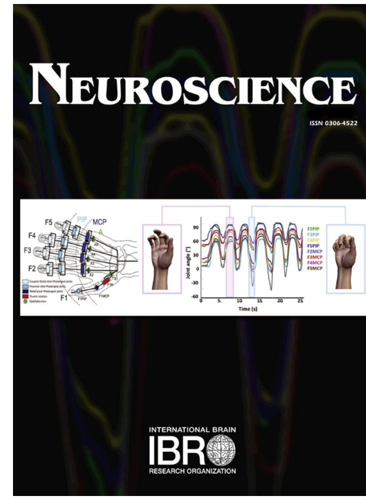

Research Article

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Bilateral prefrontal cortex anodal tDCS effects on self-reported aggressiveness in imprisoned violent offenders

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Abbreviations: BAQ, Buss-Perry Aggression Questionnaire; DLPFC, dorsolateral prefrontal cortex; tDCS, transcranial direct current stimulation; VLPFC, ventrolateral prefrontal cortex.

Abstract

Reduced activity of the frontal lobes, and particularly of the prefrontal cortex, has been related with violent behavior, aggression and crime. The causal importance of prefrontal cortex activity for aggressive behaviors and the self-perception of aggressiveness needs however to be clarified. The aim of this study was to explore the effect of an anodal transcranial direct current stimulation protocol (tDCS, 1.5 mA, 15 min), which, according to previous studies, enhances cortical excitability, applied bilaterally over the prefrontal cortex on self-reported aggressiveness. Two imprisoned violent offender cohorts, discerned by the degree of aggressiveness (murderers vs. non-murderers), were included in this single-blind sham-controlled study. Self-reported aggressiveness was recorded before and after 3 tDCS sessions (one session per day). Four dimensions of aggression were evaluated by means of the standardized Buss-Perry Aggression Questionnaire (BAQ). In both inmate groups the results revealed an aggression-reducing effect of tDCS on the Physical aggression, Anger, and Verbal aggression dimensions of the BAQ. In the Hostility dimension, tDCS significantly reduced aggression only in the group of murderers. These results suggest that modulation of prefrontal cortex excitability by 3 consecutive sessions of tDCS reduces self-reported aggressiveness similarly in murderer and non-murderer samples.

Key words: aggression, dorsolateral prefrontal cortex, inmate, murderers, transcranial direct current stimulation.

INTRODUCTION

Frontal lobe abnormalities have been proposed to be associated with violent crimes and aggression (Pincus, 1999). Clinical evidence and basic research suggest alterations of the prefrontal network as a biological factor that contributes to violent behavior (Keune et al., 2012; Potegal, 2012). In congruence with this concept, low scores on executive function tests (a group of functions canonically associated with the dorsolateral prefrontal cortex (DLPFC)) predict high rates of physically aggressive behavior (Giancola et al., 1998), and poor frontal lobe regulatory control of blood glucose levels correlates with hostile violence (Walters et al., 2016). An involvement of the DLPFC has also been observed in virtual aggressive behavior tests (Klasen et al., 2013). Moreover, imagination of aggressive behavior reduces regional cerebral blood flow in the ventromedial prefrontal cortex (Pietrini et al., 2000). Aggression has also been related to other prefrontal areas, such as the anterior cingulate gyrus and orbitofrontal cortex (Molenberghs et al., 2015). These areas are thought to be involved in judgements of the morality of behaviors (Moll et al., 2007). Thus, the DLPFC, medial prefrontal cortex, orbitofrontal cortex, and the anterior cingulate cortex are critical components of a brain network involved in diverse aspects of aggressive behaviors (Repple et al., 2017; Chester and DeWall, 2018; Klasen et al., 2018). Whereas an association between frontal lobe functions and aggression has been revealed in different clinical, behavioral and neuroimaging studies, evidence regarding the causal involvement of the prefrontal cortex, including specific frontal functions and mechanisms involved in different dimensions of aggressive behavior, is however limited.

A versatile tool to investigate the causal involvement of the prefrontal cortex in aggression is non-invasive brain stimulation, which can up-and downregulate cortical activity and excitability. An impact of such an intervention over the prefrontal cortex on aggression would support the causal relevance of prefrontal activity for aggressive behavior. Transcranial direct current stimulation (tDCS) increases or reduces cortical excitability via the application of anodal or cathodal weak direct currents, respectively, over the target area (Nitsche and Paulus, 2000). Specifically, an increase of prefrontal excitability by tDCS (Nitsche and Paulus, 2000, 2001, 2011, Nitsche et al., 2003, 2005;

Priori et al., 2009; Stagg and Nitsche, 2011) could be a mechanism to modulate aggressive behaviors through counteracting aggression-related hypofrontality.

