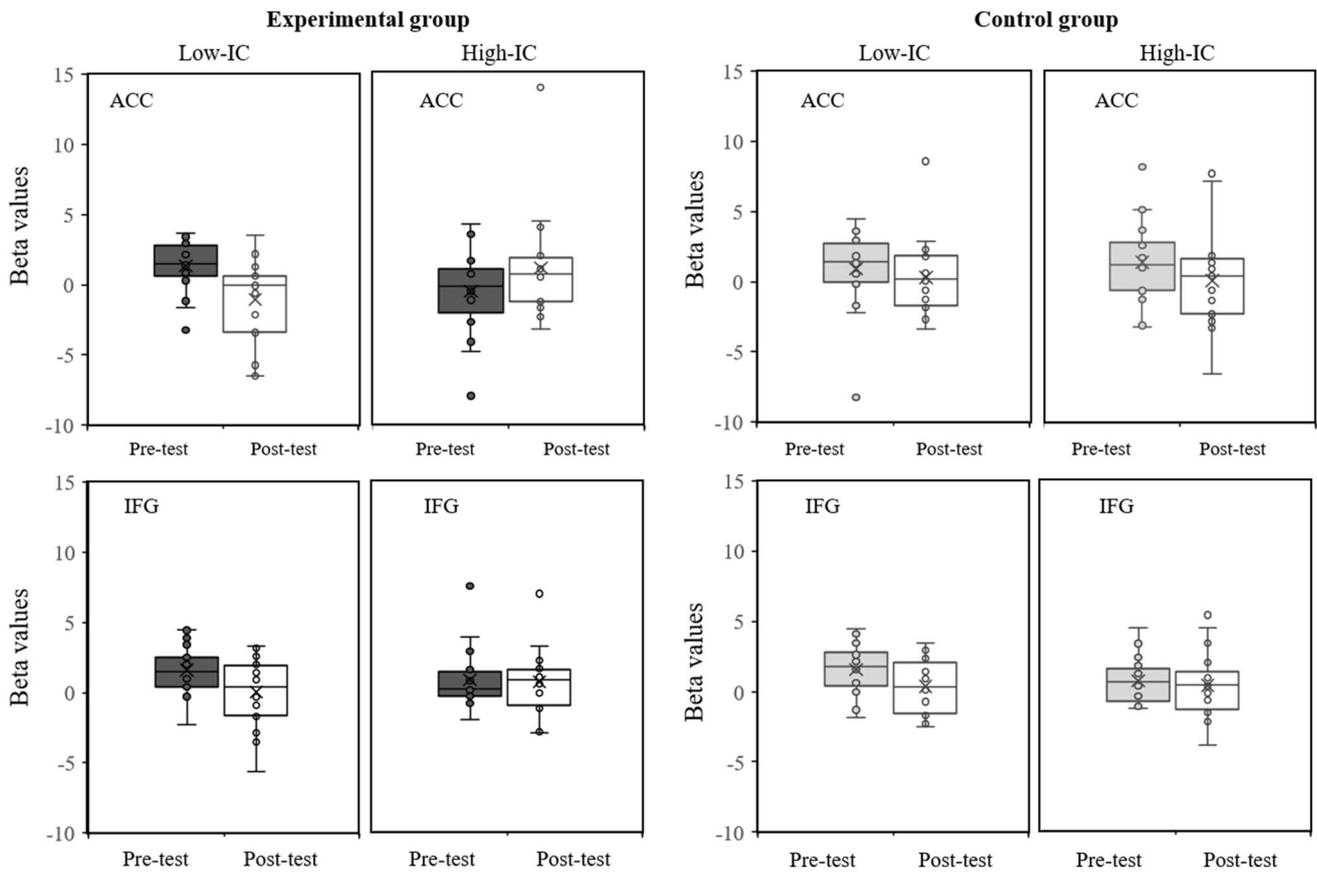
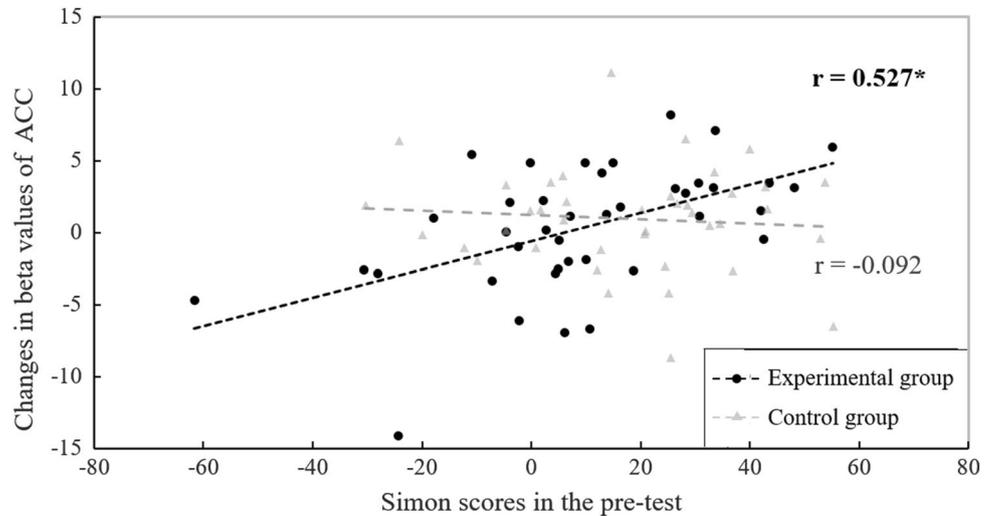


