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The role of pre-supplementary motor cortex in action control with emotional stimuli: A repetitive transcranial magnetic stimulation study

Simone Battaglia | **Claudio Nazzi** | **Chiara Di Fazio** | **Sara Borgomaneri**

Center for Studies and Research in Cognitive Neuroscience, Department of Psychology "Renzo Canestrari", Cesena Campus, Alma Mater Studiorum Università di Bologna, Cesena, Italy

Correspondence
Simone Battaglia and Sara Borgomaneri, Department of Psychology "Renzo Canestrari", University of Bologna, Viale Berti Pichat 5, Bologna 40127, Italy.
Email: simone.battaglia@unibo.it and sara.borgomaneri@unibo.it

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Abstract

Swiftly halting ongoing motor actions is essential to react to unforeseen and potentially perilous circumstances. However, the neural bases subtending the complex interplay between emotions and motor control have been scarcely investigated. Here, we used an emotional stop signal task (SST) to investigate whether specific neural circuits engaged by action suppression are differently modulated by emotional signals with respect to neutral ones. Participants performed an SST before and after the administration of one session of repetitive transcranial magnetic stimulation (rTMS) over the pre-supplementary motor cortex (pre-SMA), the right inferior frontal gyrus (rIFG), and the left primary motor cortex (lM1). Results show that rTMS over the pre-SMA improved the ability to inhibit prepotent action (i.e., better action control) when emotional stimuli were presented. In contrast, action control in a neutral context was fostered by rTMS over the rIFG. No changes were observed after lM1 stimulation. Intriguingly, individuals with higher impulsivity traits exhibited enhanced motor control when facing neutral stimuli following rIFG stimulation. These results further our understanding of the interplay between emotions and motor functions, shedding light on the selective modulation of neural pathways underpinning these processes.

KEYWORDS
action inhibition, emotion, inferior frontal gyrus, pre-supplementary motor cortex, stop signal task, transcranial magnetic stimulation

INTRODUCTION

The capacity to restrain pre-established ongoing actions is essential to avert possible detrimental behavioral consequences. Achieving this involves incorporating pertinent signals into neural processes to adapt or halt them before completion. The capacity to restrain prepotent responses is assessable through experimental inquiry using a stop signal task (SST). Tailored to offer a precise gauge, this task enables the examination of the duration it takes for the brain to curb unsuitable motor responses.[1–3] Operationally, participants are directed to initiate a response to a go stimulus and subsequently swiftly withhold their ongoing response when they encounter a stop signal. The stop signal reaction time (SSRT), as conceptualized by Logan and Cowan,[4] serves as an estimation of reactive action inhibition performance. It quantifies the duration of the inhibitory process, revealing the time needed for effective motor inhibition (i.e., longer SSRT indicates worse inhibitory performance).[4] Compared to proactive inhibition, which refers to preparatory processes that result in a response being

withheld before it is initiated, reactive inhibition refers to the cessation of a motor response that is already in progress.[5]

Effective response inhibition is currently understood to primarily rely on the functioning of several areas within the action inhibition network (AIN), which comprises the left and right inferior frontal gyrus (IFG),[6–11] dorsolateral prefrontal cortex,[8,12] anterior cingulate cortex (ACC),[9,13] pre-supplementary motor area (pre-SMA)[8,12,14,15] and other motor-related areas,[16–18] bilateral superior temporal gyri,[9] parietal cortex,[8,9,12] insula,[8,9,12] the subthalamic nucleus (STN),[19–22] basal ganglia,[7,9,12] and the cerebellum[23]—all of which have been implicated through a range of studies (see Ref. 9 for a meta-analysis). Critically, the above-mentioned functional magnetic resonance (fMRI) studies lack the ability to establish a causal link between behavioral performance and neural activation, while brain-injured patient investigations[6,24–30] do not take into consideration possible brain plasticity mechanisms. Hence, the available evidence limits the possibility to make definitive causal conclusions regarding the distinct participation of individual brain nodes within the AIN in a variety of aspects related to action control.

A potential solution to this challenge involves employing invasive (i.e., deep brain stimulation [DBS]) or noninvasive brain stimulation (NIBS) techniques to selectively modify specific cortical components of the AIN, thereby enabling an exploration of their role in inhibitory control. For instance, DBS studies have shown a crucial role of the STN in action control (for a review, see Ref. 31), and NIBS studies report that an impairment in action inhibition can occur due to temporary interference of the IFG and the pre-SMA[30,32–35] (for a comprehensive review of the NIBS studies on the topic, see Ref. 36). Although sometimes controversial due to different NIBS protocols and methodological differences in the SST paradigms, the existent findings suggest that both the pre-SMA and IFG (especially the right-IFG [rIFG]) may play a crucial role in the control of actions. Interestingly, emotions elicited by the perception of arousing/threatening stimuli are likely to impact several cognitive functions,[37–39,138] including action inhibition. In support of this notion, the observation of threatening stimuli was found to influence early motor activity[40–47] and motor behavior across a variety of tasks.[48–56] Indeed, several SST studies have demonstrated that emotions can influence action inhibition. However, they reported both the enhancement and impairment of action control (i.e., increase or decrease in SSRT) by emotions (for a review, see Ref. 57). Additionally, some evidence shows no influence of emotional stimuli in inhibitory performance.[56,58,59] One aspect that may contribute to the contrasting results is the different roles of the emotional stimulus (e.g., presented as stop signal, go signal, or presented before the go signal as a *prime*), but also the relevance of the emotional stimulus (i.e., task-relevant [requiring the explicit discrimination of the emotional stimuli][50,60] or task-irrelevant [not requiring emotion discrimination][61–63]). Therefore, a great deal of confusion characterizes our understanding of the interplay between emotions and action control. Furthermore, the neural network subtending the interaction between emotion and action control has been scarcely investigated.

Multiple pieces of evidence substantiate the presence of anatomical and functional links between the limbic cortex and motor/premotor regions.[64,65] This neural network, with a special focus on the pre-SMA, could potentially serve as the neural pathway by which emotions engage and influence the process of motor planning. In line with this hypothesis, several studies have consistently reported pre-SMA activation in different emotion-related processes, such as the observation of emotional facial[66–68] and body expressions[69] or vocal/speech emotion recognition.[70] Moreover, the pre-SMA activity was found to be critical for facial emotional expression recognition,[71,72] and its electrical stimulation was found to induce emotion-related behaviors.[73,74] From all this evidence, it is reasonable to hypothesize that the pre-SMA plays a role in inhibiting inappropriate motor responses, even more so when emotional stimuli are presented as stops. The only existent fMRI study that aimed at investigating the neural bases of action inhibition using the SST with emotional stimuli[56] reported that emotional cues during stop trials interacted with activity in limbic regions, as well as with the pre-SMA, while the IFG seemed to be involved in action inhibition but not directly involved when emotional stimuli were presented as stops. This neuroimaging evidence is in line with the idea that the SMA-complex, via direct afferents from the amygdaloid complex,[75] could be the cortical hub interfacing the limbic and the motor systems,[56,76] while the IFG may be responsible for motor inhibition with neutral or abstract stimuli.[6,56,77,78]

However, to date, no NIBS study has been conducted to test the critical role of these two frontal nodes of the AIN when action inhibition needs to be performed within an emotional context (i.e., when emotional stimuli are presented as stop signals). Herein, to investigate this issue, we performed an emotional SST with emotional and neutral stimuli serving as stop stimuli, and by using repetitive transcranial magnetic stimulation (rTMS), we interfered with the activity of the pre-SMA or rIFG. We interfered with the activity of the left primary motor cortex (lM1) as an active control site, as was done by Hsu and colleagues[33] and Kwon and Kwon.[79] Although M1 has been found to be involved in inhibitory control,[17,18] SST investigations using NIBS have reported null findings;[33,79] thus the crucial role of M1 in action suppression is still under debate.