Although electrode position over a specific area does not imply that stimulation effects are necessarily specific for this area, anodal tDCS (1.5 mA for 20 min) applied over the right ventrolateral prefrontal cortex (VLPFC) region reduced aggressive behavior elicited by violent video games in a recent study (Riva et al., 2017). Thus, ventral regions of the prefrontal cortex might be involved in eliciting aggression. Considering the role of the DLPFC in executive functions and control and evaluation of situations, this prefrontal region might also be related to aggressiveness through control-related cognitive processes relevant for aggression management. Indeed, DLPFC is a prefrontal region involved in executive functions (Kujach et al., 2018; Nejati et al., 2018; Strobach et al., 2018), in the evaluation of violations of rules (Müller-Leinß et al., 2017), affective/reward processes (Wang et al., 2017; Nejati et al., 2018; Wittkuhn et al., 2018), craving modulation (Liu et al., 2017), inhibition and impulse/emotional control (Henigsberg et al., 2017; Coplan et al., 2018; Geng et al., 2018). Moreover, neuroimaging studies have suggested an involvement of the DLPFC in Machiavellian behavior (tendencies to distrust, deceive and exploit others) (Cohen-Zimerman et al., 2017). In principal accordance, anodal tDCS, with the anode positioned over the right DLPFC, reduced proactive aggression (Dambacher et al., 2015a). Because the left DLPFC has also been implicated in violent behavior, as suggested by neuroimaging studies (Yang and Raine, 2009), a bilateral involvement of this region in aggressiveness might take place. Increasing the activity of the right and left DLPFC simultaneously by anodal stimulation, which has not been tested in this context before, may thus be suited not only to reveal the causal involvement of this region in aggressiveness, but also represent a promising intervention tool to reduce it. In addition to the study of brain mechanisms of aggressiveness via tDCS, this technique has also been used to explore the neurobiology of moral behavior (Choy et al., 2018), which is closely related to aggressive behavior. An effect of tDCS over the ventral (Fumagalli et al., 2010) or medial (Yuan et al., 2017) prefrontal cortex, the temporoparietal junction (Sellaro et al., 2015; Ye et al., 2015; Leloup et al., 2016) and DLPFC (Kuehne et al., 2015; Zheng et al., 2018) on moral behavior has been demonstrated in healthy humans by different moral tasks. There are also studies showing an effect of invasive stimulation techniques, such as deep brain stimulation (DBS), on morality and moral behavior in different medical conditions, as for example in Parkinson's disease (for a review see Santens et al., 2018).

Here we aim to analyze the effect of bilateral anodal tDCS applied over the DLPFC on aggressiveness traits in two groups of imprisoned violent offenders with suggested different levels of aggressiveness (murderers, representing a high level of aggressiveness, and non-murderers, representing a lower level of aggressiveness). We applied bilateral anodal tDCS simultaneously over both hemispheres, as has previously been described for the motor cortex (Angius et al., 2018), posterior parietal cortex (Klein et al., 2013), and DLPFC (Möller et al., 2017), in a bipolar bihemispheric electrode montage (Nasseri et al., 2015). Considering the relationship between reduced frontal activity and aggressiveness (Witte et al., 2009; Mehta and Beer, 2010; Tikàsz et al., 2017), we expected that enhancement of DLPF, or more generally, prefrontal cortex, activity by bilateral anodal tDCS results in lower scores of aggressiveness.

With this procedure we observed lower scores of aggressiveness after stimulation in both groups of prisoners. This finding suggests an involvement of the prefrontal cortex in self-perception of aggressiveness.

EXPERIMENTAL PROCEDURES

Participants

Forty-one imprisoned male violent offenders (mean age = 36.2 ± 12.3 years, minimum age = 19 years), from two different prisons of the south of Spain (located in Córdoba and Huelva), participated voluntarily in the study. They did not receive any economic compensation for participation in the study. The crimes committed by participants of the non-murderer group were all related to different kinds of robberies with violence (7 participants), fights for drug trafficking (10) or gender violence (9). Crimes of participants of the murderer group were also related to robberies with extreme violence (3), fights for drug trafficking resulting in murder (6), or gender murder (5) (except one murder case categorized as “by offer or reward”). Exclusion criteria were metal implants, diagnosis of epilepsy, psychosis or other neuropsychiatric disorders or being under central nervous system-active medication. The study was authorized by the

General Secretary of Penitentiary Institutions, Ministry of the Interior, Spain. All participants provided written informed consent prior to participation. To evaluate if the baseline aggressiveness of inmates was high, aggressiveness measures were also recorded in a group of psychology students without tDCS intervention (*n* = 51, mean age = 20.9 ± 2.04 years, 14 males and 37 females). The Ethics Committee of the University of Huelva approved the experimental procedures. The study complies with the Code of Ethics of the World Medical Association Declaration of Helsinki.

