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REPLICATION

No Evidence for Enhancements to Visual Working Memory With Transcranial Direct Current Stimulation to Prefrontal or Posterior Parietal Cortices

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The present study examined the relative contributions of the prefrontal cortex (PFC) and posterior parietal cortex (PPC) to visual working memory. Evidence from a number of different techniques has led to the theory that the PFC controls access to working memory (i.e., filtering), determining which information is encoded and maintained for later use whereas the parietal cortex determines how much information is held at 1 given time, regardless of relevance (i.e., capacity; McNab & Klingberg, 2008; Vogel, McCollough, & Machizawa, 2005). To test this theory, we delivered transcranial DC stimulation (tDCS) to the right PFC and right PPC and measured visual working memory capacity and filtering abilities both during and immediately following stimulation. We observed no evidence that tDCS to either the PFC or PPC significantly improved visual working memory. Although the present results did not allow us to make firm theoretical conclusions about the roles of the PFC and PPC in working memory, the results add to the growing body of literature surrounding tDCS and its associated behavioral and neurophysiological effects.

Keywords: visual working memory, attention control, filtering, tDCS

Working memory (WM) is a capacity-limited system responsible for maintaining and acting upon information in a goal-directed manner (Baddeley & Hitch, 1974; Cowan, 2001; Engle, 2002). Visual WM is the system responsible for the maintenance and direction of information in the visual modality. Typically, visual WM capacity limitations are reached when individuals try to maintain 3 to 4 pieces of information (Luck & Vogel, 1997; Cowan, 2001). The theoretical bases for these limitations constitute two distinct yet related abilities: the amount of information one can hold at any given time, sometimes referred to as the scope of attention or visual WM capacity (*k*), and the ability to gate access of information to WM, sometimes referred to as the *control of attention or filtering* (Cowan et al., 2005, 2006; Cowan & Morey, 2006; Vogel, McCollough, & Machizawa, 2005). Ideally, individuals can maintain and operate upon a large number of goal-relevant pieces of information in the execution of effective ongoing cognitive processes. Further, individuals must prevent irrelevant information, either from the external stimulus environment or from internal representations, from gaining access to WM.

A number of techniques have been used to examine how visual WM operates and how attention control and capacity limitations are determined. These include individual differences, electroencephalography (EEG), neuroimaging (e.g., positron emission tomography, functional MRI), lesion studies, and noninvasive manipulative techniques like transcranial magnetic stimulation (TMS) and transcranial DC stimulation (tDCS). At the level of individual differences, prior studies have shown that WM capacity correlates well with other important cognitive functions like general fluid intelligence, attention control, and long-term memory (Engle, Tuholski, Laughlin, & Conway, 1999; Unsworth & Engle, 2007; Unsworth, Fukuda, Awh, & Vogel, 2014). Furthermore, individual differences investigations have demonstrated that a critical component of visual WM capacity is the ability to gate access of information to WM (Vogel et al., 2005). In general, people who have a reduced ability to select and maintain only goal-relevant information show reductions in capacity. WM capacity is also associated with more domain-general attention control mechanisms that are responsible for goal-directed behavior (Kane, Bleckley, Conway, & Engle, 2001; Kane & Engle, 2002; Kane & Engle, 2003). EEG studies have demonstrated that sustained activity over occipital and parietal areas during maintenance of visual WM representations is predictive of capacity estimates, as well as other cognitive abilities like attention control and fluid intelligence (Unsworth, Fukuda, Awh, & Vogel, 2015; Vogel et al., 2005). Furthermore, neuroimaging using functional MRI (fMRI) has demonstrated that the prefrontal cortex (PFC) and posterior parietal cortex (PPC) are highly involved in visual WM (Courtney, Ungerleider, Kiel, & Haxby, 1997; D'Esposito & Postle, 2015; Eriksson, Vogel, Lansner, Bergström, & Nyberg, 2015; Nee et al., 2013).

Of most relevance to the current study is the dissociation between the roles of the PFC and PPC in the two major limitations of visual WM mentioned earlier—capacity and control. Several studies have specifically examined this dissociation. For example, McNab and Klingberg (2008) demonstrated that activity in the

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PFC and basal ganglia preceded filtering of irrelevant information from visual WM. Further, the level of activity in these regions correlated with individual differences in visual WM capacity when filtering was required (McNab & Klingberg, 2008). This study provided evidence that the PFC exerts control over what information is permitted access to the limited-capacity visual WM system. Vogel et al. (2005) also showed that the ability to filter irrelevant items from visual WM relates to sustained EEG signals in lateralized parietal and occipital areas, referred to as contralateral delay activity (CDA). When individuals were presented with only relevant information during encoding periods, CDA amplitude reliably predicted capacity. Further, when participants were required to filter irrelevant information, low-capacity individuals' CDA revealed unnecessary storage of irrelevant information, whereas high-capacity individuals' CDA reflected exclusive storage of relevant information. These electrophysiological data were complemented by behavioral evidence that low-capacity individuals showed significantly worse performance on trials requiring filtering. Therefore, frontal and parietal regions show complementary yet distinct roles in visual WM. Whereas activity in the PFC appears to reflect control over access to WM, parietal regions seem to reflect the amount of information being stored in WM.

Recently, noninvasive methods that transiently alter cortical activity have been developed to test theories of how the brain carries out various cognitive processes. For example, tDCS passes a fairly weak electrical signal through a region of cortex in a way that can alter the excitability of neurons in that region (Nitsche et al., 2008). By altering the activity of cortical regions using tDCS and observing associated behavioral changes, researchers can test theories about the roles of various brain regions in specific cognitive processes. Most relevant to the current study, tDCS has been used to alter WM by stimulating PFC, but the mixture of results indicate this method may not be entirely reliable (see Brunoni & Vanderhasselt, 2014, for review). Here we discuss a few recent studies that have specifically examined visual WM capacity and control over WM using tDCS. In one study, Heimrath, Sandmann, Becke, Müller, and Zaehle (2012) delivered anodal, cathodal, and sham tDCS to the right parietal cortex and had participants complete a lateralized visual WM task. Participants received each of the three stimulation types in sessions separated by at least 24 hr. Anodal tDCS significantly reduced visual WM capacity for contralateral stimuli, and cathodal tDCS increased capacity, compared with sham stimulation. For ipsilateral stimuli, both anodal and cathodal tDCS significantly reduced visual WM capacity compared with sham stimulation (Heimrath et al., 2012). Although Heimrath et al. showed that stimulation to parietal areas can significantly alter visual WM capacity, this study did not specifically examine the effect of parietal stimulation on filtering, nor did they stimulate frontal areas. In a similar study, Tseng et al. (2012) showed significant improvements in visual WM capacity following anodal stimulation to the right posterior parietal cortex (rPPC) compared with sham. In their first experiment, Tseng et al. observed an improvement in capacity at the group level. In their second experiment, Tseng et al. only observed a significant change in capacity among individuals who had low baseline WM capacity determined by a median split of 20 participants. Tseng et al. also observed a significant increase in CDA following anodal tDCS compared with sham among the low-WM group. These results suggest that effects of tDCS may only arise when participants have some room for improvement. That is, low-WM individuals may especially benefit from stimulation the PPC. However, Jones and Berryhill (2012) showed an opposite pattern of findings. Specifically, Jones and Berryhill (2012) observed a significant increase in visual WM capacity following anodal and cathodal tDCS to right PPC (compared with sham) for high-capacity individuals only. Low-capacity individuals actually showed decreases in visual WM following active tDCS (both anodal and cathodal) compared with sham. A follow-up experiment showed that high-capacity individuals demonstrated especially large improvements in capacity at larger set sizes (Jones & Berryhill, 2012).

