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The Medial Frontal Cortex Mediates Self-Other Discrimination in the Joint Simon Task

A tDCS Study

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Abstract: Interacting with other individuals confronts cognitive control systems with the problem of how to distinguish between self-generated (internally triggered) and other-generated (externally triggered) action events. Recent neuroscience studies identified two core brain regions, the anterior medial frontal cortex (aMFC) and the right temporo-parietal junction (rTPJ), to be potentially involved in resolving this problem either by enhancing self-generated versus other-generated event representations (via aMFC) and/or by inhibiting event representations that are externally triggered (via rTPJ). Using transcranial direct current stimulation (tDCS), we investigated the role of the aMFC and the rTPJ for the online control of self-generated versus other-generated event representations in a joint Simon task. In two experimental sessions, participants received anodal, cathodal, or sham tDCS (1 mA intensity applied for 20 min), while performing an auditory joint Simon task. In addition to a general performance enhancement during cathodal (inhibitory) and anodal (excitatory) stimulation with increased practice, we found a significantly increased joint Simon effect (JSE) during cathodal stimulation of the aMFC (Experiment 1), as compared to sham stimulation. No modulation of the JSE was found during stimulation of the rTPJ (Experiment 2). By enhancing self-generated event representations the aMFC seems to be crucially involved in resolving the self-other discrimination problem in the joint Simon task.

Keywords: tDCS, anterior medial frontal cortex, right temporo-parietal junction, joint action, joint Simon effect

Acting together with other people is a crucial part of our daily life. In order to fluently interact with our conspecifics we must anticipate another person's actions, integrate action event representations generated by ourselves with events generated by others, and dynamically coordinate our own actions with those of others (Knoblich & Sebanz, 2006; Liepelt & Prinz, 2011; Liepelt, Stenzel, & Lappe, 2012; Sebanz, Bekkering, & Knoblich, 2006; Sebanz & Knoblich, 2009; Sebanz, Knoblich, Prinz, & Wascher, 2006; Tsai, Kuo, Hung, & Tzeng, 2008). But having shared representational systems (Prinz, 1997) also comes with a cost. It confronts our cognitive control system with a fundamental problem, namely, the demand of discriminating between self-generated and other-generated action events (Brass, Ruby, & Spengler, 2009). Based on the assumption of a tight link between perception and action, previous research tested the question why people do not imitate all the time when observing other people's action when having shared representational systems (Brass, Bekkering, Wohlschläger, & Prinz, 2000; Brass & Heyes, 2005; Liepelt, von Cramon, & Brass, 2008). Using functional magnetic resonance imaging (fMRI), Brass, Derrfuss, and von Cramon (2005) found two brain regions to be involved in the control of imitative behavior – the anterior medial frontal cortex (aMFC) and the right temporo-parietal junction (rTPJ).

The aMFC has been related to the control of externally triggered actions when perceiving another person's action

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in an imitation-inhibition task by enforcing own action intentions (Brass et al., 2005). This interpretation is in line with other studies associating this brain region with perspective taking – referring to the tendency to adopt psychological perspectives of others (Civai, Miniussi, & Rumiati, 2014; Ruby & Decety, 2003, 2004; Spengler, von Cramon, & Brass, 2009a) and self-referential processing (Abraham, Werning, Rakoczy, von Cramon, & Schubotz, 2008; Northoff & Bermpohl, 2004; Tsakiris, Costantini, & Haggard, 2008). The rTPJ has also been related to the online control of co-activated event representations when participants have to execute, for example, a finger-lifting movement while simultaneously observing a congruent or an incongruent finger-lifting movement of another person (Brass et al., 2005). The activation foci in the aMFC and the rTPJ found in imitation-inhibition tasks have been shown to overlap with those found in Theory of Mind (TOM) tasks (Amodio & Frith, 2006; Decety & Lamm, 2007) involving mentalizing, self-referential processing, and agency processing (Brass et al., 2009). However, similar activation foci in the aMFC and rTPJ have also been found (1) in nonsocial attentional tasks (Decety & Lamm, 2007; Mitchell, 2008), (2) in tasks involving the processing of erroneous physical representations (Saxe & Kanwisher, 2003), (3) in reward processing for persons and objects alike (Harris, McClure, Van den Bos, Cohen, & Fiske, 2007), and (4) in cognitive control tasks (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004).

Together, these studies provide indirect evidence for the assumption that the control of shared representations may reflect a domain-general process for social (Adolphs, 2003; Frith & Frith, 2003; Saxe, Carey, & Kanwisher, 2004; Sebanz, Rebbechi, Knoblich, Prinz, & Frith, 2007; Spengler et al., 2009a) and nonsocial cognitive functions (Botvinick et al., 2001; Decety & Lamm, 2007; Mitchell, 2008). Following this assumption, a recent study found improved performance during an imitation-inhibition and a perspective-taking task when cortical excitability of the rTPJ was increased by anodal transcranial direct current stimulation (tDCS; Santiesteban, Banissy, Catmur, & Bird, 2012). tDCS is a noninvasive stimulation technique that stimulates the cerebral cortex with a weak direct current passing between two scalp electrodes. A weak direct current polarizes the transmembrane neural potential of the stimulated brain regions at subthreshold level. Depending on the polarity of the stimulation it can decrease (cathodal tDCS) or increase (anodal tDCS) cortical excitability of the respective brain regions (Nitsche et al., 2008). While studies testing tDCS effects in the motor and visual domains provided clear evidence for polarity-specific (i.e., cathodal = inhibitory, anodal = excitatory) tDCS effects (Antal, Kincses, Nitsche, Bartfai, & Paulus, 2004; Nitsche & Paulus, 2000, 2001; Nitsche et al., 2003a; Stagg et al., 2011), this physiological dichotomy does not translate necessarily to cognitive and behavioral effects (Santiesteban et al., 2012). This difference may depend on the initial activation level, the neuronal state of the stimulated region, and on task characteristics (Antal, Nitsche, Kincses, et al., 2004; Antal, Nitsche, Kruse, et al., 2004; Jacobson, Koslowsky, & Lavidor, 2012). tDCS is known to modulate several cognitive functions (see Kuo & Nitsche, 2012; Paulus, 2011) and it is considered to be a promising tool to infer causal relationships between activity in a particular brain region and a specific cognitive function (for reviews, see Been, Ngo, Miller, & Fitzgerald, 2007; Levasseur-Moreau, Brunelin, & Fecteau, 2013; Nitsche et al., 2008). If tDCS is applied in accordance with present safety guidelines, it can be considered as a safe technique of brain stimulation with relatively minor adverse effects such as light itching sensations, metallic taste, and slight headache (Bikson, Datta, & Elwassif, 2009; Jacobson et al., 2012; Liebetanz et al., 2009; Nitsche et al., 2008).