Procedure

tDCS

tDCS over the prefrontal cortex was delivered by two battery driven constant-current stimulators (TCT Research Ltd tDCS Stimulator, TST Kowloon, Hong Kong) (Wexler, 2015; Brennan et al., 2017) with conductive rubber electrodes placed between two saline-soaked sponges. Considering that previous studies have shown that F3/F4 locations of the international 10-20 EEG system for electrode placement (Klem et al., 1999; Herwig et al., 2003) are well-suited proxies of the anatomical position of the left and right DLPFC, respectively, as probed by neuronavigation (De Witte et al., 2018) and magnetic resonance imaging-based DLPFC localization (Wörsching et al., 2017), the anode electrodes were placed over F3 and F4. As mentioned above, bilateral anodal tDCS applied simultaneously over both hemispheres has been tested in previous studies targeting the DLPFC (Möller et al., 2017). The cathode return electrodes were placed over the supraorbital ridges (Fp2 and Fp1 according to the international 10-20 EEG system), and the minimum distance between the edges of all electrodes was 4 cm (Moliadze et al., 2010). The size of the anode and cathode electrodes was 5 × 5 cm (25 cm^2) and 5 × 7 cm (35 cm^2), respectively. The DLPFC electrode size was slightly smaller so that the (anodal) current density was larger over the target region (Nitsche et al., 2007; Bikson et al., 2010). The electrodes were fixed onto the head by a tDCS headstrap (CMUS1209, Caputron Universal Strap, USA). Stimulation was applied for 15 min with an intensity of 1.5 mA. Stimulation was gradually ramped up and down for 10 sec at the beginning and the end of stimulation, respectively. For sham tDCS at 1.5 mA, current was increased and then decreased over 10 sec at the beginning and end of the session, respectively, to ensure some tingling sensation typical for real tDCS but avoid after-effects of stimulation. Subjects were blinded for tDCS conditions. After the application of tDCS, all participants were asked if they experienced intervention-caused tingling or other skin sensations. Fig. 1 shows a representation of the position, size and distance between electrodes, as well as a picture exemplifying the electrode montage. Fig. 2 depicts the position of the electrodes according to the international 10-20 EEG system, and a dorsal view of the human brain showing the localization of the DLPFC with respect to the electrode montage.

FIGURE 1 APPROXIMATELY HERE, PLEASE

FIGURE 2 APPROXIMATELY HERE, PLEASE

Aggressiveness measure

Self-reported aggressiveness was recorded by a Spanish version (Andreu Rodríguez et al., 2002; Vigil-Colet et al., 2005) of the Buss-Perry Aggression Questionnaire (BAQ) (Buss and Perry, 1992). The BAQ is a 40 items self-rating scale that has been validated in different populations. Participants rate themselves on each statement as follows: the smallest number on the scale (1) means "extremely uncharacteristic for me", and the largest number on the scale (5) means "extremely characteristic for me". Thus, a higher aggression degree corresponds to a higher score. The BAQ includes 4 dimensions of aggressiveness (Physical aggression, Anger, Verbal aggression, and Hostility) with high internal consistency, test-retest reliability coefficients (Pan et al., 2018), and construct validity (Gerevich et al., 2007). In the general population of federal offenders (Diamond and Magaletta, 2006), higher scores on BAQ subscales were recorded in offenders with a prior history of violence, head injuries, childhood abuse, residential treatment, custody, or foster care as a child.

Design

According to the official prison records of the participants, two groups of inmates were included in the study (murderers n = 15 vs. non-murderers n = 26). In each of these groups, participants were assigned to one of two stimulation conditions (real vs. sham). The group size in the respective experimental and control conditions was: non-murderers and anodal tDCS over the DLPFC, n = 13 (NM-anodal); non-murderers and sham tDCS, $n = 13$ (NM-sham); murderers and anodal tDCS over the DLPFC, $n = 8$ (M-anodal); murderers and sham tDCS, $n = 7$ (M-sham). All participants completed the BAQ immediately before the first tDCS session (Pre-tDCS), as well as immediately after 3 tDCS sessions (Post-tDCS), one session per consecutive day. Therefore, the BAQ was completed the first and the third day of the experimental procedure. The non-prisoners sample completed the BAQ once, to compare baseline measures between the three groups.

