



# No Effect of Transcranial Direct Current Stimulation over Left Dorsolateral Prefrontal Cortex on Temporal Attention

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## Abstract

■ Selection mechanisms that dynamically gate only relevant perceptual information for further processing and sustained representation in working memory are critical for goal-directed behavior. We examined whether this gating process can be modulated by transcranial direct current stimulation (tDCS) over left dorsolateral prefrontal cortex (IDLDFC)—a region known to play a key role in working memory and conscious access. Specifically, we examined the effects of tDCS on the magnitude of the “attentional blink” (AB), a deficit in identifying the second of two targets presented in rapid succession. Thirty-four participants performed an AB task before (baseline), during and after 20 min of 1-mA anodal and cathodal tDCS in two separate sessions. On the basis of previous reports linking individual differences in AB magnitude

to individual differences in DLPFC activity and on the basis of suggestions that effects of tDCS depend on baseline brain activity levels, we hypothesized that anodal tDCS over IDLDFC would modulate the magnitude of the AB as a function of individual baseline AB magnitude. Behavioral results did not provide support for this hypothesis. At the group level, we also did not observe any significant effects of tDCS, and a Bayesian analysis revealed strong evidence that tDCS to IDLDFC did not affect AB performance. Together, these findings do not support the idea that there is an optimal level of prefrontal cortical excitability for cognitive function. More generally, they add to a growing body of work that challenges the idea that the effects of tDCS can be predicted from baseline levels of behavior. ■

## INTRODUCTION

Our senses convey a continuous stream of information to our brain. Our ability to interpret this information, however, is not constant over time. One phenomenon that brings this fact to light is the attentional blink (AB). When given the task to detect two targets among a stream of distractors presented in rapid succession, participants are often unable to identify the second target (T2) if it is presented within approximately 200–500 msec of the first target (T1; Raymond, Shapiro, & Arnell, 1992). It is as if attention temporarily “blinks.” Since its discovery, a variety of models of the AB have been proposed that differ considerably in their specific architectures (Martens & Wyble, 2010; Dux & Marois, 2009; Olivers & Meeter, 2008; Shapiro, Raymond, & Arnell, 1997). Nevertheless, working memory (WM) plays a central role in virtually all of them. Initially, many theories relied on the idea that the AB reflects a limitation in processing resources required for the encoding and consolidation of target information in WM. For example, it was postulated that, when many WM resources are devoted to the processing of T1, too few may be available for T2, rendering its representation vulnerable to distractor interference (Chun & Potter, 1995; Shapiro, Raymond, & Arnell, 1994).

However, limited-capacity theories of the AB were challenged by results showing that participants have no trouble reporting three or more consecutive targets, as long as there are no intervening distractors (Di Lollo, Kawahara, Shahab Ghorashi, & Enns, 2005; see also Olivers, van der Stigchel, & Hulleman, 2007; Kawahara, Enns, & Lollo, 2006; Kawahara, Kumada, & Di Lollo, 2006; Nieuwenstein, 2006; Nieuwenstein & Potter, 2006). This observation is difficult to explain from a limited capacity perspective alone and spurred the development of novel accounts of the AB, which assign greater importance to the first distractor stimulus after T1 and explain the AB in terms of dysfunctional gating of information into WM, rather than a capacity limitation of WM per se (Olivers & Meeter, 2008; Di Lollo et al., 2005).

Findings from neuroimaging and electrophysiological studies confirm the critical role of WM in the AB. For example, activity in brain areas crucial for WM processes, such as lateral frontal and parietal cortex, reliably differentiates between trials in which T2 is seen (no-blink trials) versus missed (blink trials; Slagter, Johnstone, Beets, & Davidson, 2010; Kranczioch, Debener, Schwarzbach, Goebel, & Engel, 2005; Gross et al., 2004; Marois, Chun, & Gore, 2000). Especially, left dorsolateral prefrontal cortex (IDLDFC) seems to be a critical node in the AB (Slagter et al., 2010; Hommel et al., 2006). Slagter et al. (2010), for example, found that, across participants, greater T2

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detection-related activity in IDLPFC was associated with a smaller AB. This is notable because IDLPFC is deemed particularly important in situations that require maintenance of target information in WM in the face of distraction (Feredoes, Heinen, Weiskopf, Ruff, & Driver, 2011; McNab & Klingberg, 2008; Postle, 2006), as is the case in the AB task. Furthermore, this finding suggests that differences in IDLPFC activation might contribute to individual differences in AB magnitude. One consistent observation in the literature is that individuals differ substantially in the size of their AB; some display a very big AB and almost always miss T2, whereas others virtually always detect T2 (i.e., display no AB; Martens, Munneke, Smid, & Johnson, 2006), with most individuals falling somewhere between these two extremes (Dale, Dux, & Arnell, 2013). Activity levels in IDLPFC may underlie these individual differences in AB magnitude and in target selection more generally.

In this study, we used transcranial direct current stimulation (tDCS) over IDLPFC to modulate the excitability of this brain region and examine if it is causally involved in the AB. The reference electrode was placed over right anterior prefrontal cortex (apFC). In tDCS, a weak and constant electric current is delivered to the brain using two electrodes attached to the scalp. Current flows from the positive (anodal) to the negative (cathodal) electrode. The excitability of cortical areas below the anodal electrode is increased, whereas areas below the cathodal electrode decrease in excitability (Nitsche & Paulus, 2000; Bindman, Lippold, & Redfearn, 1962). An important difference between tDCS and transcranial magnetic stimulation, another widely used stimulation technique, is that it does not directly induce neuronal action potentials. Rather, tDCS makes the endogenous generation of action potentials more or less likely by tonically depolarizing or hyperpolarizing the resting membrane potential (Nitsche et al., 2008). In addition to these immediate effects, long-term effects of up to 1.5 hr after stimulation have been shown for stimulation durations of 9 min and longer in the motor domain (Nitsche & Paulus, 2001). Recent work furthermore suggests that stimulation effects on performance are at least in part determined by the preexisting balance between cortical excitation and inhibition. The relation between the excitation/inhibition balance within a brain area and its efficiency is argued to follow an inverted-U shape in which functioning is optimal when excitation and inhibition interact in a way that permits both flexibility (i.e., plasticity, new learning) and stability (i.e., resistance to distraction, maintenance of information).