Fig. 7 The beta values of the left ACC and the rIFG for the low-IC and the high-IC individuals in the experimental and control groups, respectively. ACC anterior cingulate cortex, rIFG right inferior frontal gyrus

Fig. 8 Correlation results between Simon scores before training and changes in beta values of the left ACC (experimental group: $r = 0.527, p < 0.001$; control group: $r = -0.092, n.s.$)



a significant correlation in the control group ($r = -0.092, p = 1$). In addition, for the right IFG, no significant correlation between activation level changes and

individual differences was found for the experimental group ($r = 0.334, p = 0.07$) or the control group ($r = 0.164, p = 0.622$).

Discussion

The present study examined both the effects of short-term training on the neural correlates of IC and, of particular interest, the interaction between training effect and individual differences in IC ability on the neural plasticity of the IC system.

Our behavioral results showed that there was no significant difference in the Simon task between both the experimental and the control groups in the pre-test, indicating that both groups were well matched before the training. Another main finding was the reduction in global reaction times in the experimental group, despite the individual differences seen in the post-test when compared to the pre-test. However, the training sessions did not affect the Simon effect (i.e., difference in reaction times between congruent and incongruent trials) in the experimental group. The training effects on the Simon task performance are two-fold: (1) strengthening of top-down controlled inhibitory processes (e.g., Spierer et al. 2013) and (2) enhancement of task familiarity (Bürki et al. 2014). The strengthening of the top-down control mainly improved performance in the incongruent trials, which require interference suppression. In contrast, the familiarity effect improved performance in both incongruent and congruent trials. The overall improvement in the Simon task following training suggests that individuals with both high- and low-IC abilities in the experimental group further benefited from the training effect on task familiarity rather than on the top-down control.

With regard to the fMRI results for the pre-test session, the whole-brain analysis revealed that, compared to congruent trials, incongruent trials increased activation levels in the frontal and parietal brain regions, extending to subcortical areas including the thalamus and cerebellum. These findings are consistent with previous studies on the Simon effect (e.g., Liu et al. 2004; Rubia et al. 2006; Sebastian et al. 2013), suggesting that the classic fronto-parietal neural network for inhibitory control was involved in performing the Simon task in the current study. Furthermore, when considering the experimental and the control group separately, there was not a significant difference in neural activation associated with the Simon effect between the two groups at the pre-test session. This finding suggests that the neural mechanisms underlying IC performance were also comparable between the two groups before training.

However, further analyses revealed that the activation level of the SMA and bilateral thalamus was reduced by training for the low-IC individuals in the experimental group. These brain areas are critical nodes in the frontal cortical-basal ganglia neural network supporting inhibitory

control (Haber and Calzavara 2009; Hikosaka and Isoda 2010; Hung et al. 2018). Specifically, the SMA is associated with outright inhibition independent from attentional capture of unexpected events (Sharp et al. 2010) and strategies (Sebastian et al. 2017) during the inhibitory control process. Neuropathological studies have shown that the thalamus is instrumental for interference suppression in schizophrenia and attention-deficit hyperactivity disorder (Minzenberg et al. 2009; Rubia et al. 2011). The thalamic response varies as a function of task (Peterson et al. 2002), age (Rubia et al. 2006), and learning outcomes (de Bourbon-Teles et al. 2014). Our results further confirm the essential roles of both the SMA and the thalamus in inhibitory control and indicate that the neural plasticity of the two regions is more manifest for individuals with low-IC performance. This finding is mirrored by the positive correlations between individual differences in IC ability before training and neural activation changes between before and after training, and reveals that individual differences modulate the neural plasticity of IC.

