Ann. N.Y. Acad. Sci. ISSN 0077-8923

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES
Issue: *The Year in Cognitive Neuroscience*

The stimulated social brain: effects of transcranial direct current stimulation on social cognition

Roberta Sellaro,[1] Michael A. Nitsche,[2,3,4] and Lorenza S. Colzato[1]

[1]Cognitive Psychology Unit & Leiden Institute for Brain and Cognition, Leiden University, the Netherlands. [2]Department of Clinical Neurophysiology, Georg-August University Göttingen, Germany. [3]Leibniz Research Centre for Working Environment and Human Resources, Dortmund, Germany. [4]Department of Neurology, University Medical Hospital Bergmannsheil, Bochum, Germany

Address for correspondence: Roberta Sellaro, Cognitive Psychology Unit & Leiden Institute for Brain and Cognition, Leiden University, Wassenaarseweg 52, 2333 AK Leiden, the Netherlands. r.sellaro@fsw.leidenuniv.nl

Transcranial direct current stimulation (tDCS) is an increasingly popular noninvasive neuromodulatory tool in the fields of cognitive and clinical neuroscience and psychiatry. It is an inexpensive, painless, and safe brain-stimulation technique that has proven to be effective in modulating cognitive and sensory–perceptual functioning in healthy individuals and clinical populations. Importantly, recent findings have shown that tDCS may also be an effective and promising tool for probing the neural mechanisms of social cognition. In this review, we present the state-of-the-art of the field of tDCS research in social cognition. By doing so, we aim to gather knowledge of the potential of tDCS to modulate social functioning and social decision making in healthy humans, and to inspire future research investigations.

Keywords: brain stimulation; transcranial direct current stimulation; social cognition; emotion regulation; self–other representations; morality

Introduction

The last few decades have witnessed a significant advancement in noninvasive technologies for interacting safely and painlessly with the brain to induce direct and/or indirect changes in cortical excitability.[1,2] As an adjunct to imaging techniques, which deliver mainly correlational information, brain stimulation techniques allow researchers to test more directly circuit-based theories, and to infer causal relationships among a given stimulated neurotransmitter/brain area and a (assumed) related cognitive function.[1,3]

Among these techniques, transcranial direct current stimulation (tDCS)[4] has become recognized as a promising tool in neuroscience research for understanding the relationship between brain and behavior in healthy humans and clinical populations.[3] Indeed, several studies have provided converging evidence showing that tDCS is suited to modulate cognitive[5–8] and sensory–perceptual functioning,[9] and to ameliorate symptoms of several neurological and psychiatric disorders.[10] By comparison, only a limited number of studies have assessed the effects of tDCS on social cognition. Notwithstanding the paucity of research in this area, there is sufficient evidence to foresee the potential of this technique to study and modulate the physiological bases of social functioning and social decision making.

In this review, we intend to discuss the currently available findings from recent studies that have successfully applied tDCS to probe the neural underpinnings of social behavior in healthy individuals. By providing this overview, we aim to help gain a better understanding of the potential of tDCS for making a unique contribution to the field of social cognition. Before reviewing these studies, we provide a summary of the mechanisms through which tDCS presumably modulates cortical excitability and the factors that mediate its cortical and behavioral effects. We conclude with some critical remarks to point out the methodological shortcomings of the reviewed studies, with the ultimate goal of stressing

doi: 10.1111/nyas.13098

the need for researchers to use more rigorous procedures that take into consideration factors known to influence tDCS. Such a consideration would facilitate a better interpretation of the results and, possibly, provide a more homogeneous picture of the published literature. We hope to inform future studies on how to design proper tDCS experiments so as to exploit, at best, the advantages offered by this technique and to move the field forward.

Transcranial direct current stimulation

Given that previous reviews[11–13] have already extensively covered the basic principles and mechanisms of action of tDCS, here we provide only a brief recap of the current knowledge.

In the classical protocols, tDCS delivers a low-intensity constant current, varying between 1 and 2 mA, via relatively large (25–35 cm^2) electrodes that are applied on the participants' scalp above brain regions of interest for a few minutes (5–20 min). At least two electrodes with opposite polarities, a positively charged anode and a negatively charged cathode, are needed, with the resulting current flowing from the anode toward the cathode. A limited but sufficient portion of the applied current enters the brain and is capable of altering spontaneous neural activity and excitability.[13] Over the last few years, new protocols have been developed that are assumed to deliver more focal effects, or network stimulation, by aid of smaller electrodes or multielectrode arrangements often based on computational modeling.[14–16]

The current applied to the brain via tDCS is not of sufficient magnitude to generate action potentials.[13] Rather, tDCS causes a subthreshold modulation of the resting membrane potential of cortical neurons, altering their likelihood of firing and thereby affecting spontaneous cortical activity.[17–19] The tDCS-induced shifts in the resting membrane potential are largely, although not entirely (see below), determined by the polarity of the stimulation. Anodal stimulation causes a slight depolarization of the resting membrane potential, which increases the probability of neural firing and, consequently, cortical excitability.[17–19] In contrast, cathodal stimulation leads to a slight hyperpolarization of the resting membrane potential and, hence, decreased probability of neural firing and excitability. [17–19] Changes in neural activity are observed during the stimulation period, and when the current is delivered for a sufficient period of time (i.e., at least 9–10 min), such changes can remain for longer than 1 h after the stimulation.[13,19–21] This makes it possible to assess the cortical and behavioral effects of tDCS both during (online) and after (offline) stimulation. Although online and offline tDCS-induced changes in cortical excitability are associated with similar neurophysiological effects, they seem to depend on different mechanisms.[11] Broadly speaking, the primary effects of both anodal and cathodal tDCS during stimulation appear to solely depend on subthreshold membrane polarization.[22,23] Conversely, the aftereffects of tDCS seem to depend more on synaptic modulation, which is assumed to depend on strengthening (anodal tDCS) or weakening (cathodal tDCS) glutamatergic synapses, as well as a reduction of GABAergic activity independent from stimulation polarity.[22–25] However, activity of neuromodulators, including dopamine, acetylcholine, and serotonin, seems to play a role as well.[26–29]

The tDCS-induced changes in cortical excitability have been found to result in corresponding behavioral effects, the direction of which is assumed to depend on the relation between the effects of stimulation polarity and task-dependent alterations of brain physiology.[8,13] However, it is worth noting that all that is known about the physiological effects of tDCS, including the aforementioned link between tDCS-induced cortical and behavioral changes, comes primarily from studies that have focused on motor cortex excitability. Therefore, these principles do not necessarily apply one-to-one to stimulation of nonmotor areas, as the available evidence in fact would suggest.[30]

Besides the polarity of stimulation, tDCS-induced physiological and behavioral effects depend on a variety of other factors, such as electrode montage and size, current density, intensity and duration of the stimulation, as well as state-dependency (i.e., the initial brain state) and intersubject variability involving cortical anatomy, genetic polymorphisms, and/or psychological and motivational factors[30–35] (for further details, see Critical remarks section below). Among them, electrode montage is a crucial factor—and not just because it determines the polarity of the stimulation. As previously mentioned, tDCS needs at least two electrodes to work; typically, one electrode (i.e., the target electrode) is placed over the brain area of interest and the other (i.e., the return electrode) over another region (either cephalic or extracephalic). When both electrodes are placed on the scalp (i.e., bipolar cortical electrode montage),[36] not just the target electrode but also the return electrode will have a functional effect on the area where it is placed, thus introducing an important confounding factor when such a functional effect is not desired. To avoid that, researchers may opt for the use of an extracephalic electrode (i.e., monopolar extracephalic electrode montage) or a larger cephalic return electrode. Indeed, the use of a larger return electrode has been shown to be an effective and easy way to allow a functional monopolar montage because of smaller current density, when current strength is kept constant.[14] Even in that case, however, the position of the return electrode will affect the physiological effects of tDCS, because it determines current flow direction through the brain. Current flow direction in relation to neuronal orientation is critical for the effects and direction of the effects of tDCS; to be effective, the electrical field has to meet the long axis of a neuron, and electrical field orientation in relation to neuronal orientation will determine excitability-enhancing or -diminishing effects. This is the case because current has to enter and leave a given neuron to be effective. Because of higher receptor and ion channel density at the soma and axon hillock, it is assumed that current flow direction at these areas determines the effects of tDCS at the cellular level. In accordance with this, it has been demonstrated that, dependent on neuronal orientation, tDCS of identical current flow direction has antagonistic effects in hippocampal slices,[37] and that in the human brain, return electrode positions anterior and posterior to a target electrode result in different effects with identical target electrode stimulation polarity.[38,39]

tDCS effects on social cognition

The growing interest in studying social cognition has been undoubtedly fueled by the availability of neuroimaging methods, such as functional magnetic resonance imaging (fMRI), that have provided neuroscientists with the possibility to explore the neural correlates of social cognitive phenomena. Yet, because of the known limitations inherent in neuroimaging methods, our understanding of these phenomena remains far from conclusive.

Noninvasive brain stimulation techniques, such as transcranial magnetic stimulation (TMS) and tDCS, may allow a more precise investigation of the neural underpinnings of social behavior. Indeed, the use of TMS has significantly contributed to knowledge in this field.[40] Compared to TMS, however, tDCS adds some features that makes the latter technique a tool the field could really benefit from. First, tDCS is safe, painless, inexpensive, portable, and easy to apply. Second, tDCS does not produce distracting acoustic noise or muscle twitching that can interfere with task performance. Third, tDCS is associated with only mild and short-lasting skin sensations (i.e., tingling and itching sensations under the electrodes) that can be induced by short stimulation (e.g., 30 s) as well, without producing any lasting physiological effect,[19] a property that ensures the possibility to implement a reliable sham (placebo) condition. [41–44] More importantly, in the virtue of its intrinsic features, tDCS can be administered more easily than TMS to several individuals simultaneously, which offers a unique opportunity to study social behavior in real-life situations and in conditions that require the simultaneous interaction of a number of subjects.[45] In comparison to TMS, tDCS is an intrinsically neuromodulatory technique. This means that TMS, by means of induction of action potentials during task performance, can more easily than tDCS disrupt task-related activity, whereas online tDCS would influence this activity more gradually, which might be important for, for example, boosting plasticity in learning tasks.[46] On the other hand, both techniques are able to induce neuroplasticity; thus, for offline stimulation to modulate brain states both tools are similarly well suited. One advantage of TMS, as compared to tDCS, is its superior temporal and spatial resolution; however, attempts have been made to enhance the focality of tDCS effects, even though physical focality might not always translate one-to-one to physiological or functional specificity. In addition, both repetitive pulse TMS and tDCS have been shown to induce alterations of remote functional networks,[47,48] and tDCS seems to be able to modulate relatively specifically task-related connectivity.[49]

As previously mentioned, the application of tDCS in this field is still at its very beginning and, to date, only few aspects of social cognition have been tackled, and not even extensively. Below, we provide an overview of the currently available studies and

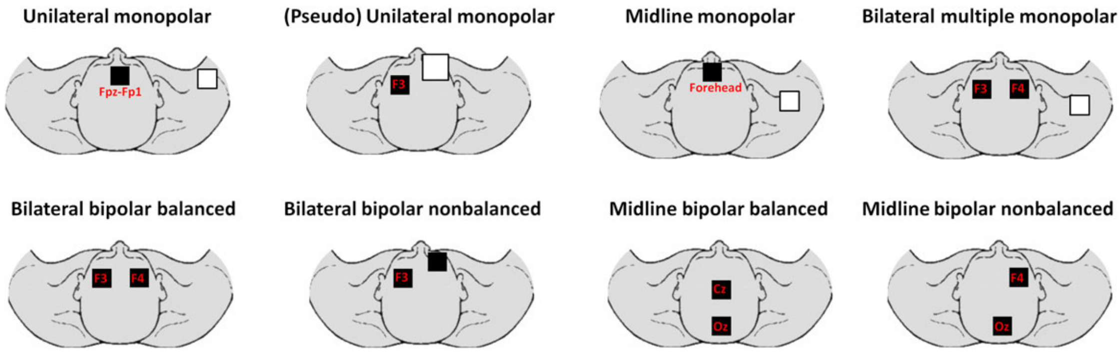

Figure 1. Schematic representations of tDCS montages used in the reviewed studies. Only one example for each type of montage is provided. For an extensive description of the different types of tDCS montages, see Nasseri *et al.*[36]

related findings. For the sake of simplicity, we divide these studies into three separate, although interconnected areas: modulation of socially provoked affective reactions, self–other representations, and morality. For each area, we provide a table with a brief description of the reviewed studies in terms of design, relevant tDCS parameters, and electrode placement (see also Fig. 1).

