

# Transcranial direct current stimulation of right dorsolateral prefrontal cortex attenuates deception-related physiological responses

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## ABSTRACT

Dorsolateral prefrontal cortex (DLPFC) is thought to play a prominent role in the cognitive aspects of deception. However, lateralization of activity reported in neuroimaging studies has not been consistent; deception has been associated with increased activity in left, right and bilateral dorsolateral prefrontal cortex. Research suggests that cognitive and emotional processing differ between left and right dorsolateral prefrontal cortex. Therefore, we hypothesized that lateralization during deception would be a function of both cognitive and emotional components in deception. We applied anodal transcranial direct current stimulation to left or right dorsolateral prefrontal cortex during a deception task to determine how enhancement of processing in one hemisphere might affect deception. We measured reaction times, heart rate and skin conductance during a simulated interrogation. We found that stimulation of right dorsolateral prefrontal cortex resulted in a general decrease in heart rate and skin conductance responses (SCRs). We suggest that right DLPFC may play a general role in top-down regulation of limbic structures involved in generating transient physiological responses that are relevant to the detection of deception.

**KEYWORDS:** heart rate, lateralization, lie detection, skin conductance, transcranial direct current stimulation

## INTRODUCTION

Deception often requires complex higher-order processes and relies heavily on the frontal executive system (for a review, see [1]). Neuroimaging studies have often demonstrated increased activity in dorsolateral prefrontal [2], ventrolateral prefrontal [3], anterior prefrontal [4, 5] and anterior cingulate cortices [6] during deception. One area that is believed to be particularly important for cognitive aspects of deception is dorsolateral prefrontal cortex. This area is well known for its roles in working memory [7] and cognitive control [8] and is likely responsible for these types of processes during deception as well [9, 10]. Interestingly, DLPFC activity during deception has not been consistently lateralized. Neuroimaging studies have reported increased activity in left DLPFC [6, 7, 11, 12], right DLPFC [4, 13, 14] and in bilateral DLPFC [9, 15, 16] when comparing deceptive to truthful responses. The nature of this inconsistent lateralization has yet to be explained.

Although neuroimaging has contributed greatly to our knowledge of the cortical areas involved in deception, due to its correlative nature, causal relationships between brain activity and behavior cannot be inferred. Non-invasive stimulation techniques such as transcranial direct current stimulation (tDCS) and repetitive transcranial magnetic stimulation (rTMS), on the other hand, can induce lasting changes in cortical excitability (for tDCS see [17]; for rTMS see [18]), and allow for causal inferences about

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cortical function. Therefore, these techniques have the potential to bring about novel insights into the functional roles of cortical areas involved in deception, and may help elucidate reasons behind lateralized DLPFC activity reported in neuroimaging studies on deception.

Numerous non-invasive stimulation studies have investigated the function of DLPFC and have provided evidence that DLPFC is involved in various cognitive and emotional processes that may be relevant to deception. For instance, stimulation of DLPFC has not only affected cognitive processes such as working memory [19] and cognitive control [20], but has also affected processes involving emotions such as risk-taking [21], social decision making [22, 23], moral judgments [24], and evaluations of valenced stimuli [25, 26]. Interestingly, a few studies have even demonstrated lateralized function of DLPFC by comparing stimulation effects of left and right hemispheres [23, 27, 28, 29, 30]. Therefore, we suspect that, during deception, DLPFC may be involved in both cognitive and emotional processing, and that this involvement may be lateralized.

Only a few studies have investigated DLPFC involvement in deception using non-invasive stimulation; unfortunately, we are not aware of any that have manipulated or measured emotion. Nevertheless, non-invasive stimulation has been used to support neuroimaging evidence suggesting the involvement of DLPFC in cognitive processing during deception. Priori *et al.* [31] and Mameli *et al.* [32] each found that excitatory (anodal) bilateral tDCS over DLPFC affected reaction times during non-emotional deception. Interestingly, they found somewhat opposing results; lies about familiar images were slowed in Priori *et al.*, whereas lies about general knowledge were accelerated in Mameli *et al.*, when comparing real to sham stimulation. Although these results seem contradictory, Mameli *et al.* proposed (as did Ganis *et al.* [4]) that different types of lies rely on different networks. However, their use of bilateral stimulation confounds inferences about these networks as well as inferences about DLPFC lateralization. Karton & Bachman [33], on the other hand, investigated left and right DLPFC separately during spontaneous deception using inhibitory (low-frequency) rTMS. In their paradigm, participants were asked to either name the color of a disk presented on a computer screen (truth) or name

a different color (lie), at will. They found differential effects of stimulation: inhibition of left DLPFC increased the number of deceptive responses, whereas inhibition of right DLPFC decreased the number of deceptive responses. Intriguingly, these results suggest that left and right DLPFC may contribute differently to the cognitive aspects of deception. It is important to note, however, that these studies focused purely on the cognitive aspects of deception, and did not measure or attempt to elicit emotion.