Statistical analysis

For each dimension of the BAQ, a one-way ANOVA was conducted to analyze baseline differences (pre-tDCS) between murderers and non-murderers. Baseline scores of both groups were also compared with those of the non-prisoner group. A mixed 2 × 2 × 2 ANOVA, with two between-subject factors, the first being the group (murderers vs. non-murderers) and the second factor the tDCS condition (real vs. sham), was conducted to analyze the scores in each of the four BAQ dimensions (dependent variable) recorded before and after stimulation (within-subject factor). In case of significant results of the ANOVAs, post-hoc Bonferroni-corrected Student's *t* tests were conducted. Correlation coefficients between age, type of crime committed, and post-stimulation effects were also calculated within each group. The chi-square test was performed to analyze possible group differences in percentages of correct identification of the respective tDCS condition (real vs. sham). The critical level of significance was set to $p < 0.05$ in all tests. The analyses were carried out using SPSS software.

RESULTS

No serious adverse effects during or after tDCS were reported by any of the participants. Tingling sensations were the only adverse effects consistently reported in both stimulation conditions. The chi-square test [$\chi^2(1, N = 41)$] revealed no significant differences between observed and expected percentages of correct identification of the stimulation condition ($p > .05$). The one-way ANOVA for baseline measures (pre-tDCS) indicated that the mean score of the murderers group was higher than that of the non-murderers group only in the Verbal aggression dimension ($F(1, 39) = 11.729$, $p = .001$, $\eta_p 2 = .231$). No baseline differences between both groups were found in the remaining aggression dimensions. When the pre-tDCS measures of the murderer and non-murderer groups were compared to the BAQ scores of the non-prisoners sample, lower baseline values were found in the non-prisoner group in the dimensions of Physical aggression ($F(2, 89) = 8.839$, $p < .001$, $\eta_p2 = .166$) and Hostility ($F(2, 89) = 56.87$, $p < .001$, $\eta_p2 = .561$). Interestingly, the scores of the non-prisoner group in the Verbal aggression dimension was higher than those of the non-murderer group ($F(2, 89) = 7.62$, $p = .001$, $\eta_p2 = .146$) (Fig. 3). The results of the repeated-measures ANOVA conducted to analyze differences between groups in each of the four BAQ dimensions are shown in Table 1. Fig. 4 shows the mean scores of each group in each BAQ dimension before and after intervention.

TABLE 1 APPROXIMATELY HERE, PLEASE

FIGURE 3 APPROXIMATELY HERE, PLEASE

FIGURE 4 APPROXIMATELY HERE, PLEASE

The mixed model ANOVA of the Physical aggression dimension showed a significant main effect of the factor time (pre-tDCS vs. post-tDCS) ($F(1, 37) = 13.233$, $p = .001$, $\eta_p2 = .263$) and the interaction between stimulation and time ($F(1, 37) = 14.307$, $p = .001$, $\eta_p2 = .279$). Post-hoc comparisons revealed significant differences between pre-anodal tDCS and post-anodal tDCS in the murderer ($p < .001$) and non-murderer ($p = .010$) groups, with higher values before stimulation in both groups. There were no significant differences between anodal and sham tDCS before stimulation in the murderer ($p = .878$) and non-murderer ($p = .463$) groups. After stimulation, values in the anodal tDCS condition were lower than those of sham tDCS in the murderer group ($p = .021$) but not in the non-murderer group ($p = .546$). In the anger dimension, the ANOVA results show a significant main effect of the factor time ($F(1, 37) = 28.505$, $p < .001$, $\eta_p2 = .435$) and a significant interaction between stimulation and time ($F(1, 37) = 27.624$, $p < .001$, $\eta_p2 = .427$). Post-hoc comparisons revealed significant differences between pre-anodal tDCS and post-anodal tDCS in the murderer ($p < .001$) and non-murderer ($p < .001$) groups, again with higher values before stimulation. No significant differences were found between anodal and sham tDCS before stimulation in the murderer group ($p = .634$), but lower values were found in the sham condition of the non-murderer group (p = .033). After tDCS, values in the anodal condition were lower than those of sham tDCS in the murderer group (p < .05) but not in the non-murderer group (p = .563). The ANOVA of the Verbal aggression dimension showed significant main effects of the factor group (F(1, 37) = 11.540, p < .002, η_p2 = .238), stimulation (F(1, 37) = 5.984, p = .019, η_p2 = .139), and time (F(1, 37) = 7.389, p < .010, η_p2 = .166). The interaction between the factors stimulation and time was also significant (F(1, 37) = 11.328, p = .002, η_p2 = .234). Post-hoc comparisons revealed significant differences between pre-anodal tDCS and post-anodal tDCS in the murderer (p < .001) and non-murderer (p = .032) groups, with higher values before stimulation in both groups. There were no significant differences between anodal and sham tDCS before stimulation in the murderer (p = .426) and non-murderer (p = .431) groups. After stimulation, values in the anodal tDCS condition were lower than those of sham tDCS in the murderer (p = .002) and non-murderer (p = .010) groups. Finally, the ANOVA of the Hostility dimension resulted in a significant main effect of the factor time (F(1, 37) = 4.142, p = .049, η_p2 = .101) and a significant interaction between stimulation and time (F(1, 37) = 4.828, p = .034, η_p2 = .115). Post-hoc comparisons revealed significant differences between pre-anodal tDCS and post-anodal tDCS in the group of murderers (p = .013), with higher values before stimulation. No significant differences were found between anodal and sham tDCS before stimulation in the murderer (p = .500) and non-murderer (p = .418) groups. After stimulation, values in the anodal tDCS condition were lower than those of sham tDCS in the murderer group (p < .05) but not in the non-murderer group (p = .824). No correlation was found between age and post-stimulation effects in the murderer (Pearson's correlation coefficient for Physical aggression, Anger, Verbal aggression, and Hostility, respectively, = -0.03, -0.19, 0.2, -0.2; p > 0.05) and non-murderer (-0.12, 0.12, -0.2, 0.03; p > 0.05) groups, or between type of crime and post-stimulation effects in the murderer (0.2, 0.11, 0.2, 0.2; p > 0.05) and non-murderer (-0.2, -0.04, -0.16, 0.2; p > 0.05) groups.