Modulation of socially provoked affective reactions

In this section, we describe recent studies that used tDCS to probe the role of prefrontal cortex areas in modulating affective reactions triggered by threat to social belongingness, interpersonal provocation, or vicariously experienced pain (see Table 1).

A first interesting line of research comprises three studies[50–52] that used tDCS to assess the possible causal role of the right ventrolateral prefrontal cortex (VLPFC) in modulating emotional reactions, such as feelings of social pain and aggressiveness[53,54] that are typically encountered when experiencing social exclusion, a condition that can be a threat to one of the fundamental human needs, that is, the need to belong.[55] In these studies, social exclusion (vs. inclusion) was manipulated during right VLPFC stimulation by engaging the participant in a virtual ball-tossing game[56] with two other alleged players who were computer-programmed to either exclude or include him/her. After completion of the game and once the stimulation ceased, reactions to social exclusion were evaluated. In the first of these studies, Riva *et al.*[50] found that excitability-enhancing (anodal) tDCS applied over the right VLPFC of socially excluded participants, compared to sham stimulation, reduced significantly levels of social pain and hurt feelings. Complementary results were observed in a follow-up study in which socially excluded participants who received excitability-reducing (cathodal) tDCS of the right VLPFC, compared to those who received either sham or cathodal tDCS to a control area, showed higher levels of social pain and hurt feelings.[51] Further converging evidence was obtained in a third study[52] in which aggressive reactions following social exclusion were tested, and socially excluded participants who received right VLPFC anodal tDCS, compared to the sham group, were found to behave less aggressively toward the alleged perpetrator of the social exclusion.

Taken together these results seem to corroborate the hypothesis that a causal link exists between right VLPFC activity and regulation of emotional reactions[57] associated with threats to social belongingness. However, as previously pointed out, the use of bipolar cortical electrode montage (see also "Critical remarks" section below) limits the specificity of stimulation effects and, thus, calls for further research to verify such a causal link.

Other studies have focused on affective reactions caused by interpersonal anger-producing provocation.[58,59] These studies exploited tDCS to corroborate the hypothesis that prefrontal hemispheric asymmetries may differentially mediate anger-related reactions.[60,61] Higher left (vs. right) or higher right (vs. left) prefrontal activity was induced by placing tDCS electrodes bilaterally over the left and right dorsolateral prefrontal cortex (DLPFC) so as to simultaneously increase cortical activity in one hemisphere and decrease it

Table 1. Summary of the design and relevant tDCS parameters

Study	Target region(s)	Location of target electrode(s)	Location of return electrode	Montage	Stimulation types	Sample size and design	tDCS protocol and stimulation parameters	Blinded to stimulation type (experimenter/participant)	Control area	Control task
Riva *et al.*[50]	Right VLPFC	F6; 25 cm^2	Left supraorbital area; 35 cm^2	Bilateral bipolar nonbalanced	Anodal, sham	79, between participants	Offline and online; 1.5 mA for 15 min (sham: 30 s)	Yes/yes	No	No
Riva *et al.*[51]	Right VLPFC or right PPC (control area; Exp. 2)	F6 or P2 (control area); 25 cm^2	Left supraorbital area; 35 cm^2	Bilateral bipolar nonbalanced	Cathodal, sham	82 (Exp. 1) and 40 (Exp. 2), between participants	Offline and online; 1.5 mA for 20 min (sham: 30 s)	Yes/yes	Yes	No
Riva *et al.*[52]	Right VLPFC	F6; 25 cm^2	Left supraorbital area; 35 cm^2	Bilateral bipolar nonbalanced	Anodal, sham	80, between participants	Offline and online; 1.5 mA for 20 min (sham: 30 s)	Yes/yes	No	No
Hortensius *et al.*[58]	Left/right DLPFC	F3/F4; 35 cm^2	n.a.	Bilateral bipolar balanced	Left anodal–right cathodal, left cathodal–right anodal, sham	60, between participants	Offline; 2 mA for 15 min (sham: 30 s)	Yes/yes	No	No
Kelley *et al.*[59]	Left/right DLPFC	F3/F4; 35 cm^2	n.a.	Bilateral bipolar balanced	Left anodal–right cathodal, left cathodal–right anodal, sham	90, between participants	Offline; 2 mA for 15 min (sham: 30 s)	Yes/yes	No	No
Boggio *et al.*[76]	Left DLPFC, left M1 (control area), V1 (control area)	F3, C3 (control area), Oz (control area); 35 cm^2	Right supraorbital area; 35 cm^2	Bilateral bipolar nonbalanced; midline bipolar nonbalanced (V1 stimulation)	Anodal, sham	23, within participants	Online; 2 mA for 5 min (sham: 30 s)	Yes/yes	Yes	No
Wang *et al.*[77]	Left DLPFC	F3; 35 cm^2	FP2; 25 cm^2	Bilateral bipolar nonbalanced	Anodal, cathodal, sham	27, between participants	Offline; 2 mA for 5 min (sham: 30 s)	Not specified/yes	No	No
Rêgo *et al.*[78]	Left/right DLPFC	F3/F4; 35 cm^2	n.a.	Bilateral bipolar balanced	Left anodal–right cathodal, left cathodal–right anodal, sham	24, between participants	Offline and online; 2 mA for 15 min (sham: 30 s)	Yes/yes	No	No

Studies are listed as they occurred in the text. VLPFC, ventrolateral prefrontal cortex; DLPFC, dorsolateral prefrontal cortex; M1, primary motor cortex; V1, visual cortex; Exp., experiment. Electrodes location was defined according to the International 10–20 electrode placement system. Montreal Neurological Institute (MNI) coordinates were provided for those studies that did not employ the International 10–20 electrode placement system. To define the electrode placement, we referred to the framework for categorizing tDCS electrode montages recently proposed by Nasseri *et al.*[36] The sample size refers to the number of participants the statistical analyses relied on.

in the contralateral one (i.e., left/right prefrontal-hemispheric dominance). Anger was elicited by exposing participants, at the end of the stimulation period, to an interpersonal insult (i.e., an insulting feedback on an essay written before the stimulation). In the study of Hortensius *et al.*,[58] participants who received stimulation to induce left prefrontal-hemispheric dominance (left anodal–right cathodal tDCS) were significantly more aggressive than those who received either stimulation to induce right prefrontal-hemispheric dominance (right anodal–left cathodal tDCS) or sham—a finding that fits with the hypothesis that higher left (vs. right) frontal cortical activity predicts angry approach-oriented responses (e.g., reactive aggression).[62] In a follow-up study, Kelly *et al.*[59] observed greater state rumination associated with anger feelings in the group of participants who received tDCS to induce right prefrontal-hemispheric dominance, as compared to participants who received either sham or tDCS to induce left prefrontal-hemispheric dominance. The finding that higher right (vs. left) prefrontal cortical activity is associated with increased anger-induced rumination is particularly noteworthy as it supports the emerging idea that the right prefrontal cortex is also involved in behavioral inhibition[60,61] and not just in avoidance motivation.[63–65]

Taken together these results support the hypothesis that prefrontal hemispheric asymmetries may play a critical role in determining reactive reactions to anger-producing provocation.[60,61] Yet, as we will argue later on, for such a conclusion to be supported it would be necessary for follow-up studies to rule out the possibility that modulation of activity in one hemisphere alone may be sufficient to produce the observed effects.

Finally, a third line of research investigated the neural processes involved in the modulation of emotional–affective reactions provoked when viewing images of other humans in pain-related situations.[66,67] All these studies focused on the role of the DLPFC, which has repetitively proven, also by means of tDCS, to be involved in emotion regulation and pain relief[68–75] and, as such, is a likely candidate area for modulating affective reactions to others' pain.[68,69] Boggio *et al.*[76] found that anodal tDCS applied over the left DLPFC while confronting participants with emotionally aversive images of human pain led them to perceive these images as less unpleasant and lowered feelings of emotional discomfort/pain, as compared to baseline pre-tDCS assessment, sham tDCS, and anodal tDCS applied over two control areas. Inconsistent results were observed by Wang *et al.*[77] They found that participants who received left DLPFC anodal tDCS before being presented with pictures of other persons experiencing painful situations judged other's pain as more intense (i.e., they showed increased pain empathy) than those who received sham or cathodal tDCS, while pain-related self-unpleasantness scores were left unaffected. Instead, Rêgo *et al.*[78] investigated the lateralized role of the DLPFC. During bilateral stimulation of the DLPFC (left anodal–right cathodal, left cathodal–right anodal, or sham), pupil dilation was measured while participants watched emotionally charged movies showing people under painful situations, which they had to rate in terms of emotional valence and arousal. At the end of the stimulation, participants filled in a self-pain perception scale requiring them to rate how much they experienced negative feelings during the video-clips presentation. Results showed that, compared to the sham group, participants who received left cathodal–right anodal tDCS, but not those who received left anodal–right cathodal tDCS, showed decreased perception of intensity and valence for other's pain and increased pupil dilation response, with the latter result possibly reflecting increased cognitive demands due to reappraisal processes.[79] In contrast, both active stimulations were found to affect self-pain perception ratings, which in both cases were lower (i.e., higher affective modulation) than ratings of the sham group. According to the authors, these results support a recent hypothesis that both left and right DLPFC mediate affective and emotional regulation, but operate according to different strategies, that is, by reinterpreting the meaning of the affective responses and by psychologically distancing the self from emotional stimuli, respectively.[71]

Taken together, the results of this last set of studies provide evidence that the DLPFC is somehow involved in modulating affective reactions to others' pain.[68,69] However, because of the heterogeneity of the observed effects, the specific contribution of the left and right DLPFC is still far from being clear. Several reasons for these heterogeneous effects may be identified, mostly related to differences pertaining to the stimulation protocol (online vs.

offline), electrode placement and montage, the specific task implemented to address tDCS effects, and the extremely low sample size that especially the last two studies[77,78] rely on. On the basis of these considerations and taking in mind the previously mentioned methodological shortcomings associated with the use of bilateral bipolar (balanced and nonbalanced) cortical electrode montage, more research is still needed to provide unequivocal support for the hypothesis that the DLPFC is causally involved in modulating affective reactions to others' pain and to shed light on its lateralized role.

All in all, the results described in this section corroborate the role of prefrontal cortex areas in modulating affective reactions triggered by different social contexts causing social distress. Therefore, these results provide a compelling example of how tDCS can be a valuable tool to corroborate hypotheses stemming from neuroimaging studies. However, as we previously mentioned and as we further discuss later on, more efforts should be made to improve the quality of scientific work, for example, by systematically varying the stimulation parameters (e.g., stimulation protocol, current intensity, and electrode montage) to gain a better understanding of the results and to account for possible inconsistences across studies.

Self–other representations

Under this area we mainly describe tDCS studies that have examined neural processes pertaining to the ability to handle mental representations of both the self and other people, a fundamental ability for humans to engage in successful social interactions[80–82] (see Table 2).

Santiesteban *et al.*[83] tested the role of the right temporoparietal junction (TPJ) in mediating the ability to distinguish and switch between concurrently activated self-related and other-related representations (i.e., self–other control).[80,81,84] In this study, anodal, cathodal, or sham tDCS was delivered over the right TPJ before participants executed three tasks, two of them requiring self–other control: a perspective taking task,[85] which requires to inhibit one's own perspective and to enhance that of the other,[80] and a control-of-imitation task,[86] which instead requires to inhibit the other person's motor representations and to enhance the motor representations of one's own intended action.[81,84] The third task was a mental state attribution task[87] that did not require self–other representations to be controlled.[a] The results showed that anodal tDCS, compared to cathodal and sham tDCS, improved online control of self–other representations in both perspective-taking and control-of-imitation tasks, without affecting performance in the mental state attribution task. These findings therefore corroborate the hypothesis that the right TPJ may enable self–other control over coactivated representations by inhibiting one's own or the other person's representations, depending on the task demands.[80,81,84,88] Moreover, the absence of any tDCS modulatory effect on mental state attribution suggests that online self–other control and mental state attribution may rely on independent processes.[81] Interestingly, these results were replicated in a follow-up study in which Santiestaban *et al.*[89] obtained evidence that the assumed role of the TPJ in self–other control is not restricted to the right TPJ, but extends to the left TPJ as well. Indeed, they found that, compared to anodal stimulation of a control area, anodal TPJ stimulation improved self–other control in both perspective-taking and control-of-imitation tasks, regardless of whether the right or left TPJ was

[a]In the perspective taking task, participants were instructed to move objects to specific locations within a virtual bookshelf based on the instructions received by another person who looked at the bookshelf from a different perspective. Proper task performance requires to suppress one's own perspective and to take the perspective of the other. In the control of imitation task, participants were instructed to perform an index or middle finger lifting movement depending on the identity of a number displayed on a computer screen. The number appeared between the fingers of a hand showing an index or middle finger lifting movement that could be either compatible or incompatible with the requested finger movement. Proper task performance requires participants to inhibit an imitative response in order to execute the correct one when such a response is incompatible with the observed movement. In the mental state attribution task (i.e., a self-referential task), on each trial, participants were preinstructed to make judgments (i.e., a mental or a physical judgment) about either themselves or another (famous) person. Compared to the previous tasks, no online control of coactivated self-related and other-related representations was required.