Moreover, the ROI analyses revealed an interaction between groups, individual variance in IC abilities, and test sessions in the activation levels of the ACC. Specifically, the training sessions decreased the activation level of the ACC in the low-IC participants of the experimental group, although the same was not observed in the high-IC participants. In addition, this pattern was not shown in the control group.

Previous neuroimaging evidence demonstrated that the ACC forms an important part of the neural substrate of the IC system, supporting various mental activities, including conflict monitoring and conflict resolution (e.g., Botvinick et al. 2001; Crottaz-Herbette and Menon 2006; Egner and Hirsch 2005; Kerns et al. 2005). When participants were asked to suppress irrelevant information that interfered with their goal-directed behaviors (e.g., performing the Simon task, the Flanker task, or the Stroop task), the ACC was increasingly activated in the incongruent, as opposed to the congruent, condition, indicating its involvement in interference suppression (e.g., Adelman et al. 2002; Fan et al. 2003; Kerns et al. 2004; Liu et al. 2004). In the present study, we found that training reduced the activation level of the ACC in low-IC participants. This finding is consistent with that of several previous IC-training studies, in which the training procedure reduced neural activation (e.g., Chavan et al. 2015, 2017). In addition, it has been suggested that a lower level of activation in a neural system indicates a higher level of efficiency (Poldrack 2000). Therefore, the finding of reduced activation in the left ACC might suggest that training in interference suppression enhanced the neural efficiency of IC in individuals with a low-IC ability. Specifically, the training sessions might have strengthened participants' IC ability

through the improvement of their conflict monitoring and interference suppression, since the training task used in the present study required monitoring potential conflicts and withholding proponents, inappropriate responses (e.g., pressing the button on the same side to where the arrow points). Therefore, the reduced activation of the left ACC might indicate that individuals in the low-IC group engaged fewer neural resources to perform the Simon task in the post-test session. In contrast, individuals with high-IC abilities might be able to efficiently harness generic attentional resources to address interference conflict, resulting in a lower possibility of neuroplasticity when compared to low-IC individuals.

The effect of individual differences in inhibitory control on the neural plasticity of the left ACC was also confirmed by the correlational analysis. Specifically, we found that there was significant positive correlation between participants' IC abilities prior to training and changes (i.e., reductions) in the activation levels in the ACC after training. In other words, participants in the experimental group with lower IC abilities exhibited more training-induced reduction in the ACC as compared to those with higher IC abilities. This result confirms the finding observed in the SMA and bilateral thalamus, and is also consistent with studies on older adults (Paxton et al. 2006) and schizophrenia patients (Edwards et al. 2010), indicating that individuals with poor baseline performance benefit better from cognitive control training.

More interestingly, when compared to the pre-test, activation of the rIFG was found to decrease in the post-test in both the experimental and the control groups. This observation is consistent with that in some previous studies (Hartmann et al. 2016; Manuel et al. 2013), in which participants performed either a go/no-go task or a stop-signal task for about an hour. They found better behavioral performances and reduced activity of rIFG at the end of the task, as opposed to its beginning. Moreover, these results revealed the sensitivity of rIFG activity to adaption and also suggested that short practice was able to improve the efficiency of the neural activity associated with response inhibition (Hartmann et al. 2016; Manuel et al. 2013). Therefore, the present finding further indicates that task repetition may induce adaptive changes in response inhibition. In addition, when compared to both the go/no-go and the signal-stop tasks, the Simon task employed in our study is more demanding with regards to monitoring both conflict and interference (Nee et al. 2007) when compared to response inhibition (Aron et al. 2014). It is reasonable that we did not find any specific change in rIFG associated with training in inference suppression, but our finding that neural correlates associated with response inhibition is likely to be enhanced by brief practice may suggest that the Simon task also involves the process of response inhibition to some extent.