However, a few non-invasive stimulation studies involving emotion have found lateralized stimulation effects on DLPFC, suggesting that left and right DLPFC are differentially involved in emotion. Therefore, we suspect that DLPFC involvement in emotion processing during deception may be lateralized as well. For instance, stimulation of right, but not left, DLPFC has affected the acceptance rates of unfair offers during the ultimatum game [22, 23, 34], a strategic decision-making game involving emotion [35]. Interestingly, Knoch *et al.* [23] found that stimulation only affected acceptance rates of unfair offers made by people; acceptance rates of unfair offers made by a computer were not affected by stimulation. Similarly, stimulation of right DLPFC seems to particularly affect risk-taking behavior as well [21, 36, 37]. Interestingly, however, left DLPFC stimulation has been shown to affect emotion processing during other tasks. For example, Peña-Gómez *et al.* [26] and Boggio *et al.* [25] each found that excitatory tDCS over left DLPFC reduced negative ratings of negatively valenced stimuli. These mixed findings demonstrate that the nature of DLPFC involvement in emotion processing has yet to be fully elucidated.

Therefore, the aim of the present study was to investigate left and right DLPFC involvement in both emotional and cognitive processing during deception. We chose to use excitatory (anodal) tDCS in order to temporarily increase left or right DLPFC activity during deception. tDCS is now a common procedure used to investigate cortical function; tDCS can easily, and with very little physical discomfort, induce a lasting yet transient change in cortical excitability (for a review, see [17]). Direct current is typically delivered through electrodes encased in 25-35 cm<sup>2</sup> saline-soaked sponges placed on the scalp, for an extended period of time. Common stimulation

procedures consist of the administration of 1-2 mA current for around 20 minutes, although stimulation of up to 30 minutes appears to be well tolerated [38, 39, 40]. Importantly, tDCS procedures allow for sham stimulation, a control stimulation procedure that has proven to be effective [41]. Other methodological advancements such as extracephalic reference electrode placements [42] and double-blind simulation procedures [43] allow for precision and control in the laboratory. Therefore, we believe that tDCS is an ideal method for investigating cortical function during deception.

The present study investigated stimulation effects on deception using three dependent measures: reaction time to infer stimulation effects on cognition, and heart rate and skin conductance to infer effects on emotion. Heart rate and skin conductance have been shown to indicate deception in laboratory settings [44]. Although these autonomic responses likely reflect arousal rather than deception *per se* [45, 46], they can often reliably differentiate deceptive from truthful responses. Specifically, lies compared to truthful responses elicit larger SCRs and transient decreases in heart rate [47, 48, 49, 50, 51].

The deception task used in the present study was a variant of the guilty knowledge test [52], in which it is assumed that only those knowledgeable of the details of a situation will show differential responses between relevant and irrelevant information. In the present study, participants were instructed to hide their knowledge of the face of one murderer while identifying another murderer during an interrogation; faces of these murderers were interspersed among a group of unfamiliar faces. Therefore, we could compare lie and truthful responses toward relevant and irrelevant information.

Here, we investigated the involvement of left and right DLPFC during deception using excitatory (anodal) tDCS with an extracephalic reference electrode, in a double-blind between-subjects design. In order to investigate cognition and emotion during deception, we recorded reaction time, phasic heart rate (pHR) and SCRs during a computer-based interrogation. Further, our paradigm was designed to mimic real-life deception. Participants learned the identity of a murderer in secret and were required to lie to an experimenter during an interrogation by pretending not to recognize the face of the murderer when it was presented on a computer screen. During

the interrogation, a video camera was directed at the face of the participants in an attempt to increase emotion. Further, we also provided motivation to try to get away with lying; participants were told they could win a certificate if they could conceal their emotions and successfully deceive the interrogator.

## MATERIALS AND METHODS

### Participants

75 participants were initially recruited for the present study using the New Mexico State University (NMSU) research participation system; 11 participants out of the 75 were not included in any analysis due to experimental error or failure to complete the deception task. The remaining 64 participants were designated to three groups based on DLPFC stimulation: 20 (6 males) in left, 23 (3 males) in right and 21 (7 males) in sham stimulation groups. All participants were NMSU students and were participating to receive class credit in a psychology course. Prior to participation, participants were screened for psychological and neurological disorders; all participants were healthy, medication free and did not have a history of neurological or psychological disorders. Participants were between 18 and 31 years old (mean age of 20), right handed and had normal or corrected-to-normal vision. This study was approved by the NMSU Institutional Review Board. Written informed consent was obtained prior to participation.

### Deception paradigm

Upon arrival, participants first learned about a murder by reading from a “wanted” poster modeled after those used by the FBI. This poster contained details about the murder and described two men who were guilty of the crime. However, the poster was incomplete; it only portrayed the face of one of the murderers (murderer 1). A blank rectangle appeared in place of the picture of the second murderer with a question mark presented where his face should have been placed. Next, participants were told they would be presented with two sealed envelopes. One would contain a completed “wanted” poster that showed the face of the second murderer (murderer 2). The other would contain a similar poster with an innocent man. However, in actuality, both envelopes contained the poster with the second murderer. Participants were told that they would

have the opportunity to open one of the envelopes in secret.