From Action Observation to Joint Action

The studies investigating the control of shared representations we have described so far had participants passively observe others performing certain actions or perceive others' actions while acting themselves. In the last decade, however, a number of studies used a so-called second-person approach (Schilbach et al., 2013) by having participants interact or coact with another person. Prominent examples for joint action tasks are modified versions of classical interference tasks such as the joint "PRP" (i.e., psychological refractory period) task (Pashler, 1984, 1994; for the joint version see Liepelt & Prinz, 2011), the joint Eriksen flanker task (Eriksen & Eriksen, 1974; for the joint version see Atmaca, Sebanz, & Knoblich, 2011; Dolk, Hommel, Prinz, & Liepelt, 2014), the joint "SNARC" (i.e., spatial numerical association of response codes) task (Dehaene, Bossini, & Giraux, 1993; for the joint version see Atmaca, Sebanz, Prinz, & Knoblich, 2008), or the joint Simon task (Simon & Rudell, 1967; for the joint version see Sebanz, Knoblich, & Prinz, 2003; Dolk et al., 2011).

The most prominent of these cognitive tasks aiming at investigating the mechanisms underlying joint action is the joint Simon task. The joint Simon task, which was developed by Sebanz and colleagues (2003), is based on the standard Simon task (Simon 1990; Simon & Rudell, 1967), where an individual participant carries out a two-choice reaction task by responding with a left key press to one stimulus (e.g., high pitched tone) and with a right key press to another stimulus (e.g., low pitched tone). In each trial one of the two stimuli is randomly presented either via a left or via a right positioned loudspeaker. In the joint Simon task, pairs of participants share performance of this task (Vlainic, Liepelt, Colzato, Prinz, & Hommel, 2010). One participant takes over one half of the task (e.g., responds to the high pitched tone with the right button) while the other participant performs the complementary half of the task (e.g., responds to the low pitched tone with the left button) – which renders the task a simple go/nogo task for each person (Hommel, 1996). Although stimulus location is irrelevant for performing the task, it has repeatedly been shown that response times (RTs) are typically longer when stimulus and response location are incompatible (i.e., stimulus assigned to a left key response is presented via the right loudspeaker) than when they are compatible (i.e., stimulus assigned to a left key response is presented via the left loudspeaker; e.g., Colzato, van den Wildenberg, & Hommel, 2013; Dittrich, Dolk, Rothe-Wulf, Klauer, & Prinz, 2013; Dolk, Hommel, Prinz, & Liepelt, 2013; Hommel, Colzato, & van den Wildenberg, 2009; Liepelt, Wenke, & Fischer, 2013; Sebanz, Knoblich, & Prinz, 2005; Vlainic et al., 2010). This stimulus-response (S-R) compatibility effect is observed when a single subject performs the entire task alone, as in the standard Simon task – the Simon effect (SE). While this effect vanishes if a participant carries out only half of the task alone by pressing one of the two keys to one of the two stimuli (the go/nogo version of the Simon task), having another participant respond to the other stimulus by pressing the other key brings back the effect – the joint Simon effect (JSE).

Ideomotor Theory and the Referential Coding Account

The most widely accepted explanation for the occurrence of the SE supposes a dimensional overlap of the irrelevant spatial stimulus dimension and the spatially defined response dimension (Hommel, Müsseler, Aschersleben, & Prinz, 2001; Kornblum, Hasbroucq, & Osman, 1990; Nicoletti & Umiltà, 1989). The irrelevant spatial feature of the stimulus automatically activates the corresponding response (automatic response activation route), which facilitates responses when the task-relevant stimulus dimension requires that response, but interferes when the task-relevant stimulus dimension requires the alternative response (indirect route; see Kornblum et al., 1990). The occurrence of dimensional overlap and hence the JSE in the joint Simon task is typically assumed to be caused by the co-representation of the coactor's task share (i.e., S-R mapping), which is considered to be functionally equivalent to the representation of one's own task share and thereby quite similar to the standard Simon task (or the demands thereof; e.g., Sebanz et al., 2005; Tsai, Kuo, Jing, Hung, & Tzeng, 2006). However, recent findings provided evidence for a crucial role of the *spatial response* (Dittrich, Rothe, & Klauer, 2012; Guagnano, Rusconi, & Umiltà, 2010) or *referential response coding* for the occurrence of the JSE (e.g., Dolk et al., 2011, 2013; Liepelt, Wenke, Fischer, & Prinz, 2011; see also Ansorge & Wühr, 2004; Hommel, 1993a for similar theoretical line for the SE). According to Ideomotor theory (James, 1890; Stock & Stock, 2004) and its extensions (Theory of Event Coding; Hommel et al., 2001), one's own (intentional) actions are controlled by the activation of feature codes representing their perceivable effects. Consequently, perceiving alternative (externally generated) action events that share feature codes with one's own action event representations leads to an action selection conflict between externally triggered and internally intended actions (i.e., their activated feature codes). By emphasizing (cf. intentionally weighting; Memelink & Hommel, 2013) action features that discriminate best between self-generated (internally triggered) and other-generated (externally triggered) action events in a given task context – referential coding (Dolk et al., 2013; Hommel, 1993a; Hommel et al., 2001), the action selection conflict can be resolved. In the joint Simon task these features relate to the relative spatial location of both alternative actions, as in the spatial Simon task space is the most discriminative event feature. Referential response coding reintroduces a match or mismatch between spatial stimulus and spatial response features and hence produces the JSE (Kornblum et al., 1990; Stenzel et al., 2014). Instead of assuming that the JSE emerges from the automatic co-representation of the coactor's actions (Sebanz et al., 2003) or task (Sebanz et al., 2005), referential coding assumes that the JSE is the product of the resolution of the action discrimination conflict arising from common coding of internally and externally activated events through intentional weighting.

In line with these theoretical assumptions (and behavioral approvals; for a review see Dolk, Hommel, Colzato, et al., 2014), an fMRI study (Sebanz et al., 2007) found the aMFC to be more active during joint as compared to individual go/nogo Simon task performance when stimuli referring to their own action alternative were presented – i.e., go-trials for the participant. Given that the aMFC is associated with the implementation of one's own motor intentions, these functional findings nicely corroborate recent structural imaging results from our laboratory (Dolk, Liepelt, Villringer, Prinz, & Ragert, 2012) showing that the size of the JSE increases with decreased gray matter volume (GM) of the aMFC.