In a more recent study, Li et al. (2017) delivered anodal tDCS to three different regions across three sessions separated by at least 48 hr: right PFC, right PPC, and visual cortex. In each session, participants completed a lateralized visual WM task that required filtering of irrelevant information on some trials. So Li et al. (2017) were able to examine the respective impacts of stimulation to right PFC (rPFC), right PPC (rPPC), and visual cortex on visual WM capacity estimates and filtering abilities. Li et al. observed a significant improvement in capacity following anodal stimulation to rPPC compared with visual cortex, and a marginally significant improvement in capacity following stimulation to rPFC compared with visual cortex. In terms of filtering abilities (computed as a difference in capacity between distractor-absent and distractor-present trials), stimulation to rPFC significantly improved filtering compared with stimulation to visual cortex. But stimulation to rPPC reduced filtering abilities (albeit not quite significantly) compared with stimulation to visual cortex. In a follow-up experiment, Li et al. observed a nearly identical pattern of results with a nonlateralized version of the visual WM task from their first experiment. In sum, Li et al. argue that the rPFC acts as a control region to gate access to visual WM (i.e., attention control), whereas the rPPC determines the amount of information that can be maintained in visual WM (i.e., attention scope).

The present study used a very similar design to Li et al. (2017) to test the relative contributions of the rPFC and rPPC to visual WM.[1] Specifically, we hypothesized that stimulation to the rPFC would specifically enhance filtering, whereas stimulation to the rPPC would specifically increase WM capacity. In Experiment 1, we delivered tDCS to rPFC, using sham stimulation to the same region in a separate session as a within-subject control. In Experiment 2, we delivered tDCS to the rPPC, again using sham stimulation in a separate session as a within-subject control. Although our design and theoretical aims were very similar to Li et al. (2017), the present set of experiments are worth consideration given the mixed results of tDCS in the literature reviewed earlier (see also Horvath, Forte, & Carter, 2015; Brunoni & Vanderhasselt, 2014, for reviews). In order to establish that tDCS can be used to reliably manipulate cortical activity in a way that systematically alters behavior in theoretically meaningful ways, we should be able to directly and conceptually replicate studies that observe specific behavioral effects of tDCS to certain brain regions.

[1] We should note that the present set of experiments were conducted and all data were collected prior to the publication of Li et al. (2017). We designed our experiments with similar theoretical issues in mind, so we try to most directly compare our results to Li et al.

Experiment 1

The goal of Experiment 1 was to manipulate activity in the rPFC via tDCS to observe its downstream effects on visual WM. As the rPFC theoretically controls access to WM, active tDCS to this region should produce greater filtering performance compared with when participants receive sham stimulation to this same area. Further, if manipulation of rPFC activity via active tDCS alters WM capacity (k), then we should also observe an increase in k estimates during/after active stimulation compared with sham stimulation. Finally, if we observe a change in filtering but not a change in k, then this would dissociate the role of rPFC in control and its role in determining visual WM capacity. If we do not observe changes in either filtering or k estimates, then tDCS may not be a reliable enough technique to test theoretical claims about the role of rPFC in visual WM.

Method

Participants and Procedure

The experimental protocol was approved by the Institutional Review Board of the University of Oregon. A total of 29 participants were recruited from the human subjects pool at the University of Oregon. After signing up through an online participation system, participants were sent an email with an informed consent form and Safety Screening Questionnaire. The informed consent outlined the study and provided participants with information regarding the nature of the study, as well as any known potential risks. The safety screening provided 12 yes/no questions concerning personal/family history of epilepsy, personal history of head injuries, metal implants, and so forth. If participants answered yes to any of the safety screening questions, or if they felt uncomfortable participating in a study that involved tDCS, they were encouraged to cancel their participation without penalty. When participants came to the lab, they completed the informed consent form, a brief demographics form, and the Safety Screening Questionnaire. If they had any additional questions at that time, they were allowed to ask the researcher for clarification. Participants first completed a colored squares change detection task to measure baseline visual WM capacity. After completing this task, participants were randomly assigned to one of two conditions. In one condition, participants received sham stimulation during their first session and active stimulation during their second session. In the other condition, participants received active stimulation during their first session and sham stimulation during their second session. Other than the counterbalancing of sham and active stimulation session across conditions, the procedures were identical for all participants. All participants then completed a filtering task during active/sham stimulation. Stimulation lasted for 20 min, regardless of how long the participant spent on the first block of the filtering task. All participants then completed a second block of the filtering task after the stimulation/sham block. Participants returned to the lab for a second session exactly one week after their first session. The second session was identical to the first session with only one difference. If participants had received active stimulation in the first session, they received sham stimulation in the second session, and vice versa. Two participants did not return for the follow-up session, and for 3 participants, we were unable to establish a connection with the tDCS device. So the final sample included 24 participants ($n = 11$ in sham/active condition, $n = 13$ in active/sham condition).

Stimulation

For our stimulation procedure we tried to most directly replicate the procedures of previous studies examining similar cognitive phenomena and similar brain regions (Jones & Berryhill, 2012; Li et al., 2017; Tseng et al., 2012), as well as some specific recommendations made in a recent review of tDCS best practices (Reinhart, Cosman, Fukuda, & Woodman, 2017). Participants were first fit with an EEG cap that fit snugly but was not overly tight. The cap served two purposes. First, it allowed us to correctly place the sponge and electrode over the rPFC using the international 10–20 EEG electrode placement system (electrode site F4). Second, it kept the sponge and electrode in place during stimulation. We first wet the sponges using a saline solution so they were wet but not dripping. Then, we placed the reference electrode on the left cheek and secured it to the head using an elastic headband. We then placed the reference electrode between the sponge (50 cm^2) and the headband, ensuring it was flush to the sponge. The anodal electrode was placed over a sponge (16 cm^2) centered under electrode site F4 on the EEG cap. We then turned on the tDCS device to ensure an electrical connection between the electrodes. If no connection could be reached, we rewet the sponges and replaced the electrodes until a connection could be reached. After a good connection was reached, we started the stimulation, which passed a constant current of 1.5 mA for a total of 20 min. Stimulation was delivered via a battery-powered tDCS constant-current stimulator (Mind Alive, Inc.). In the stimulation condition, we turned the device on, and it ran continuously for the full 20 min. The researcher sat next to the participant with the device in hand (out of the view of the participant) while the stimulation occurred. To ensure the device maintained a connection throughout the session, the experimenter monitored the device, which shows a ramping-up on a series of lights once per minute to demonstrate a continued connection. The device did not lose connection during the stimulation session for any participants. After 20 min, the device automatically shut off. In the sham condition, we turned the device on and allowed it to run for one minute (30-s ramp-up and 30-s ramp-down) to allow the participant to feel the sensation of stimulation. We then discretely turned the device off (out of the view of the participant) and left it off for 18 min. We then turned the device back on and left it on for one minute (30-s ramp-up and 30-s ramp-down). We then turned the device off again. After the sham/stimulation session, we removed the electrodes, sponges, headband, and EEG cap. Participants were given paper towels with which they could dry their face and hair. We then immediately proceeded with the second filtering task block. After the second block of the filtering task in both sessions, we gave participants a questionnaire about their perceptions during the stimulation session. Specifically, participants were asked, "Did you experience any of the following during the stimulation session? (Please circle all that apply)" Options were itching/tingling under the electrodes/sponges, dizziness, nausea, headache, blurred vision, and none of the above. Then, in both sessions we asked participants to guess their condition. Specifically, participants were told, "In this experiment, you were randomly assigned to either a stimulation/exper-

imental condition or to a sham/control condition for each session. Please indicate whether you think you were in the stimulation condition or the sham condition for this session by circling one." Participants circled stimulation or sham on the question sheet.