In summary, the present study examined both the effects of short-term training on IC and the neuroplasticity of the underlying mechanism. Our findings described training-induced functional neuroplastic changes in the SMA, left ACC, and bilateral thalamus, areas associated with IC performance. Moreover, individual differences in IC abilities prior to training modulated the neural change in these areas such that short-term training of IC enhanced neural efficiency in individuals with low IC. Overall, our results highlight the role of prior IC abilities when studying the effects of IC training, indicating another individual differences, along with age (Bunge et al. 2002), sex (Garavan et al. 2006; Li et al. 2006; Liu et al. 2015), and pre-existing experience (Chavan et al. 2017), which can play a role in constraining IC-training outcomes. For this reason, future studies on IC training should take individual variance in prior IC abilities into consideration.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All the procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

References

- Adelman NE, Menon V, Blasey CM, White CD, Warsofsky IS, Glover GH, Reiss AL (2002) A developmental fMRI study of the Stroop color-word task. *Neuroimage* 16:61–75
- Aron AR (2010) From reactive to proactive and selective control: developing a richer model for stopping inappropriate responses. *Biol Psychiatry* 69:55–68
- Aron AR, Poldrack RA (2006) Cortical and subcortical contributions to stop signal response inhibition: role of the subthalamic nucleus. *J Neurosci* 26:2424
- Aron AR, Fletcher PC, Bullmore ET, Sahakian BJ, Robbins TW (2003) Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nat Neurosci* 6(2):115–116