In line with previous evidence, we hypothesize that interfering with the activity of the pre-SMA may impact the selective ability to suppress prepotent actions in an emotional context, while targeting the rIFG may influence action control with neutral stimuli. This would be in line with the idea that threat perception may influence brain systems involved in motor control in humans through partially overlapping but also partially different pathways than those mediating voluntary inhibition. Finally, it has been found that personality traits like impulsivity[12,80–82] may impact the ability to suppress an ongoing action, even when emotional stimuli are presented[83] (but see Ref. 84 on the complex relationship between impulsivity and inhibitory control). We, therefore, tested whether impulsivity may influence the effect of the rTMS over the different nodes of the AIN during action control with emotional stimuli.

TABLE 1 Demographic data.

Group	Age	Education	Gender
pre-SMA	24.27 ± 2.21	15.23 ± 1.66	F = 13; M = 9
rIFG	24.68 ± 3.47	15.50 ± 1.92	F = 10; M = 12
lM1	24.73 ± 2.76	15.73 ± 1.49	F = 9; M = 13

Note: Age and education are reported as mean ± standard deviation, expressed in years for all groups. Gender is reported as the number of female and male participants.

MATERIALS AND METHODS

Participants

A total of 66 right-handed healthy volunteer adults participated in the present study. All participants were recruited using a snowball sampling approach via social media, mailing lists, and a general academic campaign. Prior to participation, subjects declared that they had no history of neurological or psychiatric disorders, and none of the participants was regularly taking any medication affecting the central nervous system. All participants had normal or corrected-to-normal vision. To test the hypothesis, participants were randomly divided into three groups: the pre-SMA and rIFG groups acted as experimental groups, while the lM1 group served as the active control group. In particular, 22 participants were randomly assigned to the pre-SMA group, 22 to the rIFG group, and 22 to the lM1 group. The number of participants was determined based on a power analysis, which indicated that a sample size of ∼22 participants is necessary to achieve a statistical power (1-β) of 0.95 (two-tailed $\alpha = 0.05$; effect size $f = 0.47$;[61,85–88] number of measurements = 2; correlation = 0.5; analysis performed with G∗Power software[89]). Finally, groups were matched for age ($F(2,63) = 0.17$; $p = 0.85$; $\eta_p^2 = 0.01$), years of education ($F(2,63) = 0.48$; $p = 0.62$; $\eta_p^2 = 0.02$), and gender (χ^2 (2, $N = 66$) = 1.57; $p = 0.45$; see Figure S1 and Table 1 for further demographic data).

In addition, different personality traits of the participants were investigated as previous studies have shown that SST performance, as well as reactive action inhibition, may be influenced by psychological or psychiatric conditions (i.e., anxiety, depression, impulsivity, addiction).[57,61,90] Subjective levels of anxiety were measured through the State-Trait Anxiety Inventory (STAI; Trait-scale-Y2),[91] and subjective levels of impulsivity were measured by the Barratt Impulsiveness Scale-11 (BIS-11).[92] The STAI-Y2 consists of a 20-item self-report questionnaire providing an assessment of anxiety and evaluates how often respondents experience anxiety. The BIS-11 is a questionnaire designed to assess the personality construct of impulsiveness. It is composed of 30 items assessing common impulsive or nonimpulsive behaviors. Finally, we administered the Hospital Anxiety and Depression Scale (HADS)[93] to exclude participants with high levels of anxiety and depression from our sample. The HADS is a 14-item questionnaire designed to assess the levels of anxiety and depression that a person is experiencing. It consists of seven questions for anxiety and seven for depression. The three groups did not show any significant difference in terms of anxiety (STAI-Y2: $F(2,63) = 2.19$, $p = 0.12$, $\eta_p^2 = 0.07$; HADS-anxiety: $F(2,63) = 2.12$, $p = 0.13$, $\eta_p^2 = 0.06$), HADS-depression ($F(2,63) = 0.99$, $p = 0.38$, $\eta_p^2 = 0.03$), and BIS-impulsivity ($F(2,63) = 0.03$, $p = 0.97$, $\eta_p^2 < 0.01$) scores (Figure S1; see Table 2 for further details). Importantly, data collection was anonymous, and all participants gave their informed consent electronically through our online platform before the task. Data were hosted and stored on a private server and were password-protected and accessible only by the corresponding author. The study was conducted in accordance with the ethical principles of the World Medical Association Declaration of Helsinki and was approved by the Bioethical Committee of the University of Bologna.

Experimental procedure

After their arrival, participants were required to provide written informed consent before starting any experimental procedures. The main experiment involved three consecutive phases for each participant. Participants were seated in a quiet room ensuring that their position was centered relative to the screen and maintaining a viewing distance of 50 cm from the screen. At this point, the experimenter provided participants with an overview of the control inhibition task (e.g., SST). This initial phase aimed to establish a performance baseline, assessing the inhibitory response capacity elicited by the task for each participant. Subsequently, during the neurostimulation phase, the resting motor threshold (rMT) was determined for each participant, followed by precise localization of the stimulation target on the scalp using a neuronavigation system with stereotaxic coordinates (see TMS paragraphs below for detailed procedures) and, finally, rTMS was performed. Immediately afterward, in the final phase of the experiment, the same SST task was readministered to evaluate the effect of neurostimulation on the ability to control the inhibitory response contingent upon the specific brain area that had been stimulated. Lastly, participants were asked to complete the personality traits questionnaires.

Stop signal task

The SST was implemented in MATLAB (version R2018b; The MathWorks, Inc.) on a Windows-based PC (Lenovo ThinkCentre Desktop Computer), and stimuli presentation was controlled by PsychToolbox.[94] In particular, the SST consisted of a simple reaction time (RT) task, which included both go and stop trials.[1,4,95,96] Go stimuli consisted of the presentation of a black arrow pointing left or right,

TABLE 2 Questionnaires data.

Group	STAI-Y2	HADS-anxiety	HADS-depression	BIS-11			
				Total score	Motoric impulsivity	Attentional impulsivity	Nonplanning impulsivity
pre-SMA	46.86 ± 9.13	8.68 ± 3.87	4.14 ± 2.85	65.45 ± 8.61	17.00 ± 3.75	20.45 ± 4.45	28.00 ± 3.72
rIFG	46.95 ± 8.91	7.73 ± 3.79	4.00 ± 2.85	64.91 ± 7.26	16.91 ± 3.16	20.05 ± 4.01	27.95 ± 2.97
IM1	41.91 ± 9.40	6.32 ± 3.82	3.09 ± 2.29	65.23 ± 8.27	16.82 ± 3.74	21.45 ± 3.66	26.95 ± 3.53

Note: Scores are reported as mean ± standard deviations.
Abbreviations: BIS-11, Barratt Impulsiveness Scale-11; HADS, Hospital Anxiety and Depression Scale; STAI-Y2, State-Trait Anxiety Inventory.