In the present study, we explored as to whether the ability to discriminate between self-generated (internally triggered) and other-generated (externally triggered) action events in a joint Simon task is modulated by tDCS of aMFC (Experiment 1) and/or rTPJ (Experiment 2). Based on previous research on imitation inhibition (Brass et al., 2009; Santiesteban et al., 2012) and joint action (Dolk et al., 2012; Sebanz et al., 2007) there seem to be two potential mechanisms of how this may be accomplished: By enhancing self-generated event representations via the aMFC and/or by inhibiting co-activated other-generated event representations via the rTPJ. Using tDCS, the present study tested the role of aMFC (Experiment 1) and rTPJ (Experiment 2) for self-other discrimination in the joint Simon task.

Experiment 1

In Experiment 1 we tested whether impairing the enhancement of self-generated event representations in the aMFC leads to an enlarged JSE. Previous studies have shown that the aMFC has a role in managing the conflict between intended internally triggered and externally generated event representations in an imitation-inhibition task, by enforcing one's own motor intention (Brass et al., 2005, 2009). In the present study, participants received inhibitory (cathodal), excitatory (anodal), or sham stimulation over the aMFC during the performance of an auditory joint Simon task. When assuming that the enhancement of self-generated event representations takes place in the aMFC, one should either find an increased JSE during cathodal stimulation over aMFC or a reduced JSE during anodal stimulation over aMFC (as compared to sham stimulation), or both. The modulatory tDCS influence over aMFC on the JSE may appear as a change in both, compatible trials (i.e., response facilitation) and/or incompatible trials (i.e., response interference). That is, because any change in the strength of self-generated event representations also has an impact on the automatic response activation route (Kornblum et al., 1990).

While an increased JSE would indicate an impairment of aMFC function due to a diminished self-representation during inhibitory stimulation, a reduced JSE would indicate an enhancement of aMFC function due to an enhanced self-representation during excitatory stimulation.

Material and Methods

Participants

Twenty healthy participants without any history of neurological or psychiatric disorder, intracranial metal implants or other implanted devices, migraine, or seizures took part in this experiment (9 females M_{age} = 25.0 years, SD_{age} = 2.4 years). All participants were right-handed and had normal or corrected-to-normal vision. Together with a gender- and age-matched coactor, they completed an auditory joint Simon task on two consecutive days. On both days participants (not the coactors) received tDCS using a sham protocol (see description below) that was followed by active stimulation. Active stimulation was applied on two separate days in order to prevent carry-over effects. The order of active stimulation (i.e., cathodal and anodal stimulation) applied was counterbalanced across participants. The local Ethics Committee of the University of Leipzig approved this study, which was conducted in accordance with the ethical standards laid down in the Declaration of Helsinki. All subjects gave their written informed consent before the experiment and were paid €24 as compensation for expenses.

Transcranial Direct Current Stimulation

tDCS was delivered by a battery-driven constant current stimulator (neuroConn GmbH Ilmenau, Germany) using a pair of surface rubber electrodes inserted in saline-soaked sponges. The target electrode comprised an area of 5 cm × 7 cm, while the size of the return electrode was increased comprising an area of 10 cm × 10 cm in order to reduce current density under this electrode and hence making the stimulation of this site functionally inert (Nitsche et al., 2007). To increase conductivity, we used 0.9% NaCl solution. Prior to the experiment each participant underwent an anatomical MRI scan acquired on a 3 Tesla scanner (MAGNETOM Trio, Siemens, Erlangen, Germany). T1-weighted images were acquired using a MPRAGE sequence (TR = 1.3 s; TE = 3.46 ms; flip angle = 10°; FOV = 256 mm × 240 mm; 176 slices; voxel slice = 1 × 1 × 1.5 mm). Anatomical scans were used for localization of the brain area by neuronavigation (aMFC; see Figure 1B) to be stimulated on each individual brain (Brainsight Version 2, Rogue Research Inc., Montreal, Canada). Coordinates for aMFC were averaged coordinates from earlier studies that used MRI and magnetoencephalography (MEG; Table 1). Montreal Neurological Institute (MNI) coordinates for the aMFC were (MNI: 0, 55, 13). The return electrode was placed over the rTPJ (MNI coordinates: 63, −50, and 23). As the area of the return electrode was the target area in Experiment 2, we ensured that in both experiments (Experiment 1 and Experiment 2) exactly the same electrode montage regarding the distance between electrodes was applied. Given the constant distance between electrodes and the constant relative difference in size between both electrodes we could control that both experiments differed only with respect to the location of stimulation, while the focality of stimulation delivered by the target electrode was the same.

In accordance with the standard safety limits (Nitsche et al., 2003b; Poreisz, Boros, Antal, & Paulus, 2007), a constant current of 1 mA intensity was applied for 20 min resulting in a current density of 0.0329 mA/cm^2 while the stimulated participant performed an auditory joint Simon task together with a coactor (not stimulated). At the beginning of the experimental session the current was

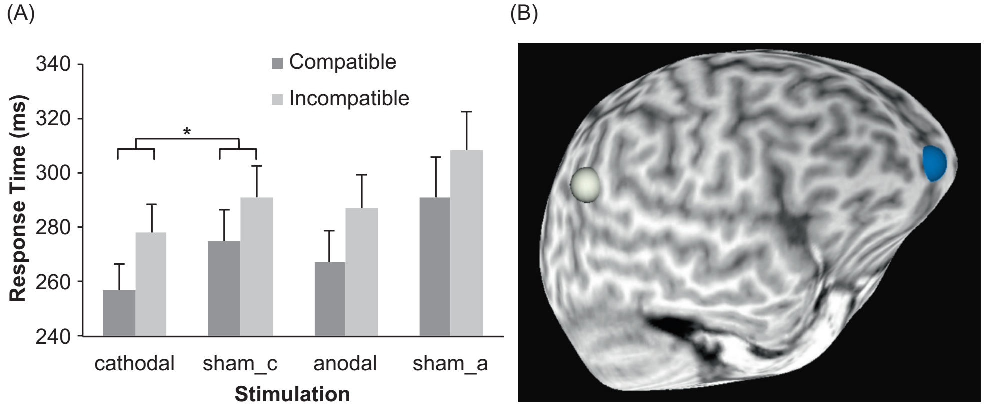

Figure 1. Mean reaction times in milliseconds (A) obtained for stimulation (cathodal, sham cathodal [sham_c], anodal and sham anodal [sham_a] stimulation) and compatibility (compatible and incompatible) for the anterior medial frontal cortex (aMFC). The right panel (B) shows the aMFC region (blue) that was the target brain region used for tDCS, and the right temporo-parietal junction (rTPJ) region (white) representing the control region projected on a 3D brain reconstruction. The asterisk ($*p < .05$) shows the significant interaction between compatibility and stimulation for cathodal stimulation conditions. Error bars represent standard errors of the mean.