Tasks

Colored squares change detection. To ensure participants in the two conditions did not differ on baseline visual WM capacity, all participants completed a colored squares change detection task at the beginning of both sessions. The task consisted of sequential arrays of colored squares on a gray background. Every trial began with a screen saying "Remember, press left for same, right for different. Are you ready? Press the space bar to continue." Once the participant pressed the spacebar, a blank gray screen appeared for 500 ms. This was followed by a 2,500-ms fixation screen upon which a black cross was centered on a gray background. A 100-ms blank screen then appeared, followed by the first presentation of the array. The array presented 4, 6, or 8 colored squares randomly selected from a set of seven colors (white, black, red, yellow, green, blue, and purple) for 250 ms. The locations of the items within the array were random with several constraints. Items appeared inside a 540 pixel × 402 pixel region centered on the screen. Items were each 20 pixels × 20 pixels in size and appeared randomly within the region with the constraint that no items were within a 55-pixel distance from each other. After a 900-ms delay, the array reappeared, and one of the colored squares had a circle around it. The participants' task was to determine if the circled square was the same color or a different color from the first array. Participants used a key labeled *S* to indicate same color and a key labeled *D* to indicate different color (the *F* and *J* keys on the keyboard). The test array remained on-screen until the participant made their response. Set size and colors were randomly selected on each trial. The color of the tested square changed on 50% of trials. The color of untested items never changed. Participants first completed six practice trials after which they were encouraged to seek clarification if needed. They then completed 60 experimental trials. There were six trial types (change/no change for each of three set sizes) that were randomly intermixed. Participants completed 10 trials of each trial type. A graphical depiction of the task is shown in Figure 1. Visual WM capacity was computed using Cowan's *k* (Cowan, 2001; Cowan et al., 2005; Pashler, 1988). *K* estimates from this task were used to ensure roughly equal baseline WM capacity across conditions and experiments.

Filtering. The filtering task was a nonlateralized version of Vogel et al.'s (2005) visual WM task. In this task, participants were presented with red and blue rectangles and asked to remember the orientation of the red rectangles and ignore the blue rectangles. Each trial began with a screen saying "Remember, press left (S) for same, right (D) for different. Are you ready? Press the space bar to continue." Once the participant pressed the space bar, a 500-ms blank gray screen appeared, followed by a 1,000-ms screen with a black fixation cross. This was followed by another 100-ms blank screen, then the initial array appeared. The array consisted of red and blue rectangles at one of four orientations: vertical, horizontal, 45° right, and 45° left. The array could consist of two red rectangles and no blue rectangles, four red rectangles

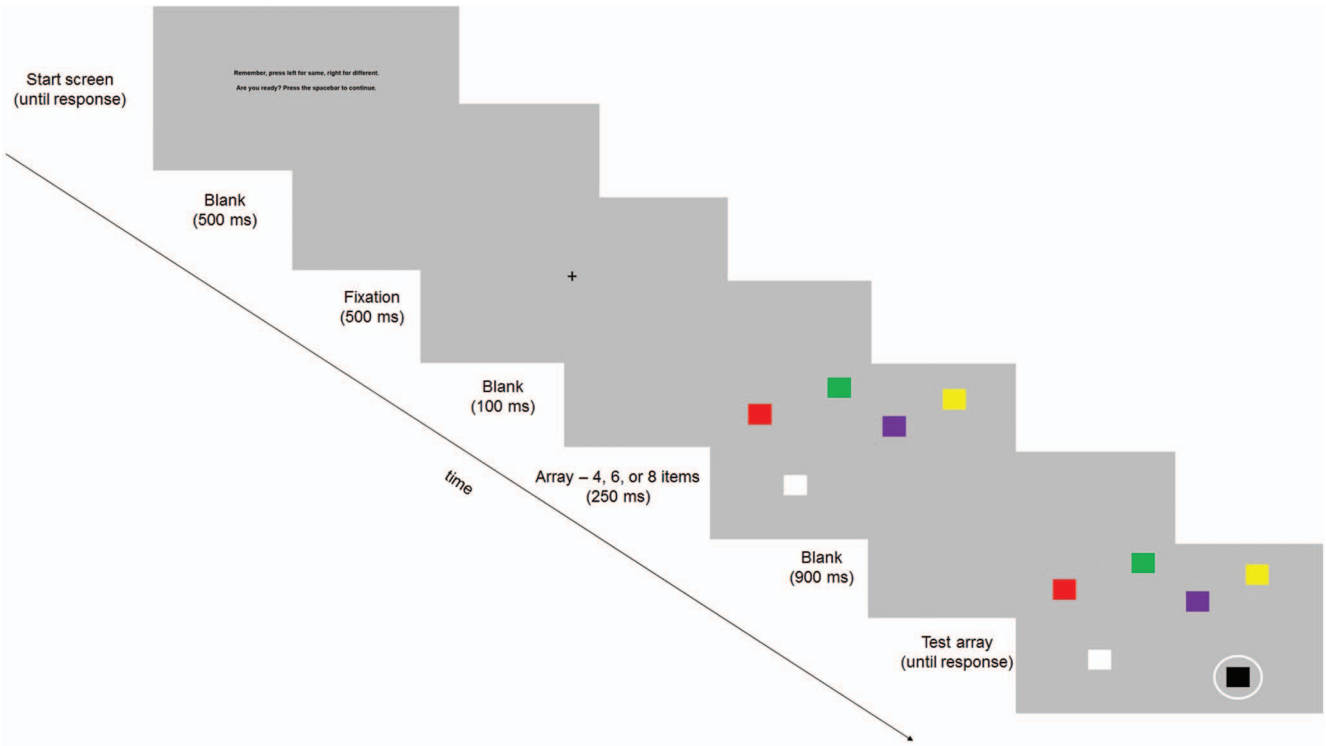

Figure 1. Graphical depiction of an example trial for the colored squares change detection task. The tested item was indicated by a white circle around one of the items. See the online article for the color version of this figure.

and no blue rectangles, two red rectangles and two blue rectangles, or four red rectangles and two blue rectangles. Items appeared within a 520 × 402 pixel region centered on the screen. Item locations were random with the constraint that no items were within a 100-pixel distance from each other. The array remained on screen for 250 ms, followed by a 900-ms blank delay. The array then reappeared, and one of the red rectangles had a white dot on it. The participants' task was to indicate whether or not the rectangles with the white dot on it had changed orientation or not. Participants indicated their response by pressing a key marked *S* for same orientation or *D* for different orientation. The test array stayed on-screen until the participant made their response. The orientation of the tested rectangle changed 50% of the time. The orientation of the untested rectangles never changed. There were eight trial-types (change/no change for each of four set sizes) which each appeared 30 times in each 240-trial block. Trial types were randomly intermixed. An example trial is shown in Figure 2. Participants completed six practice trials followed by two blocks of 240 experimental trials. Because the stimulation was not time-locked to the stimulation device, all trials were included in the final analyses, even if they occurred during the ramp-up and ramp-down phases of the stimulation/sham sessions. *K* estimates were computed using the same formula as in the colored-squares change detection task with performance on four-target/no-distractor trials. Filtering scores were computed as the difference in *k* between two-target/no-distractor trials and two-target/two-distractor trials. Active/sham stimulation occurred during the first block of trials. As soon as a good connection was reached on the tDCS device, the participants completed the practice trials. If the participant did not have any further questions regarding the task, the experimenter instructed the participant to begin the task and started stimulation. If participants finished the task before the 20-min mark, they were asked to sit and relax until the stimulation session finished. The experimenter then removed the cap and electrodes, and participants completed the second block of experimental trials.