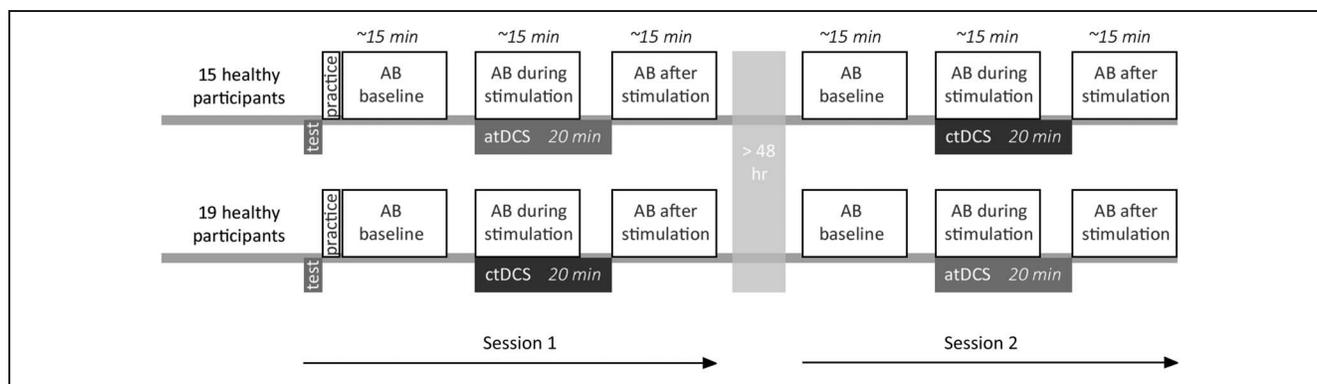
Baseline excitation/inhibition balances might, however, vary across individuals within a given brain region and determine the effect of brain stimulation depending on whether it moves the balance toward or away from its optimum. For example, if a certain brain area is functioning optimally, anodal tDCS (atDCS) would worsen its efficiency due to overexcitation. Conversely, if the area functions suboptimally because of overinhibition, atDCS would improve its efficiency. Thus, atDCS may improve

performance in individuals with suboptimal levels of cortical excitability, while impairing performance in individuals with optimal or supraoptimal cortical excitability by causing overexcitation.

Given that individual differences in AB magnitude have been linked to individual differences in IDLPFC activation (Slagter et al., 2010), and the fact that stimulation effects on performance may depend on baseline brain excitability levels, we hypothesized that atDCS over IDLPFC would modulate the AB as a function of individual baseline AB magnitude. Specifically, our main prediction was that individuals with a relatively large AB to begin with would benefit most from anodal stimulation, whereas those with a small baseline AB would benefit less or even display a decrement in performance as a result of anodal stimulation. We did not expect an effect of cathodal tDCS (ctDCS) given previous work using a similar stimulation protocol showing no differential effect of cathodal versus sham stimulation of IDLPFC on WM performance (Fregni et al., 2005).

To control for polarity-unrelated effects of tDCS, we applied both atDCS and ctDCS over IDLPFC. If the mere fact of receiving tDCS would systematically influence performance on the AB task, for example, by distracting the participant and producing a more diffuse attentional set (Arend, Johnston, & Shapiro, 2006; Olivers & Nieuwenhuis, 2006), then we would expect similar results for both stimulation conditions. If, however, only atDCS affects performance, we can exclude the possibility that such nonspecific effects contributed to our findings and safely conclude that AB performance was modulated by anodal electrical stimulation.

In addition to examining the effects of tDCS on T2 identification, we looked at how tDCS affects the ability to inhibit irrelevant distractors. Distractor inhibition appears to play an important role in the AB (Arnell & Stubitz, 2010; Olivers & Meeter, 2008; Di Lollo et al., 2005; Chun & Potter, 1995), and evidence suggests that IDLPFC may be particularly involved in situations in which target information has to be maintained in WM in the face of distraction (Feredoes et al., 2011; McNab & Klingberg, 2008; Postle, 2006). It is thus possible that any observed stimulation-induced modulations of the AB reflect a changed ability to inhibit distracting information. To explore the role of IDLPFC in distractor inhibition and enhance our understanding of how this area is involved in the AB, we used a version of the AB task designed by Dux and Marois (2008) that permitted us to quantify not only participants' AB performance but also their ability to inhibit distractor stimuli. Specifically, distractor inhibition was inferred by examining the influence of a post-T1 distractor that primed T2 on subsequent T2 identification. More effective distractor inhibition should lead to a prime-related reduction in T2 identification, because strong suppression of the prime stimulus should render reactivation of this representation by T2 more difficult. Conversely, less effective distractor inhibition should lead to a prime-related enhancement in T2 identification, because the representation of T2 benefits from residual activation of its identity



**Figure 1.** Experimental procedure. Participants completed two sessions that differed only in the polarity of the tDCS administered (atDCS or ctDCS). In each session, participants performed three blocks of 200 trials of the AB task: one before tDCS (baseline), one during the 20-min stimulation period (during tDCS), and one after the stimulation ended (after tDCS). Sessions were at least 48 hr apart.

by the prime. If anodal stimulation over IDLPFC improves or disrupts distractor inhibition, one would thus expect to see a smaller or larger priming effect, respectively. Furthermore, if distractor inhibition is an important determinant of the AB, one may predict that anodal-stimulation-induced changes in distractor inhibition lead to changes in AB performance.

Thus, the current study examined the effects of atDCS versus ctDCS over IDLPFC on the AB and on distractor inhibition. Our main hypothesis was that atDCS, but not ctDCS, over IDLPFC would modulate the AB as a function of individual baseline AB magnitude and by changing participants' ability to inhibit distracting information.

### Relation to London and Slagter (2015)

This study is a republication of the study reported in London and Slagter (2015). That paper has been retracted because of an erroneous result that affected one of the main conclusions and that was only discovered subsequent to publication. The current paper hence supersedes London and Slagter (2015).

## METHODS

### Participants

Thirty-eight participants (22 women, mean age = 22.4 years,  $SD = 2.8$ ), of which four were later excluded (see Results), took part in the study. All had normal or corrected-to-normal sight, had no history of neurological or psychiatric disorders, and were not color blind. The study was approved by the local ethics committee. Participants gave written informed consent and were compensated with course credit or money (€10 per hour). Four participants (two women) were excluded because of poor T1 identification (see below).