Table 1. Studies included for averaging aMFC coordinates

		Talairach coordinates		
aMFC	Brain area	*x*	*y*	*z*
Brass et al. (2005)	aMFC (BA10/32)	1	50	9
Brass et al. (2009)	aMFC	1	39	18
Forstmann et al. (2008)	aMFC posterior part (BA10)	−14	36	3
Moll, Eslinger, & de Oliveira-Souza (2001)	Bilateral medial frontal gyrus (BA9/10)	4	54	3
Sebanz et al. (2007)	Medial frontal gyrus (BA10)	18	64	8
Spengler, von Cramon, & Brass (2009b)	aMFC	−8	56	12
Volz et al. (2006)	aMFC	−5	54	6
Zysset, Huber, Ferstl, & von Cramon (2002)	aMFC (BA10/9)	−6	55	13
Zysset, Huber, Samson, Ferstl, & von Cramon (2003)	aMPFC (BA9/10)	5	49	16

ramped up within 30 s, followed by 19 min of stimulation at target intensity. After this period, the current was ramped down within 30 s. Prior to active stimulation, sham stimulation was applied to all participants. During sham stimulation the current was also ramped up and down within 30 s, whereas in between the current of 1 mA was maintained for 30 s only (instead of 19 min). Independently of sham or active stimulation, the application of tDCS is associated with minimal somatosensory sensations (mostly an itching sensation). The sham protocol used in this study ensured that the participants could feel the initial sensation but excluded any physiological effects of tDCS outlasting the actual stimulation duration (see Gandiga, Hummel, & Cohen, 2006; Nitsche & Paulus, 2000). All participants were blinded to the type of stimulation and the placement of the active electrode.

Stimuli and Procedure

The experiment was carried out in a dimly lit room. A monitor was placed in the middle of the desk due to instructional purposes. Participants were seated next to each other in front of the monitor. The participant who was supposed to receive tDCS always sat on the right side. Both participants and coactors were instructed to operate their response button with their right index finger, while their left hand was placed under the table on their left leg. The auditory stimuli, the trial timing, the response feedback, and the procedure were identical to those of a previous study showing an auditory JSE without applying tDCS (for details see Vlainic et al., 2010). The participant on the right side (i.e., stimulated actor) was always assigned to tone B (“chap”), while the participant on the left side (i.e., not stimulated coactor) always had to respond when tone A (“oerg”) was presented. During the instruction phase, tDCS after the sham protocol was given to the right-seated participant.

During sham stimulation, three experimental blocks were completed. Between these blocks participants had a short break of approximately 2 min. After completion of the third block participants had a break of about 5 min. Within this break the application either of the cathodal or of the anodal stimulation (referring to the target area, depending on the experimental session and order assignment of the participant) was started. Then, another three experimental blocks were accomplished, which again were separated by two short breaks. The completion of all three blocks including the two short breaks of 2 min took approximately 20 min (i.e., the duration of active stimulation). One block consisted of 128 trials with 64 go-trials for each participant and coactor consisting of 32 S-R compatible trials and 32 S-R incompatible trials.

Results

All participants tolerated the treatment of tDCS without major side effects, while most participants reported to have experienced a mild initial tingling sensation caused by tDCS, as indicated by verbal self-reports. For statistical analysis of the RT data, only data from the stimulated participant were considered. Trials with incorrect responses (1.6%; for an overview of percentage errors of the stimulated participant only, see Table 2) and trials with RTs faster than 100 ms and slower than 1,000 ms (0.1%) were excluded from further analysis (Liepelt et al., 2011; Röder, Kusmierek, Spence, & Schicke, 2007). Low number of errors reflects the ease of a simple stimulus discrimination task. Because of the low number of overall errors, error rates were not further analyzed. Mean RTs were submitted to an analysis of variance (ANOVA) with two within-subjects factors: Stimulation (active stimulation vs. sham stimulation) and S-R Compatibility (compatible vs. incompatible). We performed separate ANOVAs for each stimulation type accounting for less reliable AeCi (anodal-excitation, cathodal-inhibition) tDCS effects, as for more complex cognitive functions tDCS effects often go into the same direction for anodal and cathodal stimulation (Boggio et al., 2010; Marshall, Molle, Siebner, & Born, 2005) and tDCS effects of only one of both active stimulation types were found in previous studies (Monti et al., 2008; for an overview see Jacobson et al., 2012). We also calculated Bayesian posterior probabilities for the occurrence of the null (H_0) and the alternative (H_1) hypothesis given the obtained data (Masson, 2011; Wagenmakers, 2007). This method allows to directly quantify evidence in favor of the alternative and null hypothesis, respectively, by providing the exact probability of their occurrence, with values ranging from 0 (i.e., no evidence) to 1 (i.e., very strong evidence; see Raftery, 1995 for a classification).

Cathodal (Inhibitory) Stimulation Versus Sham Stimulation (aMFC)

We observed a significant main effect of Compatibility, $F(1, 19) = 92.45$, $p < .001$, $\eta_p^2 = 0.83$, $p(H_1|D) > 0.99$, indicating an overall JSE with faster responses in S-R compatible trials (266 ms) than in S-R incompatible trials (285 ms). The main effect of Stimulation was significant, $F(1, 19) = 18.27$, $p < .001$, $\eta_p^2 = 0.49$, $p(H_1|D) = 0.99$, indicating faster responses during cathodal stimulation (267 ms) as compared to sham stimulation (283 ms). The interaction of Stimulation and Compatibility was significant, $F(1, 19) = 5.30$, $p < .05$, $\eta_p^2 = 0.22$, $p(H_1|D) = 0.72$, showing a significantly increased JSE during cathodal stimulation (21 ms; $t(19) = 9.34$, $p < .001$), as compared to sham stimulation (16 ms; $t(19) = 6.90$, $p < .001$; Figure 1; see also Appendices A and B).

Table 2. Mean error rates (%) obtained for stimulation (cathodal, sham cathodal [sham_c], anodal and sham anodal [sham_a] stimulation) and compatibility (C, compatible and IC, incompatible) of the stimulated participants in Experiment 1 (aMFC)

aMFC	C	IC
Cathodal tDCS	0.8	0.8
Sham_c tDCS	0.5	1.5
Anodal tDCS	1.0	1.4
Sham_a tDCS	0.5	1.2

Anodal (Excitatory) Stimulation Versus Sham Stimulation (aMFC)

We found a significant main effect of Compatibility, $F(1, 19) = 48.11$, $p < .001$, $\eta_p^2 = 0.72$, $p(H_1|D) > 0.99$, showing faster responses in S-R compatible trials (279 ms) than in S-R incompatible trials (298 ms), indicating an overall JSE. Furthermore, the main effect of Stimulation was significant, $F(1, 19) = 13.14$, $p < .01$, $\eta_p^2 = 0.41$, $p(H_1|D) = 0.98$, showing faster responses during anodal stimulation (277 ms) as compared to sham stimulation (299 ms). The interaction of Stimulation and Compatibility was not significant, $F(1, 19) = 1.66$, $p = .21$, $\eta_p^2 = 0.08$, $p(H_0|D) = 0.66$ (Figure 1).