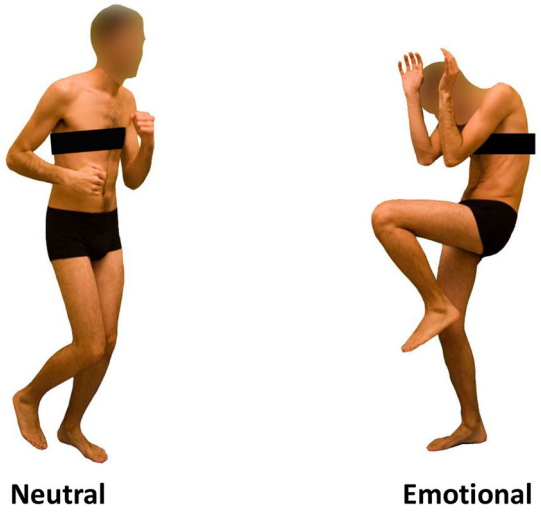

FIGURE 1 Visual stimuli used as stop signal stimuli. For all groups, stimuli consisted of two different body pictures with emotional and neutral body expression that have been previously used and validated.[41–46,134]

while the Stop stimuli could be one of two colored images of dynamic body postures (already employed in our previous work):[83,97] a fearful emotional body posture acted as an emotional stop stimulus, while an image of a dynamic (i.e., running) neutral body posture was used as a control neutral stop stimulus (see Figure 1). The use of a neutral dynamic body stimulus matched for the amount of the conveyed implied motion is fundamental to prevent factors other than the emotional content to influence our findings.

Importantly, the two different body pictures (i.e., fearful and neutral expression) were previously validated by Borgomaneri and colleagues in several studies[41,43,44,46] and have also been used as stop signals in other studies.[83,97] Stimuli were edited to have the same shape, surface, complexity, colors, and contrast ratio with Blender (Blender Foundation) and Adobe Photoshop CS6 software (Adobe).

Participants started the SST by performing a short practice block (approximately 3 min, 32 trials) to familiarize themselves with the task. Immediately afterward, they performed four experimental blocks that constituted the main task. Each block was composed of a total of 128 trials, consisting of 96 go trials (75%) and 32 stop trials (25%). Therefore, during the whole task, each participant was presented with a total of 384 go trials and 128 stop trials. In each block, the go and stop trials contained stimuli in equal proportion, that is, 96 go trials and 32 stop trials. Each trial started with the presentation of a black dot centered on a blank white screen for 400 ms (i.e., fixation point) and ended with an empty blank white screen for a random interval of between 1000 and 2000 ms, acting as an intertrial interval. In the go trials, participants had to perform a go task with their right hand by pressing the left key as fast as they could when a black arrow pointing to the left appeared, or the right key when the arrow pointed to the right. In the event of no response, the stimulus would remain on the screen for a maximum time of 750 ms. The stop trials were identical to the go trials except that a picture of a stimulus (i.e., stop signal) was presented for 100 ms after a variable stop signal delay (SSD) relative to the onset of the go stimulus (i.e., the arrow), instructing participants to suppress the imminent go response (see Figure 2). The initial value of the SSD was set to 250 ms and adjusted individually and dynamically throughout the experiment (from a minimum of 50 ms to a maximum of 750 ms), a procedure referred to as a staircase. If participants successfully inhibited their response on a stop trial, the SSD was increased by 50 ms on a subsequent stop trial, while if they failed to withhold their motor response, the SSD was reduced by 50 ms on a subsequent stop trial.[61,62,81,90,96] Importantly, the staircase was independent within-subject, as the SSD was adjusted separately for each stimulus (i.e., the staircase for one stimulus was calculated independently from the next stimulus in each participant) to ensure successful inhibition in approximately 50% of the stop trials for each stimulus.[96,98,99] Participants were instructed to respond as quickly and accurately as possible to the arrow and were asked to inhibit their response upon viewing a stimulus which followed the initial go stimulus that appeared on the screen. However, they were also instructed that sometimes it might not be possible to successfully inhibit their response and, in such cases, they should continue to perform the task irrespective of having made an error.[61,96] Furthermore, participants were asked not to hesitate or slow down to avoid increasing the chances of stopping. Overall, our task was designed based on the recommendations of Verbruggen and colleagues.[96]

Data processing and analysis

To measure the participants' performance on the SST (SSRT), an index of reactive inhibition was estimated based on Logan and Cowan's notion of the race-model.[4] SSRT is the overall latency of a chain of processes involved in stopping a response, including the detection of

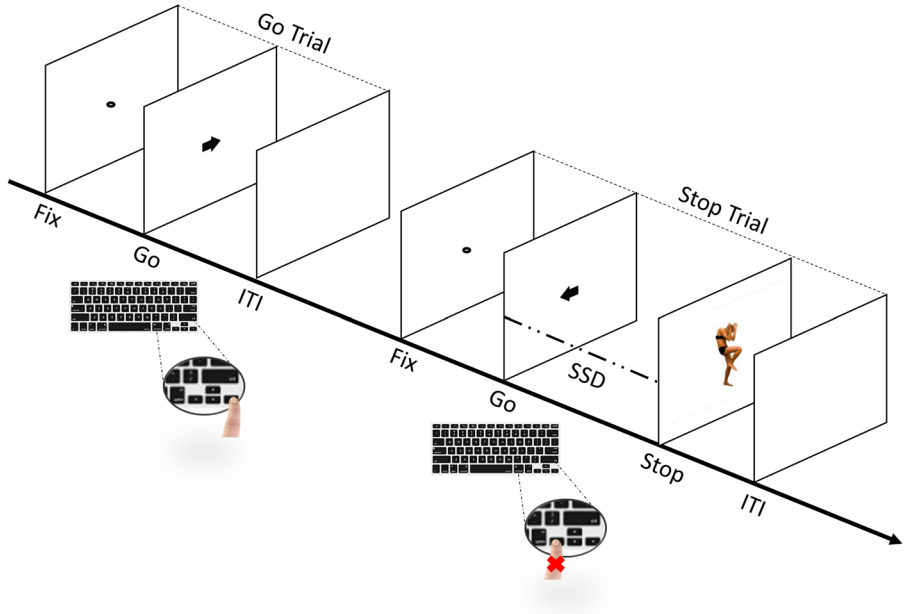

FIGURE 2 Sequence of trials in the stop signal task (SST). The experimental task includes both go and stop trials.[1,4,95,96] Participants perform a short practice block and, immediately afterward, four experimental blocks. Each block includes a total of 128 trials, of which 96 are go trials (75%) and 32 are stop trials (25%). In go trials, participants respond to the go task (i.e., the direction of the arrow that appears on the screen) by pressing the corresponding arrow key on the keyboard. In stop trials, the arrow is followed by a stop signal after a variable stop signal delay (SSD) instructing participants to suppress the imminent go response. The initial value of the SSD was set to 250 ms and adjusted individually and dynamically throughout the experiment (i.e., staircase procedure), so that if participants successfully inhibited their response on a stop trial, the SSD was increased by 50 ms in a subsequent stop trial, while if they failed to withhold their motor response, the SSD was reduced by 50 ms in a subsequent stop trial. Abbreviations: Fix, fixation duration; ITI, intertrial interval.

the stop signal. However, prior to analyzing SSRT, the reliability of the overall performance of the participants in the task was verified by calculating the inhibition rate, which must be around 50%.[95,96,98,100] Furthermore, we made sure that RTs for unsuccessful stop trials were shorter than go trial RTs.[96] Subsequently, data collected in this experiment were processed to estimate SSRTs.[96] Accordingly, data were analyzed by adopting the integration method with the replacement of go omissions. In particular, the point at which the stop process ends is estimated by integrating the RT distribution and finding the point at which the integral is equal to p(respond|signal). The ending time of the stop process corresponds to the *n*th RT, where n = the number of RTs in the RT distribution of go trials multiplied by p(respond|signal). Also, to determine the *n*th RT, all go trials with a response were considered, including go trials with a choice error and go trials with a premature response. It is important to highlight that these omissions (i.e., go trials in which participants did not respond before the end of the trial) were assigned the maximum RT to compensate for the lack of response. Moreover, premature responses in unsuccessful stop trials (i.e., responses executed before the stop signal is presented) were included in calculating p(respond|signal) and mean SSD. This version of the integration method produces the most reliable and least biased SSRT estimation (for further details and an exhaustive review, see Ref. 96).