### Experimental Procedure

Participants performed an AB task in two sessions, one in which they received anodal stimulation and one in which

they received cathodal stimulation (see Figure 1 for an overview of the experimental design). These two sessions were separated by at least 48 hr to ensure that the effects of the previous session had washed out (Nitsche & Paulus, 2001). Session order was counterbalanced across participants, such that 15 participants received atDCS in the first session and ctDCS in the second, and 19 participants received ctDCS in the first session and atDCS in the second session. At the beginning of the first session, participants received 1-mA atDCS to IDLPFC for 15 sec, ramped up and down in 30 sec, so that they could briefly experience the sensation before beginning the experiment and decide whether or not they wanted to continue. All participants decided to continue with the experiment. During each session, participants first briefly practiced the AB task and then performed the AB task before stimulation (baseline), during stimulation, and again after stimulation. After each session, 33 of 34 participants completed questionnaires on possible physical side effects of the stimulation. In addition, before and after each session, of these 33 participants, 31 completed the short form of the Activation–Deactivation Adjective Check List (AD ACL) questionnaire designed to assess various arousal states and mood at the present moment (Thayer, 1978). The test–retest reliability of its four subscales is high: energy = .92, tension = .89, calmness = .89, and tiredness = .90.

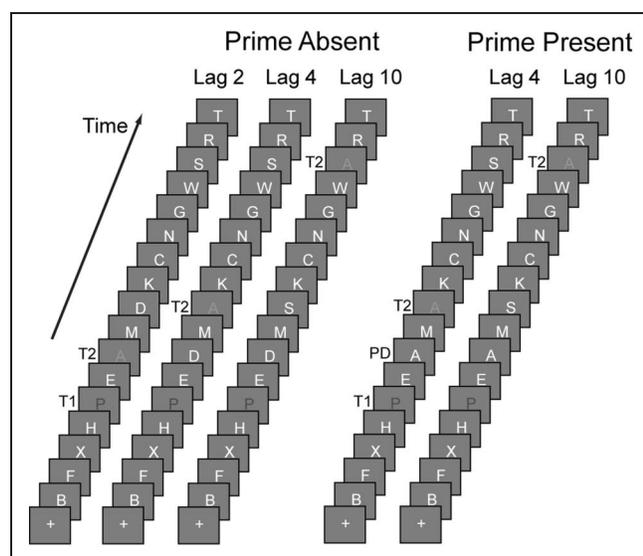
### tDCS

tDCS was delivered with a battery-driven, constant current stimulator (Neuroconn) with a maximum output of 10 mA and administered by two 35-cm<sup>2</sup> (5 × 7) galvanized rubber electrodes inside saline-soaked sponges that were placed on the scalp with rubber bands. The electrode of interest was placed over IDLPFC at the F3 position according to the 10–20 system for electroencephalogram (EEG) electrode placement (DaSilva, Volz, Bikson, & Fregni, 2011). The reference electrode was placed on the contralateral supraorbital area of the face, over right aPFC. This electrode setup is similar to previous studies, which reported effects of atDCS on WM performance (Keeser et al., 2011;

Teo, Hoy, Daskalakis & Fitzgerald, 2011; Fregni et al., 2005). In the anodal stimulation condition, participants received 1-mA stimulation over IDLPFC for 20 min. In the cathodal stimulation condition, participants received -1-mA stimulation over IDLPFC for 20 min. In both conditions, the current was ramped up in 20 sec and ramped down in 60 sec.

## Task and Design

The task was identical to the task used by Slagter and Georgopoulou (2013) and modeled after the task designed by Dux and Marois (2008) (see Figure 2). Each trial consisted of a rapid serial visual presentation (RSVP) of 17 uppercase letters excluding I, L, O, Q, U, and V (font type: Courier New, font size: 40). T1 was red; T2, green; the distractors, white; and the background, gray. Participants were instructed to detect both targets. T1 appeared at Serial Position 5, and T2 followed T1 after one, three, or nine distractors, that is, at Lag 2, Lag 4, and Lag 10, respectively. Each trial started with a fixation square presented for 480 msec, followed by the 17 letters, each presented for 92 msec. In prime-absent trials, all stimuli were different, whereas in the prime-present trials, the second distractor after T1 (i.e., at Lag 2) had the same identity as T2 (priming distractor). This distractor was presented at the lag where the AB is typically maximal, making it unlikely that it was consciously perceived. Necessarily, priming distractors could only occur in trials in which T2 was presented at Lag 4 or Lag 10. Thus, there were five trial types: prime-absent Lag 2, prime-absent Lag 4, prime-absent Lag 10,



**Figure 2.** AB task. Participants viewed RSVP streams of letters. T1 was red (here displayed in dark gray), T2 was green (here displayed in light gray), and the distractors were white. T2 could appear at Lag 2, 4, or 10. In the prime-present trials, a priming distractor with the same identity as T2 appeared at Lag 2. All stimuli had different identities in the prime-absent trials. Participants reported the identities of T1 and T2 at the end of each stream.

prime-present Lag 4, and prime-present Lag 10. At the end of each RSVP, participants were prompted to input the target identities using a keyboard. The task was programmed using Presentation software (Neurobehavioral Systems, Inc.). Participants were seated approximately 90 cm from a computer screen in a comfortable chair. The 23-in. LCD high-performance gaming monitor was driven by a standard personal computer running the Microsoft operating system XP and refreshed at 120 Hz with a resolution of 1920 × 1080 pixels in 16-bit color.

In each session, participants performed three blocks of 200 trials of the AB task (approximately 15 min per block): a block before tDCS stimulation (baseline), a block during the 20-min stimulation period (during tDCS), and a block after stimulation ended (after tDCS; see Figure 1). Trial types were equally probable and randomly intermixed within a block. Participants thus performed 40 trials in each block of each trial type. Before each baseline block, participants performed 20 practice trials. The baseline blocks were included in each session to exclude possible differences between sessions in situational factors such as amount of sleep and mood from affecting our results, as both arousal and mood have been shown to affect the magnitude of the AB (Jefferies, Smilek, Eich, & Enns, 2008; Olivers & Nieuwenhuis, 2006).

## Questionnaires

The questionnaire on the possible side effects of tDCS consisted of eight items describing physical sensations for which participants were asked to indicate to what extent each had been present during stimulation. The eight physical sensations were itching, prickling, burning, pain, headache, fatigue, dizziness, and nausea. Response options were “not,” “a little,” “somewhat,” “strongly,” or “very strongly.”

The Short-Form AD ACL consists of 20 items on four subscales for which participants had to indicate whether they felt like this “definitely not,” “not really,” “a little,” or “definitely” (Thayer, 1978). The feelings probed were active, energetic, vigorous, lively, and full of pep (energy subscale); sleepy, tired, drowsy, wide-awake, and wakeful (tired subscale); jittery, intense, fearful, clutched up, and tense (tension subscale); and placid, calm, at rest, still, and quiet (calmness subscale).