Discussion

First, we could replicate previous findings (Sebanz et al., 2003) showing an overall JSE when two persons share a Simon task. Decreasing cortical excitability of the aMFC by cathodal stimulation increased the size of the JSE as compared to sham stimulation. This finding can neither be explained by an effect of stimulation order nor by potential differences between the two sham conditions that we applied (see Appendix B). The finding of an increased JSE during cathodal aMFC stimulation as compared to sham stimulation is in line with the assumption that the aMFC is causally involved in modulating the JSE. Cathodal stimulation seems to impair the enhancement of self-generated event representations, leading to a larger JSE. Anodal stimulation did not alter the size of the JSE relative to sham level, which may suggest that undisturbed self-enhancement during sham stimulation is already optimal in this task. Alternatively, the relatively fast reaction time level in this task could have prevented the detection of further self-enhancement due to a ceiling effect.

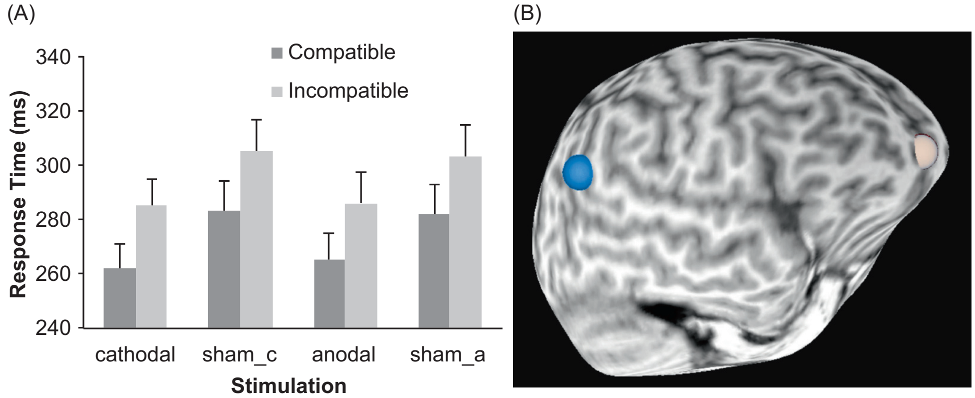

Figure 2. Mean reaction times in milliseconds (A) obtained for stimulation (cathodal, sham cathodal [sham_c], anodal and sham anodal [sham_a] stimulation) and compatibility (compatible and incompatible) for the right temporo-parietal junction (rTPJ). The right panel (B) shows the rTPJ region (blue) that was the target brain region used for tDCS, and the anterior medial frontal cortex (aMFC) region (white) representing the control region projected on a 3D brain reconstruction. Error bars represent standard errors of the mean.

Experiment 2

In Experiment 2, we tested whether impairing the inhibition of other-generated event representations in the rTPJ modulates the JSE. Previous studies show an involvement of the rTPJ in this process in imitation-inhibition tasks where a person selects a finger-lifting movement while observing a task-irrelevant incompatible movement of another person (Brass et al., 2005, 2009). A similar process has also been shown to be involved in the discrimination of self-related events and events related to others in agency tasks (Abraham et al., 2008; Decety & Grèzes, 2006; Decety & Sommerville, 2003; Farrer & Frith, 2002; Newman-Norlund, Bosga, Meulenbroek, & Bekkering, 2008; Tsakiris et al., 2008). In order to test whether the inhibition of other-generated event representations is causally involved in the JSE, we stimulated the rTPJ in the same way as in Experiment 1 with cathodal, anodal, and sham tDCS while participants performed an auditory joint Simon task together with a (not stimulated) coactor.

If the action discrimination conflict in the joint Simon task can be resolved via the inhibition of other-generated event representations, one should find either an increased JSE during cathodal stimulation over rTPJ or a reduced JSE during anodal stimulation over rTPJ (as compared to sham stimulation), or both. While the former effect would indicate an impairment of rTPJ functioning during inhibitory stimulation, the latter finding would indicate an enhancement of rTPJ function during excitatory stimulation.

Methods

Participants

Twenty-six new healthy participants without any history of neurological or psychiatric disorder, implanted metal objects, or a history of seizures took part (14 females M_{age} = 25.38 years, SD_{age} = 3.41 years) in this experiment. All participants fulfilled the same criteria and were treated the same way as participants of Experiment 1.

tDCS, Stimuli, and Procedure

tDCS was used in the same way as in Experiment 1 with the exception of the targeted brain region. Coordinates for the targeted brain area (rTPJ; see Figure 2B) were chosen from a previous transcranial magnetic stimulation (TMS) study that clearly localized the rTPJ (Tsakiris et al., 2008) with the following coordinates (MNI: 63, −50, and 23). The return electrode was placed over the brain area aMFC (MNI: 0, 55, and 13). Stimuli, task, and procedure were identical to those of Experiment 1.

Results

All participants tolerated the treatment of tDCS without any major side effects, while again a mild initial tingling sensation caused by tDCS was reported. For statistical analysis, data were treated in the same way as in Experiment 1. Trials with incorrect responses (0.9%; for an overview of percentage errors of the stimulated participant only, see Table 3) and trials with RTs faster than 100 ms and slower than 1,000 ms (0.2%) were excluded from further analysis (Liepelt et al., 2011; Röder et al., 2007).

Cathodal (Inhibitory) Stimulation Versus Sham Stimulation (rTPJ)

There was a significant main effect of Compatibility, $F(1, 25) = 101.23$, $p < .001$, $\eta_p^2 = 0.80$, $p(H_1|D) > 0.99$, showing an overall JSE with faster responses in S-R compatible trials (272 ms) than in S-R incompatible trials (295 ms). We also observed a significant main effect of Stimulation,

Table 3. Mean error rates (%) obtained for stimulation (cathodal, sham cathodal [sham_c], anodal and sham anodal [sham_a] stimulation) and compatibility (C, compatible and IC, incompatible) of the stimulated participants in Experiment 2 (rTPJ)

rTPJ	C	IC
Cathodal tDCS	0.3	1.3
Sham_c tDCS	0.5	0.8
Anodal tDCS	0.5	0.8
Sham_a tDCS	0.4	1.0

$F(1, 25) = 30.90$, $p < .001$, $\eta_p^2 = 0.55$, $p(H_1|D) = 0.99$, with faster RTs during cathodal stimulation (273 ms) as during sham stimulation (294 ms). The interaction of Stimulation and Compatibility was not significant, $F(1, 25) = 0.40$, $p = .53$, $\eta_p^2 = 0.02$, $p(H_0|D) = 0.80$ (Figure 2).