Table 2. Summary of the design and relevant tDCS parameters

Study	Target region(s)	Location of target electrode(s)	Location of return electrode	Montage	Stimulation types	Sample size and design	tDCS protocol and stimulation parameters	Blinded to stimulation type (experimenter/ participant)	Control area	Control task
Santiesteban *et al.*[83]	Right TPJ	CP6; 35 cm^2	Cz; 35 cm^2	Midline bipolar nonbalanced	Anodal, cathodal, sham	49, between participants	Offline; 1 mA for 20 min (sham: 30 s)	Not specified/ yes	No	No
Santiesteban *et al.*[89]	Right TPJ, left TPJ, occipital cortex (control area)	CP6, CP5, Oz (control area); 35 cm^2	Cz; 35 cm^2	Midline bipolar nonbalanced; midline bipolar balanced (Oz stimulation)	Anodal	44, between participants	Offline; 1 mA for 20 min	Not specified/ yes	Yes	No
Hogeveen *et al.*[90]	Right TPJ, right IFC	CP6, FC6; 35 cm^2	Cz; 35 cm^2	Midline bipolar nonbalanced	Anodal, sham	48, between participants	Offline; 1 mA for 20 min (sham: 30 s)	Not specified/ yes	No	No
Sowden *et al.*[97]	Right TPJ, occipital cortex (control area)	CP6, Oz (control area); 35 cm^2	Cz; 35 cm^2	Midline bipolar nonbalanced; midline bipolar balanced (Oz stimulation)	Anodal	33, between participants	Offline; 1 mA for 20 min	Not specified/ not specified	Yes	No
Conson *et al.*[98]	Left/right DLPFC	F3/F4; 35 cm^2	n.a.	Bilateral bipolar balanced	Left anodal–right cathodal, left cathodal–right anodal, sham	16, within participants	Offline; 1 mA for 15 min (sham: 20 s)	Not specified/ yes	No	No
Liepelt *et al.*[101]	aMFC (Exp. 1) or right TPJ (Exp. 2)	MNI (aMFC): 0, 55, 13, MNI (right TPJ): 63, -50–23; 35 cm^2	MNI (right TPJ): 63, -50–23; MNI (aMFC): 0, 55, 13; 100 cm^2	(Pseudo) unilateral monopolar	Anodal, cathodal, sham	20 (Exp. 1) and 26 (Exp. 2), between (anodal vs. cathodal) and within (active vs. sham) participants	Online; 1 mA for 20 min (sham: 30 s)	No/yes	No	No
Sellaro *et al.*[105]	MPFC	Fpz; 35 cm^2	Oz; 35 cm^2	Midline bipolar balanced	Anodal, cathodal, sham	60, between participants	Online; 1 mA for 20 min (sham: 35 s)	No/yes	No	No

Studies are listed as they occurred in the text. TPJ, temporoparietal junction; IFC, inferior frontal cortex; aMPFC, anterior medial frontal cortex; DLPFC, dorsolateral prefrontal cortex; MPFC, medial prefrontal cortex. Exp., experiment. Electrodes location was defined according to the International 10–20 electrode placement system. Montreal Neurological Institute (MNI) coordinates were provided for those studies that did not employ the International 10–20 electrode placement system. To define the electrode placement, we referred to the framework for categorizing tDCS electrode montages that was recently proposed by Nasseri *et al.*[36] The sample size refers to the number of participants the statistical analyses relied on.

stimulated. Again, they did not observe any tDCS-induced effect on performance in a task tapping the ability to infer other's mental states. Further converging evidence supporting the role of the right TPJ in the control of imitation was provided by Hogeveen *et al.*[90] In this study, following anodal tDCS of either the right TPJ or the right inferior frontal cortex (IFC), or sham stimulation, participants were confronted with two critical tasks: a control-of-imitation task,[86] in which better performance requires to inhibit imitative behavior, and a social interaction task,[91] in which higher mimicry levels signal better social interaction. Replicating the results observed by Santiesteban *et al.*,[83,89] right TPJ anodal tDCS, compared to sham, improved online control over imitative behavior, without affecting the degree of mimicry in the social interaction task. Instead, anodal tDCS of the right IFC, compared to sham tDCS, was found to have a dissociable effect on both tasks: similarly to right TPJ anodal tDCS, it improved the ability to inhibit imitation, but it also increased imitative behavior in the social interaction task. These results support the hypothesis that the right IFC, compared to the right TPJ, has a more direct impact on imitation, leading to either inhibit or enhance imitation, depending on task demands.[92–96]

Taken together, studies provide evidence for the critical role of the right TPJ in mediating the online control of concurrently activated self-related and other-related representations. Interestingly enough, such a role for the right TPJ has recently been proven to mediate the ability to detect lies as well.[97] Indeed, using an offline protocol, Sowden *et al.*[97] showed that anodal tDCS of the right TPJ, compared to anodal tDCS of a control area, improved lie-detection performance when participants were confronted with statements in which the to-be-judged opinions were in conflict with those held by the participants, a condition that in a previous experiment of the same study was found to significantly impair lie detection.

Other studies have provided evidence supporting the role of prefrontal cortex areas in handling self–other representations. For instance, Conson *et al.*[98] assessed the effects of bilateral stimulation (left anodal–right cathodal, left cathodal–right anodal, and sham) of the DLPFC on visual perspective taking.[99] They found that the ability to judge a visual scene from another person's point of view (but not from one's own perspective) was significantly impaired following left cathodal–right anodal tDCS, a finding that was taken to suggest that the right DLPFC may be involved in inhibiting shared action representations so as to prioritize self-related representations.[100]

In another study, the role of the anterior medial frontal cortex (aMFC) in self–other action discrimination was examined. Liepelt *et al.*[101] delivered anodal, cathodal, or sham stimulation over either the aMFC or the right TPJ while participants performed a joint Simon task,[102–104] a turn-taking paradigm requiring the participant and a confederate to perform complementary parts of the same task. A more pronounced joint Simon effect (i.e., reduced self–other action discrimination) was found during excitability-reducing cathodal tDCS of the aMFC, but not of the right TPJ, a finding that supports the assumed role of the aMPF in enhancing the representation of self-generated actions.[81,84,94] The absence of any tDCS effect during right TPJ stimulation instead further supports the view that this area mediates online self–other control only when self-related and other-related representations are concurrently activated, as is the case in perspective taking and control-of-imitation tasks.[80,81,84,88] As such, these results are in line with the results observed by Santiesteban *et al.*[83,89]

Finally, Sellaro *et al.*[105] investigated the possible causal role of the medial prefrontal cortex (MPFC) in counteracting stereotypes activation resulting from in-group versus outgroup categorization,[106,107] a situation in which self and other representations can be seen as polarized on a positive versus negative dimension. In this study, increased cognitive control over stereotypes activation with a resulting reduced implicit negative bias toward a social out-group was found in the group of participants who received online anodal tDCS of the MPFC, compared to participants who received cathodal or sham stimulation, a finding that speaks in favor of the idea that the MPFC may contribute to self-regulatory and cognitive-control processes implemented to overcome unwanted responses driven by stereotypes activation.[108,109] Interestingly, in the same sample of participants MPFC tDCS was not effective in modulating interpersonal trust,[110] although MPFC activity has been linked to the degree of mutual trust.[111–113]

Taken together, the studies reviewed in this section provide evidence supporting the role of the TPJ and prefrontal cortex areas in mediating several facets pertaining to the ability to handle self–other representations, and speak in favor of the possibility that the use of tDCS may represent a promising way to improve social abilities. However, important methodological limitations are present in the majority of them. These limitations mainly concern the use of bilateral bipolar cortical electrode montages, which may introduce important confounding factors. For instance, all studies investigating the role of the TPJ[86,92,93,97] placed the cathodal (return) electrode over Cz, which is very close to the primary motor cortex, an area considered to be part of the mirror neuron system[114] and, thus, likely to be involved in mediating imitative behavior.[115,116] The remaining studies also present problems related to the electrode placement, such as the use of a bilateral bipolar-balanced montage (in Conson *et al.*)[98]—which does not allow one to ascribe the observed effects to stimulation of the left or of the right DLPFC (or both)—and the use of a very large distance between the target and the return electrodes (in Sellaro *et al.*),[105] which may have prevented a selective stimulation of the target area.[117]

Although these criticisms do not apply to the study of Liepelt *et al.*,[101] who opted for the use of a larger, and thus functionally inert, return electrode, follow-up studies are needed that vary the position of the return electrode to rule out the possibility that the observed effects emerged from interactions between the target and the return electrodes.[118]

Morality

In this section, we review recent studies that have applied tDCS to study the neural correlates of different facets pertaining to morality, such as moral judgment, social norm compliance, and deception (see Table 3).

A first line of research examined the neural correlates of moral judgments. Fumagalli *et al.*[119] exploited tDCS to corroborate the causal role of the ventral prefrontal cortex (VPC) in driving moral judgments on the basis of the emotional evaluation of harmful acts.[120,121] In this study, before and after having received anodal or cathodal tDCS of the VPC or of a control area, participants were presented with moral (personal and impersonal, i.e., high vs. low conflict, respectively) dilemmas[120,122] and asked to choose between utilitarian and nonutilitarian options (e.g., saving five lives by scarifying an innocent person vs. preserving the life of an innocent person even though this would cause five people to die). Mirroring the pattern of results observed in patients with focal damage to the ventromedial prefrontal cortex (VMPFC),[121] anodal tDCS of the VPC increased the proportion of utilitarian responses, but only in female participants, a finding that the authors interpreted as evidence that, in this specific case, anodal tDCS may have reduced, instead of increased, cortical excitability of the targeted area, thereby reducing the aversive emotional reactions to harmful acts. Alternative interpretations claiming for possible anodal tDCS-induced neurochemical changes in the brain (e.g., increased in dopamine levels) were considered as well. Therefore, although the results reported by Fumagalli *et al.*[119] are of interest, they call for further research to verify these claims. Besides that, this study represents a compelling example of how the use of tDCS may potentially benefit from combining this technique with neuroimaging methods to monitor changes in brain activity, thereby enabling researchers to verify speculations about the polarity of the tDCS-induced effects.

More recently, Kuehne *et al.*[123] tested the role of the DLPFC, which is thought to drive moral judgments on the basis of the cost–benefit analysis of the consequences of a given behavior[120,122] and as such, can be considered as the rational counterpart of the VMPFC. In this study, participants received active (either anodal or cathodal) and sham tDCS of the left DLPFC while reading high-conflict moral dilemmas. The participants were asked to judge the appropriateness of utilitarian actions. Compared to sham and cathodal tDCS, during anodal tDCS of the left DLPFC participants judged utilitarian actions as more inappropriate. This result is at odds with the hypothesis that the DLPFC is involved in counteracting the aversive emotional responses inherent in utilitarian decisions,[120,122] on the basis of what one would have expected to observe DLPFC anodal tDCS to induce a reduced aversion to harming others for a greater good. According to the authors, these results support the hypothesis that the DLPFC is not only involved in implementing rational cognitive control over emotional aversion, it also plays a crucial role in integrating emotional responses induced by contextual information appraisal.[124] However, other factors such as electrode montage (and, thus, current flow direction and possible confounding effects due to concurrent stimulation of the parietal cortex), stimulation protocol, and the high-intensity current—which may invert the stimulation polarity[32]—may account for the unexpected results. Therefore, the results reported by Kuehne *et al.*[123] cannot be seen as conclusive with regard to the role of the left DLPFC in making moral judgments.