Prior to choosing an envelope, participants were informed that they would later be interrogated by the experimenter, who would present them with several faces, including the murderers and innocent men; the experimenter would try to detect whether or not they would recognize the second murderer. Participants were told that if their envelope contained the second murderer, they should lie to the experimenter by pretending not to recognize his face. On the other hand, they were told that if their envelope contained the innocent man, they should simply respond truthfully to all faces presented during the interrogation.

Further, in a ploy to improve motivation, participants were told that one's ability to lie could predict future business success and that if they had learned the identity of the second murderer, but the experimenter could not tell whether or not they were being deceptive, they would be awarded a "Certificate of Expertise" for concealing their emotions. Following these instructions, participants were then left alone in a room with a closed door to choose and open one of the two envelopes in secret. They were instructed to read the poster contained within, fold the poster and put it in their pocket, and knock on the door from the inside of the room to let the experimenter know that they had finished. Next, participants were taken into another room to begin the interrogation. Electrodes for physiological recordings and tDCS were attached upon entering the interrogation room.

The interrogation was computer-based. An E-Prime program [53] displayed faces, one at a time. Participants saw three different types of faces: murderer 1, murderer 2 and previously unfamiliar faces. Participants were instructed to respond "yes" or "no" to indicate whether or not they recognized a murderer, using a keyboard with keys labeled "y" and "n". Participants were also instructed to always identify murderer 1 as a murderer. Therefore, successful deception in this paradigm required participants to respond truthfully to murderer 1 (i.e. "yes"), truthfully to unfamiliar faces (i.e. "no") and deceptively to murderer 2 (i.e. "no"). This manipulation required participants to pay attention to every face presentation so that they would not miss responding "yes" to murderer 1. All faces were

gray-scale male faces with neutral expressions taken from the Karolinska Directed Emotional Faces database [54]; pictures of faces occupied visual angles of  $6.5^\circ \times 9^\circ$ . During the interrogation, faces were presented one after another. Faces appeared on the screen for up to 2 seconds or until a response was made. Following a keyboard response, the response itself (i.e. "yes" or "no") appeared below each face for 500 ms, after which the face and response disappeared. Face presentations were spaced 10 seconds apart (10 seconds after stimulus offset) to allow skin conductance to return to baseline. Participants responded to 72 randomized face presentations in total: 12 of murderer 1 (truth), 12 of murderer 2 (lie) and 48 unfamiliar (truth) faces; unfamiliar faces consisted of 8 previously unseen faces, each presented 6 times throughout the interrogation.

Given that the interrogation was computer-based, steps were taken to make the interrogation feel like a real life interrogation. A video camera was pointed at the face of each participant during the interrogation. Participants were told that the experimenter would be monitoring their facial expressions and electrophysiological activity during the interrogation to detect deception (however, facial expressions were not recorded or analyzed). Throughout the interrogation, the experimenter sat in an adjacent room directly behind participants with an open door between them to monitor their facial and physiological responses on television and computer monitors displaying each; these working monitors were shown to the participants to convince them that the experimenter would be actively attempting to detect deception throughout the interrogation.

Immediately following the interrogation, participants were partially debriefed; the experimenter admitted to knowing, all along, that the participants had learned the identity of the second murderer from the envelope that they chose. Next, participants were asked to perform a shorter version of the same interrogation task while responding truthfully. This served as a memory test. During this test, participants were shown 26 images: 5 of murderer 1, 5 of murderer 2 and 16 (two of each) of the unfamiliar faces used in the interrogation. Following the memory test, participants were fully debriefed. Six participants were unable to identify the second murderer during this test and were not included in

any analysis. Additionally, one participant responded truthfully throughout the first part of the interrogation; this participant was also excluded from the analysis.

### **Electrophysiological recordings**

During the interrogation, electrophysiological data were collected using a BioSemi ActiveTwo EEG system with a galvanic skin response (GSR) module sampled at 2048 Hz (www.biosemi.com). Changes in skin conductance were recorded using BioSemi GSR electrodes placed on the inner index and middle fingers of the left hand between the second and third knuckle. Heart rate was recorded with an Ag/AgCl BioSemi flat electrode placed on the manubrium (top of sternum). Prior to electrode placement, the skin was cleaned using isopropyl alcohol. Conductive gel was used to ensure conductance. Electrodes were attached at least 15 minutes prior to recording.