Results

To ensure participants were blind to conditions, we examined the postsession questionnaires to see if participants accurately guessed their condition in each session. In the first session, guessing accuracy was 70%, which was significantly different from chance guessing, $t(23) = 2.20$, $p = .04$. In the second session, guessing accuracy was 67%, $t(23) = 1.70$, $p = .10$.[2] Our next analysis was to ensure that baseline WM capacity (*k*) was not significantly different between participants in the two conditions (sham/active vs. active/sham). Mean *k* did not differ as a function of condition in the first session, $t(22) = .72$, $p = .48$. Preexperimental *k* estimates were about equal in the sham/active ($M = 2.65$, $SD = .92$) and the active/sham ($M = 2.35$, $SD = 1.09$) conditions during the first session. Preexperimental *k* did not differ in the second session either, $t(22) = .72$, $p = .38$. Again *k* values were about equal in the sham/active condition ($M = 3.16$, $SD = .72$) and the active/sham condition ($M = 2.81$, $SD = 1.11$). *K* values increased significantly from Session 1 ($M = 2.49$, $SD = 1.00$) to Session 2 ($M = 2.98$, $SD = .95$; paired-samples $t[23] = 2.54$, $p = .02$), which probably indicated a practice effect. This effect did not interact with condition, $F(1, 22) = .02$, $p = .90$. So the increase was not significantly greater for participants in either condition. Baseline *k* estimates were significantly correlated within participants across sessions ($r = .54$, $p = .01$), indicating good session-to-session reliability.

Our next set of analyses was to determine if active tDCS over rPFC significantly altered *k* values and filtering scores during the filtering task. To compute *k* in the filtering task we again used Cowan's *k* (Cowan, 2001) using performance on trials where there were four targets (red rectangles) and no distractors (blue rectangles). To examine this effect, we ran a repeated-measures analysis of variance (ANOVA) with condition (sham/active, active/sham) as a between-subjects effect and session (1, 2) and block (during stimulation/sham, after stimulation/sham) as within-subjects effects. There was no main effect of condition, $F(1, 22) = .79$, $p = .38$, partial $\eta^2 = .04$, and condition did not significantly interact with block, $F(1, 22) = 2.03$, $p = .17$, partial $\eta^2 = .09$, or session, $F(1, 22) = 3.52$, $p = .07$, partial $\eta^2 = .16$. Further, the condition by session by block interaction was not significant, $F(1, 22) = .29$, $p = .60$, partial $\eta^2 = .01$. Because there was no evidence that the order of sessions had an impact, we collapsed across conditions for our remaining analyses. This analysis revealed no main effect of session, $F(1, 23) = 2.59$, $p = .12$, partial $\eta^2 = .10$, but a marginal effect of block, $F(1, 23) = 3.89$, $p = .06$, partial $\eta^2 = .06$. Session and block did not significantly interact, $F(1, 23) = .18$, $p = .67$, partial $\eta^2 = .01$. So if anything, participants' *k* estimates increased slightly from the first block to the second block.

To further examine the magnitude of effects, we subtracted *k* after sham stimulation from *k* after active stimulation. For this analysis, we report 95% confidence intervals (CIs) around the mean differences, as well as Bayes factors in favor of the null (BF_{01}). Bayes factors were computed using JASP software (JASP Team, 2016). These can be interpreted as the ratio of the likelihood of the null hypothesis (no effect) being true compared with the alternative hypothesis. So a BF_{01} of 2 can be interpreted as the null hypothesis being twice as likely to be true given the data. This analysis revealed a nonsignificant increase in *k* when comparing performance during active stimulation to performance during sham stimulation ($M_{diff} = .22$, $SD_{diff} = .72$), $t(23) = 1.52$, $p = .14$, (95% CI [−.08, .53], $BF_{01} = 1.71$), and a nonsignificant increase in *k* when comparing performance after active stimulation to performance after sham stimulation ($M_{diff} = .16$, $SD_{diff} = .66$), $t(23) = 1.15$, $p = .26$, (95% CI [−.12, .43], $BF_{01} = 2.58$). So although the effects were in the hypothesized direction, they did not reach traditional thresholds for significance. These effects are depicted in Figure 3a.

To examine the reliability of *k* estimates, we computed correlations within and across sessions to ensure unreliability of the measures could not have accounted for our observation of null effects. *K* estimates were highly correlated between blocks in both the first session ($r = .81$, $p < .001$) and the second session ($r = .79$, $p < .001$). *K* estimates were also highly correlated across sessions ($r = .75$, $p < .001$). Collectively, these results suggest that active tDCS over rPFC did not significantly affect partici-

[2] It is not clear why participants had an inclination of what condition they were in, but because this was only significantly above chance in one session, it did not occur in Experiment 2, and there were no clear differences in behavioral performance, we do not think it had a significant impact on the results.

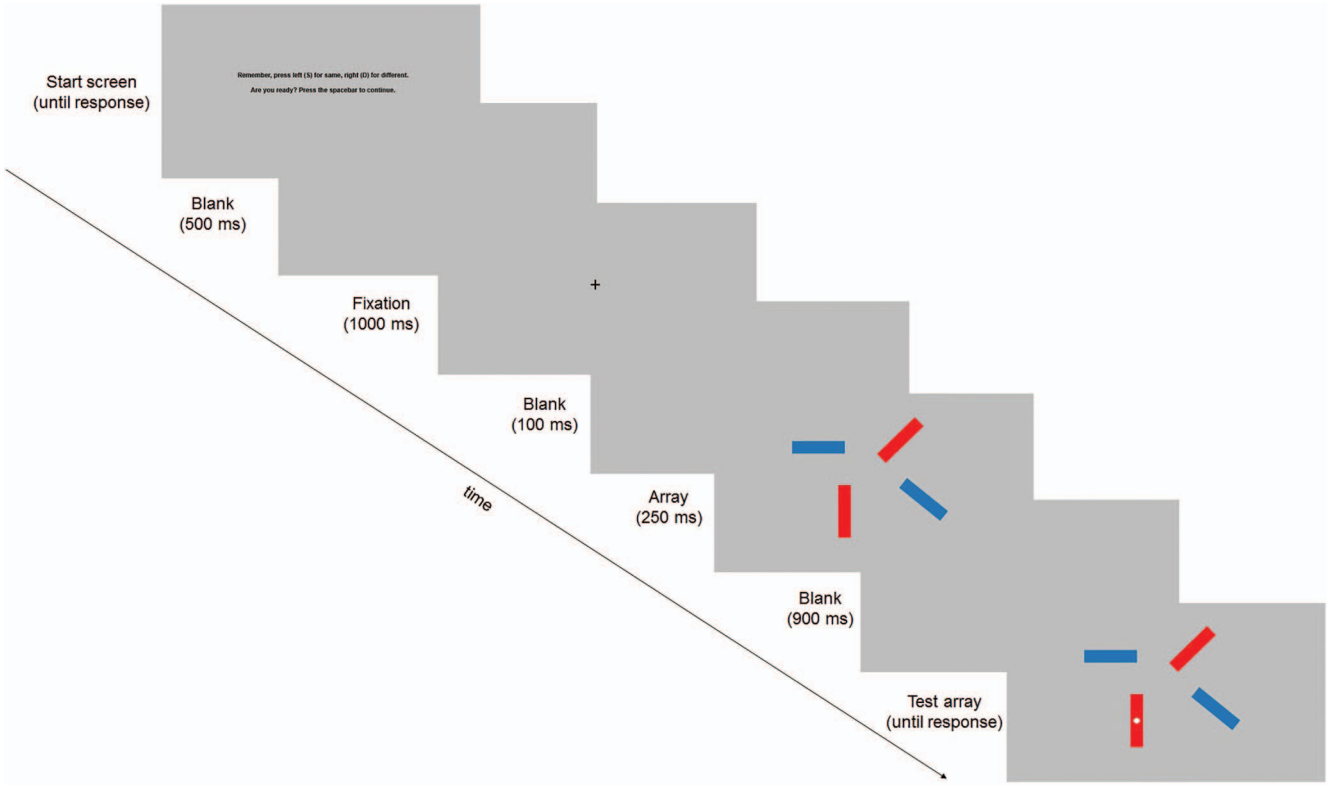

Figure 2. Graphical depiction of a filtering task trial. Arrays could contain two red (dark grey) rectangles (targets) and zero blue (light grey) rectangles (distractors), four red rectangles and zero blue rectangles, two red rectangles and two blue rectangles, or four red rectangles and two blue rectangles. The tested item was indicated by a white dot on one of the red rectangles. See the online article for the color version of this figure.

pants' *k* compared with when participants performed the same task under sham tDCS over rPFC.