- Aron AR, Monsell S, Sahakian BJ, Robbins TW (2004a) A componential analysis of task-switching deficits associated with lesions of left and right frontal cortex. *Brain* 127(7):1561–1573
- Aron AR, Robbins TW, Poldrack RA (2004b) Inhibition and the right inferior frontal cortex. *Trends Cogn Sci* 8(4):170–177
- Aron AR, Robbins TW, Poldrack RA (2014) Inhibition and the right inferior frontal cortex: one decade on. *Trends Cogn Sci* 18(4):177–185
- Berkman ET, Kahn LE, Merchant JS (2014) Training-induced changes in inhibitory control network activity. *J Neurosci* 34:149–157
- Blasi G, Goldberg TE, Weickert T, Das S, Kohn P, Zolnick B, Bertolino A, Callicott JH, Weinberger DR, Mattay VS (2006) Brain regions underlying response inhibition and interference monitoring and suppression. *Eur J Neurosci* 23:1658–1664
- Botvinick M, Nystrom LE, Fissell K, Carter CS, Cohen JD (1999) Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature* 402(6758):179–181
- Botvinick MM, Braver TS, Barch DM, Carter CS, Cohen JD (2001) Conflict monitoring and cognitive control. *Psychol Rev* 108:624–652
- Botvinick MM, Cohen JD, Carter CS (2004) Conflict monitoring and anterior cingulate cortex: an update. *Trends Cogn Sci* 8(12):539–546
- Bowley C, Faricy C, Hegarty B, Johnstone SJ, Smith JL, Kelly PJ, Rushby JA (2013) The effects of inhibitory control training on alcohol consumption, implicit alcohol-related cognitions and brain electrical activity. *Int J Psychophysiol* 89:342–348
- Bunge SA, Dudukovic NM, Thomason ME, Vaidya CJ, Gabrieli JD (2002) Immature frontal lobe contributions to cognitive control in children: evidence from fMRI. *Neuron* 33:301–311
- Bürki CN, Ludwig C, Chicherio C, De Ribaupierre A (2014) Individual differences in cognitive plasticity: an investigation of training curves in younger and older adults. *Psychol Res* 78(6):821–835
- Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD (1998) Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science* 280(5364):747–749
- Chamberlain SR, Hampshire A, Müller U, Rubia K, Campo ND, Craig K, Grant JE (2009) Atomoxetine modulates right inferior frontal activation during inhibitory control: a pharmacological functional magnetic resonance imaging study. *Biol Psychiatry* 65(7):550–555
- Chambers CD, Garavan H, Bellgrove MA (2009) Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neurosci Biobehav Rev* 33:631–646
- Chavan CF, Mouthon M, Draganski B, Van der Zwaag W, Spierer L (2015) Differential patterns of functional and structural plasticity within and between inferior frontal gyri support training-induced improvements in inhibitory control proficiency. *Hum Brain Mapp* 36:2527–2543
- Chavan C, Mouthon M, Simonet M, Hoogewoud HM, Draganski B, Van der Zwaag W et al (2017) Sustained enhancements in inhibitory control depend primarily on the reinforcement of fronto-basal anatomical connectivity. *Brain Struct Funct* 222(1):635–643
- Chein JM, Schneider W (2005) Neuroimaging studies of practice-related change: fMRI and meta-analytic evidence of a domain-general control network for learning. *Cogn Brain Res* 25(3):607–623
- Chikazoe J (2010) Localizing performance of go/no-go tasks to prefrontal cortical subregions. *Curr Opin Psychiatry* 23:267–272
- Cohen JR, Poldrack RA (2008) Automaticity in motor sequence learning does not impair response inhibition. *Psychon B Rev* 15(1):108–115
- Congdon E, Mumford JA, Cohen JR, Galvan A, Aron AR, Xue G et al (2010) Engagement of large-scale networks is related to individual differences in inhibitory control. *Neuroimage* 53:653–663
- Crottaz-Herbette S, Menon V (2006) Where and when the anterior cingulate cortex modulates attentional response: combined fMRI and ERP evidence. *J Cogn Neurosci* 18:766–780
- de Bourbon-Teles J, Bentley P, Koshino S, Shah K, Dutta A, Malhotra P, Egner T, Husain M, Soto D (2014) Thalamic control of human attention driven by memory and learning. *Curr Biol* 24(9):993–999
- Diamond A (2013) Executive functions. *Annu Rev Psychol* 64:135–168
- Duvernois S, Motamedinia S, Rugg MD (2009) The relationship between aging, performance, and the neural correlates of successful memory encoding. *Cereb Cortex* 19(3):733–744
- Edwards BG, Barch DM, Braver TS (2010) Improving prefrontal cortex function in schizophrenia through focused training of cognitive control. *Front Hum Neurosci* 4:32
- Egner T, Hirsch J (2005) The neural correlates and functional integration of cognitive control in a Stroop task. *Neuroimage* 24:539–547
- Engel S, Behnke A, Fleischhauer M, Küttler L, Kliegel M, Strobel A (2014) No evidence for true training and transfer effects after inhibitory control training in young healthy adults. *J Exp Psychol Learn Mem Cogn* 40(4):987–1001
- Fan J, Flombaum JI, Mccandliss BD, Thomas KM, Posner MI (2003) Cognitive and brain consequences of conflict. *Neuroimage* 18:42–57
- Garavan H, Ross T, Stein E (1999) Right hemispheric dominance of inhibitory control: an event-related functional MRI study. *Proc Natl Acad Sci* 96(14):8301–8306
- Garavan H, Hester R, Murphy K, Fassbender C, Kelly C (2006) Individual differences in the functional neuroanatomy of inhibitory control. *Brain Res* 1105:130–142
- Gehring WJ, Knight RT (2000) Prefrontal–cingulate interactions in action monitoring. *Nat Neurosci* 3(5):516–520
- Haber SN, Calzavara R (2009) The cortico-basal ganglia integrative network: the role of the thalamus. *Brain Res Bull* 78(2–3):69–74
- Hartmann L, Sallard E, Spierer L (2016) Enhancing frontal top-down inhibitory control with Go/NoGo training. *Brain Struct Funct* 221(7):3835–3842
- Heilbronner SR, Hayden BY (2016) Dorsal anterior cingulate cortex: a bottom-up view. *Annu Rev Neurosci* 39:149–170
- Hikosaka O, Isoda M (2010) Switching from automatic to controlled behavior: cortico-basal ganglia mechanisms. *Trends Cogn Sci* 14(4):154–161
- Hung Y, Gaillard SL, Yarmak P, Arsalidou M (2018) Dissociations of cognitive inhibition, response inhibition, and emotional interference: voxelwise ALE meta-analyses of fMRI studies. *Hum Brain Mapp* 39(10):4065–4082
- Kerns JG, Cohen JD, Cho RY, Stenger VA, Carter CS (2004) Anterior cingulate conflict monitoring and adjustments in control. *Science* 303:1023–1026
- Kerns JG, Cohen JD, Rd MDA, Johnson MK, Stenger VA, Aizenstein H et al (2005) Decreased conflict- and error-related activity in the anterior cingulate cortex in subjects with schizophrenia. *Am J Psychiatry* 162(10):1833–1839
- Lenartowicz A, Verbruggen F, Logan GD, Poldrack RA (2011) Inhibition-related activation in the right inferior frontal gyrus in the absence of inhibitory cues. *J Cogn Neurosci* 23:3388–3399
- Li CSR, Huang C, Constable RT, Sinha R (2006) Gender differences in the neural correlates of response inhibition during a stop signal task. *Neuroimage* 32:1918–1929
- Liu X, Banich MT, Jacobson BL, Tanabe JL (2004) Common and distinct neural substrates of attentional control in an integrated Simon and spatial Stroop task as assessed by event-related fMRI. *Neuroimage* 22(3):1097–1106
- Liu H, Rossi S, Zhou H, Chen B (2014) Electrophysiological evidence for domain-general inhibitory control during bilingual language switching. *PLoS ONE* 9(10):e110887