DISCUSSION

The present study included an imprisoned men sample, in which aggression levels, according to the crimes committed, are suggested to be considerably larger than in a standard non-reclusive population. The baseline differences found between prisoners and non-prisoners in two of the four aggression dimensions of the BAQ are compatible with such an increased level of aggressiveness of the imprisoned groups. The baseline aggressiveness in both inmate groups did not differ in 3 of the 4 dimensions. Only in the Verbal aggression dimension the scores of the murderer group were higher compared to the non-murderer group. In the two inmate groups, multiple sessions of tDCS applied bilaterally over the prefrontal cortex reduced self-perception of aggressiveness, as compared to baseline. This effect was consistent throughout the 4 aggression dimensions evaluated in the group of murderers, and in 3 of the 4 dimensions in the group of non-murderers. When anodal tDCS was compared to sham, lower values were found in all BAQ dimensions after real stimulation only in the murderer group. The non-murderer group also showed lower scores after real stimulation, compared to sham, but only in the Verbal aggression dimension. Thus, the application of anodal tDCS was associated with a more general effect in the murderer group, involving all aggressiveness dimensions evaluated. This slight difference in the aspects of aggression influenced by the stimulation procedure may be due to baseline differences between both groups regarding the measures of aggressiveness (significant baseline differences were found only in the Verbal aggression dimension, although in that dimension, anodal tDCS reduced the post-stimulation scores in both groups). Alternatively, this slight difference may also be attributed to a different response of the prefrontal cortex to stimulation in the group with apparently greater aggression levels. Regardless of these differences between both groups, these findings are consistent with previous studies focused on the role of the prefrontal cortex in aggressiveness (Witte et al., 2009; Dambacher et al., 2015a).

The effects found in this study are specifically in accordance with other studies exploring the involvement of the DLPFC in aggression. A respective effect of non-invasive brain stimulation over the right or left DLPFC on reactive and proactive aggressive behavior has been reported in a repetitive transcranial magnetic stimulation (rTMS) study using the Social Orientation Paradigm (Perach-Barzilay et al., 2013). Considering that bilateral tDCS (1.5 mA, for about 21 min) over the inferior frontal gyrus (F7 and F8), with different polarities over the hemispheres, did not have an effect on aggression (Dambacher et al., 2015b), the effect of the study protocol applied in the present study is fairly specific. This specificity might be based on the specific frontal area stimulated, and/or different stimulation polarities over the hemispheres.

Regarding proposed mechanisms underlying this effect of tDCS over the DLPFC on aggression, the prefrontal cortex is an important anatomical substrate of the cortical network involved in aggression (Repple et al., 2017), and the DLPFC is a relevant component of this network (Klasen et al., 2013). Particularly, the interaction between the DLPFC and the amygdala is involved in the control of aggressive behaviors. In a neuroimaging study, administration of quetiapine (an atypical antipsychotic) increased functional connectivity of this aggression-related network, and was associated with anti-aggressive effects (Klasen et al., 2013). The respective functional connectivity between the prefrontal cortex and the amygdala is also involved in the regulation of negative emotions (Banks et al., 2007), which could be a potential mechanism for the control of aggressive behavior. Beyond these connections, intra-prefrontal networks are also relevant for aggressive reactions (Chester and DeWall, 2018). It can be inferred that reduced activity of the prefrontal cortex and its connectivity increases aggression, because prefrontal cortex "top-down" control to modulate aggressive behaviors becomes limited (Siever, 2008). Therefore, we hypothesized that enhancement of DLPFC excitability and activity via tDCS might be promising to reduce aggressive tendencies. The findings of the present study are congruent with this hypothesis. The reduced scores in the BAQ observed after stimulation over the prefrontal cortex indicate a modulatory effect of tDCS over the DLPFC (or perhaps more generally over the prefrontal cortex/frontal lobe) on aggression.