Our next set of analyses sought to test whether tDCS over rPFC had a significant impact on filtering. To compute filtering costs for each individual we subtracted their *k* estimates from the two-target/two-distractor trials from their *k* estimates on the two-target/no-distractor trials. A larger filtering costs represents a greater effect of distractor presence. So lower costs reflect better filtering. We ran a repeated-measures ANOVA on filtering costs with condition (sham/active, active/sham) as a between-subjects effect and session (1, 2) and block (during stimulation/sham, after stimulation/sham) as within-subjects effects. There was no main effect of condition, $F(1, 22) = .04$, $p = .84$, partial $\eta^2 = .002$, and condition did not interact with either session, $F(1, 22) = .51$, $p = .51$, partial $\eta^2 = .02$) or block, $F(1, 22) = .09$, $p = .76$, partial $\eta^2 = .004$. Finally, the session by block by condition interaction was not significant, $F(1, 22) = .31$, $p = .58$, partial $\eta^2 = .01$, so we collapsed across conditions for our remaining analyses. There was no main effect of session, $F(1, 23) = .38$, $p = .54$, partial $\eta^2 = .02$, or block, $F(1, 23) = .04$, $p = .84$, partial $\eta^2 = .002$, and session and block did not interact, $F(1, 23) = .26$, $p = .62$, partial $\eta^2 = .01$. Just as with the analysis of *k,* we wanted to further examine the magnitude of effects. We computed the difference in filtering costs between sham and active stimulation by subtracting filtering costs during/after sham stimulation from filtering costs during/after active stimulation. This analysis revealed a nonsignificant difference in filtering between active and sham stimulation both during ($M_{diff} = -.003$, $SD = .32$, $p = .97$; 95% CI [−.14, .13], $BF_{01} = 4.65$) and after stimulation ($M = -.04$, $SD = .23$, $p = .35$; 95% CI [−.14, .05], $BF_{01} = 3.09$). Therefore, we did not observe any evidence that filtering costs significantly changed based on active stimulation over the rPFC. These results are depicted in Figure 3b. Descriptive statistics for *k* estimates and filtering costs for all trial types, separated by condition, session, and block are shown in Table 1.

Discussion

Experiment 1 sought to test the idea that manipulating activity in the rPFC, a region that theoretically controls access visual WM, would systematically alter performance in a visual WM task. Our analyses showed that although participants' *k* estimates increased as a function of practice with the task, as they showed increases across blocks within sessions and across sessions, there was no evidence that the tDCS significantly altered *k*. If this had been the case, we would have observed significantly greater *k* estimates when participants received active tDCS over rPFC compared with when they received sham tDCS over this same region. The results indicated that the active and sham stimulation did not produce significantly different *k* estimates, either during the actual stimulation session or immediately following stimulation. Similarly, we

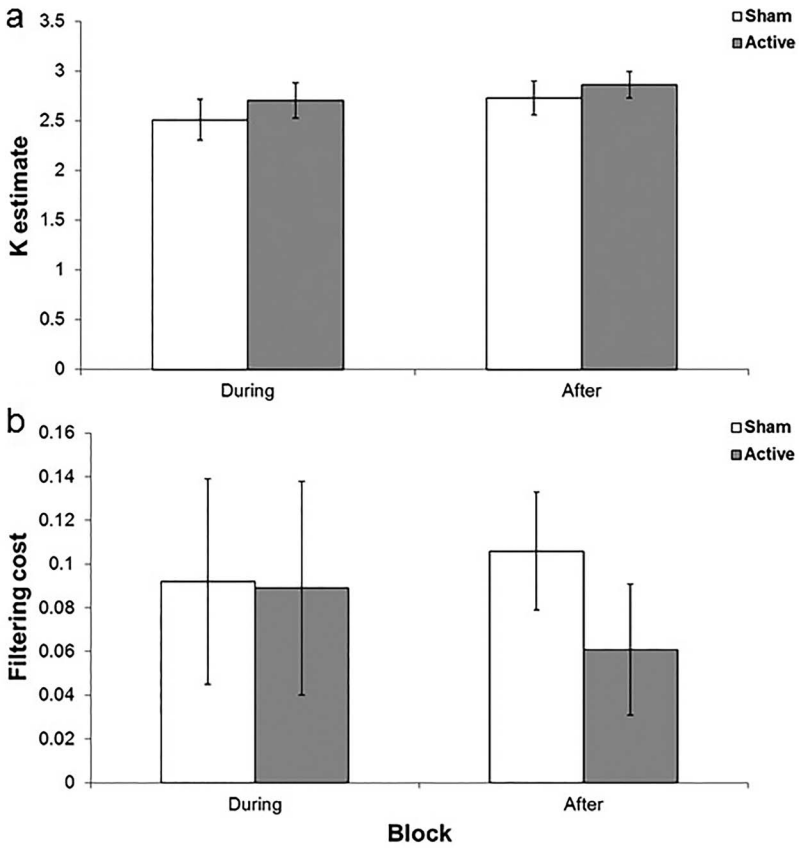

Figure 3. a) Visual working memory capacity (*k*) estimates as a function of stimulation session and block, and b) filtering cost as a function of stimulation session and block. Error bars represent ± one standard error of the mean.

observed no evidence that active stimulation significantly altered filtering abilities compared with sham stimulation.

Experiment 2

The goal of Experiment 2 was to test whether manipulating activity in the rPPC would substantially alter visual WM. To do so, we ran a very similar design to Experiment 1 with only one alteration. Rather than receiving active/sham stimulation over the rPFC, participants received stimulation over the rPPC. Theoretically, this area controls the number of items an individual can hold in visual WM (McNab & Klingberg, 2008; Vogel et al., 2005). If this is the case, we should observe an effect of active tDCS on *k* estimates. If stimulation to the rPPC also changes filtering costs relative to sham stimulation, this would provide evidence that the rPPC is also involved in controlling access to WM. Finally, if stimulation to rPPC alters *k* estimates but not filtering scores, then this would dissociate the roles of the rPPC in determining the capacity of and allowing access to visual WM. If we do not observe any differences in capacity or filtering during or after active tDCS compared with sham stimulation, then tDCS may not be a reliable technique to causally test theoretical claims about the role of the rPPC in visual WM.

Method

Participants and Procedure

A total of 33 participants were recruited from the undergraduate human subject pool at the University of Oregon. All screening procedures, counterbalancing of stimulation/sham sessions, stimulation protocol, timing of sessions, and behavioral tasks were identical to Experiment 1. Six participants did not return for the second session, 1 participant did not complete a second block of filtering trials in the second session, 1 participant did not follow task instructions for the first session, and we could not secure an adequate connection for 2 participants, leaving a final sample of 24 participants ($N = 11$ in sham/active condition, $N = 13$ in active/sham condition). No participants in Experiment 2 had participated in Experiment 1.