### **Transcranial direct current stimulation (tDCS)**

Three minutes before the interrogation, participants began receiving either anodal (excitatory) or sham tDCS, over left or right DLPFC using the “study mode” function built into our NeuroConn DC Stimulator Plus (NeuroConn GmbH, Ilmenau, Germany). “Study mode” allowed for a double-blind procedure. “Study mode” uses five-digit codes to command the device to administer real or sham stimulation. The experimenter was not familiar with these codes; therefore, neither the experimenter nor the participant knew whether or not the device would administer real or sham stimulation. During real stimulation, 2 mA of direct current was delivered through conductive rubber electrodes encased in 25 cm<sup>2</sup> saline-soaked (1% NaCl) sponges. At the beginning of stimulation, current was gradually increased to 2 mA during the first 10 seconds. During sham stimulation, current was ramped up in 10 seconds, remained on for 15 seconds and ramped down in 10 seconds (35 seconds total). For all stimulation conditions, the impedance limit was set to 50 k $\Omega$ ; if impedance surpassed this limit, the device was programmed to automatically shut off. Transcranial DC stimulation did not begin for three participants due to an inability to reduce impedance below this level (we believe that this was the result of very thick hair; these participants were not included in the study); impedance never

surpassed this limit after stimulation began for the other subjects. The anode was placed over left or right DLPFC, while the cathode was placed over the contralateral upper-arm. During sham stimulation, the placement of the anode was counterbalanced between left and right DLPFC. Dorsolateral PFC electrode locations were determined by measuring 5 cm anterior to a point 20% of the auricular measurement down from the vertex toward the auricular area (area F3 or F4 of the 10-20 EEG system [55]). Stimulation began approximately 3 minutes before the interrogation and remained on throughout the interrogation. During the three pre-interrogation minutes, participants were required to report their physical sensations to stimulation using the physical pain descriptors used in Clark *et al.* [40], numbered as 0) no sensation, 1) cold, 2) some tingling, 3) warm, 4) lots of tingling/some itching, 5) very warm, 6) lots of itching, 7) burning (like a sunburn), 8) burning (like scalding water), and 9) hurts a lot. Despite a willingness to continue, stimulation was terminated for one participant who reported a 7; this participant was not included in the study. Otherwise, stimulation was well tolerated. If participants reported an itching sensation during pre-interrogation stimulation, additional saline was applied to the sponge electrodes using a plastic pipette; verbal confirmation indicated that the addition of saline sufficiently alleviated discomfort in these participants.

### **Data processing**

Accuracy and reaction time data were collected using E-prime [53]. Prior to analysis, data were inspected for outliers. One participant with reaction times more than three standard deviations from the mean was not included in reaction time analysis.

MATLAB (version R2012a, MathWorks Inc., Natick, MA) was used to process physiological data. Heart rate data were down-sampled offline to 200 Hz and were divided into 11-second epochs (3 seconds pre- and 8 seconds post-stimulus) using EEGLAB (v.11.0.4.3b, [56]). During sham stimulation, the NeuroConn “study mode” procedure passed a weak current every 550 ms in order to check the impedance; impedance checking put a spike artifact in sham data. This artifact was successfully removed, but heart beats that overlapped the artifact were not salvaged; this resulted in a loss of about 16% of

heart beats in the sham condition. Heartbeats were detected using custom MATLAB scripts and converted to beats-per-minute (BPM) based on heartbeat R-R intervals. BPM scores were divided and averaged over 8 discrete time points, corresponding to 7 post-stimulus seconds (beginning with zero). BPM scores of heartbeats that occurred within 500 ms of each post-stimulus second were averaged into each time point (for example, heartbeat BPM scores that were averaged into the time point “1” occurred between 500 and 1500 ms post-stimulus). The average BPM of heartbeats occurring 3 seconds pre-stimulus within each epoch were used as baseline values, which were subtracted from each discrete time point average within each epoch. The resultant values (indicating phasic heart rate change) were used in the analyses; negative values represent deceleration relative to baseline. Five participants were not included in heart rate analysis: four due to poor recording and one for having an average heart rate change more than three standard deviations from the mean.

Skin conductance data were down-sampled offline to 10 Hz and divided into 10-second epochs (2 seconds pre- and 8 seconds post-stimulus). Epochs that did not contain a behavioral response or contained a response error were removed. Particularly noisy trials and trials that contained overlapping SCRs were removed via visual inspection by an experimenter who was blind to stimulation conditions. SCR amplitude was determined by the difference between onset and peak of SCRs that began between 1 and 4 seconds post-stimulus; trials where no skin conductance increase was observed were scored as zero. Raw SCR data distributions were strongly positively skewed; data exploration suggested that this skew was the result of a few highly responsive participants. Data were standardized by converting to z-scores; following standardization, data distributions were normal and did not contain any outliers. Standard difference scores were calculated by subtracting SCRs to unfamiliar faces from those to murderers 1 and 2; these difference scores were used in the analysis.

## RESULTS

### Behavioral results

We observed very high accuracy during the interrogation (>95%); on occasion, participants

mistakenly responded “no” to murderer 1, when they should have responded “yes”. A univariate analysis of variance (ANOVA) confirmed that accuracy did not differ between stimulation groups ( $F_{2,60} = 0.247, p = 0.782, \eta_p^2 = 0.01$ ). Given that Priori *et al.* [31] and Mameli *et al.* [32] each found stimulation effects on reaction times during deception, our primary behavioral interest was in reaction times.