Some limitations of this study should be taken into account. The study was single-blinded, since only one researcher was allowed to enter the prison and a device for coded blinding was not available. Furthermore, we only applied anodal tDCS to induce excitability alterations of the DLPFC. A cathodal DLPFC tDCS condition would have been advantageous to demonstrate specificity of the results. If aggression is related to DLPFC activity, cathodal tDCS over this region might however enhance aggressiveness, because it reduces cortical excitability (Nitsche and Paulus, 2000, 2001; Batsikadze et al., 2013), which would have been an effect we aimed to avoid because of the specific participant group. Moreover, since in our stimulation protocol the cathodes were placed over the frontopolar cortex, the prefrontal stimulation effect might be due not only to the respective excitability alterations of the DLPFC, but also due to an influence of the cathodal electrodes over frontopolar areas or to an extended effect of the electric field over other prefrontal regions, such as the ventromedial prefrontal cortex. Although the specificity of electrode placement is important to reproduce tDCS effects, the results of the current study nevertheless show, in a more general way, the possibility of modulating the perception of aggressiveness by non-invasive brain stimulation methods. In future studies, the implementation of more focal tDCS procedures, such as HD tDCS, could provide valuable information on the specific cortical areas involved in the effects of stimulation. Besides, the effect of prefrontal vs stimulation remote from frontal areas could be directly compared to elucidate the functional involvement of specifically the prefrontal cortex in aggressiveness with larger certainty. On the other hand, data acquisition in this study was restricted to the aggression questionnaire monitoring self-perceived aggression, and it can be argued that reactive/proactive aggression and self-reported aggression do not necessarily depend on the same mechanisms. However, the impact of DLPFC stimulation might not be restricted to perceived aggression, because anodal tDCS (2mA, 12.5 min) applied over the right DLPFC (with the cathode placed over the left DLPFC) reduced proactive aggression in men as measured by the Taylor aggression paradigm in another study (Dambacher et al., 2015a). This possibility should be explored systematically in future studies. Furthermore, in the present study we applied bilateral tDCS over the DLPFC, following the logic of previous studies suggesting a bihemispheric involvement of this area in aggression. This however makes it impossible to explore the relative contribution of the left and right hemisphere to the reduction of aggression. Thus, the functional relevance of both hemispheres in aggressiveness needs to be explored systematically in future studies contrasting the efficacy of unilateral and bilateral DLPFC tDCS. Exploration of the physiological effects of DLPFC stimulation would have been advantageous to evaluate mechanisms of the respective effects on those aggression measures, but was not possible in this specific environment. In addition, the inclusion of a non-imprisoned population as an added tDCS control group might have further strengthened the results and helped to answer the question if this intervention is only efficient for reduction of pathologically enhanced aggression or has a more general impact. Finally, long-term effects of stimulation were not obtained, thus future studies should include measures at different time-points to explore the duration of respective effects.

In conclusion, three consecutive sessions of bilateral prefrontal cortex tDCS reduced self-perceived aggressiveness as measured by the BAQ in men with high aggression potential. This effect was constant throughout the different aggression dimensions and similar in both groups of prisoners, murderers and non-murderers, although an effect on all dimensions was only evident in the group of murderers. The results of this pilot study encourage future studies to explore the mechanistic aspects of these effects into larger detail, including application of anodal and cathodal stimulation, both unilateral and bilateral, and to evaluate application-relevant features, such as exploring its effects on real aggressive behavior and on clinical syndromes.

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FIGURE CAPTIONS

Fig. 1. A, Picture showing the electrode montage of this study. B, Representation of the electrode montage showing the position (10-20 EEG system), size and distances between electrodes. Anodes 5 × 5 cm (F3/F4), cathodes 5 × 7 cm (Fp1/Fp2). The resulting distance between electrode edges was not less than 4 cm.

Fig. 2. Bipolar bihemispheric electrode montage according to the international 10-20 EEG system (anodes over F3 and F4, and cathodes over Fp1 and Fp2). A, Dorsal view of the human brain showing the localization of the dorsolateral prefrontal cortex (DLPFC) in red color (in the online version). B, Localization of the DLPFC with respect to the electrode montage. Images obtained from the NIC 2.0 Neuroelectrics free software.