In another recent study, Sellaro *et al.*[125] used tDCS to corroborate the assumed role of the right TPJ in mediating mental state reasoning during moral judgments.[126,127] Before and after receiving anodal, cathodal, or sham tDCS over the right TPJ, participants were asked to judge the moral permissibility of hypothetical situations in which protagonists produced either a negative or neutral outcome, on the basis of either a negative or neutral belief that they were causing harm or no harm, respectively.[127] The results showed that right TPJ anodal tDCS, compared to cathodal and sham tDCS, enhanced the influence of belief information on moral

Table 3. Summary of the design and relevant tDCS parameters

Study	Target region(s)	Location of target electrode(s)	Location of return electrode	Montage	Stimulation types	Sample size and design	tDCS protocol and stimulation parameters	Blinded to stimulation type (experimenter/participant)	Control area (yes/no)	Control task (yes/no)
Fumagalli *et al.*[119]	VPC, occipital cortex (control area)	Forehead (bilaterally); occipital cortex (control area); 54 cm^2	Right deltoid; 64 cm^2	Midline monopolar	Anodal, cathodal	78, between participants	Offline; 2 mA for 15 min	Not specified/yes	Yes	No
Kuene *et al.*[123]	Left DLPFC	F3; 35 cm^2	P4; 35 cm^2	Bilateral bipolar nonbalanced	Anodal, cathodal, sham	54, between (anodal vs. cathodal) and within (active vs. sham) participants	Online; 2 mA for 20 min (sham: 30 s)	Not specified/yes	No	No
Sellaro *et al.*[125]	Right TPJ (Exp. 1) or left supraorbital area (Exp. 2); 35 cm^2	CP6 (Exp. 1) or left supraorbital area (Exp. 2); 35 cm^2	Left supraorbital area; 35 cm^2 (Exp. 1); or right TPJ (Exp. 2); 100 cm^2	Bilateral bipolar nonbalanced (Exp. 1), (pseudo) unilateral monopolar (Exp. 2)	Anodal, cathodal, sham (Exp. 1); cathodal (Exp. 2)	60, between participants	Offline; 1 mA for 20 min (sham: 35 s)	No/yes	No	No
Knoch *et al.*[45]	Right DLPFC	F4; 35 cm^2	Left supraorbital area; 100 cm^2	(Pseudo) unilateral monopolar	Cathodal, sham	64, between participants	Online; 1 mA for less than 15 min (sham: 30 s)	Not specified/yes	No	No
Civai *et al.*[136]	MPFC	Fpz-Fp1; 35 cm^2	Right arm; 35 cm^2	Unilateral monopolar	Cathodal, sham	40, between participants	Online; 2 mA for 15–20 min (sham: 30 s)	Not specified/yes	No	No
Ruff *et al.*[141]	Right LPFC	MNI: 52, 28, 14; 35 cm^2	Cz; 35 cm^2	Midline bipolar nonbalanced	Anodal, cathodal, sham	63, between participants	Online; 1 mA for 15–20 min (sham: 30 s)	Yes/yes	No	Yes
Nihonsugi *et al.*[142]	Right DLPFC	MNI: 44, 34, 22; 35 cm^2	Oz; 35 cm^2	Midline bipolar nonbalanced	Anodal, sham	22, within participants	Online; 2 mA for 15–20 min (sham: 30 s)	Not specified/yes	No	No
Priori *et al.*[151]	Bilateral DLPFC	F3/F4; 32 cm^2	Right deltoid; 64 cm^2	Bilateral multiple monopolar	Bilateral anodal, bilateral cathodal, sham	15, within participants	Offline; 1.5 mA for 10 min (sham: 10 s)	Not specified/yes	No	No
Mameli *et al.*[152]	Bilateral DLPFC	F3/F4; 32 cm^2	Right deltoid; 64 cm^2	Bilateral multiple monopolar	Bilateral anodal, sham	20, within participants	Offline; 2 mA for 15 min (sham: 10 s)	Yes/yes	No	Yes
Fecteau *et al.*[155]	Left/right DLPFC	F3/F4; 35 cm^2	n.a.	Bilateral bipolar balanced	Left anodal–right cathodal, left cathodal–right anodal, sham	36, between participants	Offline; 2 mA for 20 min (sham: 30 s)	Yes/yes	No	Yes
Karim *et al.*[156]	Right aPFC	PF2; 24 cm^2	PO3; 24 cm^2	Bilateral bipolar nonbalanced	Cathodal, sham (Exp. 1); anodal, sham (Exp. 2); cathodal, sham (Exp. 3: control task)	22 (Exp. 1) and 22 (Exp. 2) and 20 (Exp. 3), within participants	Online; 1 mA for 13 min (sham: 30 s)	Yes/yes	No	Yes

Studies are listed as they occurred in the text. VPC, ventral prefrontal cortex; DLPFC, dorsolateral prefrontal cortex; LPFC, lateral prefrontal cortex; TPJ, temporoparietal junction; MPFC, medial prefrontal cortex; aPFC, anterior prefrontal cortex; Exp., experiment. Electrodes location was defined according to the International 10–20 electrode placement system. Montreal Neurological Institute (MNI) coordinates were provided for those studies that did not employ the International 10–20 electrode placement system. To define the electrode placement, we referred to the framework for categorizing tDCS electrode montages that was recently proposed by Nasseri *et al.*[36] The sample size refers to the number of participants the statistical analyses relied on.

judgments, leading this group of participants to assign less blame to accidental harms (belief neutral, outcome negative), a result that confirms the hypothesis that right TPJ recruitment during moral judgments reflects reliance on the agent's innocent intention.[126,128] Importantly, in a follow-up experiment, Sellaro *et al.*[125] showed that this outcome was specific to stimulation of the right TPJ and could not be attributed to the position of the return electrode, which was placed over the contralateral supraorbital area. However, also in this case, follow-up studies employing an extracephalic montage and comparing offline versus online stimulation would be appropriate to restrict the observed effects to stimulation of the right TPJ, and to assess possible state-dependent tDCS effects, with the goal to elucidate the effects of variations in experimental parameters on tDCS outcomes.

Taken together, these results show that tDCS, applied over prefrontal cortex areas or over the right TPJ, can affect people's decisions in making moral judgments, but they also highlight the need to use more rigorous approaches to assess the effects of stimulation parameters and to restrict tDCS outcomes to the target area in the attempt to achieve an unequivocal interpretation of the results. Moreover, it would be of interest for future studies to compare DLPFC and TPJ stimulation when performing different moral judgment tasks to better elucidate the functional role of these areas.

A second line of research investigated the neural processes underpinning social norm compliance. Complying to social norms often requires people to override self-interest in favor of normatively valued goals.[129] A striking example of nonutilitarian behavior is provided by performance typically observed in the ultimatum game (UG):[130] people tend to reject offers that are viewed as a violation of the fairness norm to punish the proposer, although such a decision comes at the expense of their self-interest as well.[131]

Knoch *et al.*[45] assessed the possible causal role of the DLPFC in mediating reactions to fairness. In this study, participants in groups of 18 (6 responders and 12 proposers) performed the UG while those playing the role of the responder received cathodal or sham tDCS of the right DLPFC. The results showed that cathodal tDCS, compared to sham, increased the probability of accepting unfair offers, a finding that can be taken to suggest that cortical excitability reduction of the right DLPFC may have helped participants to override fairness motives so as to accomplish self-interest.[132–135] Similar results were found by Civai *et al.*[136] who, however, stimulated the left MPFC, another area previously linked to rejection of unfair offers.[132,137–140] In this study, a modified version of the UG was used in which participants (all acting in the role of the responder) played both for themselves and on behalf of an unknown person. Compared to sham tDCS, excitability-diminishing cathodal stimulation increased the probability of accepting unfair offers, but only when participants played for themselves (i.e., when they were the target of the unfairness). These results favor the hypothesis that the MPFC plays a specific role in evaluating self-damaging unfairness,[138–140] rather than being involved in the general evaluation of fairness.[132,137] Given the overlap in findings between the studies of Knoch *et al.*[45] and Civai *et al.*,[136] it would be of interest to conduct further experiments to compare stimulation of different prefrontal cortex areas during and/or before the execution of different tasks tapping reactions to fairness, while using identical stimulation parameters and an extracephalic montage (as in Civai *et al.*[136]).

Ruff *et al.*[141] examined the role of the LPFC in mediating social norm compliance as a function of the threat to be punished. During stimulation (anodal, cathodal, or sham) of the right LPFC, groups of 12 participants (all receiving tDCS stimulation) performed an economic game requiring them to decide how much money to share with a partner. Two different conditions were compared, one in which making an unfair offer to the partner had no consequence (baseline condition: voluntary norm compliance), and one in which the partner could punish the participant if s/he deemed the offer unfair (punishment condition: sanction-induced norm compliance). The results showed that, compared to the sham group, anodal tDCS increased sanction-induced norm compliance and decreased voluntary norm compliance, whereas cathodal tDCS decreased sanction-induced norm compliance and increased voluntary norm compliance. Recently, Nihonsugi *et al.*[142] extended these findings by showing that the right DLPFC is implicated in social-norm compliance not just when sanctions are imposed, but whenever the intentions and beliefs of others are taken into consideration and the individual is motivated to minimize guilt aversion.

Participants received anodal or sham tDCS of the right DLPFC while performing a modified version of the trust game[143] suitable to assess the relative impact of intention-based (guilt-aversion) and outcome-based (iniquity-aversion) decisions. The results showed that, compared to sham stimulation, anodal tDCS selectively increased intention-based decisions, without affecting outcome-based decisions, which represents the first evidence that guilt aversion and inequity aversion may rely on different neural substrates. Yet, because of the large distance between the target and the return electrodes, it is difficult to say whether the observed effects were due to selective modulation of the right DLPFC or rather due to a more widespread and nonselective modulation over the cortex.[117]

Taken together, tDCS studies on social norm compliance suggest a broader role for the right DLPFC that is likely not restricted to the top-down regulation of self-interest[133–135] and may extend to the modulation of the relative weight of intention-based economic decisions. But again, because of critical differences across studies in terms of stimulation parameters, electrode montage, and employed tasks, further research is needed to address the functional role of different, but nearby, prefrontal cortex areas in mediating social norm compliance, as well as to shed light on the possible role of different stimulation parameters.

Finally, a third line of research used tDCS to investigate the neural correlates of deception, a complex process previously linked to two prefrontal cortex areas, the DLPFC[144–150] and the anterior prefrontal cortex (aPFC).[146,150] In the study of Priori *et al.*,[151] before and after anodal, cathodal, or sham tDCS delivered bilaterally over the DLPFC, participants performed a computer task instructing them to answer truthfully or to produce two types of lies: denying a fact that really happened (i.e., denying to have seen a picture when they had been previously presented with that picture) and producing a false response about an event that did not happen (i.e., pretending to have seen a picture when they had not been previously presented with that picture). Compared to cathodal and sham tDCS, anodal tDCS reduced the speed of lie production for the former, but not for the latter, type of lie, thus suggesting that the two types of lies are likely modulated by different neural substrates. Mameli *et al.*[152] extended these findings by testing the effects of tDCS delivered bilaterally over the DLPFC on general knowledge and personal information deception. Before and after tDCS (anodal and sham), participants performed a modified version of the guilty knowledge task,[153,154] requiring them to produce truthful or lie responses to questions referring to personal information and general knowledge. Anodal tDCS was found to speed-up lie responses concerning general knowledge information, but not personal information, thereby suggesting that different brain networks are devoted for different types of lies. Importantly, no effects of tDCS were observed on performance in the control task. Evidence that tDCS of the DLPFC can also affect lies concerning personal information was provided by a recent study of Fecteau *et al.*,[155] who used an electrode montage different from that used in the studies of Priori *et al.*[151] and Mameli *et al.*[152] Before and after tDCS of the DLPFC (right anodal–left cathodal, right cathodal–left anodal, or sham), participants performed two tasks, each requiring to produce different types of lies: untruthful responses about daily personal information and untruthful responses about personal past experience with either memorized lies or spontaneous lies. Two control tasks were implemented to confirm that possible tDCS-induced effects were confined to deception. Both active tDCS conditions were found to affect deception, but not performance in the control tasks. Specifically, right anodal–left cathodal DLPFC tDCS reduced the latency for generating lies about daily personal information and memorized lies, but not spontaneous lies about past experience. In contrast, right cathodal–left anodal DLPFC tDCS reduced the latency for generating spontaneous and memorized lies about past experience, but did not affect the latency for generating untruthful answers about daily personal information.

Finally, Karim *et al.*[156] assessed the possible causal role of the right aPFC in lie production, in a paradigm in which participants were let to decide which questions they would answer truthfully and which ones with a lie, with the further instruction to appear a "skillful liar." After being involved in a mock crime, participants received tDCS (cathodal or sham in experiment 1; anodal or sham in experiment 2) of the aPFC while undergoing an interrogation[153,154] with an investigator they had to deceive. Compared to sham tDCS, improved deceptive behavior (i.e., shorter reaction times in telling the lie but not the truth) associated with reduced skin-conductance response (measured during the interrogation) and lower feelings of guilt (assessed at the end of the interrogation) was observed during cathodal, but not anodal aPFC tDCS. Notably, cathodal aPFC tDCS was ineffective in modulating performance in a control task (experiment 3), thereby confirming that tDCS-induced effects were confined to deceptive behavior.