Finally, to characterize changes in inhibitory control resulting from neurostimulation for each specific TMS-stimulated site, we created an inhibitory performance index of SSRT (ΔSSRT). Specifically, ΔSSRT were calculated for each participant by considering SSRT collected after neurostimulation minus the SSRT collected during baseline (i.e., before stimulation) separately for emotional and neutral stimuli. This approach allows us to have a direct assessment of changes in inhibitory capacity associated with the precise cortical region targeted by rTMS.

Data were analyzed offline using custom-made MATLAB scripts (The MathWorks, Inc.) estimating SSRT as described, and all statistical analyses were performed with STATISTICA (StatSoft STATISTICA 13). Mixed-design analyses of variance (ANOVAs) were used to investigate differences within and between groups. All the post-hoc analyses were conducted with the Duncan test and the significance threshold was set at $p < 0.05$. Mean percentage of the stop performance and correct responses on go trials were not normally distributed (as shown by the Shapiro–Wilk test). Thus, we further implemented nonparametric Bonferroni-corrected Friedman ANOVAs, one for each group.

Neuronavigation and transcranial magnetic stimulation

Brain target areas were identified with the EMS SofTaxic Navigator system, which automatically estimates coordinates in Talairach space from a magnetic resonance imaging-constructed stereotaxic template. Skull landmarks and ~80 points providing a uniform representation of the scalp were digitized by means of a Northern Digital Polaris Vicra

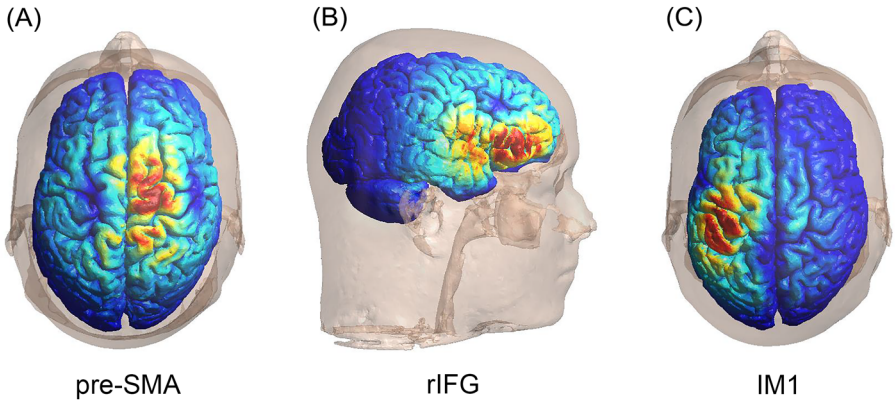

FIGURE 3 Computational simulation of the estimated electric field distribution from rTMS targeting the brain. The volumetric spread of magnetic field simulation was created using SimNIBS v4.0.1. Conductivities for different tissue compartments were set as follows: 0.465 Siemens per meter (S/m) (skin), 0.01 S/m (skull), 0.5 S/m (eyeballs), 1.654 S/m (cerebrospinal fluid), 0.275 S/m (gray matter), and 0.126 S/m (white matter). The estimation was carried out by simulating a figure-of-eight 70 mm coil with stimulation intensity set at the mean stimulation intensity of each group. The coil was placed according to the mean coordinates of each area. The estimated computational simulation showed an accurate propagation of the TMS stimulation over the chosen sites, hence supporting the rTMS setup used. Abbreviations: IM1, left motor cortex; pre-SMA, pre-supplementary motor cortex; rIFG, right inferior frontal gyrus; rTMS, repetitive transcranial magnetic stimulation; TMS, transcranial magnetic stimulation.

digitizer.[101–105] An individual estimated magnetic resonance image (MRI) was obtained for each subject through a 3D warping procedure fitting a high-resolution MRI template with the participant's scalp model and craniometric points. This procedure ensures a global localization accuracy of ~5 mm.[101] We targeted the pre-SMA and rIFG using the following Talairach coordinates, respectively: $x = 9, y = 6, z = 49$, and $x = 51, y = 15, z = 1$.[56] The targeted area IM1 was defined as the point where stimulation consistently evoked the largest motor-evoked potentials (MEPs) in the right first dorsal interosseous (FDI).[106–108]

The SofTaxic Navigator system was used to estimate the projection of the targeted scalp positions on the brain surface, confirming correct coil placement for all of the sites.[101–105] The estimated Talairach coordinates for the IM1 (i.e., the FDI optimal scalp position) were (mean ± SD): $x = -43.9 \pm 6.4$, $y = -23.1 \pm 12$, $z = 50.2 \pm 6.78$; the brain surface Talairach coordinates for the pre-SMA were: $x = 9.2 \pm 1.9$, $y = 4.6 \pm 4.6$, $z = 64 \pm 3.1$; and the rIFG coordinates were: $x = 49.4 \pm 2.5$, $y = 15.1 \pm 2.6$, $z = 2.45 \pm 6.7$.

Finally, TMS was applied with a Magstim super rapid[2] magnetic stimulator and a figure-of-eight coil with an outer winding diameter of 70 mm (Magstim Company Limited). We set rTMS intensity at 110% of the rMT (see the next paragraph) and applied a single train of low-frequency rTMS at 1 Hz for a total duration of 20 min (1200 pulses), a protocol that suppresses cortical excitability beyond the duration of the rTMS application itself.[109] Furthermore, SimNIBS v4.0.1[110] was used to estimate the electric field distribution induced by TMS and for automatic skull segmentation from MR images[111] (Figure 3).

Resting motor threshold

After participants had completed the first phase of the experiment, the intensity of the rTMS protocol was determined by assessing the individual rMT.[112] We placed the coil tangentially to the scalp on the region overlying the left motor cortex with the coil handle pointing backward and laterally at a 45° angle away from the midline. The left motor cortex was stimulated in line with previous noninvasive brain stimulation studies[33] because the SST task was performed with the right hand. Using a suprathreshold pulse intensity (approximately 120%–130% of the rMT[113]), the coil was moved over the scalp to determine the optimal position from which maximal MEP amplitudes could be elicited in the contralateral FDI muscle—corresponding to the hand area in the motor cortex. From that position, we assessed the rMT, which was defined as the minimal intensity of the stimulator output that produces MEPs with amplitudes of at least 50 millivolts (mV) with 50% probability.[114] A one-way ANOVA on rMT intensity showed no significant effect of group ($F(2,63) = 0.53$; $p = 0.59$; $\eta_p^2 = 0.02$; pre-SMA: mean ± SD: 60.23 ± 12.96; rIFG: 57.1 ± 8.22; IM1: 59.09 ± 8.29; Figure S1).