Anodal (Excitatory) Stimulation Versus Sham Stimulation (rTPJ)

We found a significant main effect of Compatibility, $F(1, 25) = 96.15$, $p < .001$, $\eta_p^2 = 0.79$, $p(H_1|D) > 0.99$, with faster responses in S-R compatible trials (274 ms) than in S-R incompatible trials (294 ms), indicating a JSE irrespective of stimulation. Furthermore, RTs during anodal stimulation (276 ms) were faster as during sham stimulation (292 ms), $F(1, 25) = 12.90$, $p = .001$, $\eta_p^2 = 0.34$, $p(H_1|D) = 0.97$. The interaction effect of Stimulation and Compatibility was not significant, $F(1, 25) = 0.01$, $p < .93$, $\eta_p^2 < 0.001$, $p(H_0|D) = 0.83$ (Figure 2; see also Appendix C).

Discussion

In Experiment 2, we tested whether the inhibition of co-activated other-generated events mediated by the rTPJ is crucially involved in the joint Simon task. We could replicate the finding of a significant JSE as found in Experiment 1. Both, cathodal (inhibitory) and anodal (excitatory) stimulation, led to an overall speed-up in response times, which may, however, be due to a practice effect. Active stimulation of the rTPJ (anodal and cathodal) did not modulate the size of the JSE, a finding that cannot be explained by potential differences between the two sham conditions that we tested (see Appendix C). While the rTPJ has been shown to be involved in the inhibition of other-generated events in the imitation-inhibition task, our findings indicate no such role in the joint Simon task.

General Discussion

Recent research shows that a central problem people are facing when interacting with other agents consists in discriminating between self- and other-generated events (Dolk et al., 2013; Hommel et al., 2009; Liepelt et al., 2011; Sellaro, Dolk, Colzato, Liepelt, & Hommel, 2015; for a recent review on the joint Simon task see Dolk, Hommel, Colzato, et al., 2014). Using tDCS, the present study tested the role of the aMFC (Experiment 1) and the rTPJ (Experiment 2) in resolving the self-other discrimination problem in a joint Simon task. While the aMFC has been shown to be involved in the enhancement of one's own motor intentions (Brass et al., 2005, 2009; Spengler et al., 2009a), the TPJ has previously been associated with the inhibition of online co-activated externally generated event representations (Ruby & Decety, 2004; Santiesteban et al., 2012).

In two experiments, we could replicate previous observations of a JSE when two people share a Simon task (Sebanz et al., 2003), as indicated by slower response times in incompatible as compared to compatible trials. During both, cathodal and anodal tDCS stimulation, responses were faster as compared to sham stimulation, which may, however, be due to a practice effect and not to the active stimulation. Only cathodal (inhibitory) stimulation of the aMFC increased the size of the JSE. In line with the prediction that decreasing the excitability of the aMFC should impair self-other discrimination, we observed an increase of the JSE during cathodal aMFC stimulation. This finding is in line with previous fMRI findings showing a stronger activation of aMFC when participants had to distinguish between their own action and another, concurrently perceived action (Brass et al., 2005) in an imitation-inhibition task (i.e., during incompatible trials).

The Present Findings in Light of the Referential Coding Account

In a joint Simon task participants operate in a turn-taking mode. On go-trials, they do not simultaneously perceive the coactor's action in terms of visual or auditory action effects while selecting their own response. However, we argue that the frequent presence of alternative responses produces the need to discriminate between internally activated action events and externally co-activated events. One way to resolve the self-other discrimination problem during joint action is to enhance self-generated versus externally activated event representations. Cathodal stimulation over the aMFC seems to impair the enhancement of self-generated events (i.e., increasing the discrimination problem) leading to a larger JSE. From the perspective of referential coding (Dolk et al., 2013; see Dolk, Hommel, Colzato, et al., 2014 for a review), the actor needs to select task-relevant action representations from the pool of all concurrently activated event representations. This requires a focus on task features that discriminate best between task-relevant (self-generated) and task-irrelevant (other-generated) event representations – intentional weighting principle (Dolk et al., 2013; Hommel, 1993a; Hommel et al., 2001; Memelink & Hommel, 2013). In the joint Simon task, the best discriminating feature is the horizontal location leading to a spatial coding of the participant's response and hence to a dimensional overlap with the spatial stimulus code producing the JSE (Dolk et al., 2013; see Dolk, Hommel, Colzato, et al., 2014 for a review). The present tDCS findings suggest that the functioning of aMFC and the intentional weighting process interact, so that stronger weighting (of discriminative action features) can compensate for the failure of self-enhancement via aMFC. If so, aMFC could be considered to serve a domain-general function (i.e., enhancement of self-representations) that plays a crucial role in both social and nonsocial circumstances.

The absence of modulatory tDCS effects on the JSE that we found for the rTPJ may be due to the turn-taking characteristic of the joint Simon task. In turn-taking tasks, participants do not observe the behavior of the other while preparing their own response. This may reduce the demands of keeping apart self- and other-related motor representations as compared to situations when self and other actions take place simultaneously (Brass et al., 2009). For example, Brass et al. (2009) used a modified version of the imitation-inhibition task and had the imperative stimulus and the observed movement appear either simultaneously or the observed movement appeared after the response to the imperative stimulus was given. For rTPJ this study found a Delay × Compatibility interaction showing that the compatibility effect was present for the simultaneous condition, but absent for the delay condition. This finding is in line with the previously suggested role of the rTPJ in visual perspective taking (Decety & Grèzes, 2006) and action discrimination in the imitation-inhibition task (Brass et al., 2009) where action planning takes place during action observation. In line with this view of the TPJ, Santiesteban et al. (2012) showed that anodal tDCS of the rTPJ reduces the interference effect in the imitation-inhibition task, which requires the online discrimination of internally and externally activated action events. Together with our findings showing that active rTPJ stimulation had no modulatory effect on the size of the JSE in a turn-taking task, these findings support the view that the TPJ is involved in keeping self and other perspectives apart, a function that is also needed in mentalizing and TOM tasks (Decety & Grèzes, 2006). Furthermore, our findings suggest that the cognitive and neural mechanisms enabling action discrimination in the imitation-inhibition task and the joint Simon task may at least partially involve different cognitive and neural processes.