Stimulation

The stimulation procedures were identical to Experiment 1, with the exception that stimulation was delivered over the rPPC. To do so, we centered the active electrode under P4 on the EEG cap according to the international 10–20 electrode system. Participants

Table 1
Descriptive Statistics for Experiment 1

Condition	Session	Measure	During stim	After stim
Sham/active	Sham	2 target/0 distractor *k*	1.74 (.23)	1.80 (.15)
		2 target/2 distractor *k*	1.65 (.33)	1.67 (.22)
		4 target/0 distractor *k*	2.42 (.88)	2.85 (.64)
		4 target/2 distractor *k*	2.36 (.98)	2.76 (.74)
		Filtering cost	.09 (.19)	.13 (.14)
	Active	2 target/0 distractor *k*	1.87 (.13)	1.81 (.14)
		2 target/2 distractor *k*	1.78 (.22)	1.79 (.18)
		4 target/0 distractor *k*	2.99 (.58)	3.14 (.48)
		4 target/2 distractor *k*	2.72 (.76)	2.65 (.67)
		Filtering cost	.09 (.16)	.02 (.16)
Active/sham	Active	2 target/0 distractor *k*	1.63 (.36)	1.76 (.26)
		2 target/2 distractor *k*	1.54 (.53)	1.66 (.31)
		4 target/0 distractor *k*	2.51 (.97)	2.63 (.70)
		4 target/2 distractor *k*	2.21 (1.11)	2.53 (.78)
		Filtering cost	.09 (.29)	.10 (.13)
	Sham	2 target/0 distractor *k*	1.65 (.66)	1.63 (.43)
		2 target/2 distractor *k*	1.55 (.55)	1.54 (.51)
		4 target/0 distractor *k*	2.58 (1.13)	2.58 (1.04)
		4 target/2 distractor *k*	2.31 (1.28)	2.59 (1.00)
		Filtering cost	.09 (.27)	.09 (.12)

Note. Sample sizes were as follows: $n = 11$ in sham/active condition, and $n = 13$ in active/sham condition. During stim = trials performed during stimulation/sham; After stim = trials performed after stimulation/sham; *k* = visual working memory capacity estimate using Cowan's *k* formula (Cowan, 2001); Filtering cost = *k* estimate on 2 target/0 distractor trials minus *k* estimate on 2 target/2 distractor trials, computed within each participant and then averaged across participants.

were randomly sorted into two conditions. In one condition, participants received sham stimulation during their first session and active stimulation during their second session. In the other condition, participants received active stimulation during their first session and sham stimulation during their second session. Participants received the same postsession questionnaire as in Experiment 1.

Tasks

Colored squares change detection. See Experiment 1.

Filtering. See Experiment 1.

Results

To ensure participants were blind to conditions, we examined the postsession questionnaires to see if participants accurately guessed their condition in each session. In both sessions, guessing accuracy was 56%, which was not significantly different from chance guessing, $t(24) = .59$, $p = .56$. Our next analysis ensured there were no baseline differences in *k* across participants in the two conditions. Participants did not differ in baseline *k* estimates in either Session 1, $t(23) = .72$, $p = .48$, or Session 2, $t(23) = .68$, $p = .48$. So *k* values were about equal across conditions on average. Baseline *k* estimates increased from Session 1 ($M = 2.46$, $SD = .72$) to Session 2 ($M = 2.74$, $SD = .63$; $t(23) = 2.16$, $p = .04$), similar to Experiment 1. These *k* estimates were significantly correlated across sessions ($r = .55$, $p = .005$), indicating good session-to-session reliability.

Our next set of analyses tested whether active stimulation to the rPPC affected *k* estimates during or after stimulation. Just as in Experiment 1, we used performance on trials where there were four targets and no distractors to estimate *k*. We ran a repeated-measures ANOVA with within-subjects factors of session (sham, active) and block (during, after).[3] The analysis revealed no main effects of session, $F(1, 23) = 1.24$, $p = .28$, partial $\eta^2 = .05$, or block, $F(1, 23) = 1.25$, $p = .28$, partial $\eta^2 = .05$, and session and block did not significantly interact, $F(1, 23) = .90$, $p = .35$, partial $\eta^2 = .04$. To examine the magnitude of effects, we computed the difference in *k* between active and sham stimulation (during and after) by subtracting active *k* from sham *k*. We again report 95% confidence intervals and Bayes factors in favor of the null hypothesis (BF_{01}). This analysis revealed a nonsignificant difference in *k* during active stimulation compared with sham ($M_{diff} = -.04$, $SD_{diff} = .53$), $t(23) = -.35$, $p = .73$, (95% CI [−.26, .19], $BF_{01} = 4.40$). The difference in *k* after active stimulation compared with after sham stimulation was also nonsignificant ($M_{diff} = -.07$, $SD_{diff} = .61$), $t(23) = -.53$ $p = .60$, (95% CI [−.32, .19], $BF_{01} = 4.09$). So we observed no evidence that active tDCS to rPPC significantly altered *k* estimates, either during the stimulation session or afterward. These effects are depicted in Figure 4a.

Similar to Experiment 1, we examined within-session and between-session reliability. *K* estimates from four-target/no-distractor trials were significantly correlated from the first block of trials to the second block of trials in both the first session ($r = .55$, $p = .004$) and the second session ($r = .64$, $p = .001$). Further, *k* estimates from this measure were significantly correlated across sessions (collapsing across blocks, $r = .54$, $p = .007$). These correlations indicate good within-session and between-session reliability of *k* estimates.

Our next set of analyses focused on how stimulation to rPPC affected filtering. We first computed a filtering cost for each participant for each session and block of trials by subtracting *k* estimates from two-target/two-distractor trials from *k* estimates from two-target/no-distractor trials. As a reminder, filtering costs closer to zero indicate better filtering. We ran a repeated-measures ANOVA on filtering costs with within-subjects factors of session (1, 2) and block (during, after).[4] We observed no main effect of session, $F(1, 23) = 1.06$, $p = .531$, partial $\eta^2 = .04$. There was a main effect of block, $F(1, 23) = 4.30$, $p = .05$, partial $\eta^2 = .16$, suggesting that filtering costs dropped within a session, but the session by block condition was not significant, $F(1, 23) = .26$, $p = .62$, partial $\eta^2 = .01$, so this effect was about equal across stimulation and sham sessions. To examine the magnitude of these effects, we computed the difference in filtering costs during and after active stimulation compared with sham stimulation for each participant. On average, filtering costs were not significantly different during active stimulation compared with sham ($M_{diff} = .05$,

[3] Similar to Experiment 1, we also included condition as a between-subjects factor. There were no significant effects or interactions with condition (all Fs < 2), so we collapsed across conditions for the remaining analyses.

[4] We observed one significant effect of condition, as the condition by session interaction was significant. Whereas participants in the sham/active condition showed a marginal increase in filtering costs across sessions, $t(10) = 2.17$, $p = .06$, participants in the active/sham condition did not, $t(12) = .83$, $p = .42$.