- Liu Q, Zhu X, Ziegler A, Shi J (2015) The effects of inhibitory control training for preschoolers on reasoning ability and neural activity. *Sci Rep* 5:14200
- Manuel AL, Bernasconi F, Spierer L (2013) Plastic modifications within inhibitory control networks induced by practicing a stop-signal task: an electrical neuroimaging study. *Cortex* 49(4):1141–1147
- Minzenberg MJ, Laird AR, Thelen S, Carter CS, Glahn DC (2009) Meta-analysis of 41 functional neuroimaging studies of executive function in schizophrenia. *Arch Gen Psychiatry* 66(8):811–822
- Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD (2000) The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: a latent variable analysis. *Cogn Psychol* 41(1):49–100
- Nee DE, Wager TD, Jonides J (2007) Interference resolution: insights from a meta-analysis of neuroimaging tasks. *Cogn Affect Behav Neurosci* 7(1):1–17
- Niendam TA, Laird AR, Ray KL, Dean YM, Glahn DC, Carter CS (2012) Meta-analytic evidence for a superordinate cognitive control network subserving diverse executive functions. *Cogn Affect Behav Neurosci* 12(2):241–268
- Nigg JT (2000) On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and a working inhibition taxonomy. *Psychol Bull* 126:220–246
- Paxton JL, Barch DM, Storandt M, Braver TS (2006) Effects of environmental support and strategy training on older adults’ use of context. *Psychol Aging* 21(3):499
- Peterson BS, Kane MJ, Alexander GM, Lacadie C, Skudlarski P, Leung H-C, May J, Gore JC (2002) An event-related functional MRI study comparing interference effects in the Simon and Stroop tasks. *Cogn Brain Res* 13(3):427–440
- Poldrack RA (2000) Imaging brain plasticity: conceptual and methodological issues—a theoretical review. *Neuroimage* 12:1–13
- Rubia K, Smith AB, Woolley J, Nosarti C, Heyman I, Taylor E et al (2006) Progressive increase of frontostriatal brain activation from childhood to adulthood during event-related tasks of cognitive control. *Hum Brain Mapp* 27(12):973–993
- Rubia K, Halari R, Cubillo A, Smith AB, Mohammad A-M, Brammer M, Taylor E (2011) Methylphenidate normalizes fronto-striatal underactivation during interference inhibition in medication-naïve boys with attention-deficit hyperactivity disorder. *Neuropsychopharmacology* 36(8):1575–1586
- Sebastian A, Baldemann C, Feige B, Katzev M, Scheller E, Hellwig B et al (2013) Differential effects of age on subcomponents of response inhibition. *Neurobiol Aging* 34(9):2183–2193
- Sebastian A, Rössler K, Wibral M, Mobascher A, Lieb K, Jung P, Tüscher O (2017) Neural architecture of selective stopping strategies: distinct brain activity patterns are associated with attentional capture but not with outright stopping. *J Neurosci* 37(40):9785–9794
- Sharp DJ, Bonnelle V, De Boissezon X, Beckmann CF, James SG, Patel MC, Mehta MA (2010) Distinct frontal systems for response inhibition, attentional capture, and error processing. *Proc Natl Acad Sci* 107(13):6106–6111
- Spierer L, Chavan CF, Manuel AL (2013) Training-induced behavioral and brain plasticity in inhibitory control. *Front Hum Neurosci* 7(2):427
- Van Boxtel GJM, Van der Molen MW, Jennings JR, Brunia CHM (2001) A psychophysiological analysis of inhibitory motor control in the stop-signal paradigm. *Biol Psychol* 58:229–262
- Wolf RC, Walter H (2005) Evaluation of a novel event-related parametric fMRI paradigm investigating prefrontal function. *Psychiatry Res* 140(1):73–83

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