## Analyses

### Individual Differences

To examine whether the effects of tDCS depended on individual differences in baseline AB performance and/or distractor inhibition ability, we first calculated two parameters: AB magnitude and T2 priming. This was done for each participant separately for each Block (before, during, after tDCS) and Stimulation session (anodal, cathodal). AB magnitude was calculated as T2 accuracy given that T1 was correct (T2 | T1) in Lag 10 versus Lag 2 prime-absent trials. T2 |

T1 accuracy was assessed in prime-absent trials to get a measure of AB magnitude that was independent of the prime (Slagter & Georgopoulou, 2013; Dux & Marois, 2008). T2 priming, from which the amount of distractor inhibition was inferred, was quantified as T2|T1 in Lag 4 prime-present trials versus Lag 4 prime-absent trials. T2 priming was only assessed at Lag 4, and not at Lag10, because of the short duration of RSVP priming (Maki, Frigen, & Paulson, 1997). To assess if the effects of tDCS depended on individual differences in baseline AB performance or baseline distractor inhibition ability, we then calculated the change from baseline, separately for AB magnitude and T2 priming, and the “during” and “after” atDCS and ctDCS blocks, by subtracting the respective baseline scores from the scores in each of these four blocks. Partial Pearson correlations between these change measures (e.g., AB magnitude during atDCS – before atDCS) and their corresponding baselines (i.e., AB magnitude before atDCS) were then used to estimate if effects of tDCS on performance during and after stimulation depended on initial performance. In these analyses, we controlled for session order and T1 performance to exclude the possibility that observed effects could simply be explained by practice effects or an overall change in target identification, respectively. Alpha (.05) was divided by 4 to account for the number of correlation tests used to assess the relationships between baseline performance and stimulation-related change in performance for both AB magnitude and T2 priming. Thus, an alpha of .0125 was applied.

Finally, measurement error is always a concern in analyses examining the relationship between a baseline measure and the change in this measure over time. It is well known that, if the variable has an extreme value when first measured, it will tend to be closer to the mean when measured on a second occasion and, if it has an extreme value during the second measurement, it will tend to have been closer to the mean at first. This problem is known as regression to the mean. Moreover, even when no relationship exists between the baseline ( $x$ ) and the change ( $y-x$ ), the fact that  $x$  is present in both terms leads to an expected correlation of  $-.7$  between  $x$  and  $y-x$  because of mathematical coupling (Archie, 1981). To address these issues and rule them out as alternative explanations, we applied a test for equality of variances to those variables of interest that were significantly correlated (Tu & Gilthorpe, 2007; Jin, 1992; Myrtek & Foerster, 1986).

### Group Level

**AB task.** We next examined if tDCS also affected AB performance and/or distractor inhibition at the group level. For each participant, Lag (2, 4, and 10), Block (baseline, during tDCS, after tDCS), and Stimulation condition (atDCS, ctDCS) separately, the percentage of trials in which T1 was accurately identified (regardless of T2 performance) and the percentage of trials in which both targets were correctly identified (T2|T1 accuracy) were calculated.

To investigate the effect of tDCS on the AB, a repeated-measures ANOVA with T2|T1 accuracy in prime-absent trials as the dependent variable was conducted with Lag (2, 4, 10), Block (baseline, during, after), and Stimulation (atDCS, ctDCS) as within-participant factors and Session order (atDCS first, ctDCS first) as a between-participant factor. A similar analysis was run with T1 accuracy as the dependent variable to assess effects of stimulation on T1 performance. The effect of tDCS on distractor inhibition was tested with a repeated-measures ANOVA with Prime (absent, present), Lag (4, 10), Block (baseline, during, after), and Stimulation (atDCS, ctDCS) as within-participant factors and Session order (atDCS first, ctDCS first) as a between-participant factor.

To provide more information regarding the likelihood of our data given the null versus alternative hypotheses regarding the effect of tDCS on the AB (a three-way interaction between Stimulation, Lag, and Block) and on performance in general (an interaction between Stimulation and Block), we conducted an analogous Bayesian analysis using the JASP software package (JASP Team, 2019). Bayes factors are reported in terms of evidence for the null hypothesis ( $BF_{01}$ ) and evidence for the alternative hypothesis ( $BF_{10}$ ). When there are multiple factors in the design, such as is the case here, this yields a very large amount of models. Therefore, we combined the evidence for or against each effect across all models, by using Bayesian model averaging (Rouder, Morey, Verhagen, Swagman, & Wagenmakers, 2017; Wagenmakers et al., 2017). Specifically, we calculated an “inclusion Bayes factor across matched models” as implemented in JASP. We used the classification proposed by van Doorn et al. (2020) to label the size of the Bayes factors as weak (1–3), moderate (3–10), or strong ( $>10$ ).

**Questionnaires.** To examine whether there were systematic differences in physical sensations between atDCS and ctDCS, paired-sample  $t$  tests were conducted for each of the eight items on the tDCS side-effects questionnaire. To determine whether there was a difference in the effects of atDCS versus ctDCS on arousal states, scores on each of the four subscales were calculated before and after stimulation for each stimulation session separately and subsequently subtracted from each other to obtain a measure of the effect of electrical stimulation. For each subscale separately, a paired-sample  $t$  test was then conducted comparing the resulting difference scores between the atDCS and ctDCS conditions. A Bonferroni correction was applied to account for multiple comparisons for both questionnaires, separately resulting in an alpha of  $.05/8 = .0063$  for the tDCS side-effects questionnaire and an alpha of  $.05/4 = .0125$  for the Short-Form AD ACL questionnaire.

## RESULTS

Four participants (two women) were excluded because of poor T1 identification ( $>2$  SDs below the mean). In the

remaining 34 participants, average T1 accuracy over both baseline measurements was 86%.