Taken together, tDCS studies on deception corroborate the hypothesis that a causal link may exist between prefrontal cortex activity and processes involved in the generation of untruthful answers. Moreover, the results of these studies seem to support the hypothesis that different types of deception may rely on a similar neural network involving the DLPFC.[144–150] However, because of the electrode montages used in the reviewed studies and given the absence of any objective measure of tDCS-induced changes in brain activity, the hemispheric contribution of this area to deceptive processes remains to be determined. For instance, the use of a multiple monopolar montage, as in the studies of Priori *et al.*[151] and Mameli *et al.*,[152] has been found to stimulate brain networks rather than a specific brain region.[16] Therefore, it would be of interest for future studies to replicate and extend these results to investigate functional connectivity among brain regions. Likewise, as previously pointed out, the use of a bilateral bipolar-balanced montage, as in Fecteau *et al.*,[155] does not allow one to establish whether tDCS effects on deception were due to modulation of activity in either the left or the right DLPFC, or were rather due to changing the balance of activity between the two hemispheres. Thus, follow-up studies are necessary to shed light on this issue. Finally, the study of Karim *et al.*[156] suggests that areas other than the DLPFC may be causally linked to deception. However, in this study a bilateral bipolar montage was used, which makes it difficult to unequivocally ascribe the observed effects to stimulation of the target area (i.e., the right aPFC). Given that the aPFC is quite close to the DLPFC, and because of tDCS limitations with regard to focality, further research is required to compare the effect of stimulation of different cortical areas in mediating different types of lies, as well as to address the possible modulatory effect of different stimulation parameters.

In sum, the results reviewed in this last section provide striking examples of the potential of tDCS to alter very complex social processes, such as moral decision making, social norm compliance, and deception. However, the aforementioned considerations about the reviewed studies also suggest that more systematic studies should be carried out to assess the role of stimulation parameters and protocols, and to identify optimal electrode placements that guarantee selective stimulation of the target areas.

Critical remarks

The available studies show that tDCS is able to affect social functioning and social decision-making processes. Notably, the studies of Knoch *et al.*[45] and Ruff *et al.*[141] provide an excellent demonstration, but sadly unique in this kind, of how tDCS, in contrast with other techniques (e.g., fMRI, TMS), can be successfully used in contexts requiring the simultaneous interaction of many subjects. This paves the way for being able, in the near future, to study the neural correlates of social cognition outside the research laboratory by immersing participants in real-life situations. As such, this technique may be seen as a highly useful add-in tool for social neuroscience research aimed at gaining a better understanding of the neural correlates of social behavior. Moreover, by virtue of its proven property to modify social behavior, tDCS may be a useful tool to treat social deficits typically observed in clinical conditions such as schizophrenia, autism, and psychopathy. However, it is important to acknowledge that for such a possibility to be realized, extensive research is needed to verify whether the observed tDCS-induced changes in social behavior are maintained over time. Previous studies on cognitive functioning have suggested that repetitive sessions of tDCS can increase the effects of stimulation,[4] but it remains to be established whether the same applies to social functioning. Therefore, systematic studies assessing the impact of multiple stimulation sessions and the risk of incurring potential side effects are compulsory.

It is interesting to note that the majority of the studies reviewed above assessed the role of prefrontal cortex areas, in particular the DLPFC. Taken together, the results of these studies seem to suggest that the DLPFC is involved in mediating a variety of social phenomena, such as reactions to interpersonal provocation, pain empathy, perspective taking, moral judgment, social norm compliance, and deception. Therefore, it is tempting to conclude that these phenomena may share common neural processes, a conclusion that seems reasonable especially if one considers that a clear distinction between the three areas we divided the reviewed studies does not exist. However, because of the heterogeneity across different studies in terms of study design and tDCS parameters, and of the methodological shortcomings of the reviewed studies (see also below), such a conclusion is premature and it may probably be wrong.

As we have already pointed out, in some occasions, the studies described in the present review produced results inconsistent to each other and/or with previous literature. These discrepancies are likely (but not necessarily) due to differences across studies in relevant aspects of their method, including stimulation parameters (e.g., intensity and duration of the stimulation, online vs. offline stimulation, electrode size and number, scalp placement), study design, and the specific task/questionnaire used to assess tDCS-induced behavioral changes.[30–34] Therefore, more research is needed to elucidate the reasons for these discrepancies.

Several studies have shown that variations in stimulation parameters can cause different amounts of electrical current to be delivered and thus can produce different tDCS effects. For instance, there is evidence that prolonged stimulation duration and higher current intensity can invert the polarity of the stimulation.[32] Likewise, on some occasions, online and offline tDCS have been found to produce different and even opposite effects.[11,157] Also, although tDCS effects are not focal, electrode positioning is critical. Studies addressing tDCS-induced physiological changes, and computational modelling studies of the expected current flow, have found significant differences in the amount and distribution of current delivered to the brain depending on the relative positions of the electrodes.[158–160] For instance, it has recently been shown that a drift of just 1 cm in electrode position causes a significant alteration of the distribution of the current flow and of the intensity of stimulation delivered to the brain.[160] This is not surprising given that tDCS physiological and behavioral effects depend on the relation between current flow direction and neuronal orientation in the target area,[37] and thus variations in electrode position are likely to alter such a relation. Importantly, this means that undesired drifts in electrode position during stimulation, due to inappropriate placement of the electrodes, can seriously undermine reproducibility of the effects. Therefore, more effort should be made to create optimized procedures that guarantee stable placement of electrodes on the scalp.

In addition, possible confounding factors arising from the position of the return electrode need to be considered. With regard to this issue, it is worth noting that the majority of the studies reviewed above used bipolar cortical electrode montages, which do not allow one to ascertain whether the observed behavioral outcomes are due to stimulation of the target area, or they emerged from an interaction between target and return electrodes.[118] This issue assumes even more importance if one considers that in the majority of the aforementioned studies, the return electrode was placed over the contralateral supraorbital area, which is located over the frontal poles. Given that most of tDCS studies, including those reviewed above, have reported effects mainly associated with stimulation of areas within the prefrontal cortex,[5,6] placing the return electrode over supraorbital regions is likely to introduce a substantial confounding factor. As previously mentioned, a potential solution is to use a larger return electrode that, because of the lower current density beneath it, is likely to be functionally ineffective[14] and, hence, allows to achieve a unipolar stimulation.[161] Another possible solution is to use a monopolar montage with an extracephalic return electrode that, however, requires higher stimulation intensity.[162] Yet, except for a few studies,[45,101,119,136] no others opted for these solutions. Researchers need to be aware that tDCS effects will depend on the relative current flow direction/electrical field orientation with respect to neuronal orientation in the target area.[37] Thus, the effects observed when using a larger return electrode (i.e., functional monopolar cephalic electrode arrangement) are likely to differ from the effects observed when using an extracephalic electrode (i.e., monopolar extracephalic electrode arrangement). Alternative solutions, such as HD electrode positions,[14–16,163] should be considered and tested.

Possible shortcomings may also be identified for those studies that used bilateral stimulation to alter hemispheric balance.[58,59,78,98,155] Indeed, it is worth noting that when the effects associated with a bilateral stimulation are not compared with those involving a unilateral stimulation, one cannot conclusively establish whether changing the balance of activity between the two hemispheres is really necessary for the observed effects to be found, or whether modulation of activity in one hemisphere alone is sufficient to produce the observed effects. Future studies need to better consider these issues in order to avoid undermining the possibility of achieving a univocal and clear interpretation of the observed results.

Extensive and systematic studies also must be carried out to probe the role played by different stimulation parameters, including the timing of the stimulation and electrode placement, with the goal of assessing whether, and to what extent, variations in stimulation parameters affect tDCS-induced changes in social behavior. Additionally, researchers should be encouraged to publish null effects that, besides reducing publication bias, might inform future studies on how to best design tDCS experiments and provide an objective assessment of tDCS effects. To this end, preregistered confirmatory research, in which researchers disclose methods, hypotheses, and the to-be-performed statistical analyses before data collection,[164] is highly recommended.

Another and related important consideration pertains to the role of individual differences in determining response to tDCS. Computational models have suggested that individual anatomical differences can affect the current flow through the brain during the stimulation.[31,165] Moreover, hair thickness may contribute to the observed variability in response to tDCS, as it may lead researchers to use a large amount of saline solution to saturate thicker hair.[166] Oversaturation of the electrode sponges can cause saline solution to spill over the sponges, and the area of the scalp being covered in saline will receive stimulation. Obviously, such a condition would severely undermine reproducibility of tDCS application and effects. Also, tDCS effects have been found to depend on the baseline status of the brain, which can substantially vary across individuals.[167,168] There is also evidence that other factors, such as age, gender, genetic polymorphisms, and psychological/motivational factors, can influence the direction and the extent of cortical and behavioral modulation.[34,35] Notably, of the reviewed studies mentioned above, only a few examined interindividual variability in response to tDCS but they limited the investigation to gender-related variability;[65,98,119] interestingly, the results of these studies extend previous observations suggesting that tDCS sensitivity may be gender specific.[169–172] To improve our understanding of whether, and to what extent, tDCS can modulate social behavior, it would be necessary for future studies to consider the role of interindividual differences. This implies testing sufficiently large samples to enable a deeper investigation of tDCS effects that take into consideration, and control for, the role of interindividual variability. Moreover, computer modeling of the current flow through the brain can provide valuable information to be used to create optimal electrode montages for a given target area and head anatomy.[173] A detailed investigation of the factors that may influence tDCS effects, in terms of both stimulation parameters and interindividual differences, is highly advisable, especially given the recent controversy about the effectiveness of tDCS in modulating cognitive processes.[174–178]

Another important issue that deserves attention is the fact that none of the reviewed studies mentioned above combined behavioral measures with either electrophysiological or neuroimaging methods, such as electroencephalography or fMRI, to monitor the impact of tDCS on brain activity, either independently or concurrently with task execution.[49,179–182] Such a combination would enable a more detailed interpretation of the results and would reduce uncertainty about the neural substrate that is modulated by tDCS. Indeed, given the lack of focality inherent in tDCS, it is important to ascertain whether, and how, stimulation delivered to the target area affects activity of adjacent and distal areas. This is essential for assessing the functional specificity of different and nearby areas in mediating a given behavior. Moreover, monitoring tDCS-induced changes in brain activity may reduce the tendency to advance circular interpretations by which the polarity of the stimulation is interpreted on the basis of the observed behavioral outcome (see, e.g., the study of Fumagalli *et al.*[119]). That said, studies aimed at replicating and extending the available findings by monitoring tDCS-induced changes in brain activity are highly recommended.

Additional critical considerations refer to the fact that only few of the reviewed studies implemented a control task[119,152,156] and/or stimulated a control area.[51,76,89,119] Introducing control tasks and stimulating control areas would guarantee a more precise investigation such to verify the specificity of the stimulation as well as the validity of the results and rule out plausible alternative interpretations. Moreover, stimulating a control area that is known to be irrelevant for the process under investigation may represent a useful alternative to the use of sham stimulation. It has been shown that when current is delivered at 1 mA the sham stimulation is reliable to blind participants;[41,43] however, higher stimulation intensities may compromise blinding, especially when using within-subjects designs.[44,183] Given that most of the reviewed studies used stimulation intensities of 1.5 mA or higher, doubts arise about whether participants were really blinded to the type of stimulation. Hence, it would be appropriate for future studies to assess explicitly the validity of the blinding procedure.

Taken together, the above-mentioned considerations suggest the necessity of conducting additional extensive research before concluding that tDCS is ready to be used to achieve a better understanding of social phenomena. Efforts aimed at enhancing the methodological quality of research (for a recent guide, see Ref. 184) within this emerging field may provide researchers with the unique opportunity to identify possible common neural processes underlying different social phenomena.

Conclusions

This review discusses how tDCS can modulate social behavior and social decision-making processes, and that it is a highly valuable tool for probing the neural underpinnings of social phenomena. However, our discussion also shows that more research is needed to exploit the potential of this technique of making a unique contribution to this field.

Conflicts of interest

M.A.N. is a member of the advisory board of Neuroelectrics, Barcelona, Spain.