RESULTS

Verification of the correct assumptions underlying the SST data collected

First, we verified the correct assumptions of the independent race model.[96] In particular, we assessed whether the mean RT on unsuccessful stop trials (i.e., trials in which participants could not desist from performing an action even though a stop signal was presented) was shorter than the mean RT for go trials. In particular, we performed a 3×2×3 ANOVA on RT with Trial type (Go/Unsuccessful emotional/Unsuccessful neutral) and Session (Pre/Post) as within-subjects factors and Group (pre-SMA/rIFG/IM1) as a between-subject factor. The analysis revealed a significant effect of Trial type ($F(2,126) = 399.49, p < 0.01, \eta_p^2 = 0.86$). Post-hoc analyses highlighted that RTs

TABLE 3 Behavioral data for SST administered pre and post TMS.

	pre-SMA		rIFG		lM1	
SST pre-TMS	**Negative**	**Neutral**	**Negative**	**Neutral**	**Negative**	**Neutral**
Inhibition Rate (%)	49.93 ± 1.77	49.79 ± 1.69	50.36 ± 2.59	50.21 ± 3.17	49.36 ± 1.15	49.50 ± 1.70
SSD (ms)	241.37 ± 74.42	244.64 ± 79.42	288.85 ± 76.96	287.68 ± 79.92	230.26 ± 72.24	232.03 ± 74.21
SSRT (ms)	238.76 ± 21.21	236.36 ± 25.72	222.14 ± 28.02	223.45 ± 28.06	237.13 ± 36.71	234.79 ± 35.88
Unsucc RT (ms)	435.85 ± 59.76	438.75 ± 66.61	453.99 ± 82.86	448.48 ± 84.32	420.95 ± 61.55	419.91 ± 62.40
Go RT (ms)	487.50 ± 71.60		513.87 ± 87.21		468.85 ± 72.91	
Correct Go (%)	94.60 ± 5.23		91.50 ± 5.37		96.06 ± 3.69	
	pre-SMA		**rIFG**		**lM1**	
SST post-TMS	**Negative**	**Neutral**	**Negative**	**Neutral**	**Negative**	**Neutral**
Inhibition Rate (%)	49.50 ± 2.61	49.15 ± 2.14	50.21 ± 1.76	50.85 ± 1.65	49.57 ± 1.68	49.22 ± 1.34
SSD (ms)	235.65 ± 66.15	225.89 ± 60.58	293.64 ± 86.24	307.63 ± 87.74	223.37 ± 69.49	228.91 ± 73.47
SSRT (ms)	231.71 ± 24.08	242.71 ± 21.21	224.30 ± 25.84	209.60 ± 29.69	237.58 ± 35.49	232.91 ± 39.68
Unsucc RT (ms)	426.89 ± 57.24	423.53 ± 53.88	463.08 ± 85.05	473.42 ± 87.48	413.74 ± 64.71	416.48 ± 64.04
Go RT (ms)	471.65 ± 65.21		516.88 ± 85.97		463.07 ± 73.08	
Correct Go (%)	96.86 ± 2.50		93.61 ± 4.51		97.37 ± 2.75	

Note: Descriptive performance of the stop signal task (SST) is reported as means ± standard deviations. In particular, Inhibition Rate, Stop Signal Delay (SSD), Stop Signal Reaction Time (SSRT), Unsuccessful Reaction Time (Unsucc RT), Go Reaction Time (Go RT), and Correct Go responses are depicted in the table for each group. Abbreviations: lM1, left primary motor cortex; pre-SMA, pre-supplementary motor cortex; rIFG, right inferior frontal gyrus; TMS, transcranial magnetic stimulation.

for go trials (Mean ± SD = 486.97 ± 77.96) were significantly longer than RTs for both unsuccessful emotional (435.59 ± 70.35, $p < 0.01$, $d = 0.69$) and unsuccessful neutral trials (436.76 ± 72.26, $p < 0.01$, $d = 0.69$). The interaction between Trial type by Session was also significant ($F(2,126) = 5.87$, $p < 0.01$, $\eta_p^2 = 0.09$). Subsequent post-hoc analyses revealed that RTs for go trials were significantly higher than both unsuccessful emotional and neutral trials, and in both sessions. Additionally, RTs for go trials were faster after the stimulation (Pre Go: 490.07 ± 78.59, Unsuccessful emotional: 436.60 ± 69.16, Unsuccessful neutral: 435.71 ± 71.64; Post Go: 483.87 ± 77.80, Unsuccessful emotional: 434.57 ± 72.05, Unsuccessful neutral: 437.82 ± 73.40; all $ps < 0.01$, all $ds \geq 0.61$). Moreover, the analysis revealed a significant interaction between Trial type, Session, and Group ($F(4,126) = 5.63$, $p < 0.01$, $\eta_p^2 = 0.15$). Post-hoc analyses showed that, across all groups, RTs for go trials were significantly higher than both unsuccessful emotional and neutral trials, and in both sessions (all $ps < 0.01$, all $ds \geq 0.50$; see Table 3 for descriptive data). The three-way interaction is referred to as nonrelevant comparisons. Hence, we can conclude that the assumption that RTs for unsuccessful stop trials were lower than RTs for go trials has been verified and was true for all groups and sessions.

Subsequently, we ensured that the staircase procedure was successful, ascertaining that the inhibition rate (i.e., percentage of stop performance when a stop signal is presented) was approximately 50% for all stimuli during both sessions (see Table 3 for descriptive SST data). To investigate differences across groups, a 2×2×3 ANOVA on the percentage of the stop performance (i.e., inhibition rate) was carried out, with Stimulus (Emotional/Neutral) and Session (Pre/Post) as within-subject factors, and Group (pre-SMA/rIFG/IM1) as a between-subject factor. The analysis revealed that the inhibition rate did not differ between Groups ($F(2,63) = 2.11$, $p = 0.13$, $\eta_p^2 = 0.01$), nor was it influenced by the emotional content of the Stimulus ($F(1,63) = 0.11$, $p = 0.74$, $\eta_p^2 < 0.01$) or by the Sessions ($F(1,63) = 0.28$, $p = 0.61$, $\eta_p^2 < 0.01$). Moreover, no interactions were found to be significant (all $F(2,63) \leq 2.31$, $p \geq 0.11$, $\eta_p^2 \geq 0.04$). The Friedman ANOVAs on the percentage of the stop performance (i.e., inhibition rate) were not significant (all $\chi^2 \leq 4.77$, $p \geq 0.57$). These results indicated that the percentage of the stop performance, when the stop signal is presented, was comparable for the two stimuli in all groups.

Likewise, we investigated the percentage of correct responses on go trials across groups using a 2×3 ANOVA, with Session (Pre/Post) as a within-subject factor and Group (pre-SMA/rIFG/IM1) as a between-

subject factor. The analysis revealed a main effect of Session ($F(1,63) = 15.70$, $p < 0.01$, $\eta_p^2 = 0.15$), but no Session by Group interaction ($F(2,63) = 0.38$, $p = 0.68$, $\eta_p^2 = 0.01$), suggesting that all participants regardless of the group had a similar correct performance in discriminating the direction of the arrow presented as the go signal. The main effect of Session revealed that correct go responses were significantly higher overall after neurostimulation (see Table 3 for descriptive SST data), suggesting a generic improvement attributable to learning. The Friedman ANOVA on the percentage of correct responses on go trials confirmed the improvement for the lM1 group ($\chi^2 = 8.05$, $p = 0.01$) and for the IFG and pre-SMA groups, although in these groups we noticed only a nonsignificant trend (all $\chi^2 \leq 4.55$, $p \geq 0.09$).