### Individual Differences

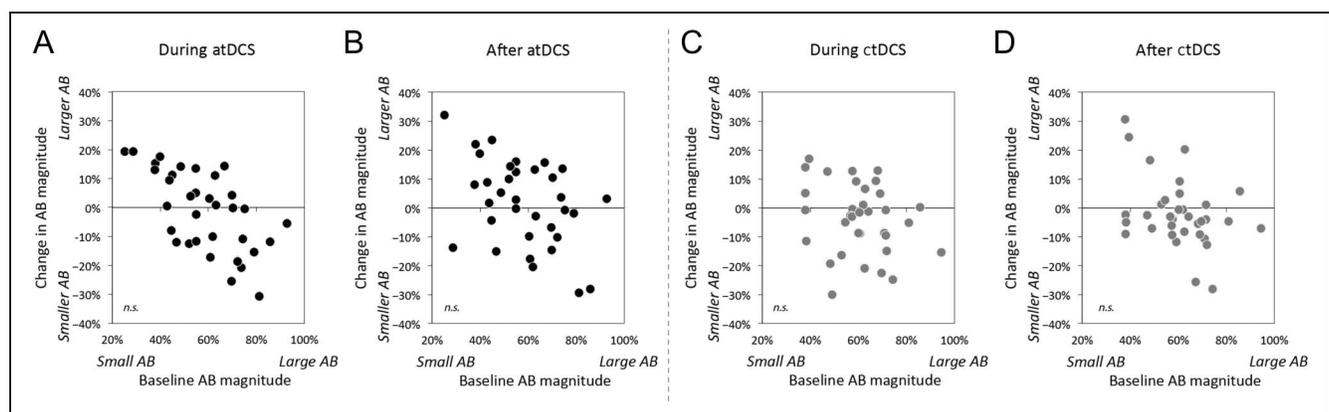
Replicating previous reports, large individual differences in AB magnitude (Dale et al., 2013) and distractor inhibition (Slagter & Georgopoulou, 2013; Dux & Marois, 2008) were observed.

#### *Individual Differences in Baseline AB Magnitude Did Not Predict the Effect of tDCS on AB Performance*

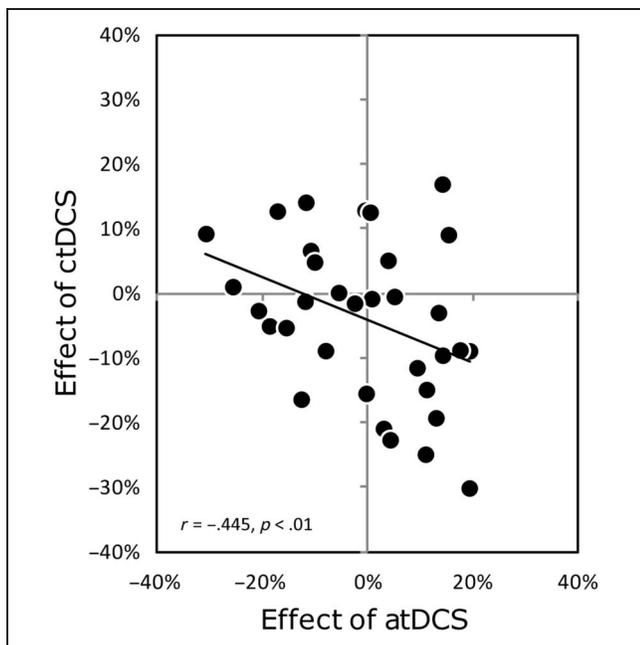
Our main prediction was that anodal stimulation would decrease the AB in individuals who have a relatively large AB to begin with, whereas it would increase the AB in individuals with a relatively small baseline AB. To test this prediction, we correlated individual baseline AB magnitude with the change in AB magnitude during (or after) stimulation versus baseline, while controlling for session order and possible stimulation-related changes in T1 accuracy at Lag 2, separately for atDCS and ctDCS. Initially, it seemed as though we found a significant inverse relation between baseline AB magnitude and change in AB magnitude during atDCS,  $r(30) = -.676, p < .001$  (see Figure 3A). However, as shown by a test of the equality of variances between the two conditions, this relationship between baseline AB magnitude and the effect of atDCS on the AB could be explained by regression to the mean. If participants with a large AB had indeed benefited from atDCS whereas participants with a small AB suffered, the variance of AB scores during baseline testing should be significantly higher than those during atDCS (Tu & Gilthorpe, 2007; Jin, 1992; Myrtek & Foerster, 1986). In contrast, if there is no effect of stimulation, then variance should remain the same from one measurement to the next. We found that variance of baseline AB scores (0.026) was not significantly higher than variance of AB scores during atDCS (0.017),  $t(32) =$

1.56,  $p < .128$ . Thus, critically, we cannot rule out regression to the mean as an explanation for our observation that individuals who had a relatively large baseline AB benefited from atDCS over IDLPFC in combination with ctDCS over right aPFC, whereas those with a small baseline AB benefited less or even exhibited a decrement in performance. No significant correlations were found between baseline AB magnitude during ctDCS or after ctDCS/atDCS. Although a modest correlation was observed between baseline AB and the post-anodal-stimulation change in AB magnitude,  $r(30) = -.427, p = .019$  (Figure 3B), it did not survive correction for multiple comparisons. Moreover, the test for equality of variances was not significant,  $t(32) = 0.23, p = .822$ , rendering it unclear if it reflects a true relationship or regression to the mean. In the cathodal condition, the correlation between baseline AB and poststimulation effect was also not significant after correction for multiple comparisons,  $r(30) = -.381, p = .038$  (Figure 3D).

To summarize, individual difference analyses revealed that neither atDCS over IDLPFC with ctDCS over right aPFC nor ctDCS over IDLPFC with atDCS over right aPFC affected the AB in individuals based on their baseline AB. The idea that there is an inverted U-shaped relationship between excitability and performance could also lead to the prediction that the effects of anodal and cathodal stimulation on AB magnitude for a given individual would go in opposite directions and that this direction would depend on where the participants find themselves on the curve before initiating tDCS. We explored this in a post hoc analysis in which we correlated the atDCS-induced change in AB magnitude with the ctDCS-induced change in AB magnitude across participants, while controlling for session order. This analysis revealed a significant negative relationship,  $r(31) = -.445, p < .01$  (see Figure 4), indicating that individuals who benefited from atDCS generally worsened because of ctDCS, whereas individuals who worsened because of atDCS tended to perform better because of ctDCS.



**Figure 3.** Baseline AB magnitude did not predict the effects of atDCS on the AB across participants during atDCS (A) or after atDCS (B). No significant relationship between baseline AB magnitude and effects of cathodal stimulation on the AB was observed either during (C) or after (D) cathodal stimulation. *n.s.* = nonsignificant.



**Figure 4.** The effects of atDCS and ctDCS opposed each other. Participants who improved with atDCS tended to get worse with ctDCS, and vice versa.