References

1. George, M.S. & G. Aston-Jones. 2009. Noninvasive techniques for probing neurocircuitry and treating illness: vagus nerve stimulation (VNS), transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS). *Neuropsychopharmacology* **35:** 301–316.
2. Fox, D. 2011. Brain buzz. *Nature* **472:** 156–159.
3. Filmer, H.L., P.E. Dux & J.B. Mattingley. 2014. Applications of transcranial direct current stimulation for understanding brain function. *Trends Neurosci.* **37:** 742–753.
4. Nitsche, M.A. & W. Paulus. 2011. Transcranial direct current stimulation—update 2011. *Restor. Neurol. Neurosci.* **29:** 463–492.
5. Kuo, M.F. & M.A. Nitsche. 2012. Effects of transcranial electrical stimulation on cognition. *Clin. EEG Neurosci.* **43:** 192–199.
6. Kuo, M.F. & M.A. Nitsche. 2015. Exploring prefrontal cortex functions in healthy humans by transcranial electrical stimulation. *Neurosci. Bull.* **31:** 198–206.
7. Cohen-Kadosh, R. 2015. Modulating and enhancing cognition using brain stimulation: science and fiction. *J. Cogn. Psychol. (Hove)* **27:** 141–163.
8. Shin, Y.I., Á. Foerster & M.A. Nitsche. 2015. Transcranial direct current stimulation (tDCS)—application in neuropsychology. *Neuropsychologia* **69:** 154–175.
9. Costa, T.L., O.M. Lapenta, P.S. Boggio & D.F. Ventura. 2015. Transcranial direct current stimulation as a tool in the study of sensory–perceptual processing. *Atten. Percept. Psychophys.* **77:** 1813–1840.
10. Brunoni, A.R., M.A. Nitsche, N. Bolognini, *et al.* 2012. Clinical research with transcranial direct current stimulation (tDCS): challenges and future directions. *Brain Stimul.* **5:** 175–195.
11. Stagg, C.J. & M.A. Nitsche. 2011. Physiological basis of transcranial direct current stimulation. *Neuroscientist* **17:** 37–53.
12. Medeiros L.F., I.C.C. de Souza, L.P. Vidor, *et al.* 2012. Neurobiological effects of transcranial direct current stimulation: a review. *Front. Psychiatry* **3:** 110.
13. Nitsche, M.A., L.G. Cohen, E.M. Wassermann, *et al.* 2008. Transcranial direct current stimulation: state of the art 2008. *Brain Stimul.* **1:** 206–223.
14. Nitsche, M.A., S. Doemkes, T. Karakoese, *et al.* 2007. Shaping the effects of transcranial direct current stimulation of the human motor cortex. *J. Neurophysiol.* **97:** 3109–3117.
15. Edwards, D., M. Cortes, A. Datta, *et al.* 2013. Physiological and modeling evidence for focal transcranial electrical brain stimulation in humans: a basis for high-definition tDCS. *Neuroimage* **74:** 266–275.
16. Ruffini, G., M.D. Fox, O. Ripolles, *et al.* 2014. Optimization of multifocal transcranial current stimulation for weighted cortical pattern targeting from realistic modeling of electric fields. *Neuroimage* **89:** 216–225.
17. Bindman, L.J., O.C. Lippold & J.W.T. Redfearn. 1964. The action of brief polarizing currents on the cerebral cortex of the rat during current flow and in the production of long-lasting after-effects. *J. Physiol.* **172:** 369–382.
18. Purpura, D.P. & J.G. McMurtry. 1965. Intracellular activities and evoked potential changes during polarization of motor cortex. *J. Neurophysiol.* **28:** 166–185.
19. Nitsche, M.A. & W. Paulus. 2000. Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. *J. Physiol.* **527:** 633–639.
20. Nitsche, M.A. & W. Paulus. 2001. Sustained excitability elevations induced by transcranial DC motor cortex stimulation in humans. *Neurology* **57:** 1899–1901.

21. Nitsche, M.A., D. Liebetanz, A. Antal, *et al.* 2003. Modulation of cortical excitability by weak direct current stimulation—technical, safety and functional aspects. *Suppl. Clin. Neurophysiol.* **56:** 255–276.
22. Nitsche, M.A., K. Fricke, U. Henschke, *et al.* 2003. Pharmacological modulation of cortical excitability shifts induced by transcranial direct current stimulation in humans. *J. Physiol.* **553:** 293–301.
23. Nitsche, M.A., A. Seeber, K. Frommann, *et al.* 2005. Modulating parameters of excitability during and after transcranial direct current stimulation of the human motor cortex. *J. Physiol.* **568:** 291–303.
24. Nitsche, M.A., W. Jaussi, D. Liebetanz, *et al.* 2004. Consolidation of human motor cortical neuroplasticity by D-cycloserine. *Neuropsychopharmacology* **29:** 1573–1578.
25. Stagg, C.J., J. Best, M. Stephenson, *et al.* 2009. Polarity-sensitive modulation of cortical neurotransmitters by transcranial stimulation. *J. Neurosci.* **29:** 5202–5206.
26. Kuo, M.F., J. Grosch, F. Fregni, *et al.* 2007. Focusing effect of acetylcholine on neuroplasticity in the human motor cortex. *J. Neurosci.* **27:** 14442–14447.
27. Monte-Silva, K., M.F. Kuo, N. Thirugnanasambandam, *et al.* 2009. Dose-dependent inverted U-shaped effect of dopamine (D_2-like) receptor activation on focal and nonfocal plasticity in humans. *J. Neurosci.* **29:** 6124–6131.
28. Nitsche, M.A., M.F. Kuo, J. Grosch, *et al.* 2009. D_1-receptor impact on neuroplasticity in humans. *J. Neurosci.* **29:** 2648–2653.
29. Nitsche, M.A., F. Müller-Dahlhaus, W. Paulus, *et al.* 2012. The pharmacology of neuroplasticity induced by noninvasive brain stimulation: building models for the clinical use of CNS active drugs. *J. Physiol.* **590:** 4641–4662.
30. Jacobson, L., M. Koslowsky & M. Lavidor. 2012. tDCS polarity effects in motor and cognitive domains: a meta-analytical review. *Exp. Brain Res.* **216:** 1–10.
31. Datta, A., D. Truong, P. Minhas, *et al.* 2012. Inter-individual variation during transcranial direct current stimulation and normalization of dose using MRI-derived computational models. *Front. Psychiatry* **3:** 91.
32. Batsikadze, G., V. Moliadze, W. Paulus, *et al.* 2013. Partially non-linear stimulation intensity-dependent effects of direct current stimulation on motor cortex excitability in humans. *J. Physiol.* **591:** 1987–2000.
33. Tremblay, S., J.F. Lepage, A. Latulipe-Loiselle, *et al.* 2014. The uncertain outcome of prefrontal tDCS. *Brain Stimul.* **7:** 773–783.
34. Krause, B. & R. Cohen-kadosh. 2014. Not all brains are created equal: the relevance of individual differences in responsiveness to transcranial electrical stimulation. *Front. Syst. Neurosci.* **8:** 25.
35. Li, L.M., K. Uehara & T. Hanakawa. 2015. The contribution of interindividual factors to variability of response in transcranial direct current stimulation studies. *Front. Cell. Neurosci.* **9:** 181.
36. Nasseri, P., M.A. Nitsche & H. Ekhtiari. 2015. A framework for categorizing electrode montages in transcranial direct current stimulation. *Front. Hum. Neurosci.* **9:** 54.
37. Kabakov, A.Y., P.A. Muller, A. Pascual-Leone, *et al.* 2012. Contribution of axonal orientation to pathway-dependent modulation of excitatory transmission by direct current stimulation in isolated rat hippocampus. *J. Neurophysiol.* **107:** 1881–1889.
38. Antal, A., T.Z. Kincses, M.A. Nitsche, *et al.* 2004. Excitability changes induced in the human primary visual cortex by transcranial direct current stimulation: direct electrophysiological evidence. *Invest. Ophthalmol. Vis. Sci.* **45:** 702–707.
39. Accornero, N., P.L. Voti, M. La Riccia, *et al.* 2007. Visual evoked potentials modulation during direct current cortical polarization. *Exp. Brain Res.* **178:** 261–266.
40. Hétu, S., V. Taschereau-Dumouchel & P.L. Jackson. 2012. Stimulating the brain to study social interactions and empathy. *Brain Stimul.* **5:** 95–102.
41. Gandiga, P.C., F.C. Hummel & L.G. Cohen. 2006. Transcranial DC stimulation (tDCS): a tool for double-blind sham-controlled clinical studies in brain stimulation. *Clin. Neurophysiol.* **117:** 845–850.
42. Poreisz, C., K. Boros, A. Antal, *et al.* 2007. Safety aspects of transcranial direct current stimulation concerning healthy subjects and patients. *Brain Res. Bull.* **72:** 208–214.
43. Ambrus, G.G., H. Al-Moyed, L. Chaieb, *et al.* 2012. The fade-in–short stimulation–fade out approach to sham tDCS–reliable at 1 mA for naive and experienced subjects, but not investigators. *Brain Stimul.* **5:** 499–504.
44. Palm, U., E. Reisinger, D. Keeser, *et al.* 2013. Evaluation of sham transcranial direct current stimulation for randomized, placebo-controlled clinical trials. *Brain Stimul.* **6:** 690–695.
45. Knoch, D., M.A. Nitsche, U. Fischbacher, *et al.* 2008. Studying the neurobiology of social interaction with transcranial direct current stimulation—the example of punishing unfairness. *Cereb. Cortex* **18:** 1987–1990.
46. Nitsche, M.A., A. Schauenburg, N. Lang, *et al.* 2003. Facilitation of implicit motor learning by weak transcranial direct current stimulation of the primary motor cortex in the human. *J. Cogn. Neurosci.* **15:** 619–626.
47. Bestmann, S., J. Baudewig, H.R. Siebner, *et al.* 2005. BOLD MRI responses to repetitive TMS over human dorsal premotor cortex. *Neuroimage* **28:** 22–29.
48. Polanía, R., W. Paulus, A. Antal, *et al.* 2011. Introducing graph theory to track for neuroplastic alterations in the resting human brain: a transcranial direct current stimulation study. *Neuroimage* **54:** 2287–2296.
49. Polanía R, M.A. Nitsche & W. Paulus. 2011. Modulating functional connectivity patterns and topological functional organization of the human brain with transcranial direct current stimulation. *Hum. Brain Mapp.* **32:** 1236–1249.
50. Riva, P., L.J. Romero Lauro, C.N. DeWall, *et al.* 2012. Buffer the pain away stimulating the right ventrolateral prefrontal cortex reduces pain following social exclusion. *Psychol. Sci.* **23:** 1473–1475.
51. Riva, P., L.J. Romero Lauro, A. Vergallito, *et al.* 2015. Electrified emotions: modulatory effects of transcranial direct stimulation on negative emotional reactions to social exclusion. *Soc. Neurosci.* **10:** 46–54.
52. Riva, P., L.J. Romero Lauro, C.N. DeWall, *et al.* 2015. Reducing aggressive responses to social exclusion using transcranial direct current stimulation. *Soc. Cogn. Affect. Neurosci.* **10:** 352–356.