Fig. 3. Baseline **(**Pre-tDCS) mean scores of the murderer (M), non-murderer (NM), and non-prisoner (NP) groups in each dimension of the Buss-Perry Aggression Questionnaire (Buss and Perry, 1992) (error bars show standard deviation). PA, Physical aggression; A, Anger; VA, Verbal aggression; H, Hostility. (*), significant differences in the mean scores with respect to the other two groups in each dimension (*p* < .05).

Fig. 4. Mean scores of both inmate groups (murderers –M vs. non-murderers –NM) in each dimension of the Buss-Perry Aggression Questionnaire (Buss and Perry, 1992) (error bars show standard deviation). A, Physical aggression; B, Anger; C, Verbal aggression; D, Hostility. (*) and (**), significant differences between pre-and post-stimulation ($p < .05$ and $p < .001$, respectively). (#), significant differences between anodal and sham stimulation within each group ($p < .05$).

Table 1. Results of the repeated-measures ANOVAs.

	d.f.	***F*-value**	***p*-value**	η_p^2	**Statistical Power (1 – β)**
Physical aggression					
2 × 2 × 2 ANOVA					
Group	1	1.556	.220	.040	.229
Stimulation	1	2.274	.140	.058	.312
Measure	1	13.223	.001*	.263	.943
Group × Stimulation	1	3.238	.080	.080	.418
Group × Measur	1	0.735	.397	.019	.133
Stimulation × Measure	1	14.307	.001*	.279	.958
Group × Stimulation × Measure	1	3.602	.066	.089	.456

Anger					
2 × 2 × 2 ANOVA					
Group	1	0.124	.727	.003	.064
Stimulation	1	0.189	.666	.005	.071
Measure	1	28.505	< .001*	.435	.999
Group × Stimulation	1	2.681	.110	.068	.358
Group × Measure	1	0.090	.766	.002	.060
Stimulation × Measure	1	27.624	< .001*	.427	.999
Group × Stimulation × Measure	1	0.745	.394	.020	.134
Verbal aggression					
2 × 2 × 2 ANOVA					
Group	1	11.540	.002*	.238	.911
Stimulation	1	5.984	.019*	.139	.664
Measure	1	7.389	.010*	.166	.754
Group × Stimulation	1	0.591	.447	.016	.116
Group × Measure	1	1.327	.257	.035	.202
Stimulation × Measure	1	11.328	.002*	.234	.906
Group × Stimulation × Measure	1	1.272	.267	.033	.196

Hostility					
2 × 2 × 2 ANOVA					
Group	1	0.350	.558	.009	.089
Stimulation	1	0.584	.450	.016	.116
Measure	1	4.142	.049*	.101	.509
Group × Stimulation	1	2.237	.143	.057	.308
Group × Measure	1	0.408	.527	.011	.095
Stimulation × Measure	1	4.828	.034*	.115	.572
Group × Stimulation × Measure	1	0.642	.428	.017	.122

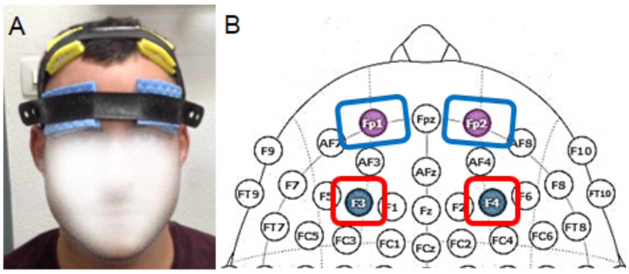
A mixed three-way ANOVA (group × stimulation condition × pre/post measure) was calculated to analyze the pre/post-stimulation mean scores of each group recorded in each of the four BAQ factors. *$p < .05$. d.f., degrees of freedom.

Highlights

*3 sessions of bilateral prefrontal cortex tDCS reduce self-reported aggressiveness