Reaction time data were analyzed with a 3 (stimulation [left, right, or sham]) x 3 (image [murderer 1, murderer 2, unfamiliar]) mixed repeated measures ANOVA. This analysis revealed a main effect of image only ( $F_{2,120} = 32.06, p < 0.001, \eta_p^2 = 0.35$ ); there was no effect of stimulation ( $F_{2,60} = 0.28, p = 0.754, \eta_p^2 = 0.01$ ) or an interaction between stimulation and image ( $F_{4,120} = 1.49, p = 0.209, \eta_p^2 = 0.05$ ). Paired t-tests indicated that responses to murderer 1 were significantly slower than to both murderer 2 ( $p < 0.001$ ) and unfamiliar faces ( $p < 0.001$ ). Critically, however, reaction times to murderer 2 and unfamiliar faces did not differ reliably ( $p = 0.264$ ). These results suggest that stimulation did not affect reaction times, and an effect of deception was not observed among reaction time data.

### Skin conductance response results

Skin conductance response data were analyzed with a 3 (stimulation [left, right, sham]) x 2 (image [murderer 1, murderer 2]) mixed repeated measures ANOVA; the SCRs to murderer 1 and murderer 2 reflect their standard difference from SCRs to unfamiliar faces. There was no significant main effect of image ( $F_{1,61} = 0.18, p = 0.675, \eta_p^2 < 0.01$ ) or an interaction between image and stimulation ( $F_{2,61} = 1.87, p = 0.162, \eta_p^2 = 0.06$ ). Therefore, difference scores of murderer 1 and murderer 2 did not differ from each other and did not vary as a function of stimulation. However, a main effect of stimulation was observed ( $F_{1,61} = 5.17, p = 0.008, \eta_p^2 = 0.15$ ). Therefore, difference scores for both murderers differed as a function of stimulation (see Figure 1). Post hoc paired t-tests revealed that SCRs during right DLPFC stimulation were smaller than both left ( $p = 0.004$ ) and sham ( $p = 0.017$ ), while left and sham did not differ ( $p = 0.586$ ). Next, in order to test an effect of deception in each stimulation group, t-tests were used to determine if SCR difference scores for murderer 2 were greater than zero. These tests revealed that difference scores

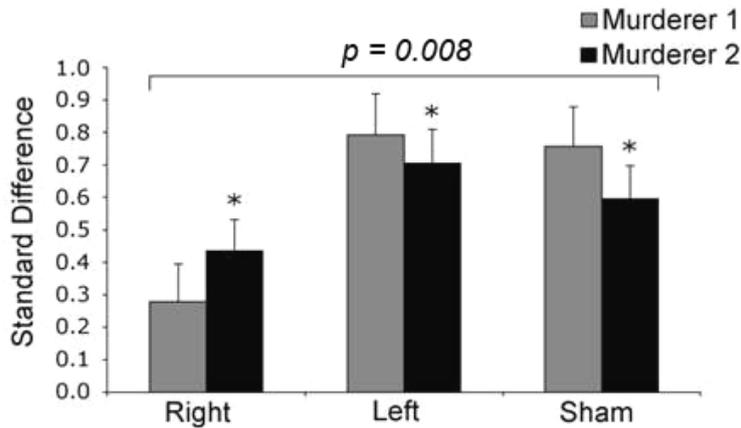
for murderer 2 were greater than zero in all stimulation groups: right ( $t_{22} = 4.92, p < 0.001$ ), left ( $t_{19} = 6.84, p < 0.001$ ) and sham ( $t_{20} = 5.41, p < 0.001$ ). Therefore, although tDCS affected SCRs, an effect of deception was still observed in all stimulation groups; SCRs in response to murderer 2 were greater than SCRs in response to unfamiliar faces.

**Phasic heart rate results**

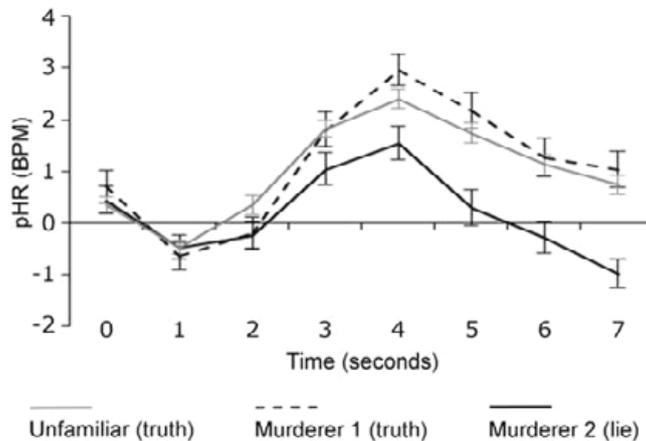
Phasic heart rate data were analyzed with a 3 (stimulation [left, right, sham]) X 3 (image [murderer 1, murderer 2, unfamiliar]) X 8 (time [0, 1, 2, 3, 4, 5, 6, 7]) repeated measures multivariate ANOVA (MANOVA). Given that we collected heart rate across 8 time points,

we suspected that the repeated measures ANOVA assumption of sphericity would be violated. Therefore, we chose the MANOVA because it does not assume sphericity [57]. Further, given that sham group data contained slightly fewer heart beats than left and right groups, we report *F*-values associated with *Pillai's Trace*, as this statistic is known to be robust when group variances differ [58].