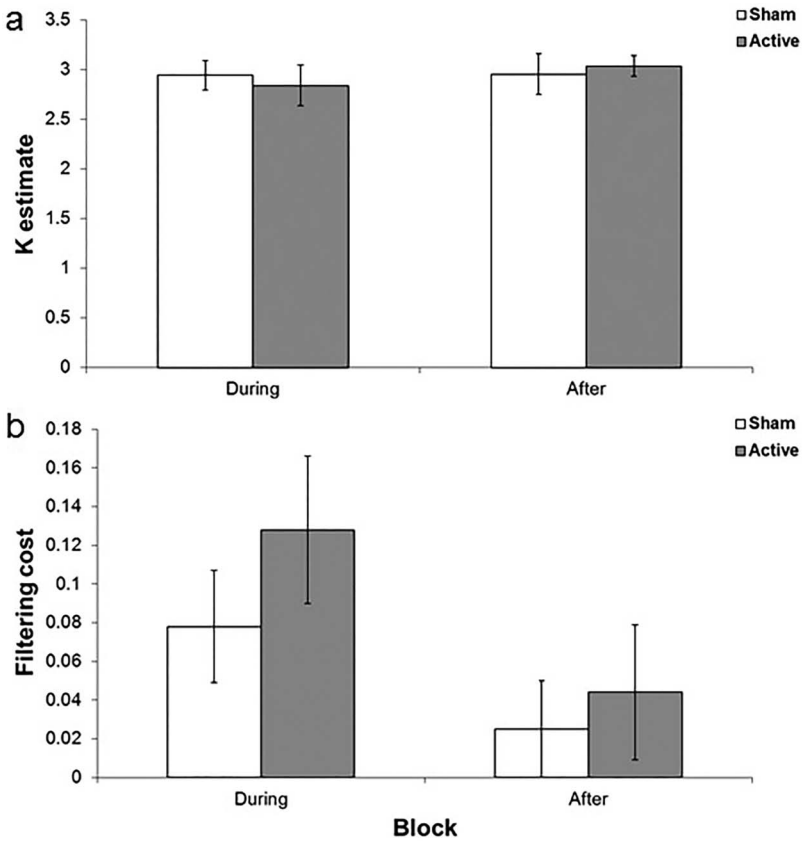

Figure 4. a) Visual WM capacity estimate (*k*) as a function of stimulation session and block and b) filtering costs as a function of stimulation session and block. Error bars represent ± one standard error of the mean.

SD_{diff} = .22), $t(23) = 1.11$, $p = .28$, (95% CI [−.04, .14], BF_{01} = 2.70), nor were they different after active stimulation compared with after sham (M_{diff} = .02, SD_{diff} = .22), $t(23) = .43$, $p = .67$, (95% CI [−.07, .11], BF_{01} = 4.28). Collectively, there was no evidence to suggest that active tDCS to the rPPC affected filtering scores compared with sham stimulation. These effects are depicted in Figure 4b. Descriptive statistics for all trial types separated by condition, session, and block are shown in Table 2.

Cross-Experimental Analyses

Because we had two specific a priori predictions for how stimulation to the rPFC and rPPC would affect visual WM capacity and filtering abilities, we combined the data from Experiments 1 and 2 to specifically test those predictions. We hypothesized, on the basis of the theoretical roles of the PFC and PPC in visual WM, that (1) stimulation to rPFC would significantly reduce filtering costs but have no effect on capacity in distractor-free conditions, and (2) stimulation to rPPC would increase capacity in distractor-free conditions but have no effect on filtering. To test these hypotheses, we ran a repeated-measures ANOVA on capacity and filtering with stimulation site (rPFC, rPPC) as a between-subjects variable and session (sham, active) and block (during, after) as within-subjects variables. The analysis of capacity revealed no main effects of session, $F(1, 46) = .97$, $p = .33$, partial $\eta^2 = .02$, block, $F(1, 46) = 2.62$, $p = .11$, partial $\eta^2 = .05$, or stimulation site, $F(1, 46) = 2.81$, $p = .10$, partial $\eta^2 = .06$. There was also no significant Session × Stimulation Site interaction, $F(1, 46) = 3.05$, $p = .09$, partial $\eta^2 = .06$, Session × Block interaction, $F(1, 46) = .15$, $p = .70$, partial $\eta^2 = .003$, or three-way interaction among session, block, and stimulation site, $F(1, 46) = .03$, $p = .87$, partial $\eta^2 = .001$. So we did not find any evidence that tDCS affected capacity and filtering in the specific ways we had hypothesized.

Some prior studies (Jones & Berryhill, 2012; Tseng et al., 2012) have suggested that stimulation is differentially effective for high- and low-capacity individuals. To test this notion, we examined correlations between preexperimental *k* estimates and the effects of active tDCS on capacity and filtering in each experiment. In Experiment 1, preexperimental *k* did not significantly correlate with the magnitude of the change in capacity or filtering either during or after stimulation (all *p*s > .21). Similarly in Experiment 2, preexperimental *k* did not significantly correlate with the magnitudes of the effects on either capacity or filtering either during or after stimulation (all *p*s > .32). Therefore, we did not observe any evidence that the effects of active stimulation to rPFC or rPPC on visual WM change as a function of baseline capacity differences.

Discussion

The goal of Experiment 2 was to test whether active tDCS to the rPPC would impact visual WM capacity or filtering abilities.

Table 2
Descriptive Statistics for Experiment 2

Condition	Session	Measure	During stim	After stim
Sham/active	Sham	2 target/0 distractor *k*	1.80 (.17)	1.77 (.26)
		2 target/2 distractor *k*	1.75 (.21)	1.74 (.22)
		4 target/0 distractor *k*	3.15 (.66)	3.22 (.81)
		4 target/2 distractor *k*	2.83 (.79)	2.81 (.83)
		Filtering	.04 (.13)	.00 (.13)
	Active	2 target/0 distractor *k*	1.82 (.21)	1.82 (.23)
		2 target/2 distractor *k*	1.65 (.29)	1.72 (.27)
		4 target/0 distractor *k*	3.19 (.76)	2.98 (.92)
		4 target/2 distractor *k*	3.03 (.73)	2.74 (.92)
		Filtering	.17 (.17)	.10 (.19)
Active/sham	Active	2 target/0 distractor *k*	1.78 (.19)	1.76 (.16)
		2 target/2 distractor *k*	1.68 (.22)	1.76 (.19)
		4 target/0 distractor *k*	2.84 (.73)	3.03 (.37)
		4 target/2 distractor *k*	2.59 (.48)	2.74 (.48)
		Filtering	.09 (.19)	.00 (.15)
	Sham	2 target/0 distractor *k*	1.78 (.25)	1.77 (.19)
		2 target/2 distractor *k*	1.68 (.28)	1.72 (.28)
		4 target/0 distractor *k*	2.94 (.63)	2.95 (.73)
		4 target/2 distractor *k*	2.90 (.67)	2.48 (.84)
		Filtering	.11 (.15)	.05 (.12)

Note. Sample sizes were as follows: $n = 11$ in sham/active condition, and $n = 13$ in active/sham condition. During stim = trials performed during stimulation/sham; After stim = trials performed after stimulation/sham; k = visual working memory capacity estimate using Cowan's k formula (Cowan, 2001); Filtering cost = k estimate on 2 target/0 distractor trials minus k estimate on 2 target/2 distractor trials, computed within each participants and then averaged across participants.

These results largely replicated Experiment 1. We observed no significant effects of stimulation on visual WM capacity or on filtering costs either during or following active tDCS to the rPPC compared with sham stimulation to the same area.

General Discussion

The goal of the present set of experiments was to examine the respective roles of the rPFC and rPPC in two aspects of visual WM: control and capacity. Theoretically, the frontal and parietal cortices play distinct yet related roles in visual WM. On one hand, the frontal cortex, and more specifically the PFC, theoretically controls access to visual WM, filtering out irrelevant information to ensure it does not interfere with goal-relevant information. On the other hand, the parietal cortex, and more specifically the PPC, theoretically determines capacity limitations. So at a basic level, the PFC handles the what of visual WM, and the PPC determines how much. In the present study, we sought to manipulate activity in these two regions and examine the downstream impacts on capacity and filtering abilities.