#### Individual Differences in Baseline Distractor Inhibition Are Not Predictive of the Effect of Stimulation on Distractor Inhibition

We next investigated whether effects of stimulation on distractor inhibition depended on an individual's baseline ability to inhibit distractors. To this end, we tested the relation between baseline T2 priming and the changes in T2 priming during and after atDCS and ctDCS, controlling for session order and for change in T1 accuracy at Lag 4 averaged over prime-present and prime-absent trials. Significant negative correlations were obtained for all tests conducted: baseline distractor priming, change in distractor priming during atDCS,  $r(30) = -.723, p < .001$ , during ctDCS,  $r(30) = -.578, p < .005$ , after atDCS,  $r(30) =$

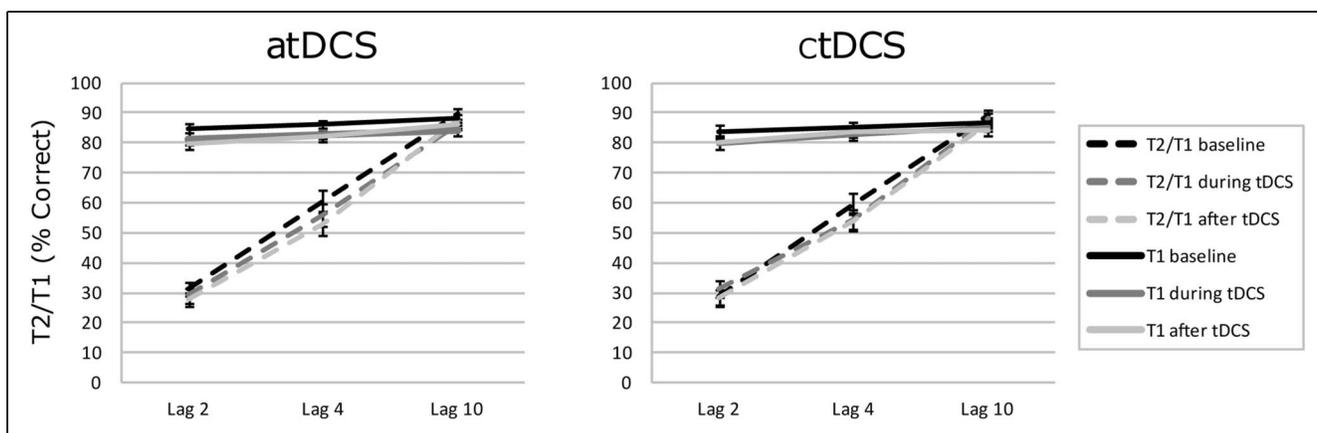
$-.543, p < .005$ , and after ctDCS,  $r(30) = -.789, p < .001$ . However, none of these correlations survived the test for equality of variances ( $t(32) = 0.84, p = .406$ ;  $t(32) = -0.50, p = .617$ ;  $t(32) = 0.12, p = .908$ ; and  $t(32) = 0.80, p = .430$ , respectively), indicating that we cannot exclude the possibility that these correlations can simply be explained by regression to the mean.

Furthermore, we post hoc examined the reliability of our measure of distractor inhibition. To this end, we correlated the baseline priming effect measured in the two separate sessions across participants using a Pearson correlation test. Surprisingly, the baseline priming effect was not correlated between sessions,  $r(31) = -.089, p = .64$  (controlling for session order), suggesting that this measure does not reflect a stable trait and calling for caution when interpreting the results of analyses including this measure. Given the unreliability of the priming measure, the fact that observed stimulation-induced changes in priming may simply reflect regression to the mean, and the fact that priming did not predict AB magnitude in a previous study (Slagter & Georgopoulou, 2013), we did not further examine if stimulation-induced changes in AB magnitude could be explained by stimulation-induced changes in distractor inhibition ability, as indexed by this priming measure. Importantly, baseline AB magnitude was reliably correlated between sessions,  $r(31) = .583, p < .001$  (controlling for session order), replicating previous reports that AB performance is stable over time (Dale et al., 2013).

#### Group Results

##### Effects of tDCS on the AB

Next, we investigated effects of tDCS on the magnitude of the AB at the group level. As can be seen in Figure 5, on average, participants displayed a clear AB, both before, during, and after atDCS and ctDCS (significant main effect of Lag:  $F(2, 64) = 311.80, p < .001$ ), but at the group level, tDCS did not modulate the size of the AB: No differential effect of atDCS versus ctDCS on the AB was observed, as



**Figure 5.** Group effects of atDCS and ctDCS on T1 performance (solid lines) and T2/T1 performance (dotted lines) in prime-absent trials. At the group level, neither anodal nor cathodal stimulation affected T1 accuracy or the AB. A Bayesian analysis confirmed strong evidence for no effect of tDCS on the AB.

indexed by a nonsignificant interaction between Stimulation, Lag, and Block,  $F(4, 128) = 0.829, p = .509$ . Session order did not influence the effect of stimulation on the AB but did affect T2 performance in general (regardless of Lag), as indicated by a significant interaction between Session order and Stimulation,  $F(1, 32) = 17.05, p < .001$ . Specifically, participants who received atDCS in the first session, on average, showed a greater improvement in T2|T1 accuracy (regardless of Lag) from the first session to the second than those who received ctDCS in the first session. Moreover, a main effect of Block,  $F(2, 64) = 9.14, p < .001$ , was found reflecting a decrease in T2|T1 performance over time. Yet, importantly, this effect was not modulated by the type of stimulation applied (nonsignificant interaction between Block and Stimulation:  $F(2, 64) = 1.50, p = .231$ ).

To follow up on the group analyses of the effect of tDCS on the AB and on performance in general, and to provide more information regarding the likelihood of our data under the null versus alternative hypotheses, we conducted an analogous Bayesian analysis using JASP (JASP Team, 2019). The frequentist ANOVA had shown that the interaction effect between Stimulation, Lag, and Block did not allow us to reject the null hypothesis. In line with this, Bayesian model averaging yielded strong evidence for the null hypothesis (inclusion Bayes factor of 30.3), under which there is no effect of tDCS on the AB. Furthermore, confirming the lack of a significant interaction between Stimulation and Block, there was also strong evidence that there was no effect of tDCS on performance across lags (inclusion Bayes factor of 16.39).