The 3 x 3 x 8 MANOVA revealed a main effect of image (*Pillai's Trace* = 0.30,  $F_{2,55} = 11.85, p < 0.001, \eta_p^2 = 0.30$ ) and a main effect of time (*Pillai's Trace* = 0.84,  $F_{7,50} = 37.89, p < 0.001, \eta_p^2 = 0.84$ ), but did not show a main effect of stimulation ( $F_{2,56} = 1.63, p = 0.204, \eta_p^2 = 0.06$ ). Figure 2 depicts pHR following each type of image when averaging over



**Figure 1.** Skin conductance responses (SCRs) following murderer 1 and murderer 2, relative to SCRs following previously unfamiliar faces. \*Effect of deception (lie > unfamiliar truth),  $p < 0.05$ .



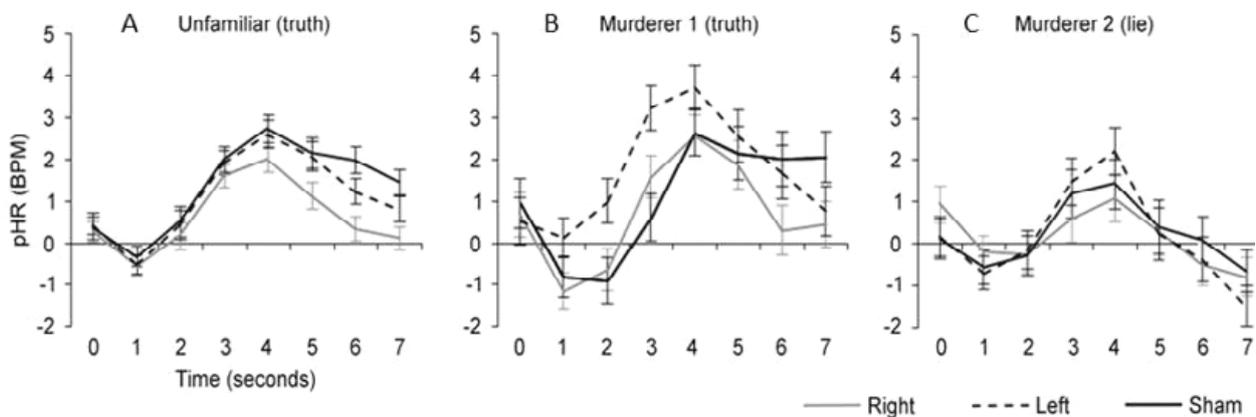
**Figure 2.** Main effect of deception on phasic heart rate (pHR).

stimulation groups. Heart rate following deceptive responses was slower than heart rate following both types of truthful responses. Interaction analysis from the  $3 \times 3 \times 8$  MANOVA revealed a significant 2-way interaction between image and time (*Pillai's Trace* = 0.67,  $F_{14,43} = 6.21$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.67$ ), a marginal 2-way interaction between stimulation and time (*Pillai's Trace* = 0.38,  $F_{14,102} = 1.68$ ,  $p = 0.071$ ,  $\eta_p^2 = 0.19$ ), and most importantly, a significant 3-way interaction between image, stimulation and time (*Pillai's Trace* = 0.68,  $F_{28,88} = 1.64$ ,  $p = 0.044$ ,  $\eta_p^2 = 0.34$ ). This 3-way interaction suggests that stimulation significantly affected pHR.