In addition, to assess the sequential effects on RTs following go trials, a 2×3 ANOVA on the go RTs was performed, with Session (Pre/Post) as a within-subject factor and Group (pre-SMA/rIFG/lM1) as a between-subject factor. No differences in RTs were found for Group ($F(2,63) = 2.69$, $p = 0.08$, $\eta_p^2 = 0.08$), Session ($F(1,63) = 1.23$, $p = 0.27$, $\eta_p^2 = 0.02$), and Session by Group interaction ($F(2,63) = 0.95$, $p = 0.39$, $\eta_p^2 = 0.03$). In conclusion, given these analyses, the SST data collected in both experimental sessions can be considered reliable and the assumption of correct inhibition rate has been verified. Thus, it is possible to reliably estimate the SSRT values.[96]

Neurostimulation and emotional content of stimuli specifically change the ability to suppress ongoing actions

Before the main analysis, it was verified that the absence of SSRT differences among groups prior to neurostimulation using a 2×3 ANOVA, with Stimulus (Emotional/Neutral) and Group (pre-SMA/rIFG/lM1) as between-subject factors. The analysis revealed that the SSRT did not differ for Group ($F(2,63) = 1.79$, $p = 0.17$, $\eta_p^2 = 0.05$), nor was it influenced by the emotional content of the Stimulus ($F(1,63) = 0.26$, $p = 0.60$, $\eta_p^2 < 0.01$). Moreover, the interaction was not found to be significant ($F(2,63) = 0.31$, $p = 0.73$, $\eta_p^2 = 0.01$).

To verify the main hypothesis of the present study, SSRT data were analyzed using a 2×2×3 ANOVA with Stimulus (Emotional/Neutral) and Session (Pre/Post) as within-subject factors, and Group (pre-SMA/rIFG/lM1) as between-subject factor. The analysis revealed a Stimulus by Group interaction ($F(2,63) = 4.40$, $p = 0.02$, $\eta_p^2 = 0.12$), but crucially revealed a significant Stimulus by Session by Group interaction ($F(2,63) = 8.15$, $p < 0.01$, $\eta_p^2 = 0.21$). Thus, to investigate these results more deeply, we analyzed ΔSSRT calculated for each participant by considering SSRT collected after neurostimulation minus the SSRTs collected before the neurostimulation (i.e., baseline session) for emotional and neutral stimuli separately. In particular, ΔSSRT data were analyzed using a 2×3 ANOVA with Stimulus (Emotional/Neutral) as a within-subject factor and Group (pre-SMA/rIFG/lM1) as a between-subject factor. The analysis revealed a Stimulus by Group interaction ($F(2,63) = 8.14$, $p < 0.01$, $\eta_p^2 = 0.21$). A post-hoc analysis on ΔSSRT data revealed a significant difference ($p = 0.02$, $d = 0.74$) in the pre-SMA group between the emotional and the neutral stimulus after the rTMS application. Similarly, a significant difference ($p < 0.01$, $d = 0.57$) between neutral and emotional stimuli emerged in the rIFG group (see Table 3 for descriptive SST data). To further investigate the effect of neurostimulation and the stimulus on SSRT data, we compared ΔSSRT with 0 (indicating no variation between Sessions, Pre vs. Post) using one-sample *t*-tests. These tests revealed that the pre-SMA group showed significantly reduced SSRT when presented with the emotional stop stimulus ($t(21) = -2.63$, $p = 0.03$, $d = 0.48$), while no changes in SSRT were observed when presented with the neutral stimulus ($t(21) = 1.42$, $p = 0.17$, $d = 0.30$). On the contrary, the analysis revealed that the rIFG group showed significantly reduced SSRT when presented with the neutral stimulus ($t(21) = -2.58$, $p = 0.01$, $d = 0.55$), while no changes in SSRT were observed when presented with the emotional stop stimulus ($t(21) = 0.33$, $p = 0.74$, $d = 0.07$). The analysis of the SSRT collected in the lM1 control group revealed an absence of variation both when emotional ($t(21) = 0.99$, $p = 0.92$, $d = 0.02$) and neutral stimuli ($t(21) = -0.59$, $p = 0.55$, $d = 0.13$) were presented as stop stimuli (Figure 4). Finally, to determine that stimulation effects on response inhibition were not due to a general effect on the speed of motor responses, we performed a correlation analysis between go RTs and SSRTs in the post-stimulation session. Specifically, both the correlation between go RTs and SSRTs for the emotional stimulus in the pre-SMA group ($r = 0.01$, $p = 0.97$) as well as the correlation between go RTs and SSRTs for the neutral stimulus in the IFG group ($r = 0.12$, $p = 0.60$) were not significant. Thus, the selective effect on SSRT without any influence on go RT disproved a possible practice effect in the pre-SMA and rIFG groups.[34]

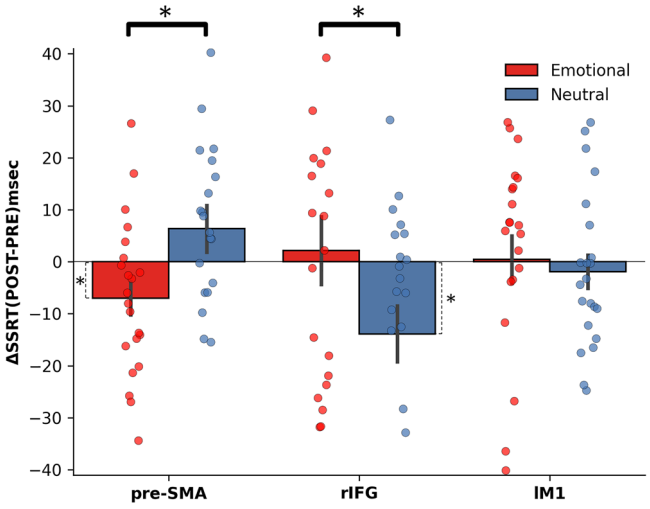

FIGURE 4 Bar graph of the experimental results. The graph shows the mean difference between the post- and pre-stimulation stop signal reaction time (SSRT) to the two different stimuli in each group. The pre-SMA group showed better inhibitory performance when facing emotional stimuli after rTMS with respect to the neutral stop stimuli, while the opposite is true for the rIFG stimulation. Meanwhile, the lM1 group shows no difference in inhibitory performance after the rTMS session. Asterisks indicate significant comparisons ($p < 0.05$), and error bars represent SEM. The picture was created using the Matplotlib and Seaborn libraries in Python. Abbreviations: ΔSSRT, SSRT inhibitory performance index; lM1, left motor cortex; pre-SMA, pre-supplementary motor cortex; rIFG, right inferior frontal gyrus.

Crucially, these results showed a difference in inhibition performance depending on the content of the stimuli (i.e., neutral vs. emotional) only after rTMS in both experimental groups. The absence of a difference in stopping ability within emotional contexts compared to neutral ones were found before applying any rTMS manipulation (i.e., in the Pre Session) is in line with previous findings.[56,58,59] However, our data demonstrated specific roles of the areas targeted by neurostimulation in the ability to withhold responses. In particular, our results causally demonstrated that rTMS over the pre-SMA improves the inhibitory process when specifically observing emotionally negative arousing body postures. On the other hand, neurostimulation over the rIFG produced the opposite pattern of results, as participants were able to better inhibit responses when the neutral stimulus was presented as the stop signal. Importantly, no significant modulations were shown for the lM1 group both before and after neurostimulation and independently of the stimulus presented as a stop signal.