The finding of overall faster response times during active tDCS as compared to sham stimulation – be it through the stimulation itself or due to an effect of practice – may be taken to challenge our interpretation of the cathodal aMFC stimulation effect in terms of impaired self-representation. Instead, one may argue that the increased JSE reflects a more indirect tDCS (or practice) effect producing faster response times during active stimulation conditions. However, the finding of the distribution analysis that we performed showed that the JSE increases with slower response times (i.e., it decreases with faster RTs), which rules out that the increased JSE during cathodal aMFC stimulation is due to a general practice effect and a speed-up of response times (see Appendix A).

Self-Other Control or Task-Relevance Gradients in MFC and TPJ?

A recent study of Nicolle et al. (2012) testing the neural mechanisms of value-based decision making with a computational fMRI approach revealed that dorsal and ventral regions of the MFC and TPJ may actually not distinguish between action values relating to self and other, but differentiate whether an action is task-relevant or task-irrelevant (Cook, 2014). Even though the assumption of an agent-independent axis in the medial prefrontal and the temporo-parietal cortex appears surprising at first, it may provide an interesting view on the present findings. Along this framework our observations could be interpreted as follows: The ventral part of the aMFC has been associated with the processing of task-relevant representations (Nicolle et al., 2012). In the Simon task, a strengthening of task-relevant information along the indirect route increases the impact of this route over the automatically activated spatial route (Kornblum et al., 1990) when the wrong response is activated thus decreasing the Simon effect (Liepelt & Fischer, in press). The inhibition of the aMFC with cathodal tDCS would lead to a lowered activation level of the relevant task representations, and thus enhances the impact of the task-irrelevant spatial location in triggering automatic response activation. Accordingly, the task-relevance framework could also explain the finding of an increased JSE during cathodal stimulation of the aMFC that we observed in our study.

Even though this reasoning for our findings is speculative at present, explaining our tDCS findings with the idea of a task-relevance organization in MFC would be in line with recent attempts to explain socio-cognitive processing in Theory of Mind (TOM) tasks and joint action phenomena like the JSE with domain-general (attentional) processes (Doneva & Cole, 2014; Liepelt, 2014; Mitchell, 2008) and referential coding (Dolk, Hommel, Colzato, et al., 2014; Dolk et al., 2013).

Our tDCS findings are in line with and extend previous observations from fMRI (Sebanz et al., 2007) and voxel-based morphometry (VBM) studies (Dolk et al., 2012), by providing the first direct (causal) evidence for an involvement of the aMFC in a joint Simon task. While our findings show a clear modulation of the JSE by aMFC stimulation, it is important to point out that due to the relatively distant electrode montage we used, we cannot determine how specific this effect is to aMFC. Previous studies provide evidence for the assumption of a network of brain areas, including the aMFC and the rTPJ, involved in the control of shared representations (e.g., Frith & Frith, 2003; Saxe et al., 2004; Spengler et al., 2009a) and for an inhibitory influence of MFC on TPJ (Schuwerk et al., 2014). With the present findings, we cannot rule out that the effect of aMFC stimulation on the JSE that we found may have emerged from interactions between the target and the return electrodes (Datta, Elwassif, Battaglia, & Bikson, 2008). However, considering the increased size of the return electrode in relation to the target electrode, it can be assumed that the current density under the return electrode was too low to have any physiological effect on the area under the electrode (Nitsche et al., 2007, 2008). The return electrode can therefore be considered to be functionally inert, which comes closer to achieving unipolar stimulation (Faria, Hallett, & Miranda, 2012). Although the electrodes were quite large, sufficient spatial specificity might be assumed, as brain areas supposed to be stimulated were located for each individual brain via neuronavigation. A finding that indicates the relative specificity of our inhibitory tDCS effect over the aMFC on the JSE is that we did not observe unspecific modulatory tDCS effects when stimulating the same brain regions in the inverse way (Experiment 2).

Recent patient studies showed that schizophrenia patients have a deficit in self-other integration in the joint Simon task (de la Asuncion, Bervoets, Morrens, Sabbe, & de Bruijn, in press; Liepelt, Schneider, et al., 2012). Liepelt and colleagues provided evidence for a reduced joint Simon effect in schizophrenia patients as compared to healthy controls, which may be due to an abnormally increased level of self-representation in these patients. On a neural level this could be the result of a dysfunctional cortical activation of aMFC and/or rTPJ or of their respective cortical connectivity. Adapting the physiological excitability of the disturbed brain region with tDCS might have beneficial remedial effects through an adjustment of the relative strength between internally and externally generated event representations in joint action.

Conclusions

Taken together, the findings of the present study contribute to the understanding of the role of the aMFC in joint action. We found evidence for a crucial involvement of the medial frontal cortex in modulating the relative strength of internally and externally activated event representations. Enhancing self-generated events may be crucial for enabling successful joint action, but also when individuals try to follow their own intentions under conditions of distraction through external events in the nonsocial world.

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Ethics and Disclosure Statements

All participants of the study provided written informed consent and the study was approved by the Ethics Committee of the University of Leipzig.

All authors disclose no actual or potential conflicts of interest including any financial, personal, or other relationships with other people or organizations that could inappropriately influence (bias) their work.

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Appendix A

As the Simon effect in the standard Simon task is typically found to vary in size with shortened RTs (Hommel, 1993b; Liepelt et al., 2011), we additionally ran a distributional analysis in order to rule out that the increased JSE we observed during cathodal aMFC stimulation as compared to sham stimulation was indirectly driven by the significant main effect of Stimulation (i.e., the shorter RTs during cathodal stimulation as compared to sham stimulation). RT distributions were computed for each subject and for each cell combination of Stimulation and Compatibility. The distributions were divided into quartiles (bins) from the fastest to the slowest (De Jong, Liang, & Lauber, 1994). Then we conducted an ANOVA including the factors Stimulation (cathodal vs. sham_c), Compatibility (compatible vs. incompatible), and Quartile. As in our main analyses, the main effects of Stimulation and Compatibility and the interaction of both factors were significant. We further obtained a significant main effect of Quartile, $F(1, 57) = 447.02$, $p < .001$, $\eta_p^2 = 0.96$, $p(H_1|D) > 0.99$, and a significant interaction of Compatibility and Quartile, $F(1, 57) = 14.47$, $p < .001$, $\eta_p^2 = 0.43$, $p(H_1|D) > 0.99$, showing increased JSEs with longer RTs (12 ms JSE for Quartile one, 15 ms for Quartile two, 18 ms for Quartile three, and 28 ms for Quartile four). The three-way interaction was not reliable, $F(1, 57) = 2.15$, $p = .11$, $\eta_p^2 = 0.10$, $p(H_0|D) = 0.95$. The finding of the distributional analyses showing that the JSE increased with longer RTs indicates that the increase of the JSE during cathodal stimulation was not due to a speed-up of RTs from sham to cathodal stimulation.