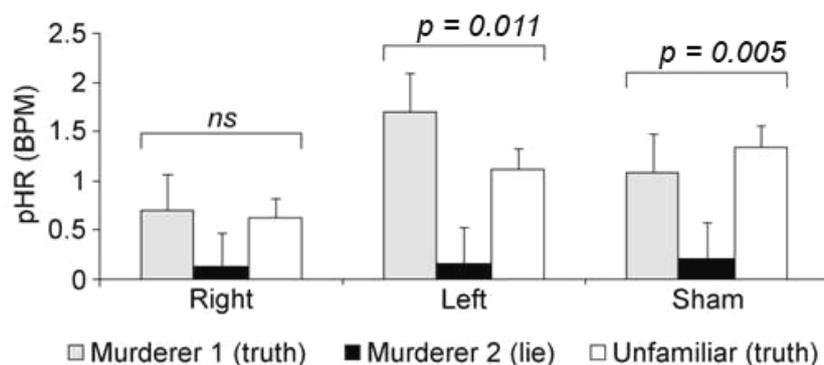
In order to further investigate the three-way interaction, we conducted 3 (stimulation)  $\times$  8 (time) MANOVAs on pHR following each image. When investigating pHR differences following unfamiliar faces (truth), we observed a main effect of stimulation ( $F_{2,56} = 3.81$ ,  $p = 0.028$ ,  $\eta_p^2 = 0.12$ ) and time (*Pillai's Trace* = 0.87,  $F_{7,50} = 46.14$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.87$ ), while the interaction was not significant (*Pillai's Trace* = 0.28,  $F_{14,102} = 1.19$ ,  $p = 0.294$ ,  $\eta_p^2 = 0.14$ ). Figure 3A demonstrates that heart rate following unfamiliar face stimuli was slowest in the right stimulation group. When investigating pHR differences following murderer 1 (truth), we observed a significant main effect of time (*Pillai's Trace* = 0.70,  $F_{7,50} = 17.17$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.70$ ) and a significant interaction between stimulation and time (*Pillai's Trace* = 0.47,  $F_{14,102} = 2.25$ ,  $p = 0.010$ ,  $\eta_p^2 = 0.24$ ), while the main effect of stimulation was not significant ( $F_{2,56} = 1.83$ ,  $p = 0.169$ ,  $\eta_p^2 = 0.06$ ).

The significant interaction suggests temporally different pHR following the presentation of murderer 1 between stimulation groups. Figure 3B depicts pHR following murderer 1; pHR in the left stimulation group accelerated sooner than both right and sham conditions.

Interestingly, when investigating pHR differences following murderer 2 (lie), we observed a main effect of time only (*Pillai's Trace* = 0.58,  $F_{7,50} = 9.98$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.58$ ); we did not observe a main effect of stimulation ( $F_{2,56} = 0.01$ ,  $p = 0.987$ ,  $\eta_p^2 < 0.01$ ) or an interaction between stimulation and time (*Pillai's Trace* = 0.19,  $F_{14,102} = 0.78$ ,  $p = 0.685$ ,  $\eta_p^2 = 0.10$ ). Therefore, stimulation did not specifically affect pHR following lie responses (see Figure 3C). Next, however, in order to investigate an effect of deception within each stimulation group, one-way repeated measures MANOVAs were used to compare average phasic heart rate change in response to each image within each stimulation group (Figure 4). A main effect of image was observed in left (*Pillai's Trace* = 0.41,  $F_{2,17} = 6.00$ ,  $p = 0.011$ ,  $\eta_p^2 = 0.41$ ) and sham stimulation groups (*Pillai's Trace* = 0.48,  $F_{2,16} = 7.43$ ,  $p = 0.005$ ,  $\eta_p^2 = 0.48$ ), but not in the right stimulation group (*Pillai's Trace* = 0.16,  $F_{2,20} = 1.95$ ,  $p = 0.168$ ,  $\eta_p^2 = 0.16$ ). Therefore, although stimulation did not specifically affect lie responses, right DLPFC stimulation seemed to attenuate heart rate change in general, resulting in reduced differentiability between lie and truthful responses.



**Figure 3.** Phasic heart rate differences between stimulation groups following unfamiliar stimuli (A), murderer 1 (B), and murderer 2 (C).



**Figure 4.** Effect of deception on pHR within each stimulation group.

## DISCUSSION

### Behavioral results

Interestingly, we did not observe an effect of stimulation on reaction times. These results do not support Priori *et al.* [31] and Mameli *et al.* [32] who each found that bilateral DLPFC stimulation affected reaction times during deception. However, our lack of effect may be the result of paradigm differences. Priori *et al.* and Mameli *et al.* each used a within-subjects design. As we used a between-subjects design, their analyses were likely more powerful than ours. However, another possibility could be a result of differences in stimulation parameters; Priori *et al.* and Mameli *et al.* each used bilateral stimulation (simultaneously increasing left and right DLPFC activity). It may be that unilateral stimulation is not sufficient to alter cognitive processing during deception. The latter interpretation is somewhat supported by Verschuere *et al.* [59] who also did not find a significant effect of unilateral inhibitory theta burst TMS of inferior frontal sulcus on reaction times during deception.

### Skin conductance response results

We observed a main effect of stimulation on skin conductance data. Those who received anodal tDCS over right DLPFC showed smaller skin conductance response differences between murderers and unfamiliar stimuli when compared to both left and sham stimulation conditions. Therefore, stimulation of right DLPFC slightly reduced the differentiability between lie and truthful responses compared to the other groups. However, despite reduced SCRs in the right stimulation condition, each group still showed a strong effect of deception; SCRs to

murderer 2 were much larger than SCRs to unfamiliar faces in all stimulation groups. This effect is consistent with the deception literature [44, 47, 50, 60]. Therefore, although stimulation of right DLPFC reduced skin conductance responses during deception, deceptive responses were still distinguishable via SCR.