Motor impulsivity predicts correct inhibition after IFG neurostimulation

To explore the relations between the better reactive action inhibition when facing emotional stimuli and personality traits, we performed an analysis of covariance (ANCOVA) with the alpha criteria set at 0.05. An index representing the inhibition for negative stimuli relative to the neutral ones collected after the rTMS session (i.e., SSRT of the negative stimuli minus the SSRT of the neutral stimuli, ΓSSRT) was used as the dependent variable, and the scores for the STAI-Y2 and BIS11 questionnaire subscales (Motor Impulsivity/Attentional Impulsivity/Non-Planning Impulsivity) were entered as covariates, whereas to test between-subjects effects, Group (pre-SMA/rIFG/lM1) was entered as independent variable.

The ANCOVA resulted in a significant corrected model ($F(14,65) = 3.37$, $p < 0.01$, $\eta_p^2 = 0.48$). In particular, the analysis revealed a significant Motor Impulsivity by Group interaction ($F(2,65) = 5.92$, $p < 0.01$, $\eta_p^2 = 0.19$), whereas no main effects (all $p \geq 0.13$, $\eta_p^2 \leq 0.05$) or other interactions were found to be significant (all $p \geq 0.01$, $\eta_p^2 \leq 0.08$). Therefore, a subsequent parameter estimates analysis derived from the same ANCOVA revealed that ΓSSRT data for only the rIFG group held a significant effect with the Motor Impulsivity (MI) subcomponent of BIS11 scores ($b = 4.64$, $t(21) = 3.41$, $p < 0.01$, $\eta_p^2 = 0.19$). Accordingly, the MI component held a significant positive correlation with the rIFG group ΓSSRT index ($b = 0.44$; $p = 0.04$; see Figure 5). The result of this analysis highlights a significant relationship between MI (i.e., the tendency of acting out without thinking) and enhanced inhibition on facing neutral stimuli following neurostimulation of the rIFG. This outcome is particularly noteworthy, as it suggests that individuals with higher MI scores were more likely to exhibit improved inhibitory control after rIFG stimulation when presented with the neutral stop stimulus compared to the emotional one.

DISCUSSION

The ability to regulate one's motor behavior is a fundamental aspect of survival, and it becomes especially crucial when applied in emotionally charged situations where emotions can significantly impact this capability. In a typical scenario, a person is driving on a busy city street and can stop the car if a pedestrian suddenly crosses the road. However, this ability can be influenced by just having heard negative news on the radio, affecting the driver's performance in stopping the car. Despite the significance of this subject, there has been limited research into the neural foundations of this ability. Herein, taking advantage of the ability of the rTMS to noninvasively stimulate a specific cortical region and transiently disrupt the underlying information processing, we interfered with the activity of specific AIN regions in separate groups of participants. Our aim was to investigate possible changes in the participants' ability to inhibit actions upon facing emotionally salient (i.e., negative) or neutral human body postures presented as stop signals in an SST, before and after the application of rTMS over the pre-SMA, rIFG, and lM1. We found a selective improvement in reactive action control (i.e., shorter SSRT) on facing emotionally negative arousing stimuli after the application of one session of inhibitory rTMS over the pre-SMA. Such a behavioral effect has been previously found when the emotional stimuli were task-relevant (see, e.g., Refs. 50 and 115) or when they were presented as task-irrelevant stop signals relative to when they were primed before the go signal.[61–63] On the other hand, a selective improvement in reactive action control (i.e., shorter SSRT) on facing neutral body postures was found after the administration of rTMS over the rIFG. No significant changes were observed after the stimulation of the lM1.

The findings of this study support the existence of emotional effects on motor control systems and provide causal evidence that such emotional effects may involve (at least partially) separate neural pathways distinct from those associated with motor inhibition in neutral contexts[137]. These findings are in line with previous results that suggested the pre-SMA to have a specific role in action control when facing emotional stimuli[56] and that the pre-SMA could play a role in the control of movements triggered by visual stimuli with emotional content.[76] However, such previous fMRI investigations lack causality information and did not report any clear interaction between emotions and action control since no effects were observed contrasting the SSRT in an emotional versus neutral context,[56] or did not directly test motor control.[76] Importantly, our results are in line with TMS findings, which demonstrated that it is possible to induce a paradoxical enhancement of motor control after inhibitory rTMS over the pre-SMA.[34,35,116]

The initial study that sought to explore the key role of the pre-SMA in action control was conducted by Obeso and collaborators.[34] In their study, the authors employed a combination of rTMS (specifically, continuous theta burst stimulation [cTBS]) targeting the pre-SMA and positron emission tomography while participants were engaged in the SST using neutral stimuli as stop signals. The results revealed that cTBS applied to the pre-SMA led to an enhancement in the effective-

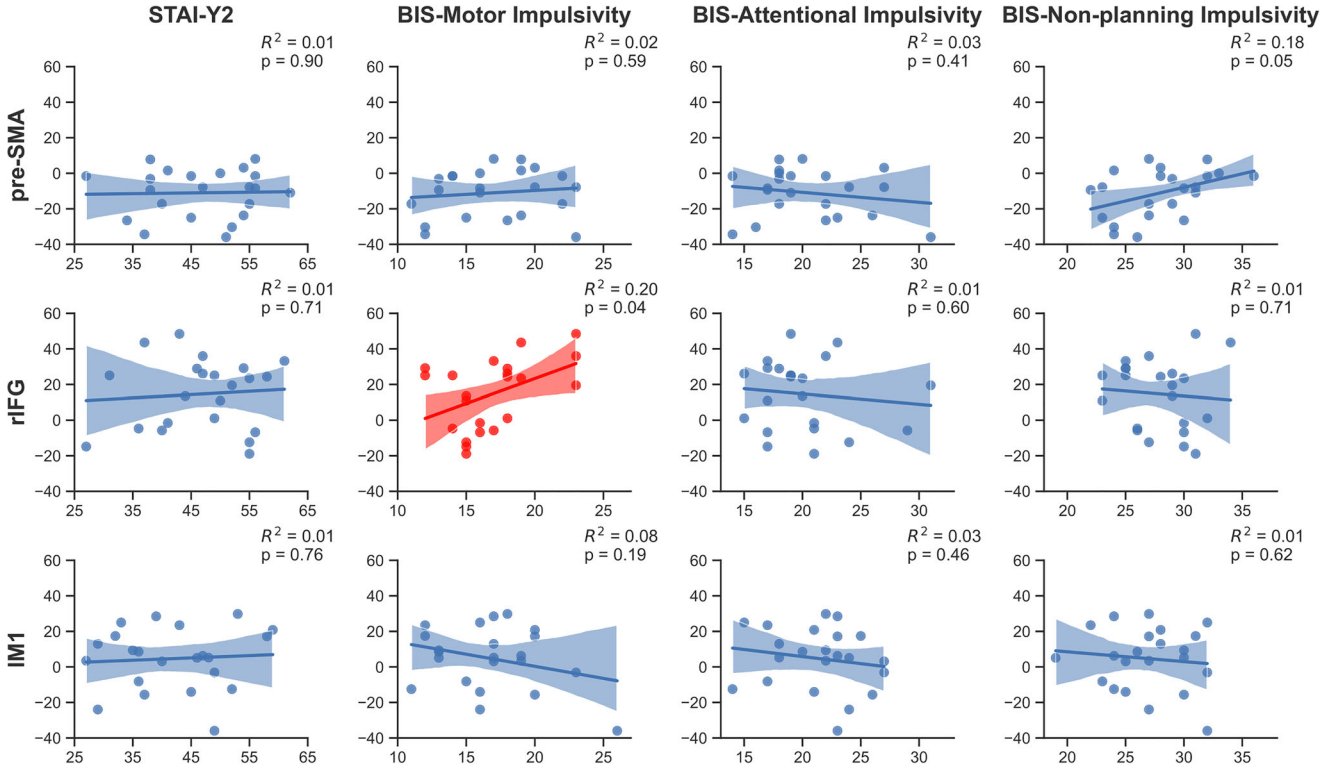

FIGURE 5 Correlation plots between questionnaire scores (on the *x*-axes) and the difference between emotional and neutral SSRT data (ΓSSRT on the *y*-axes) after neurostimulation. Highlighted in red is the significant correlation between Motor impulsivity (MI) scores and ΓSSRT in the rIFG group, which suggests that higher levels of MI correspond to greater inhibition efficiency when viewing neutral stimuli after rTMS. Data reported in the plots represent the results of individual linear regressions. Shaded areas represent confidence intervals. The picture was created using the Matplotlib and Seaborn libraries in Python.