53. Eisenberger, N.I., M.D. Lieberman & K.D. Williams. 2003. Does rejection hurt? An fMRI study of social exclusion. *Science* **302:** 290–292.
54. Riva, P., J.H. Wirth & K.D. Williams. 2011. The consequences of pain: the social and physical pain overlap on psychological responses. *Eur. J. Soc. Psychol.* **41:** 681–687.
55. Baumeister R.F. & M.R. Leary. 1995. The need to belong: desire for interpersonal attachments as a fundamental human motivation. *Psychol. Bull.* **117:** 497–529.
56. Williams, K.D., C.K. Cheung & W. Choi. 2000. Cyberostracism: effects of being ignored over the internet. *J. Pers. Soc. Psychol.* **79:** 748–762.
57. Cohen, J.R., E.T. Berkman & M.D. Lieberman. 2013. "Intentional and incidental self-control in ventrolateral prefrontal cortex." In *Principles of Frontal Lobe Function.* D.T. Stuss & R.T. Knight, Eds.: 417–444. New York: Oxford University Press.
58. Hortensius, R., D.J. Schutter & E. Harmon-Jones. 2012. When anger leads to aggression: induction of relative left frontal cortical activity with transcranial direct current stimulation increases the anger–aggression relationship. *Soc. Cogn. Affect. Neurosci.* **7:** 342–347.
59. Kelley, N.J., R. Hortensius & E. Harmon-Jones. 2013. When anger leads to rumination induction of relative right frontal cortical activity with transcranial direct current stimulation increases anger-related rumination. *Psychol. Sci.* **24:** 475–481.
60. Wacker, J., M.L. Chavanon, A. Leue, *et al.* 2008. Is running away right? The behavioral activation–behavioral inhibition model of anterior asymmetry. *Emotion* **8:** 232–249.
61. Harmon-Jones, E., P.A. Gable & C.K. Peterson. 2010. The role of asymmetric frontal cortical activity in emotion-related phenomena: a review and update. *Biol. Psychol.* **84:** 451–462.
62. Harmon-Jones, E., J.D. Sigelman, A. Bohlig, *et al.* 2003. Anger, coping, and frontal cortical activity: the effect of coping potential on anger-induced left frontal activity. *Cogn. Emot.* **17:** 1–24.
63. Harmon-Jones, E. 2004. Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biol. Psychol.* **67:** 51–76.
64. Carver, C.S. & E. Harmon-Jones. 2009. Anger is an approach-related affect: evidence and implications. *Psychol. Bull.* **135:** 183–204.
65. Dambacher, F., T. Schuhmann, J. Lobbestael, *et al.* 2015. Reducing proactive aggression through non-invasive brain stimulation. *Soc. Cogn. Affect. Neurosci.* **10:** 1303–1309.
66. Singer, T., B. Seymour, J. O'Doherty, *et al.* 2004. Empathy for pain involves the affective but not sensory components of pain. *Science* **303:** 1157–1162.
67. Jackson, P.L., A.N. Meltzoff & J. Decety. 2005. How do we perceive the pain of others? A window into the neural processes involved in empathy. *Neuroimage* **24:** 771–779.
68. Lorenz, J., S. Minoshima & K.L. Casey. 2003. Keeping pain out of mind: the role of the dorsolateral prefrontal cortex in pain modulation. *Brain* **126:** 1079–1091.
69. Godinho, F., M. Magnin, M. Frot, *et al.* 2006. Emotional modulation of pain: is it the sensation or what we recall? *J. Neurosci.* **26:** 11454–11461.
70. Peña-Gómez, C., D. Vidal-Piñeiro, I.C. Clemente, *et al.* 2011. Down-regulation of negative emotional processing by transcranial direct current stimulation: effects of personality characteristics. *PLoS One* **6:** e22812.
71. Ochsner, K.N., J.A. Silvers & J.T. Buhle. 2012. Functional imaging studies of emotion regulation: a synthetic review and evolving model of the cognitive control of emotion. *Ann. N.Y. Acad. Sci.* **1251:** E1–E24.
72. Brunoni, A.R., M.A. Vanderhasselt, P.S. Boggio, *et al.* 2013. Polarity- and valence-dependent effects of prefrontal transcranial direct current stimulation on heart rate variability and salivary cortisol. *Psychoneuroendocrinology* **38:** 58–66.
73. Wolkenstein, L., M. Zeiller, P. Kanske, *et al.* 2014. Induction of a depression-like negativity bias by cathodal transcranial direct current stimulation. *Cortex* **59:** 103–112.
74. Feeser, M., K. Prehn, P. Kazzer, *et al.* 2014. Transcranial direct current stimulation enhances cognitive control during emotion regulation. *Brain Stimul.* **7:** 105–112.
75. Maeoka, H., A. Matsuo, M. Hiyamizu, *et al.* 2012. Influence of transcranial direct current stimulation of the dorsolateral prefrontal cortex on pain related emotions: a study using electroencephalographic power spectrum analysis. *Neurosci. Lett.* **512:** 12–16.
76. Boggio, P.S., S. Zaghi & F. Fregni. 2009. Modulation of emotions associated with images of human pain using anodal transcranial direct current stimulation (tDCS). *Neuropsychologia* **47:** 212–217.
77. Wang, J., Y. Wang, Z. Hu, *et al.* 2014. Transcranial direct current stimulation of the dorsolateral prefrontal cortex increased pain empathy. *Neuroscience* **281:** 202–207.
78. Rêgo, G.G., O.M. Lapenta, L.M. Marques, *et al.* 2015. Hemispheric dorsolateral prefrontal cortex lateralization in the regulation of empathy for pain. *Neurosci. Lett.* **594:** 12–16.
79. Granholm, E. & S.R. Steinhauer. 2004. Pupillometric measures of cognitive and emotional processes. *Int. J. Psychophysiol.* **52:** 1–6.
80. Decety, J. & J.A. Sommerville. 2003. Shared representations between self and other: a social cognitive neuroscience view. *Trends Cogn. Sci.* **7:** 527–533.
81. Spengler, S., D.Y. von Cramon & M. Brass. 2009. Control of shared representations relies on key processes involved in mental state attribution. *Hum. Brain Mapp.* **30:** 3704–3718.
82. Sowden, S. & P. Shah. 2014. Self–other control: a candidate mechanism for social cognitive function. *Front. Hum. Neurosci.* **8:** 789.
83. Santiesteban, I., M.J. Banissy, C. Catmur, *et al.* 2012. Enhancing social ability by stimulating right temporoparietal junction. *Curr. Biol.* **22:** 2274–2277.
84. Brass, M., P. Ruby & S. Spengler. 2009. Inhibition of imitative behaviour and social cognition. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **364:** 2359–2367.
85. Keysar, B., D.J. Barr, J.A. Balin, *et al.* 2000. Taking perspective in conversation: the role of mutual knowledge in comprehension. *Psychol. Sci.* **11:** 32–38.
86. Brass, M., H. Bekkering, A. Wohlschläger, *et al.* 2000. Compatibility between observed and executed finger move-

ments: comparing symbolic, spatial, and imitative cues. *Brain Cogn.* **44:** 124–143.
87. Lombardo, M.V., B. Chakrabarti, E.T. Bullmore, *et al.* 2010. Atypical neural self-representation in autism. *Brain* **133:** 611–624.
88. Ruby, P. & J. Decety. 2004. How would you feel versus how do you think she would feel? A neuroimaging study of perspective-taking with social emotions. *J. Cogn. Neurosci.* **16:** 988–999.
89. Santiesteban, I., M.J. Banissy, C. Catmur, *et al.* 2015. Functional lateralization of temporoparietal junction: imitation inhibition, visual perspective-taking and theory of mind. *Eur. J. Neurosci.* **42:** 2527–2533.
90. Hogeveen, J., S.S. Obhi, M.J. Banissy, *et al.* 2015. Task-dependent and distinct roles of the temporoparietal junction and inferior frontal cortex in the control of imitation. *Soc. Cogn. Affect. Neurosci.* **10:** 1003–1009.
91. Chartrand, T.L. & J.A. Bargh. 1999. The chameleon effect: the perception–behavior link and social interaction. *J. Pers. Soc. Psychol.* **76:** 893–910.
92. Iacoboni, M., R.P. Woods, M. Brass, *et al.* 1999. Cortical mechanisms of human imitation. *Science* **286:** 2526–2528.
93. Heiser, M., M. Iacoboni, F. Maeda, *et al.* 2003. The essential role of Broca's area in imitation. *Eur. J. Neurosci.* **17:** 1123–1128.
94. Brass, M., J. Derrfuss & D.Y. von Cramon. 2005. The inhibition of imitative and over-learned responses: a functional double dissociation. *Neuropsychologia* **43:** 89–98.
95. Spengler, S., D.Y. von Cramon & M. Brass. 2010. Resisting motor mimicry: control of imitation involves processes central to social cognition in patients with frontal and temporo-parietal lesions. *Soc. Neurosci.* **5:** 401–416.
96. Sowden, S. & C. Catmur. 2015. The role of the right temporoparietal junction in the control of imitation. *Cereb. Cortex* **25:** 1107–1113.
97. Sowden, S., G.R. Wright, M.J. Banissy, *et al.* 2015. Transcranial current stimulation of the temporoparietal junction improves lie detection. *Curr. Biol.* **25:** 2447–2451.
98. Conson, M., D. Errico, E. Mazzarella, *et al.* 2015. Transcranial electrical stimulation over dorsolateral prefrontal cortex modulates processing of social cognitive and affective information. *PLoS One* **10:** e0126448.
99. Mazzarella E., A. Hamilton, L. Trojano, *et al.* 2012. Observation of another's action but not eye gaze triggers allocentric visual perspective. *Q. J. Exp. Psychol.* **65:** 2447–2460.
100. Rizzolatti G. & L. Craighero. 2004. The mirror-neuron system. *Annu. Rev. Neurosci.* **27:** 169–192.
101. Liepelt, R., B. Klempova, T. Dolk, *et al.* 2015. The medial frontal cortex mediates self–other discrimination in the Joint Simon task: a tDCS study. *J. Psychophysiol.* 10.1027/0269-8803/a000158.
102. Sebanz, N., G. Knoblich & W. Prinz. 2003. Representing others' actions: just like one's own? *Cognition* **88:** B11–B21.
103. Hommel, B., L.S. Colzato & W.P.M. van den Wildenberg. 2009. How social are task representations? *Psychol. Sci.* **20:** 794–798.
104. Dolk, T., B. Hommel, W. Prinz, *et al.* 2013. The (not so) social Simon effect: a referential coding account. *J. Exp. Psychol. Hum. Percept. Perform.* **39:** 1248–1260.
105. Sellaro, R., B. Derks, M.A. Nitsche, *et al.* 2015. Reducing prejudice through brain stimulation. *Brain Stimul.* **8:** 891–897.
106. Allport, G.W. 1979. *The Nature of Prejudice*. Reading, MA: Addison Wesley Publishing Company.
107. Greenwald, A.G. & M.R. Banaji. 1995. Implicit social cognition: attitudes self-esteem and stereotypes. *Psychol. Rev.* **102:** 4e27.
108. Amodio, D.M. & C.D. Frith. 2006. Meeting of minds: the medial frontal cortex and social cognition. *Nat. Rev. Neurosci.* **7:** 268–277.
109. Amodio, D.M., J.T. Kubota, E. Harmon-Jones, *et al.* 2006. Alternative mechanisms for regulating racial responses according to internal vs external cues. *Soc. Cogn. Affect. Neurosci.* **1:** 26–36.
110. Colzato, L.S., R. Sellaro, W.P.M. van den Wildenberg, *et al.* 2015. tDCS of medial prefrontal cortex does not enhance interpersonal trust. *J. Psychophysiol.* **29:** 131–134.
111. McCabe, K., D. Houser, L. Ryan, *et al.* 2001. A functional imaging study of cooperation in two-person reciprocal exchange. *Proc. Natl. Acad. Sci. U.S.A.* **98:** 11832–11835.
112. Delgado, M.R., R.H. Frank & E.A. Phelps. 2005. Perceptions of moral character modulate the neural systems of reward during the trust game. *Nat. Neurosci.* **8:** 1611–1618.
113. Krajbich, I., R. Adolphs, D. Tranel, *et al.* 2009. Economic games quantify diminished sense of guilt in patients with damage to the prefrontal cortex. *J. Neurosci.* **29:** 2188–2192.
114. Tkach, D., J. Reimer & N.G. Hatsopoulos. 2007. Congruent activity during action and action observation in motor cortex. *J. Neurosci.* **27:** 13241–13250.
115. Heiser, M., M. Iacoboni, F. Maeda, *et al.* 2003. The essential role of Broca's area in imitation. *Eur. J. Neurosci.* **17:** 1123–1128.
116. Catmur, C. & C. Heyes. 2011. Time course analyses confirm independence of imitative and spatial compatibility. *J. Exp. Psychol. Hum. Percept. Perform.* **37:** 409–421.
117. Neuling, T., S. Wagner, C.H. Wolters, *et al.* 2012. Finite-element model predicts current density distribution for clinical applications of tDCS and tACS. *Front. Psychiatry* **3:** 83.
118. Datta, A., M. Elwassif, F. Battaglia, *et al.* 2008. Transcranial current stimulation focality using disc and ring electrode configurations: FEM analysis. *J. Neural Eng.* **5:** 163–174.
119. Fumagalli, M., M. Vergari, P. Pasqualetti, *et al.* 2010. Brain switches utilitarian behavior: does gender make the difference? *PLoS One* **5:** e8865.
120. Greene, J.D., R.B. Sommerville, L.E. Nystrom, *et al.* 2001. An fMRI investigation of emotional engagement in moral judgment. *Science* **293:** 2105–2108.
121. Koenigs, M., L. Young, R. Adolphs, *et al.* 2007. Damage to the prefrontal cortex increases utilitarian moral judgements. *Nature* **446:** 908–911.
122. Greene, J.D., L.E. Nystrom, A.D. Engell, *et al.* 2004. The neural bases of cognitive conflict and control in moral judgment. *Neuron* **44:** 389–400.