### Phasic heart rate

We observed a significant 3-way interaction between stimulation, image and time. Therefore, stimulation affected phasic heart rate. Curiously, this effect was not particular to deceptive responses. Rather, right DLPFC stimulation caused a general attenuation of pHR. This reduced the differentiability between lie and truthful responses in the right stimulation group. Therefore, it appears that right DLPFC stimulation attenuated both SCR and pHR responses during the interrogation.

### tDCS effects on DLPFC

The present findings suggest a general decrease in physiological responsiveness during anodal stimulation of right DLPFC. From these results, it is difficult to determine particular stimulation effects on emotion. Rather, we suggest that DLPFC plays a more general role in controlling limbic structures that may be involved in emotion. Other research has suggested this as well. For instance, right DLPFC seems to be particularly involved with the stress response system as demonstrated by studies investigating stimulation effects on posttraumatic stress disorder (PTSD) [61]. An example of this involvement can be found in Boggio *et al.* [62] who investigated the treatment effects of excitatory (high frequency- 20 Hz) rTMS over left and right

DLPFC on PTSD symptoms over a 10-day clinical trial. They found that increasing activity in right DLPFC was most effective in relieving the symptoms of PTSD, when compared to left DLPFC and sham stimulation. Importantly, they also found that right DLPFC stimulation particularly decreased anxiety. Similarly, others have suggested a right prefrontal specialization for control over the HPA-axis stress response system in animal models [63]. Further, one study on healthy subjects has provided evidence of a mechanism by which right DLPFC stimulation may decrease the stress response [64]. Baeken *et al.* [64] administered excitatory (10 Hz) rTMS over left and right DLPFC to participants prior to viewing neutral and negative images during functional magnetic resonance imaging (fMRI). They found that increasing activity in right, but not left, DLPFC reduced amygdala reactivity in response to negative images. Therefore, it appears that right DLPFC is involved in down-regulating the activity of the amygdala. This interpretation fits our results; we observed reduced autonomic responses during excitatory stimulation of right DLPFC. Given that the amygdala is heavily influential over autonomic activity [65], it is plausible that stimulation of right DLPFC in the present study reduced amygdala reactivity to stimuli during the interrogation.

Another physiological mechanism may also explain stimulation effects on SCR and pHR. Cechetto and Shoemaker [65] proposed lateralized prefrontal control over sympathetic and parasympathetic nervous systems. Therefore, stimulation effects observed in the present study may be a result of altered sympathetic and parasympathetic control. In support of this interpretation, Brunoni *et al.* [66] found lateralized effects of stimulation over left and right DLPFC on high-frequency heart rate variability (a measure of parasympathetic activity [67]), in response to neutral and negative stimuli. Using bilateral bipolar tDCS (i.e. anode over left DLPFC and cathode over right DLPFC, or vice versa), they found that the largest differences in heart rate variability between neutral and negative stimuli were observed while increasing activity in left DLPFC and decreasing activity in right DLPFC; interestingly, heart rate variability could not reliably differentiate negative from neutral

stimuli when the tDCS polarity was reversed (inhibition of left and excitation of right DLPFC). These results are somewhat comparable to ours; we did not observe a significant difference in average HR between stimuli during excitatory stimulation of right DLPFC, while these differences were largely apparent during stimulation of left DLPFC and sham.

### Limitations

Given that our study did not involve neuroimaging, we cannot make assumptions about network activity. We suspect that social cognitive/emotional processes, such as those involved in deception, likely involve a myriad of complex processes incorporating numerous cortical and subcortical brain regions. Abe [1] proposed that deception commonly recruits areas involved in executive control, emotion and motivation, including prefrontal cortex, anterior cingulate, amygdala and the striatum. We observed DLPFC stimulation effects on autonomic activity. DLPFC does not have many direct anatomical connections with the amygdala or the hypothalamus [68]. Therefore, we suspect altered autonomic responsiveness was the result of DLPFC interactions with other cortical and subcortical regions. As a consequence, the precise involvement of DLPFC in emotion processing within a deception network remains unknown. Future research should combine cortical stimulation with neuroimaging in order to get a clear understanding of the networks involved.

### CONCLUSION

Here, we have demonstrated lateralized stimulation effects of right DLPFC on physiological responses during an interrogation to detect deception. Excitatory stimulation of right DLPFC decreased physiological responses throughout the interrogation. We suggest that right DLPFC administers top-down regulatory control over limbic structures involved in generating transient physiological responses which are often attributable to emotion during deception. Neuroimaging studies have indicated a role of right DLPFC in deception, and the present findings suggest that right DLPFC may be involved in regulating physiological processes reflective of emotion during deception. Therefore, interpretations of neuroimaging results during

deception that include right DLPFC may now include speculations about emotion as well as cognition.

### CONFLICT OF INTEREST STATEMENT

None to declare.

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