*This effect is observed in all dimensions of the BAQ in a sample of murderers

*In a sample of non-murderers the effect is observed in 3 of the 4 BAQ dimensions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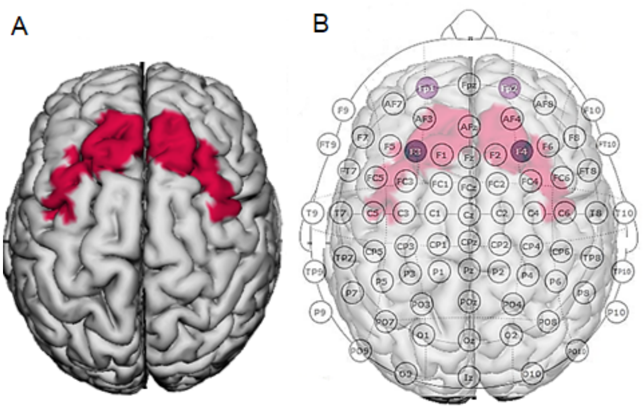
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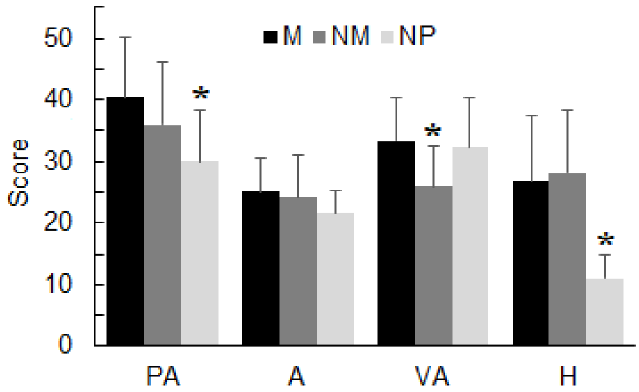
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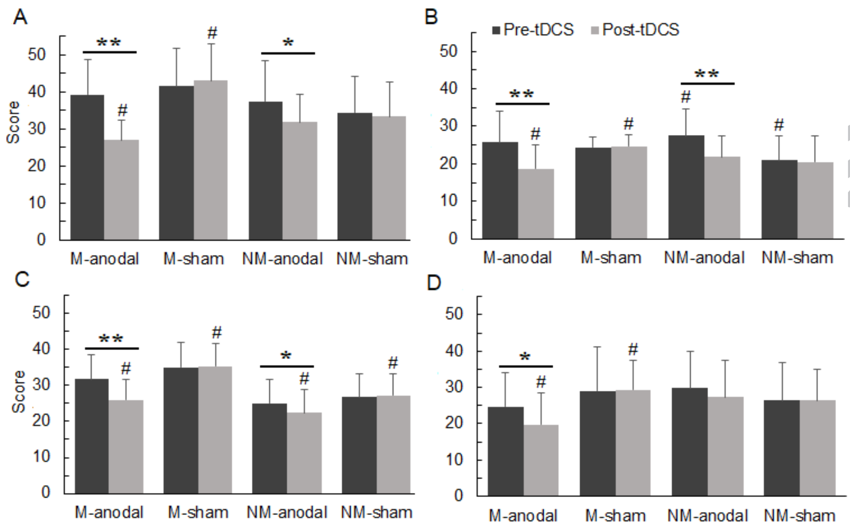
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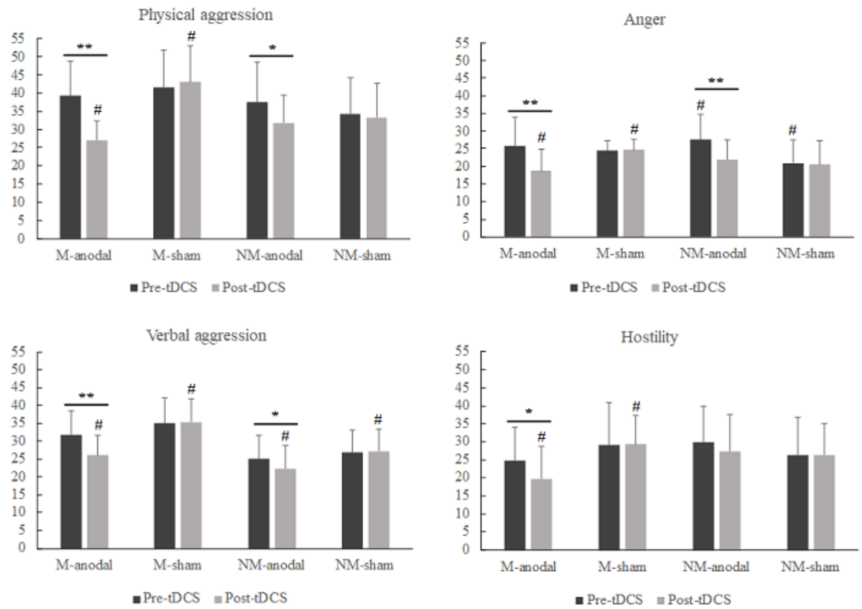
Graphical abstract



Three sessions of bilateral prefrontal cortex anodal tDCS reduce aggressiveness in both murderer (as revealed by scores in the four BAQ dimensions of aggressiveness) and non-murderer (as revealed by scores in all BAQ dimensions except in Hostility) samples.