With regard to T1 accuracy, as can be seen in Figure 5, atDCS also did not affect T1 performance at the group level, as reflected in nonsignificant interaction effects between Block and Stimulation,  $F(2, 64) = 0.084, p = .919$ , and Stimulation, Lag, and Block,  $F(4, 128) = 1.17, p = .329$ . Again, a main effect of Block,  $F(2, 64) = 11.90, p < .001$ , was found, reflecting decreasing T1 accuracy over time, which was also observed regardless of the type of stimulation applied (reflected in the nonsignificant interaction between Block and Stimulation). Thus, T1 and T2 performance both displayed a general decline over time, perhaps reflecting a time-on-task effect and/or polarity-unrelated effects of tDCS, such as fatigue or headache (Brunoni et al., 2011; Poreisz, Boros, Antal, & Paulus, 2007). T1 performance was also affected by Lag (significant main effect of Lag:  $F(2, 64) = 20.56, p < .001$ ), but this effect was not modulated by the polarity of stimulation as reflected by the nonsignificant interaction between Stimulation, Lag, and Block,  $F(4, 128) = 1.17, p = .329$ . To summarize these findings, at the group level, neither atDCS nor ctDCS over IDLPFC affected the performance for T1 or T2.

#### *tDCS Side Effects and Effects on Arousal*

To rule out the possibility that participants were differentially affected by the physical sensations brought on by the

two stimulation conditions, we compared self-reported intensities of eight physical sensations during atDCS and ctDCS. Paired-sample  $t$  tests revealed that participants did not experience significantly different physical side effects during atDCS compared to ctDCS: itching,  $t(32) = -0.571, p = .572$ ; prickling,  $t(32) = 2.390, p = .023$ ; burning,  $t(32) = 0.865, p = .394$ ; pain,  $t(32) = 1.75, p = .090$ ; headache,  $t(32) = 0.000, p = 1$ ; fatigue,  $t(32) = -0.130, p = .897$ ; dizziness,  $t(32) = 0.000, p = 1$ ; and nausea,  $t(32) = -1.000, p = .325$ .

Next, we examined if there was a different effect on arousal levels of atDCS compared to ctDCS. No significant differences were found on any of the subscales: energy,  $t(30) = 0.345, p = .733$ ; tired,  $t(30) = -0.635, p = .530$ ; tension,  $t(30) = -0.879, p = .387$ ; and calmness,  $t(30) = -0.894, p = .378$ .

## DISCUSSION

The current study examined the effects of tDCS on the AB and on distractor inhibition. Our main hypothesis was that atDCS, but not ctDCS, over IDLPFC would modulate the AB as a function of individual baseline AB magnitude and by modifying participants' ability to filter out distracting information. We did not find evidence to support these hypotheses. No effects of tDCS were observed at the group level either. In fact, Bayesian analyses revealed strong evidence that tDCS to IDLPFC did not affect the AB. These results are surprising in light of findings from previous tDCS studies that used a similar stimulation protocol and observed changes in performance on WM tasks (e.g., Zaehle, Sandmann, Thorne, Jäncke, & Herrmann, 2011; Boggio et al., 2006; Fregni et al., 2005). Yet, a growing number of studies also report null effects of tDCS (Mancuso, Ilieva, Hamilton, & Farah, 2016; Horvath, Forte, & Carter, 2015a, 2015b). Hence, outcomes across studies appear inconsistent, likely because of variability in study designs (Hurley & Machado, 2018; Reteig, Talsma, van Schouwenburg, & Slagter, 2017) and low statistical power (Medina & Cason, 2017; Minarik et al., 2016). tDCS did not affect distractor inhibition either, but post hoc analyses revealed low test-retest reliability of our priming measure, rendering it difficult to interpret this result. Finally, a post hoc analysis revealed a significant negative correlation between the effect of atDCS and ctDCS: Individuals who benefited from atDCS generally worsened because of ctDCS, whereas individuals who worsened because of atDCS tended to perform better because of ctDCS. Below, these findings are discussed in more detail.

Our main finding was that atDCS over IDLPFC did not affect AB performance at the group level, nor as a function of baseline performance. It must be noted that this does not necessarily imply that IDLPFC is not involved in the AB. Functional magnetic resonance imaging and EEG studies indicate a critical role for the prefrontal cortex in the AB, and IDLPFC is likely to be an especially important structure (e.g., Slagter et al., 2010; Hommel et al., 2006;

Gross et al., 2004). What then might be some of the most likely reasons that tDCS to IDLPFC did not affect AB performance in our study?

One possibility is that our stimulation protocol did not reliably target IDLPFC. Studies modeling current flow show that the electric field generated by tDCS can be highly nonspecific, involving widespread effects that are difficult to predict without explicit modeling based on anatomical data (e.g., Bikson & Datta, 2012; Datta, Truong, Minhas, Parra, & Bikson, 2012). Differences in brain anatomy between individuals may have also led to differences across participants in the exact path the current took and thereby the structures affected. Although electrode placement at the F3 site made it more likely that activity in IDLPFC was indeed modulated (Sadleir, Vannorsdall, Schretlen, & Gordon, 2010), we certainly should consider the possibility that, at least in some individuals, nearby structures were also and perhaps even more strongly affected by tDCS than IDLPFC.

On the basis of previous studies showing effects of 1-mA IDLPFC tDCS on WM performance (e.g., Talsma, Kroese, & Slagter, 2017; Zaehle et al., 2011; Fregni et al., 2005), we choose 1 mA as stimulation strength. This amperage has been safely used in many previous studies and has been associated with relatively minor adverse effects, such as a mild tingling sensation, moderate fatigue, or light itching, in both healthy humans and patients (Poreisz et al., 2007). Yet, this relatively low current strength, together with likely partial shunting of the current through the skull, may not have been strong enough to elicit a functionally measurable effect. A recent study found that 10 min of 1.5-mA tDCS over IDLPFC modulated the AB, suggesting that indeed perhaps a higher amperage is necessary for inducing effects (Sdoia, Conversi, Pecchinenda, & Ferlazzo, 2019). It also used a different AB task in which targets were not identified by their color but by numbers and distractor were letters, which may have placed differential attentional demands on DLPFC. However, this study used a between-participant design with only 17 participants per group, and their findings hence also warrant replication. Future studies are necessary to better understand what may drive these opposing results. Currently, little is known about the effects of tDCS dosage (Dedoncker, Brunoni, Baeken, & Vanderhasselt, 2016), and one study even reported an effect of 1-mA anodal IDLPFC tDCS, but not 2 mA, on WM performance (Hoy et al., 2013). Of additional note, a recent study reported that, although neither 1 nor 2 mA of IDLPFC atDCS affected WM performance, the amplitude of the concurrently measured P3 event-related potential was affected by current strength (Nikolin, Martin, Loo, & Boonstra, 2018). Thus, although no behavioral effects were observed in the current study, it is possible that our stimulation did have neurophysiological effects, which may have been too subtle to modulate behavioral performance. Future studies that systematically vary current strength and combine tDCS with, for example, EEG are necessary to identify optimum tDCS dosage for behavioral effects.