123. Kuehne, M., K. Heimrath, H.J. Heinze, *et al.* 2014. Transcranial direct current stimulation of the left dorsolateral prefrontal cortex shifts preference of moral judgments. *PLoS One* **10:** e0127061.
124. Tassy, S., O. Oullier, Y. Duclos, *et al.* 2012. Disrupting the right prefrontal cortex alters moral judgement. *Soc. Cogn. Affect. Neurosci.* **7:** 282–288.
125. Sellaro, R., B. Güroğlu, M.A. Nitsche, *et al.* 2015. Increasing the role of belief information in moral judgments by stimulating the right temporoparietal junction. *Neuropsychologia* **77:** 400–408.
126. Young, L. & R. Saxe. 2009. Innocent intentions: a correlation between forgiveness for accidental harm and neural activity. *Neuropsychologia* **47:** 2065–2072.
127. Young, L., J.A. Camprodon, M. Hauser, *et al.* 2010. Disruption of the right temporoparietal junction with transcranial magnetic stimulation reduces the role of beliefs in moral judgments. *Proc. Natl. Acad. Sci. U.S.A.* **107:** 6753–6758.
128. Koster-Hale, J., R. Saxe, J. Dungan, *et al.* 2013. Decoding moral judgments from neural representations of intentions. *Proc. Natl. Acad. Sci. U.S.A.* **110:** 5648–5653.
129. Boyd, R. & P.J. Richerson. 2005. "Solving the puzzle of human cooperation." In *Evolution and Culture.* S.C. Levinson & P. Jaisson, Eds.: 105–132. Cambridge, MA: MIT Press.
130. Camerer, C.F. 2003. *Behavioral Game Theory: Experiments in Strategic Interaction.* Princeton, NJ: Princeton University Press.
131. Güth, W., R. Schmittberger & B. Schwarze. 1982. An experimental analysis of ultimatum bargaining. *J. Econ. Behav. Organ.* **3:** 367–388.
132. Sanfey, A.G., J.K. Rilling, J.A. Aronson, *et al.* 2003. The neural basis of economic decision-making in the ultimatum game. *Science* **300:** 1755–1758.
133. van't Wout, M., R.S. Kahn, A.G. Sanfey, *et al.* 2005. Repetitive transcranial magnetic stimulation over the right dorsolateral prefrontal cortex affects strategic decision-making. *Neuroreport* **16:** 1849–1852.
134. Knoch, D., A. Pascual-Leone, K. Meyer, *et al.* 2006. Diminishing reciprocal fairness by disrupting the right prefrontal cortex. *Science* **314:** 829–832.
135. Baumgartner, T., D. Knoch, P. Hotz, *et al.* 2011. Dorsolateral and ventromedial prefrontal cortex orchestrate normative choice. *Nat. Neurosci.* **14:** 1468–1474.
136. Civai, C., C. Miniussi & R. Rumiati. 2015. Medial prefrontal cortex reacts to unfairness if this damages the self: a tDCS study. *Soc. Cogn. Affect. Neurosci.* **10:** 1054–1060.
137. Güroğlu, B., W. van den Bos, S.A.R.B. Rombouts, *et al.* 2010. Unfair? It depends: neural correlates of fairness in social context. *Soc. Cogn. Affect. Neurosci.* **5:** 414–423.
138. Civai, C., C. Corradi-Dell'Acqua, M. Gamer, *et al.* 2010. Are irrational reactions to unfairness truly emotionally-driven? Dissociated behavioural and emotional responses in the Ultimatum Game task. *Cognition* **114:** 89–95.
139. Civai, C., C. Crescentini, A. Rustichini, *et al.* 2012. Equality versus self-interest in the brain: differential roles of anterior insula and medial prefrontal cortex. *Neuroimage* **62:** 102–112.
140. Corradi-Dell'Acqua, C., C. Civai, R. Rumiati, *et al.* 2013. Disentangling self- and fairness-related neural mechanisms involved in the ultimatum game: an fMRI study. *Soc. Cogn. Affect. Neurosci.* **8:** 424–431.
141. Ruff, C.C., G. Ugazio & E. Fehr. 2013. Changing social norm compliance with noninvasive brain stimulation. *Science* **342:** 482–484.
142. Nihonsugi, T., A. Ihara & M. Haruno. 2015. Selective increase of intention-based economic decisions by noninvasive brain stimulation to the dorsolateral prefrontal cortex. *J. Neurosci.* **35:** 3412–3419.
143. Charness, G. & M. Dufwenberg. 2006. Promises and partnership. *Econometrica* **74:** 1579–1601.
144. Spence, S.A., T.F. Farrow, A.E. Herford, *et al.* 2001. Behavioural and functional anatomical correlates of deception in humans. *Neuroreport* **12:** 2849–2853.
145. Langleben, D.D., L. Schroeder, J.A. Maldjian, *et al.* 2002. Brain activity during simulated deception: an event-related functional magnetic resonance study. *Neuroimage* **15:** 727–732.
146. Ganis, G., S.M. Kosslyn, S. Stose, *et al.* 2003. Neural correlates of different types of deception: an fMRI investigation. *Cereb. Cortex* **13:** 830–836.
147. Phan, K.L., A. Magalhaes, T.J. Ziemlewicz, *et al.* 2005. Neural correlates of telling lies: a functional magnetic resonance imaging study at 4 Tesla. *Acad. Radiol.* **12:** 164–172.
148. Langleben, D.D., J.W. Loughead, W.B. Bilker, *et al.* 2005. Telling truth from lie in individual subjects with fast event-related fMRI. *Hum. Brain Mapp.* **26:** 262–272.
149. Nunez, J.M., B.J. Casey, T. Egner, *et al.* 2005. Intentional false responding shares neural substrates with response conflict and cognitive control. *Neuroimage* **25:** 267–277.
150. Abe, N., M. Suzuki, T. Tsukiura, *et al.* 2006. Dissociable roles of prefrontal and anterior cingulate cortices in deception. *Cereb. Cortex* **16:** 192–199.
151. Priori, A., F. Mameli, F. Cogiamanian, *et al.* 2008. Lie-specific involvement of dorsolateral prefrontal cortex in deception. *Cereb. Cortex* **18**, 451–455.
152. Mameli, F., S. Mrakic-Sposta, M. Vergari, *et al.* 2010. Dorsolateral prefrontal cortex specifically processes general—but not personal—knowledge deception: multiple brain networks for lying. *Behav. Brain Res.* **211:** 164–168.
153. Lykken, D.T. 1959. The GSR in the detection of guilt. *J. Appl. Psychol.* **43:** 385–388.
154. Lykken, D.T. 1960. The validity of the guilty knowledge technique: the effects of faking. *J. Appl. Psychol.* **44:** 258–262.
155. Fecteau, S., P.S. Boggio, F. Fregni, *et al.* 2013. Modulation of untruthful responses with non-invasive brain stimulation. *Front. Psychiatry* **3:** 97.
156. Karim, A.A., M. Schneider, M. Lotze, *et al.* 2010. The truth about lying: inhibition of the anterior prefrontal cortex improves deceptive behavior. *Cereb. Cortex* **20:** 205–213.
157. Pirulli, C., A. Fertonani & C. Miniussi. 2013. The role of timing in the induction of neuromodulation in perceptual learning by transcranial electric stimulation. *Brain Stimul.* **6:** 683–689.
158. Minhas, P., M. Bikson, A.J. Woods, *et al.* 2012. Transcranial direct current stimulation in pediatric brain: a

computational modeling study. *Conf. Proc. IEEE Eng. Med. Biol. Soc.* **2012:** 859–862.
159. Kessler, S.K., P. Minhas, A.J. Woods, *et al.* 2013. Dosage considerations for transcranial direct current stimulation in children: a computational modeling study. *PLoS One* **8:** e76112.
160. Woods, A.J., V. Bryant, D. Sacchetti, *et al.* 2015. Effects of electrode drift in transcranial direct current stimulation. *Brain Stimul.* **8:** 515–519.
161. Faria, P., M. Hallett & P.C. Miranda. 2012. A finite element analysis of the effect of electrode area and inter-electrode distance on the spatial distribution of the current density in tDCS. *J. Neural Eng.* **8:** 066017.
162. Moliadze, V., A. Antal & W. Paulus. 2010. Electrode-distance dependent after-effects of transcranial direct and random noise stimulation with extracephalic reference electrodes. *Clin. Neurophysiol.* **121:** 2165–2171.
163. Minhas, P., V. Bansal, J. Patel, *et al.* 2010. Electrodes for high-definition transcutaneous DC stimulation for applications in drug delivery and electrotherapy, including tDCS. *J. Neurosci. Methods* **190:** 188–197.
164. Wagenmakers, E.-J., R. Wetzels, D. Borsboom, *et al.* 2012. An agenda for purely confirmatory research. *Perspect. Psychol. Sci.* **7:** 632–638.
165. Truong, D.Q., G. Magerowski, G.L. Blackburn, *et al.* 2013. Computational modeling of transcranial direct current stimulation (tDCS) in obesity: impact of head fat and dose guidelines. *Neuroimage Clin.* **2:** 759–766.
166. Horvath, J.C., O. Carter & J.D. Forte. 2014. Transcranial direct current stimulation: five important issues we aren't discussing (but probably should be). *Front. Syst. Neurosci.* **8:** 2.
167. Antal, A., D. Terney, C. Poreisz & W. Paulus. 2007. Towards unravelling task-related modulations of neuroplastic changes induced in the human motor cortex. *Eur. J. Neurosci.* **26:** 2687–2691.
168. Benwell, C.S., G. Learmonth, C. Miniussi, *et al.* 2015. Non-linear effects of transcranial direct current stimulation as a function of individual baseline performance: evidence from biparietal tDCS influence on lateralized attention bias. *Cortex* **69:** 152–165.
169. Kuo, M.F., W. Paulus & M.A. Nitsche. 2006. Sex differences in cortical neuroplasticity in humans. *Neuroreport* **17:** 1703–1707.
170. Boggio, P.S., R.R. Rocha, M.T. da Silva, *et al.* 2008. Differential modulatory effects of transcranial direct current stimulation on a facial expression go-no-go task in males and females. *Neurosci. Lett.* **447:** 101–105.
171. Chaieb, L., A. Antal & W. Paulus. 2008. Gender-specific modulation of short-term neuroplasticity in the visual cortex induced by transcranial direct current stimulation. *Vis. Neurosci.* **25:** 77–81.
172. Lapenta, O.M., F. Fregni, L.M. Oberman, *et al.* 2012. Bilateral temporal cortex transcranial direct current stimulation worsens male performance in a multisensory integration task. *Neurosci. Lett.* **527:** 105–109.
173. Ruffini, G., F. Wendling, I. Merlet, *et al.* 2013. Transcranial current brain stimulation (tCS): models and technologies. *IEEE Trans. Neural Syst. Rehabil. Eng.* **21:** 333–345.
174. Horvath, J.C., J.D. Forte & O. Carter. 2015. Evidence that transcranial direct current stimulation (tDCS) generates little-to-no reliable neurophysiologic effect beyond MEP amplitude modulation in healthy human subjects: a systematic review. *Neuropsychologia* **66:** 213–236.
175. Horvath, J.C., J.D. Forte & O. Carter. 2015. Quantitative review finds no evidence of cognitive effects in healthy populations from single-session transcranial direct current stimulation (tDCS). *Brain Stimul.* **8:** 535–550.
176. Antal, A., D. Keeser, A. Priori, *et al.* 2015. Conceptual and procedural shortcomings of the systematic review "evidence that transcranial direct current stimulation (tDCS) generates little-to-no reliable neurophysiologic effect beyond MEP amplitude modulation in healthy human subjects: a systematic review" by Horvath and co-workers. *Brain Stimul.* **8:** 846–849.
177. Chhatbar, P.Y. & W. Feng. 2015. Data synthesis in meta-analysis may conclude differently on cognitive effect from transcranial direct current stimulation. *Brain Stimul.* **8:** 974–976.
178. Price, A.R. & Hamilton R.H. 2015. A re-evaluation of the cognitive effects from single-session transcranial direct current stimulation. *Brain Stimul.* **8:** 663–665.
179. Keeser, D., F. Padberg, E. Reisinger, *et al.* 2011. Prefrontal direct current stimulation modulates resting EEG and event-related potentials in healthy subjects: a standardized low resolution tomography (sLORETA) study. *Neuroimage* **55:** 644–657.
180. Shafi, M.M., M.B. Westover, M.D. Fox, *et al.* 2012. Exploration and modulation of brain network interactions with noninvasive brain stimulation in combination with neuroimaging. *Eur. J. Neurosci.* **35:** 805–825.
181. Schestatsky, P., L. Morales-Quezada & F. Fregni. 2013. Simultaneous EEG monitoring during transcranial direct current stimulation. *J. Vis. Exp.* **76:** 50426.
182. Weber, M.J., S.B. Messing, H. Rao, *et al.* 2014. Prefrontal transcranial direct current stimulation alters activation and connectivity in cortical and subcortical reward systems: a tDCS–fMRI study. *Hum. Brain Mapp.* **35:** 3673–3686.
183. O'Connell, N.E., J. Cossar, L. Marston, *et al.* 2012. Rethinking clinical trials of transcranial direct current stimulation: participant and assessor blinding is inadequate at intensities of 2 mA. *PLoS One* **7:** e47514.
184. Woods, A.J., A. Antal, M. Bikson, *et al.* 2016. A technical guide to tDCS, and related non-invasive brain stimulation tools. *Clin. Neurophysiol.* **127:** 1031–1048.