ness of inhibitory control over ingrained ongoing responses, which was accompanied by an increase in regional cerebral blood flow in the left IFG. Thus, the authors interpreted the boost in performance as due to increased activation in the contralateral (i.e., left) hemisphere during the right-hand task performance, with faster SSRT induced by the disinhibitory effect triggered by cTBS. Such an increase in performance may, therefore, be related to compensation from distant sites with connectivity to the right pre-SMA across the left hemisphere network (i.e., left pre-SMA, left IFG).[117] Herz and coworkers[116] replicated these behavioral findings, demonstrating that inhibitory rTMS over the pre-SMA can improve reactive action control with neutral stimuli as stop signals, but this effect was observed only when participants were explicitly rewarded for fast and accurate responses—suggesting the existence of an interaction between the level of motivation and motor control in the pre-SMA. Moreover, the authors combined rTMS with fMRI and noted that such a boost in performance was mediated by enhanced activation and connectivity of the IFG–subthalamic nucleus (IFG–STN) pathway, as well as by the pre-SMA connections to the striatum—a pathway shown to mediate fast and urgent behaviors.[118] This result is in line with recent findings showing striatal changes after inhibitory rTMS over the pre-SMA.[119] Interestingly, no effects were observed on SSRT after inhibitory stimulation of the rIFG.[35]

In our study, we shed new light on the way different stop signals may be processed by partially separate parts of the AIN by demonstrating that the pre-SMA and rIFG are devoted to reactive action control in differential contexts, namely, emotional or neutral, respectively. Certainly, while the rIFG and pre-SMA are components of the AIN, the IFG and SMA proper are likely responsible for orchestrating and carrying out particular actions. In contrast, the pre-SMA may play a role in preparing and selecting suitable actions, as well as suppressing unsuitable actions, as suggested by previous research.[120,121] Indeed, the involvement of the pre-SMA in emotional stimuli elaboration has been widely demonstrated.[65–67,69–72,74] As an example, Rodigari and Oliveri[65] showed that the administration of inhibitory rTMS over the pre-SMA increased the perceived valence of threatening visual stimuli, while no changes were observed for neutral visual stimuli. This result suggests that the inhibition of the pre-SMA could release this inhibitory control, resulting in an increased perception of the emotional value of a negative stimulus. It may be possible to speculate that such increased emotional value may trigger increased action control mediated by compensatory circuits, such as the IFG–STN.

Notably, we also found that MI scores, which reflect the inability to suppress a behavioral response, predicted increased action control when facing neutral stimuli after inhibitory rTMS over the rIFG, highlighting the importance of individual personality traits in modulating the impact of neurostimulation on inhibitory performance. The link between the IFG and MI has been already established in previous studies.[122–124] Interestingly, in a morphological connectivity study,

the ACC–rIFG morphological connectivity was reported to be specifically correlated with the MI and not the attentional subscale of the BIS11.[122] Similarly, another study reported a significant negative relationship between the volume of the IFG and MI.[123] In line with our data showing that individuals with higher MI scores may benefit significantly from IFG stimulation; these results suggest that the IFG may be a potential target for interventions aimed at improving inhibitory control in individuals with high levels of MI. Nevertheless, our findings partially diverge from fMRI studies using go/no-go tasks that reported motor inhibition in response to emotionally salient stimuli activates the rIFG together with subcortical limbic structures (amygdala and ventral caudate). However, rIFG activation is specifically found in conditions where emotional information was task-relevant for motor decisions, and this may have potentially conflated inhibition with target detection and task switching.[78] Moreover, most of these studies adopted go and no-go trials based on the emotional valence of stimuli (positive vs. negative) so that the emotional information was task-relevant and response inhibition was confounded with emotion recognition.[125–127]

Finally, no effect on either emotional or neutral stimuli was reported on testing action control after inhibiting the IMI. M1 is considered to be a part of the final common path for voluntary action,[128] and it receives input from the pre-SMA.[104,129,130] For example, Zandbelt and coworkers[131] found that rIFC and pre-SMA stimulation induced shorter SSRT, while increasing M1 deactivation. Thus, although necessary for action control, how action inhibition commands reach M1 is still controversial.

A potential limitation of this study is that our TMS coil and stimulation protocol do not guarantee that we successfully targeted the right pre-SMA alone but could potentially have influenced the medial section including the left pre-SMA. Hence, it is possible that our results reflect a combination of right and to some extent left pre-SMA stimulation. Another potential limitation is that we selectively stimulated the rIFG, while the left IFG has also been found to have a role in action control.[10,11] Future studies will investigate the role of both left and right IFG by comparing possible differences in action control and additionally investigating possible differences between reactive and proactive inhibition. Another intriguing possibility for future studies is the use of multiple emotional stimuli. Finally, another potential brain region involved both in emotional processing as well as in action control is the insula.[132,133] An interesting future possibility is to also investigate the role of this area in action control in emotional context.

Overall, the implications of our research extend beyond the realms of cognitive neuroscience by unraveling the intricate relationship between emotions and motor control. Our study may offer valuable insights for individuals dealing with various clinical conditions that affect their ability to regulate motor behavior in emotionally charged situations. One particular population that stands to benefit from our research findings comprises individuals with anxiety disorders, post-traumatic stress disorder (PTSD), or other related mental health conditions. These individuals often experience heightened emotional states that can significantly impair their ability to maintain control over their actions, particularly in high-stress situations. Understanding the neural pathways involved in emotional influences on motor control could pave the way for targeted therapeutic interventions.[135,136] Furthermore, the insights gleaned from our study may inform the development of interventions or treatments tailored to address the specific challenges faced by individuals in clinical populations. For instance, the application of different NIBS protocols to modulate the neural circuits identified in our research could prove effective in helping individuals with anxiety or PTSD regain control over their motor responses during emotionally charged episodes. Notwithstanding, the clinical and therapeutic implications of our research also point to the need for further investigations. Future studies could delve deeper into the development and testing of interventions that leverage the neural insights provided by our research, ultimately aiming to enhance the quality of life for individuals struggling with motor control.

To conclude, the present research corroborates the presence of emotional influences on motor control systems[83,97] and provides initial critical evidence that these emotional effects might, to some extent, engage distinct neural pathways apart from those linked to motor inhibition in neutral circumstances.

AUTHOR CONTRIBUTIONS

Conceptualization, S. Battaglia and S. Borgomaneri; methodology, data analysis, and visualization, S. Battaglia; data collection, questionnaire scoring, and analysis, C.D.F. and C.N.; writing of the original draft, S. Battaglia and S. Borgomaneri; writing, review, and editing, C.D.F. and C.N.; funding acquisition, S. Battaglia and S. Borgomaneri; supervision, S. Borgomaneri. All authors approved the final version of the manuscript for submission.

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COMPETING INTERESTS

The authors declare no competing interests.

PEER REVIEW

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