We found that individuals who benefited from atDCS over IDLPFC generally worsened because of ctDCS over IDLPFC, whereas individuals who worsened because of atDCS over IDLPFC tended to perform better because of ctDCS over IDLPFC. This finding is in line with the idea that the balance between cortical excitation and inhibition can modulate the effect of tDCS in a nonlinear fashion (Krause, Márquez-Ruiz, & Cohen Kadosh, 2013). Specifically, the relation between the excitation/inhibition balance within a brain area on the one hand and its efficiency on the other is suggested to follow an inverted-U shape in which optimal performance is achieved when excitation and inhibition interact in a way that permits both flexibility and stability (see also Cools & D'Esposito, 2011; Arnsten, 1998; Zahrt, Taylor, Mathew, & Arnsten, 1997; Williams & Goldman-Rakic, 1995). The idea that atDCS enhances cortical excitation and thereby performance (Nitsche & Paulus, 2001) and that ctDCS reduces cortical excitability and thereby may impair performance (Nitsche et al., 2003) might be therefore be overly simplistic. Indeed, a growing number of studies report that the effects of stimulation on behavior can vary considerably from individual to individual (Benwell, Learmonth, Miniussi, Harvey, & Thut, 2015; Krause & Cohen Kadosh, 2014; López-Alonso, Cheeran, Río-Rodríguez, & Fernández-del-Olmo, 2014; for a review, see Li, Uehara, & Hanakawa, 2015). In line with these ideas, we found that individuals who benefited from atDCS generally worsened because of ctDCS, whereas individuals who worsened because of atDCS tended to perform better because of ctDCS. However, this finding is based on a post hoc analysis and thus demands replication in a confirmatory study. Moreover, we did not find the expected relationship between individual AB magnitude and the effect of atDCS on AB performance.

tDCS over IDLPFC did not affect distractor inhibition processes, as indexed by T2 priming, either. We found, however, that T2 priming may not provide a reliable measure of distractor inhibition ability at the individual participant level, as no significant correlation was observed between T2 priming in the baseline blocks of the two sessions. Dux and Marois (2008) and Slagter and Georgopoulou (2013) previously reported a relationship between this measure of distractor inhibition (T2/T1 at Lag 4 in prime-present vs. prime-absent trials) and recovery from the AB (i.e., T2/T1 at Lag 10 – Lag 4 in prime-absent trials). Note, however, that this correlation may be spurious because these two indices share a variable (i.e., T2/T1 at Lag 4 in no-prime trials). Nonindependent variables such as AB recovery and T2 priming can be expected to show an average correlation of .50 (in case of equal variances), even in the complete absence of a true relationship (Archie, 1981; see also Elliott & Giesbrecht, 2015). Moreover, Slagter and Georgopoulou (2013) did not observe a relationship between distractor inhibition and AB magnitude (i.e., T2/T1 at Lag 10 – Lag 2). The low test-retest reliability of the T2 priming measure, together with the possibly spurious correlation between T2 priming

and AB recovery, casts serious doubt on the previously reported relationship between distractor inhibition and AB recovery (Slagter & Georgopoulou, 2013; Dux & Marois, 2008). Nevertheless, findings from several studies using independent measures of distractor inhibition do confirm a role for individual differences in distractor inhibition ability in the AB (Arnell & Stubitz, 2010; Martens & Valchev, 2009), and it is clear that at least the distractor immediately after T1 plays a critical role in the AB (Olivers & Meeter, 2008; Di Lollo et al., 2005; Chun & Potter, 1995). Here, the priming distractor was presented at Lag 2, so the priming effect may have also been weakened because of strong suppression triggered by the Lag 1 distractor and/or resource depletion because of T1 processing.

To conclude, we found that tDCS over IDLPFC does not modulate the AB as a function of baseline AB magnitude, nor at the group level. In fact, Bayesian analyses revealed convincing evidence that tDCS to IDLPFC did not affect AB performance at the group level. A post hoc analysis did show that the direction of the effect of tDCS on temporal attention varied over participants, with an inverse relationship between the effects of ctDCS and atDCS. This pattern of results may be explained by an inverted U-shaped relationship between prefrontal excitability and AB magnitude but demands replication. Our results add to a growing body of work describing null effects of tDCS on a variety of cognitive effects (Medina & Cason, 2017; Horvath et al., 2015a, 2015b).

## Acknowledgments

This work was supported by a Marie Curie IRG grant and a VIDJ grant by the Netherlands Scientific Organization for Research to H. A. S. We thank Chris Olivers for helpful comments on an earlier draft of this paper. We thank Leon Reteig for bringing to our attention an error in a freely available online tool that we used for our tests of the equality of variances, which led us to draw an incorrect conclusion in a previous publication of these data (London & Slagter, 2015), which has been retracted and for which this paper is the corrected replacement. We also thank him for his help with the Bayesian analysis and helpful comments on this paper.

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Raquel E. London: Conceptualization; Formal analysis; Investigation; Methodology; Project administration; Writing-original draft. Heleen A. Slagter: Conceptualization; Funding acquisition; Project administration; Supervision; Writing-review & editing.

## Funding Information

Marie Curie IRG grant, grant number: IRG grant. NWO, grant number: VIDJ grant.

## Diversity in Citation Practices

A retrospective analysis of the citations in every article published in this journal from 2010 to 2020 has revealed a persistent pattern of gender imbalance: Although the proportions of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the *Journal of Cognitive Neuroscience (JoCN)* during this period were  $M(an)/M = .408$ ,  $W(oman)/M = .335$ ,  $M/W = .108$ , and  $W/W = .149$ , the comparable proportions for the articles that these authorship teams cited were  $M/M = .579$ ,  $W/M = .243$ ,  $M/W = .102$ , and  $W/W = .076$  (Fulvio et al., *JoCN*, 33:1, pp. 3–7). Consequently, *JoCN* encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article's gender citation balance. The authors of this paper report its proportions of citations by gender category to be:  $M/M = .54$ ;  $W/M = .222$ ;  $M/W = .127$ ;  $W/W = .111$ .

## Note

1. This study is a republication, with some new analyses, of the data originally reported in London and Slagter (2015; [https://doi.org/10.1162/jocn\\_a\\_00867](https://doi.org/10.1162/jocn_a_00867)), which has been retracted because of an error in an analysis that affected a result of principal interest. This paper hence supersedes London and Slagter (2015).

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