Appendix B

To test if the increase of the JSE during cathodal aMFC stimulation as compared to sham stimulation is based on unequal sham conditions, we calculated an additional ANOVA including the factors Stimulation (sham_a vs. sham_c) and Compatibility (compatible vs. incompatible). The analysis revealed a significant main effect of Compatibility, $F(1, 19) = 46.99$, $p < .001$, $\eta_p^2 = 0.71$, $p(H_1|D) > 0.99$, showing longer RTs on incompatible trials (299 ms) as compared to compatible trials (283 ms). Importantly, the main effect of Stimulation, $F(1, 19) = 1.61$, $p = .22$, $\eta_p^2 = 0.08$, $p(H_0|D) = 0.66$, and the interaction of Stimulation and Compatibility were not significant, $F(1, 19) = 0.21$, $p = .65$, $\eta_p^2 = 0.01$, $p(H_0|D) = 0.80$. The finding of this additional analysis indicates that the increase of the JSE during cathodal aMFC stimulation was not due to a difference regarding sham stimulation.

Since we observed no differences between both sham conditions, we collapsed those two conditions and calculated an additional ANOVA including the factors Stimulation (sham_collapsed vs. cathodal vs. anodal) and Compatibility (compatible vs. incompatible). Again, the analysis revealed significant main effects of Compatibility, $F(1, 19) = 79.09$, $p < .001$, $\eta_p^2 = 0.81$, $p(H_1|D) > 0.99$, and Stimulation, $F(2, 38) = 6.13$, $p < .01$, $\eta_p^2 = 0.24$, $p(H_1|D) = 0.87$. The interaction of Stimulation and Compatibility was marginally significant, $F(2, 38) = 2.59$, $p = .088$, $\eta_p^2 = 0.12$, $p(H_1|D) = 0.86$. The Bayes analysis indicated a high probability (86%) for a significant interaction of Stimulation and Compatibility. Therefore, we calculated a separate ANOVA including Stimulation (sham_collapsed vs. cathodal) and Compatibility (compatible vs. incompatible), which revealed significant main effects of Compatibility, $F(1, 19) = 78.24$, $p < .001$, $\eta_p^2 = 0.81$, $p(H_1|D) > 0.99$, with longer RTs in S-R incompatible trials (289 ms) as compared to S-R compatible trials (270 ms) and of Stimulation, $F(1, 19) = 14.55$, $p = .001$, $\eta_p^2 = 0.43$, $p(H_1|D) = 0.99$, with longer RTs during sham stimulation (291 ms) as compared to cathodal stimulation (267 ms). We found a significant two-way interaction of these two factors, $F(1, 19) = 6.90$, $p < .05$, $\eta_p^2 = 0.27$, $p(H_1|D) = 0.99$, with a larger JSE during cathodal stimulation (21 ms) as compared to sham stimulation (16 ms). The separate ANOVA with the factors Stimulation (sham_collapsed vs. anodal) and Compatibility (compatible vs. incompatible) revealed significant main effects of Compatibility, $F(1, 19) = 62.89$, $p < .001$, $\eta_p^2 > 0.99$, $p(H_1|D) = 0.99$, with longer RTs in S-R incompatible trials (293 ms) as compared to S-R compatible trials (275 ms) and Stimulation, $F(1, 19) = 8.31$, $p = .01$, $\eta_p^2 = 0.30$, $p(H_1|D) = 0.89$, with longer RTs during sham stimulation (291 ms) as compared to anodal stimulation (277 ms). The two-way interaction was not reliable, $F(1, 19) = 2.34$, $p < .142$, $\eta_p^2 = 0.11$, $p(H_0|D) = 0.58$.

Further, we tested if the increase of the JSE during cathodal aMFC stimulation as compared to sham stimulation may be due to an order effect of active stimulation. Therefore, we added the between-subjects factor order of active stimulation (anodal first day/cathodal second day vs. cathodal first day/anodal second day) to our main analyses including the within-subjects factors Stimulation (cathodal stimulation vs. sham stimulation) and Compatibility (compatible vs. incompatible). As in our main analyses, we found significant main effects of Stimulation and Compatibility and a significant interaction of both factors showing the increased JSE during cathodal stimulation as compared to sham stimulation (all *p*s < .05). Importantly, the main effect of Order of stimulation, $F(1, 18) = 0.93$, $p = .347$, $\eta_p^2 = 0.05$, $p(H_0|D) = 0.73$, as well as all interactions with this factor were not significant (Order of active stimulation and Compatibility, $F(1, 18) = 0.82$, $p = .376$, $\eta_p^2 = 0.04$, $p(H_0|D) = .74$; Order of active stimulation and Stimulation, $F(1, 18) = 1.17$, $p = .294$, $\eta_p^2 = 0.06$, $p(H_0|D) = 0.70$; the three-way interaction, $F(1, 18) = 0.01$, $p = .937$, $\eta_p^2 < 0.001$, $p(H_0|D) = 0.82$). These findings indicate that the increased JSE during cathodal aMFC stimulation as compared to sham stimulation cannot be explained by an effect of order of active stimulation.

Appendix C

We performed an additional ANOVA including the factors Stimulation (sham_a vs. sham_c) and Compatibility (compatible vs. incompatible) in order to rule out any differences between the sham conditions. The analysis revealed a significant main effect of Compatibility, $F(1, 19) = 127.11$, $p < .001$, $\eta_p^2 = 0.84$, $p(H_1|D) > 0.99$, showing longer RTs on incompatible trials (304 ms) as compared to compatible trials (283 ms). Importantly, we neither found a significant main effect of Stimulation, $F(1, 19) = 0.02$, $p = .89$, $\eta_p^2 = 0.001$, $p(H_0|D) = 0.83$, nor an interaction of Stimulation and Compatibility, $F(1, 19) = 0.10$, $p = .75$, $\eta_p^2 = 0.004$, $p(H_0|D) = 0.80$. These findings indicate that the absence of rTPJ stimulation effects on the JSE cannot be due to potential differences in RTs between the two sham stimulation conditions.

Since no differences between both sham conditions were observed, both conditions were collapsed and included in an additional ANOVA with the factors Stimulation (sham_collapsed vs. cathodal vs. anodal) and Compatibility (compatible vs. incompatible). The analysis revealed significant main effects of Compatibility, $F(1, 25) = 108.06$, $p < .001$, $\eta_p^2 = 0.81$, $p(H_1|D) > 0.99$, and Stimulation, $F(2, 50) = 6.06$, $p < .01$, $\eta_p^2 = 0.20$, $p(H_1|D) = 0.66$. The interaction of Stimulation and Compatibility was not significant, $F(2, 50) = 0.63$, $p = .538$, $\eta_p^2 = 0.03$, $p(H_0|D) = 0.97$.