In Experiment 1, we delivered tDCS to the rPFC during a visual WM task that required maintenance of relevant information (orientation of red rectangles) on all trials and filtering of irrelevant information (blue rectangles) on some trials. From this task, we can estimate visual WM capacity (k) using a formula that adjusts for biased responding (Cowan, 2001; Pashler, 1988). Using this formula, we estimated how many items individuals were able to hold on the various trial types. Our primary estimate of capacity came from trials with four targets and zero distractors. We then analyzed how active stimulation affected each individual's capacity compared with an identical session (one week earlier or later) during which we delivered sham stimulation to the same region. Participants were randomly assigned to receive active stimulation during their first session or second session, and sham stimulation in the other session. Further, participants completed the same filtering task in two blocks. One block occurred during the delivery of the active/sham stimulation, the other block occurred immediately thereafter. Our results indicated that capacity estimates were slightly but nonsignificantly higher during and after active stimulation compared with sham stimulation. Participants showed slight but significant practice effects both within and across sessions. We then estimated filtering scores by subtracting k estimates on trials on which there were two targets and two distractors from k estimates from trials in which there were two targets and no distractors. This filtering cost estimates the impact of irrelevant information on visual WM performance. A greater cost indicates a greater impact, so scores closer to zero indicate better filtering of irrelevant information. Overall, we found no evidence that active stimulation to the rPFC significantly altered filtering abilities compared with sham stimulation.

In Experiment 2, we ran an identical procedure to Experiment 1 with one crucial exception: tDCS was delivered over the rPPC rather than the rPFC. We used the same task, counterbalancing of sessions, timing and magnitude of stimulation, device, and so forth, as Experiment 1. Results showed capacity estimates did not increase either during or after active stimulation compared with sham stimulation. Just as in Experiment 1, there were no significant differences in filtering abilities either during or after active stimulation compared with sham stimulation. Overall, the two experiments yielded no evidence that manipulating the activity of the rPFC or rPPC with tDCS can significantly alter visual WM performance.

Any time a study finds a null result, it is hard to come to firm conclusions about what the data is actually telling us. Specifically, the present set of results fails to replicate some prior studies that have observed significant alterations to visual WM performance following active tDCS to similar regions (Heimrath et al., 2012; Jones & Berryhill, 2012; Li et al., 2017; Tseng et al., 2012). Specifically, Li et al. (2017) found that active stimulation to the rPFC specifically increased filtering abilities, and active stimulation to the rPPC specifically increased capacity, compared with active stimulation to primary visual cortex. Because this study is the most directly comparable to the present set of experiments, we will address some of the subtle differences that may have accounted for our failure to conceptually replicate. First, we used a different tDCS device than Li et al. But the device used in the present study has been used in previous investigations and produced significant behavioral differences (Reinhart & Woodman, 2015; Reinhart, Zhu, Park, & Woodman, 2015). So we felt confident that this device was adequate to produce behavioral differences. Second, Li et al. (2017) used a lateralized filtering task in their Experiment 1. Our filtering task, which was otherwise nearly identical, was not lateralized. However, in Experiment 2, Li et al. (2017) replicated their findings with a nonlateralized version of the filtering task. Further, the only difference between the tasks used in the present study and Li et al. (2017) was that our study included a fourth trial type that included four targets and two distractors, in addition to the four-target/no-distractor, two-target/no-distractor, and two-target/two-distractor trial types used in both the present

study and Li et al. Third, we used different control conditions. Li et al. used a within-subjects design in which participants received active stimulation to right frontal (F4), right parietal (P4), and central occipital (Oz) sites in three separate sessions separated by 48 hours. In Experiment 1 of the present study, participants received active stimulation to right frontal (F4) in one session and sham stimulation to the same location in a second session separated by exactly one week (the order of sessions was counterbalanced across participants). It is possible, although not entirely clear why, this difference in controls would produce substantially different patterns of results. Theoretically, neither sham stimulation to PFC/PPC nor active stimulation to visual cortex should produce behavioral differences in visual WM. So both of these controls seem appropriate. Finally, the present study gave participants a colored squares change detection task before the filtering task in both sessions. This was included to ensure roughly equal baseline *k* estimates across conditions and experiments. Li et al. (2017) did not include such a task. Again, it is not clear why adding this task would have explained the differences in results, but it is worth noting. In sum, it remains unclear why the present study did not produce the same pattern of findings as Li et al. (2017), and future work may be necessary to investigate these differences.

We should also note that the present study did not entirely replicate Heimrath et al. (2012), who observed significant changes in visual WM following anodal and cathodal tDCS to the right parietal cortex. However, Heimrath et al. observed a pattern of findings that are in contrast to Li et al. (2017). Heimrath et al. showed a significant drop in WM capacity for information presented on the left side of the screen (contralateral hemifield) following anodal tDCS compared with sham to the right posterior cortex, but they observed a significant increase in WM capacity for contralateral information following cathodal tDCS compared with sham. Both cathodal and anodal tDCS significantly decreased WM capacity for ipsilateral information compared with sham. So while Li et al. (2017) observed a significant increase in WM capacity following anodal tDCS to the right parietal cortex, Heimrath et al. (2012) observed the exact opposite. Because our task was not lateralized, it is difficult to directly compare our results to Heimrath et al. Furthermore, some studies have shown that only low-capacity individuals benefit from tDCS to PPC (Tseng et al., 2012), whereas others have demonstrated the exact opposite—that only high-capacity individuals benefit from tDCS to PPC (Jones & Berryhill, 2012). Taken together, the results are heterogeneous and do not show a clear, consistent, theoretically defensible pattern. If anything, our results lie somewhere in the middle of all the research reviewed earlier.

One possible reason for why our study failed to find any significant findings is a lack of statistical power, given our sample size. Although we did not specify our sample size based on an a priori estimation of effect sizes, we wanted to rule out a lack of power as an explanation. To do so, we estimated the size of the effects observed by Li et al. (2017), as this study was most similar to the current study. We then computed our power to detect such effects using G*Power software (Faul, Erdfelder, Lang, & Buchner, 2007). We used the reported means and standard deviations for attention control (i.e., filtering) and attention scope (i.e., *k*) and assumed a within-subject correlation of .5 for pre- and poststimulation performance. Li et al. (2017) did not report this correlation, but this assumption is based on our observed within-session correlations (average $r \sim .7$). Doing so resulted in an effect size of .69 for the effect of stimulation to rPPC on attention scope and an effect size of .79 for the effect of stimulation to rPFC on attention control. With our sample size of 24 in both experiments, we had power of .90 to detect the effect of stimulation to rPPC on attention scope and power of .96 to detect the effect of stimulation to rPFC on attention control. Therefore, we do not believe the current study was significantly underpowered, and we do not believe a lack of power led to our observation of several null effects.

Although the present study did not find any results that would allow us to make firm conclusions about the respective roles of the frontal and parietal cortices in visual WM, we feel the present study makes a valuable contribution to the ongoing research into the cognitive effects of tDCS. These and similar types of causal manipulations to cortical activity (e.g., TMS) can be valuable tools for testing cognitive theories. However, as with any experimental manipulation or measurement tool, we need to assess the reliability of the effects produced by tDCS. It is certainly possible that the tDCS delivered over the brain regions in the present study did not have an appreciable physical impact on these regions. Unfortunately, in the current study the only way to infer changes in brain activity due to the stimulation is by analyzing the associated changes in behavior. Recent reviews of the behavioral and neurophysiological results of studies using tDCS has demonstrated that the results are mixed, at best, and there is considerable controversy about how reliably tDCS can be used (Brunoni & Vanderhasselt, 2014; Horvath et al., 2015; but see Reinhart et al., 2017). It is not our goal to dismiss tDCS as a technique entirely. Rather, we hope that future research can incorporate our study into the existing and growing body of literature to assess the reliability of tDCS as a useful scientific tool. We believe our set of experiments were well-grounded in psychophysical and cognitive neuroscientific theory, and were well-designed, well-controlled, and well-executed. In the end, we did not observe any theoretically meaningful effects due to tDCS. However, this does not mean the present data are entirely useless. Future research can take our findings and combine them with other studies of similar cognitive processes and make educated decisions about the use of tDCS. Furthermore, researchers can balance the existing evidence showing significant and null effects of tDCS to come to more well-rounded decisions about theoretical claims based on studies leveraging tDCS as a technique.

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